# Schein's Common Sense Emergency Abdominal Surgery

Moshe Schein
Paul N. Rogers
Ahmad Assalia
*Editors*

Robert Lane
*Editorial Adviser*

Third Edition

Springer

"The thinking surgeon". Oil on canvas. By Dan Schein
http://danschein.com/home.html

Moshe Schein • Paul N. Rogers • Ahmad Assalia
(Editors)

# Schein's Common Sense Emergency Abdominal Surgery

An unconventional book for trainees and thinking surgeons

Third Edition

With 133 Figures and 30 Tables

Springer

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*MS dedicates this book to his late father Karl Schein, a surgeon on the Eastern front during World War II and later in Haifa, Israel.*

*Karl Schein, 1911–1974*

# Editors' Note

This book has been assembled—in pieces—during long years of intensive personal involvement, clinical and academic, urban and rural, with emergency abdominal surgery in South Africa, Israel, USA, UK, and Australia.

A long line of good old friends from all around the world were helpful in generating this book and its two preceding editions. For the foundations in this noble surgical field, MS is indebted to George G. Decker of Johannesburg. Drs. Asher Hirshberg and Adam Klipfel contributed to the first edition. Dr. Alfredo Sepulveda of Santiago, Chile, edited the Spanish translation (first edition); Dr. Francesco Vittorio Gammarota of Rome, Italy, edited the Italian translation (second edition), and we will remember with affection the late Professor Boris Savchuk of Moscow who edited the Russian translation (first edition). We are grateful to the many members of SURGINET, who over the years have stimulated our brains with their constant international feedback. Thanks also to Dr. Slava Ryndine, South Africa, for the help with the caricatures, to Roger Saadia for his stimulating criticism, and to Rob Lane for superb editorial input. Thanks to Dr. Evgeny (Perya) Perelygin who drew additional caricatures for this edition and to Dan Schein who painted the image on one of the pages.

Special thanks to ladies Gabriele Schroeder and Stephanie Benko of Springer, Heidelberg, for their immense support. Most of the aphorisms and quotations used to decorate this book were retrieved from *Aphorisms & Quotations for the Surgeon* (2002) and *A Companion to Aphorisms & Quotations for the Surgeon* (2008), edited by MS and published by Nikki Bramhill's tfm Publishing, Harley, UK.

The reader will find that there are a few duplications scattered throughout the book. We did this on purpose, as repetition of important points is crucial in adult education. Any reader who has a question or a comment about anything to do with this book is invited to e mail us directly: mschein1@mindspring.com, pn.rogers@btinternet.com, assaliaa@gmail.com. We will respond.

Finally, we are indebted to our loving wives, Heidi, Jackie, and Anat and our children Omri, Yariv, Dan, Lucy, Michael, Razi, May, and Adam, for their patience and sacrifice.

January 2010

Moshe Schein, Wisconsin
Paul N. Rogers, Glasgow
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# Preface to the Third Edition

"In literature, as in love, we are astonished as what is chosen by others." (Andre Maurois, 1885-1967)

We are proud to present the third edition of this book, which since it first appeared almost 10 years ago, has become a favorite with surgeons having to deal with emergency abdominal surgery.

Is there anything new in this field that merits the revision and update of such a book every 4–5 years? Yes. Our practice has been gradually changing for the better and worse. Where we practice—in the "developed world"—the volume and spectrum of emergency surgery are declining and becoming narrower. Where any abdominal grumble is followed by a CT scan or any fart by a colonoscopy, ruptured aortic aneurysm and acute malignant colonic obstruction are becoming rarities. When most asymptomatic inguinal hernias undergo elective repair, one does not see many strangulated or obstructed groin hernias. When the entire population is being fed, or buys at the counter, anti-ulcer medications, operations for bleeding, or perforated ulcer are hardly ever performed. This, however, may not be true in other parts of the world where you have the fortune (or misfortune) to practice.

The way we practice emergency surgery has also been rapidly evolving. With almost unlimited access to abdominal imaging, we can rapidly pinpoint the diagnosis and avoid an unnecessary operation, or perform an indicated operation instead of engaging in a prolonged period of uncertainty. We are gradually becoming more selective and cautious—understanding that everything we do involves wielding a double-edged sword, and that in emergency surgery usually doing less is better but occasionally doing more may be life-saving. Meanwhile fancy diagnostic modalities are used chaotically by our nonsurgical colleagues (and some of our surgical ones)—producing *red herrings* or new "image diseases," "incidentalomas," and adding to the general confusion.

In this brave new world of changes, we need constantly to update ourselves. We have to re-learn how to deal with the old s**t—which is becoming rare—even when its odor is masked by the perfume of modern practice. And this is what we

continue doing in this new edition—reciting the written-in-stone sacred, old basics but also showing how to integrate them in the evolving modern world.

**What is new in the Third Edition?** We have a new co-editor (AA), an advanced laparoscopic surgeon, who took it upon himself to emphasize the "laparoscopic perspective" of the various chapters. We added new chapters (viz., complications of laparoscopic surgery, complications of bariatric surgery, complications of peritoneal dialysis, leaks after colorectal surgery, urological emergencies, abdominal emergencies in the "third world"), and "invited commentaries," by new contributors. All contributors are well known to us personally as experts in their field. All existing chapters have been revised, expanded, or re-written by the old or new contributors and/or the editors. Each chapter has been carefully scrutinized by us, its style and tone tuned to conform to the overall "voice" of the book.

From the beginning, we knew that a book like this—written in practical, colloquial, and direct in-your-face style will be either loved or hated. And indeed, a few reviewers—appalled by dogmas that clash with their own, and language not exactly conforming to Strunk and White's *The Elements of Style*—almost killed it. But many more loved it. In the preface to the second edition, we have cited from the many complimentary reviews and the enthusiastic feedback we have received from surgical readers around the world—we will not cite from it again. Even now, after 9 years in print, we continue to receive enthusiastic feedback from satisfied new readers. For example:

- "I have personally to admit that *Schein's Common Sense Emergency Abdominal Surgery* was on many surgical calls often my best and only friend. Furthermore, it helps me till today to guide my own teaching to students and residents…" (Pascal O. Berberat Staff Surgeon/Faculty, Klinikum rechts der Isar, Munich).
- "I just wanted to express my gratefulness for your "Common Sense." I am an attending surgeon in a General Hospital in the South of Germany and bought your book as soon as I could get hold of it (which is quite difficult—always sold out!), and was never able to read in whole myself—because it is always, always, always with the residents who "live" on it. You and your team became our "surplus" senior attending colleagues and taught a lot—the residents and us!" (Margita Geiger, Attending Surgeon, Ansbach, Bavaria, Germany).
- "In your book I read this sentence: 'Surgeons are internists who operate.' I just wish to tell you that such a sentence has definitely illuminated my brain and the wisdom and brightness of your philosophy about the surgical method (against the frequent stupidity of the usual surgical mind, at least in my country) are guiding my working days as a young emergency surgeon." (Carlo Bergamini, MD, PhD, Florence, Italy).

By popular demand the first edition of this book has been translated into Spanish and Russian and the second edition into Italian (by Dr. Francesco V. Gammarota).

German and Czech translations are underway. Motivated by the enthusiasm with which the book is being received across the world—particularly among those practicing "real surgery" in the "real world"—we set about enhancing it to produce a text that should be palatable to all of you—wherever you try to save lives—be it in Mumbai, Karachi, Cairo, Belgrade, Soweto, Mexico City, Kiev, Copenhagen, Philadelphia, Glasgow, Krakow, and yes, even in Paris (we hope there are French surgeons who can, and want to, read English☺

If you are a surgeon who practices the way he was trained 20 or 30 years ago, you will hate this book; if you are being trained by such a surgeon then you desperately need to read this book.

Dr. Anton Chekhov said: "Doctors are just the same as lawyers; the only difference is that lawyers merely rob you, whereas doctors rob you and kill you, too." Our chief aim in writing this book was to help you not kill your patients. This nonorthodox book is not yet another tedious, full-of-details textbook. We do not need more of these. It is aimed at you, the young practicing surgeon who desires a focused and friendly approach to emergency abdominal surgery. We hope and believe that this modest book will be of some value to you.


The Editors: Rogers is the one in the kilt; the one puffing on a stogie is Assalia.

Moshe Schein
Paul N. Rogers
Ahmad Assalia

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# 1 General Philosophy

Moshe Schein · Paul N. Rogers · Ahmad Assalia

*Surgeons are internists who operate …*
*"Wisdom comes alone through suffering."* (Aeschylus, Agamemnon)

At this moment—just as you pick up this book and begin to browse through its pages—there are thousands of surgeons around the world facing a patient with an abdominal catastrophe. The platform on which such an encounter occurs differs from place to place—be it a modern emergency department in London, a shabby casualty room in the Bronx, or a doctor's tent in the African bush—but the scene itself is amazingly uniform. It is always the same—you confronting a patient, the patient suffering, in pain, and anxious. And, you are anxious as well—anxious about the diagnosis, concerned about choosing the best management, troubled about your own abilities to do what is correct. We are in the twenty-first century—but this universal scenario is not new. It is as old as surgery itself. You are perhaps too young to know how little certain things have changed—or how other things did change—over the years. Yes, your hospital may be in the forefront of modern medicine; its emergency room has standby, state-of-the-art spiral computed tomography and magnetic resonance imaging machines, but, practically, something has not changed: it is the patient and you (often with the entire "system" against you)—you who are duty bound to provide a correct management plan and execute it.

## The "Best" Management of an Abdominal Emergency

It is useful to compare the emergency abdominal surgeon to an infantry officer (› Fig. 1.1). Away from the limelight and glory that surrounds cardiac or neurological surgeons, emergency abdominal surgery is closer to infantry than it is to airborne action. A war cannot be won by remote control with cruise missiles but with infantry on the ground. To achieve the final victory, someone has to agonize, sweat, bleed, and wet his or her hands in human secretions and excreta. Likewise, technological gimmicks have a limited place in emergency abdominal surgery, which is the domain of the surgeon's brain and hands. Some readers may object to this military metaphor, but the truth of the matter is that,

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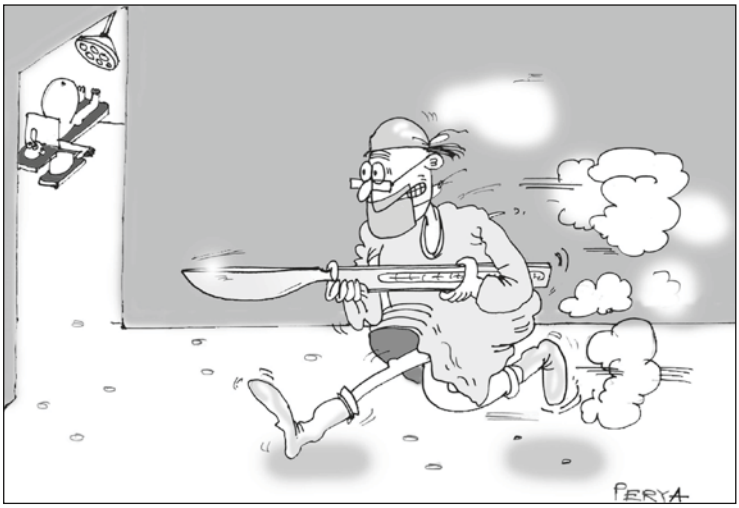


**Fig. 1.1.** "Think as an infantry soldier…"

as with infantry action, emergency abdominal surgery shares a few simple rules—developed in the trenches and during offensives—rules that are the key to victory and survival. Such a code of battle echoes the "best" management of abdominal emergencies.

| | **Infantry action** | **Emergency abdominal surgery** |
|---|---|---|
| Rule 1 | Destroy your enemy before he destroys you | Save lives |
| Rule 2 | Spare your own men | Reduce morbidity |
| Rule 3 | Save ammunition | Use resources rationally |
| Rule 4 | Know your enemy | Estimate severity of disease |
| Rule 5 | Know your men | Understand the risk-benefit ratio of your therapy |
| Rule 6 | Attack at "soft" points | Tailor your management to the disease and the patient |
| Rule 7 | Do not call for air force support in a hand-to-hand battle | Do not adopt useless gimmicks—use your mind and hands |
| Rule 8 | Conduct the battle from the front line—not from the rear | Do not take and accept decisions over the phone |
| Rule 9 | Take advice from the generals, but the decision is yours | Procure and use consultation from "other specialties" selectively |
| Rule 10 | Avoid friendly fire | Reduce iatrogenesis |
| Rule 11 | Maintain high morale among your troops | Be proud in providing the "best" management |

There are many ways to skin a cat and you know from your various surgical mentors that different clinical pathways may arrive at a similar outcome. However, only one of the diverse pathways is the "correct one"—thus, the best.

To be considered as such, the "preferred pathway" has to save life and decrease morbidity in the most efficient way. Look at this example: You can manage perforated acute appendicitis using two different pathways—both leading to an eventual recovery, and both considered absolutely appropriate.

| Pathway 1 | Pathway 2 |
|---|---|
| Young male—right lower quadrant peritonitis | Young male—right lower quadrant peritonitis |
| | CT scan |
| | Attempted laparoscopic appendectomy |
| | Conversion to open appendectomy |
| Appendectomy for gangrenous appendicitis—3 hrs after admission | Appendectomy for gangrenous appendicitis—24 hrs after admission |
| Primary closure of the wound | Wound left open |
| 24 hrs of postoperative antibiotics | 5 days of postoperative antibiotics |
| | Secondary closure of wound |
| Discharge home on the second postoperative day | Discharge home on the seventh postoperative day |

Both pathways are OK, right? Yes, but pathway 1 clearly is the "best" one: safer, faster, and cheaper.

Today, many options exist to do almost anything. Just by clicking open Medline you are overwhelmed with articles that can prove and justify almost any management pathway, with people practicing "surgical acrobatics" for the mere sake of doing so. Data and theory are everywhere—the sources are numerous, but what you really need is *wisdom* to enable you to apply correctly the knowledge you already have and constantly gather.

## General Philosophy (Fig. 1.2)

"There is nothing new in the story…" Winston Churchill said, "want of foresight, unwillingness to act when action would be simple and effective, lack of clear thinking, confusion of counsel until the emergency comes, until self preservation strikes its jarring gong…" How true is this Churchillian wisdom when applied to emergency surgery. How often do we forget old—written in stone—principles while reinventing the wheel?

**The best management in each section of this book is based on the following elements:**

- Old, established principles (don't reinvent the wheel)
- Modern, scientific understanding of inflammation and infection
- Evidence-based surgery (see "Evidence" section)
- Personal experience


**Fig. 1.2.** "General philosophy…"

## The Inflamed Patient (Fig. 1.3)

Think about your patient as being *inflamed* by myriad inflammatory mediators, generated by the primary disease process, be it inflammatory, infectious, or traumatic. Those local (e.g., peritonitis) and systemic mediators (systemic inflammatory response syndrome, SIRS) are the ones that may lead to organ dysfunction or failure and the eventual demise of your patient. The greater the inflammation, the sicker the patient and the higher the expected mortality and morbidity. Consider also that anything you do in attempting to halt your patient's inflammation may in fact contribute to it—adding wood

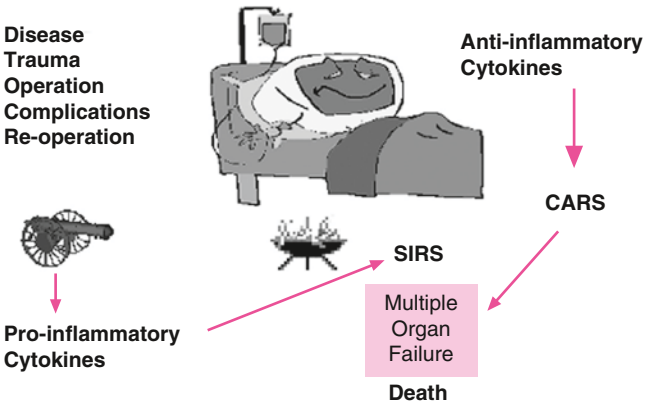


**Fig. 1.3.** The inflamed surgical patient. *SIRS* systemic inflammatory response syndrome; *CARS* compensatory anti-inflammatory syndrome. (Read the 1996 classic by the late Bone)

to the inflammatory fire. Excessive surgery, inappropriately performed and too late, just adds nails to your patient's coffin. Remember also that SIRS is antagonized by CARS (compensatory anti-inflammatory syndrome), mediated by anti-inflammatory cytokines, which in turn promotes the immune suppression and infections that are so common after major operations and severe trauma. The philosophy of treatment that we propose maintains that to cure or minimize the inflammatory processes and the anti-inflammatory response, management should be accurately tailored to the individual patient's disease. As the punishment should fit the crime, so should the remedy fit the disease. A well-trained foot soldier does not fire indiscriminately in all directions.

## Evidence

A few words about what we mean when we talk about "evidence":

| Evidence level | Description |
|---|---|
| I | A scientifically sound randomized controlled trial |
| II | Randomized controlled trial with methodological "problems" |
| III | Nonrandomized concurrent cohort comparison |
| IV | Nonrandomized historical cohort comparison |
| V | A case series without controls |
| **To this "official" classification, we wish to add another three categories frequently used by surgeons around the world:** | |
| VI | "In my personal series of X patients [never published], there were no complications." |
| VII | "I remember that case …." |
| VIII | "This is the way I do it, and it is the best." |

Note that level V studies form the bulk of surgical literature dealing with abdominal emergencies, whereas levels VI and VII evidence is the main form of evidence used by surgeons in general (think about your departmental meetings), and level VIII may remind you of your chair.

You should educate yourself to think in terms of levels of evidence and resist local dogmas. We believe that support for much of what we write here is available in the published literature, but we choose not to cite it here because it is not that kind of book. When high-level evidence is not available, we have to use an individual approach and common sense, and that is much of what this book is about.

You can get away with a lot … but not always. Most patients treated according to the above-mentioned pathway 2 will do well, but a few will not. The following pages will help you to develop your own judgment—pointing to the correct pathway in any situation. This is obviously not a "bible," but it is based on

thorough knowledge of the literature and vast personal experience. So, wherever you are—in India, Norway, Chile, Canada, or Palestine and whatever your resources—the approach to emergency abdominal surgery is the same. So, come and join us: to save lives, decrease morbidity, do it "correctly"—and attain glory.

"The glory of surgeons is like that of actors, which lasts only for their own lifetime and can no longer be appreciated once they have passed away. Actors and surgeons … are all heroes of the moment." (Honore de Balzac, 1799–1850)

"The operation is a silent confession to the surgeon's inadequacy." (John Hunter, 1728–1793)

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# 2 A Brief History of Emergency Abdominal Surgery

Harold Ellis

*"In the study of some apparently new problems we often make progress by reading the work of the great men of the past."* (Charles H. Mayo, 1865–1939)

From the earliest days until comparatively modern times, surgeons were ignorant about **the** causes of the vast majority of acute abdominal emergencies and equally ineffectual in their treatment. They were, of course, well familiar with abdominal trauma and the dire consequences of perforating injuries of the belly, the great majority of which would be fatal. Thus, in the Bible we read in the Book of Judges:

> But Ehud made him a dagger, which had two edges of a cubit length, and he did gird it under the raiment of his right thigh. And he brought the present unto Eglon, King of Moab. And Eglon was a very fat man. … And Ehud put forth his left hand and took the dagger from his right thigh, and thrust it into his belly. And the haft went in after the blade and the fat closed over the blade, so that he could not draw the blade out of his belly; and the dirt came out. … And behold their Lord was fallen down dead on the earth.

Occasionally, a faecal fistula would form, and the patient survived. That great sixteenth century French military surgeon, Ambroise Paré, recorded in his *Case Reports and Autopsy Records*:

> In time I have treated several who recovered after having had wounds by sword or pistol pass through their bodies. One of these, in the town of Melun, was the steward of the Ambassador of the King of Portugal. He was thrust through with a sword, by which his intestines were wounded, so when he was dressed a great deal of fecal matter drained from the wound, yet the steward was cured.

Occasionally, a prolapsed loop of bowel, projecting through a lacerated abdominal wound, might be successfully reduced. Still less often, an enterprising surgeon might suture a laceration in such a loop and thus save the patient's life.

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In 1676 Timothy Clark recorded the case of a butcher who attempted suicide with his butcher's knife in the village of Wayford in the country of Somerset, located in the southwest corner of England. Three days later, a surgeon who Clark does not name replaced the prolapsed gut, removed extruded omentum and prolapsed spleen and the patient recovered. Clark, himself, in 1633 had removed the spleen of a dog with survival, thus showing that the organ was not essential to life and confirming an observation made by Vesalius a century beforehand.

*Strangulated hernias* were also well known to ancients. Treatment usually consisted of forcible manipulative reduction, which was aided by hot baths, poultices, and the use of the head-down, feet-up position. Sometimes their efforts succeeded, but there was, of course, a dire risk of rupture of the gut, especially in advanced cases. William Cheselden in 1723 reported the case of a woman in her 73rd year with a strangulated umbilical hernia. At operation, he resected 26 in. of gangrenous intestine. She recovered with, of course, a persistent fecal fistula. The extreme danger of strangulated hernia is well demonstrated by the fact that Queen Caroline, wife of George II of England, died of a strangulated umbilical hernia at the age of 55 in 1736.

*Acute abdominal emergencies* have no doubt affected humankind from its earliest existence, yet it has only been in comparatively recent times—the past couple of hundred years—that the pathology and then the treatment of these conditions were elucidated. This is because over many centuries post-mortem examinations were either forbidden or frowned on in most societies. Operations on the abdomen were performed rarely, if at all, until the beginning of the nineteenth century. So, what Berkeley Moynihan called "the pathology of the living", the pathology of the abdominal cavity as revealed in the operating theatre, awaited to a large extent the development of anaesthesia in the 1840s and antiseptic surgery in the 1870s.

Knowledge of the causes of the acute abdomen advanced little in the 2,000 years following the days of Hippocrates in the fifth century BC. The Greek and Roman doctors were keen clinical observers. They recognized that, from time to time, a deep abdominal abscess might discharge spontaneously or be amenable to surgical drainage with recovery of the patient. Every other serious abdominal emergency was given the name of "ileus" or "iliac passion" and was considered to be due to obstruction of the bowels. Of course, the fatal abdominal emergencies they were seeing were indeed due either to mechanical obstruction or to the paralytic ileus of general peritonitis. Thus in Hippocrates we read:

> In ileus the belly becomes hard, there are no motions, the whole abdomen is painful, there are fever and thirst and sometimes the patient is so tormented that he vomits bile. … Medicines are not retained and enemas do not penetrate. It is an acute and dangerous disease.

Over the centuries there was little to offer the patient beyond poultices to the abdomen, cupping, bleeding, purgation and enemas, all of which probably did more harm than good. It was not until 1776 that William Cullen, of Edinburgh, coined the term *peritonitis* for inflammation of the lining membrane of the abdominal cavity and its extensions to the viscera. However, he did not think exact diagnosis of great importance since "when known, they do not require any remedies besides those of inflammation in general".

## Appendicitis

Lorenz Heister, of Helmstadt in Brunswig, must be given credit for the first description of the appendix as the site of acute inflammation, reporting this at an autopsy in 1755. For more than a century after this there were occasional autopsy reports, but most cases were unrecognized or labelled "typhilitis", "perityphilitis" or "iliac passion".

In 1848 Henry Hancock, of Charing Cross Hospital, London, reported the drainage of an appendix abscess in a young woman who was 8 months pregnant. She recovered, but in spite of Hancock's plea, so fixed was the idea that it was useless to operate once peritonitis was established that his advice was ignored for some 40 years. Indeed, it was a physician, not a surgeon, who advised appendicectomy and early diagnosis. This was Reginald Fitz, professor of medicine at Harvard, who, in 1886, published a review of 257 cases, which clearly described the pathology and clinical features and advised removal of the acutely inflamed organ or, in the presence of an abscess, surgical drainage. Fitz's advice was taken up rapidly in the United States. Thomas Morton of Philadelphia was the first to report, in 1887, the correct diagnosis and successful removal of a perforated appendix (although Robert Lawson Tait as early as 1880 had a similar case, he did not report this until 1890). The surge in early diagnosis and operative treatment was particularly pioneered by Charles McBurney of the Roosevelt Hospital, New York, who described "McBurney's point" and devised the muscle split incision, and J.B. Murphy of Chicago, who emphasized the shift in pain in "Murphy's sequence". In 1902 Fredrick Treves, of the London Hospital, drained the appendix abscess of King Edward VII, 2 days before the coronation, and did much to raise the general public's awareness of the disease.

## The Ruptured Spleen

The spleen is the most commonly injured viscus in closed abdominal trauma, yet there was surprising diffidence among the pioneer abdominal surgeons to perform a splenectomy on these exsanguinating patients—in spite of the

fact that Jules Péan of Paris had performed a successful splenectomy on a girl with a massive splenic cyst in 1867. Two unsuccessful attempts to save life in splenic rupture were reported in 1892 by Sir Arbuthnot Lane of Guy's Hospital, London, and three more fatal cases were recorded by Friedrich Trendelenburg in Leipzig the following year. The wording of these case reports strongly suggests that had blood transfusion been available, the patients might well have survived.

It fell to Oskar Riegner in Breslau to perform the first splenectomy for a pulped spleen with survival in 1893. The patient, a lad of 14, was found to have the spleen completely severed and there were 1.5 L of blood in the abdomen. Normal saline was given subcutaneously into all four limbs. His recovery was complicated by gangrene of the left foot, which required amputation, but he left the hospital, complete with artificial limb, 5 months after his splenectomy.

## Intestinal Obstruction

Not surprisingly, early attempts to deal with large bowel obstruction (usually due to a left-sided colonic cancer) comprised performance of a colostomy. The first attempt to do this was made by Pillore of Rouen in 1776. He actually carried out a cecostomy on a wine merchant with gross abdominal distension due to a recto-sigmoid growth. The operation produced great relief, but the patient died on the 28th day because of necrosis of a loop of jejunum, brought about by the large amounts of mercury given in the pre-operative attempts to overcome the obstruction. It remained for Pierre Fine of Geneva, in 1797, to perform a successful transverse colostomy. The patient, a lady of 63 with an obstructing sigmoid growth, died 14 weeks later with ascites.

Not until the introduction of anaesthesia and antisepsis could routine resection of bowel cancers be performed, the first success in this era being reported by Vincent Czerny in Heidelberg in 1879. It was soon realized that resection of the obstructed colon was very likely to result in a fatal anastomotic leak. Exteriorization of the growth, with formation of a double-barrelled colostomy and its subsequent closure was introduced by Frank Thomas Paul of Liverpool in 1895, and by Johannes von Mikulicz-Radecki of Breslau a little later. This procedure, the Paul-Mikulicz operation, was shown by the latter to reduce morality in his own cases from 43% with primary resection to 12.5% with the exteriorization method.

With its vivid clinical features of intestinal obstruction in a baby, passage of red current jelly stools, a palpable abdominal mass and sometimes a prolapsing mass to be felt per rectum or even seen to protrude through the anal verge, it is not surprising that intussusception in children was one of the earliest specific pathologies of the acute abdomen to be recognized. Treatment was expectant, with the use of enemas or rectal bougies, in attempts to reduce the mass. Surgeons were encouraged to do this by very occasional reports of success and still rarer

accounts of recovery following the passage of the sloughed gangrenous bowel per rectum. The first operative success was reported by Sir Jonathan Hutchinson, of the London Hospital, in 1871. His patient, a girl aged 2, had her intussusception reduced through a short mid-line incision, the operation requiring just a few minutes. Hutchinson's meticulous report tabulated 131 previously recorded cases, which make sad reading indeed.

There was a downside to this new abdominal surgery. It was not long after this new era commenced that the first reports appeared of small bowel obstruction due to post-operative adhesions. Thomas Bryant of Guy's Hospital recorded the first example in 1872—a fatal case following an ovariotomy. A second fatality, 4 years after removal of an ovarian mass, was reported in 1883 by William Battle of London. Today, post-operative adhesions and bands account for some three-quarters of all cases of small bowel obstructions in the Western World.

## Perforated Peptic Ulcer

Untreated, a perforated peptic ulcer nearly always results in fatal peritonitis. Unsuccessful attempts at repair were made by Mikulicz-Radecki in 1884 and by Czerny in 1885 and subsequently by a number of other surgeons. This depressing series came to an end under most difficult circumstances. In 1892, Ludwig Heusner of Wuppertal, Germany, repaired a perforated gastric ulcer high up on the lesser curve in a 41-year-old businessman with a 16 hour history; the operation was performed in the middle of the night by candlelight! The convalescence was complicated by a left-sided empyema, which required drainage. Two years later, Thomas Morse, in Norwich, published the successful repair of a perforation near the cardia in a girl of 20. With these two successes, operation for this condition became routine. Interestingly, gastric ulcer at the turn of the twentieth century was far commoner than duodenal ulcer and was especially found in young women.

## Ruptured Ectopic Pregnancy

Until 1883 a ruptured ectopic pregnancy was a death sentence. This is surprising because the early pioneers of abdominal surgery, going back to pre-anaesthetic era, were, in the main, concerned with removal of ovarian masses. Indeed, the first elective abdominal operation for a known pathology was the removal of a massive ovarian cyst by Ephraim McDowell in Danville, Kentucky, in 1809. Yet, for some inexplicable reason, the surgeon would stand helplessly by the bedside and watch a young woman, in the most useful time of her existence, exsanguinate from her ruptured tube.

The first surgeon to perform successful surgery in this condition was Robert Lawson Tait, of Birmingham, whom we have already mentioned performing a successful appendicectomy in 1880. Tait was asked to see a girl with a ruptured ectopic pregnancy by Dr. Hallwright, a general practitioner. Hallwright suggested that Tait should remove the ruptured tube. Tait recorded:

> The suggestion staggered me and I am afraid I did not receive it favourably. I declined to act and a further haemorrhage killed the patient. A post-mortem examination revealed the perfect accuracy of the diagnosis. I carefully inspected the specimen that was removed and found that if I had tied the broad ligament and removed the tube I should have completely arrested the haemorrhage, and I now believe that had I done this the patient's life would have been saved.

Eighteen months later, Tait operated on a clearly dying patient, the first occasion in which such an operation was performed. The patient, in those pre-transfusion days, died of exsanguination. Finally, in March 1888, Tait performed a successful salpingectomy on such a case, who survived even though, at operation, the abdomen was full of clot. Years later, he was able to report 39 cases, with but two deaths, including the first.

## Envoi

Even today, the acute abdomen presents a diagnostic and therapeutic challenge to the surgeon. This is in spite of the fact that we have the ancillary aids of radiology, imaging, biochemical and haematological studies to help the diagnosis and blood transfusion, fluid replacement, nasogastric suction, antibiotics and skilled anaesthetists to assist with therapy.

> "Let us therefore look back with a mélange of amazement, pride, and humility at the efforts of our surgical forefathers as they paved the way for us in the management of this fascinating group of diseases." (Harold Ellis)

## Editorial Comment

We are proud to offer this chapter by Professor Ellis of London: a renowned surgeon, educator, writer, editor, anatomist, and surgical historian. Among his many books, we would particularly recommend *Operations That Made History* and *A Brief History of Surgery*.

# Before the Operation

A

# 3 The Acute Abdomen[1]

Moshe Schein

*"For the abdominal surgeon it is a familiar experience to sit, ready scrubbed, and gowned, in a corner of the quiet theatre, with the clock pointing midnight. … In a few minutes the patient will be wheeled in and another emergency laparotomy will commence. This is the culmination of a process which began a few hours previously with the surgeon meeting with and examining the patient, reaching a diagnosis, and making a plan of action."* (Peter F. Jones)

*"The general rule can be laid down that the majority of severe abdominal pains which ensue in patients who have been previously fairly well, and which last as long as six hours, are caused by conditions of surgical import."* (Zachary Cope, 1881–1974)

Simply stated, the term *acute abdomen* refers to abdominal pain of short duration that requires a decision regarding whether an urgent intervention is necessary. This clinical problem is the most common cause for you to be called to provide a surgical consultation in the emergency room (ER) and serves as a convenient gateway for a discussion of the approach to abdominal surgical emergencies.

## The Problem

Most major textbooks contain a long list of possible causes for acute abdominal pain, often enumerating 20–30 "most common" etiologies. These "big lists" usually go from perforated peptic ulcer down to such esoteric causes as porphyria and black widow spider bites. The lists are popular with medical students but totally useless for practical guys like you.

The experienced surgeon called on to consult a patient with acute abdominal pain in the ER in the middle of the night simply does not work this way. He or she does not consider the 50 or so "most likely" causes of acute abdominal pain from the list and does not attempt to rule them out one by one. Instead, the smart surgeon tries to identify a *clinical pattern* and to decide on a course of action from a *limited menu* of management options. This chapter demonstrates how the multiple etiologies for acute abdominal pain actually converge into a

[1]Asher Hirshberg, MD, contributed to this chapter in the first edition of this book.

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small number of easily recognizable clinical patterns. Once recognized, each of these patterns dictates a specific course of action.

## The Acute Abdomen: Clinical Patterns and Management Menus

### *The Management Options*

Seeing a patient with an acute abdomen in the ER, you have only the four possible management options listed in Table 3.1. The last option (discharge) deserves some consideration. Many patients with acute abdominal pain undergo a clinical examination and a limited workup—which today in many centers may include a computed tomographic (CT) scan—only to be labeled as having "nonspecific abdominal pain" (NSAP) and then discharged. NSAP is a clinical entity, albeit an ill-defined one. It is a type of acute abdominal pain that is severe enough to bring a patient to seek medical attention (Fig. 3.1). The patient's physical examination and diagnostic workup are negative, and the pain is self-limiting and usually does not recur. It is important to keep in mind that, in an ER setting, more than half the patients presenting with acute abdominal pain have NSAP, with acute appendicitis, acute cholecystitis, and "gynecological causes" the most common "specific" conditions. But, the exact pathology you see depends, of course, on your geographical location and pattern of practice. Just remember that patients discharged home labeled with the diagnosis of NSAP have an increased probability of a subsequent diagnosis of abdominal cancer. Therefore, referral for elective investigations may be indicated.

### *The Clinical Patterns*

The acute abdomen usually presents as one of the five distinct and well-defined clinical patterns listed in Table 3.2. Two additional patterns (trauma and gynecological) are addressed elsewhere in this volume. Occasionally, a mixed picture of obstruction/peritonitis may present. Each of these clinical patterns dictates a specific management option from the menu. Your task is to identify the specific pattern to know how to proceed.

**Table 3.1.** Management options

| |
|---|
| Immediate operation ("surgery now") |
| Preoperative preparation and operation ("surgery tomorrow morning") |
| Conservative treatment (active observation, intravenous fluids, antibiotics, etc.) |
| Discharge home |

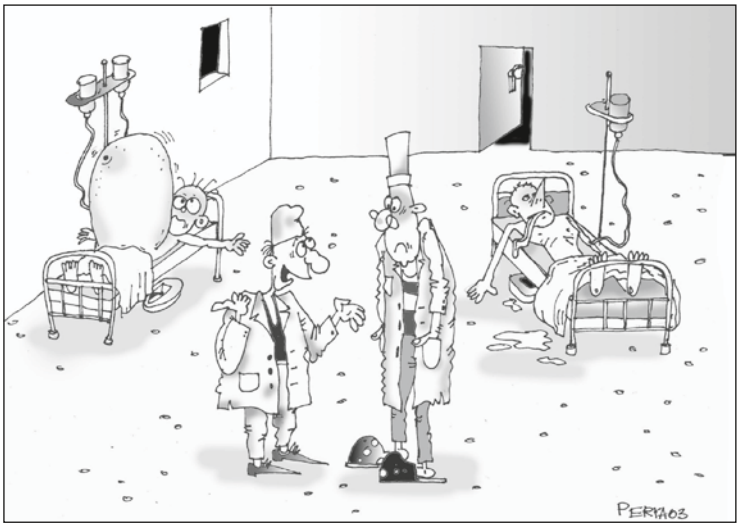


**Fig. 3.1.** "Which of them has an 'acute abdomen'?"

**Table 3.2.** Clinical patterns

| |
|---|
| Abdominal pain and shock |
| Generalized peritonitis |
| Localized peritonitis (confined to one quadrant of the abdomen) |
| Intestinal obstruction |
| "Medical" illness |

### Abdominal Pain and Shock

Abdominal pain and shock offer the most dramatic and least-common clinical pattern of the acute abdomen. The patient typically presents pale and diaphoretic, in severe abdominal pain, and with hypotension, the so-called abdominal apoplexy. The two most common etiologies of this clinical pattern are a **ruptured ectopic pregnancy** and a **ruptured abdominal aortic aneurysm** (› Chaps. 33 and 41, respectively). Here, the only management option is immediate surgery—now. No time should be wasted on "preparations" and on ancillary investigations. Losing a patient with abdominal apoplexy in the CT scanner is a cardinal, and unfortunately not too rare, sin. Note that other abdominal emergencies may also present with abdominal pain and shock due to fluid loss into the "third space." This is not uncommon in patients with **intestinal obstruction** (› Chap. 21), **acute mesenteric ischemia** (› Chap. 23), or **severe acute pancreatitis** (› Chap. 19)—particularly if neglected or superimposed on a marginal or premorbid cardiovascular system.

### Generalized Peritonitis

The clinical picture of generalized peritonitis consists of diffuse severe abdominal pain in a patient who looks sick and toxic. The patient typically lies motionless and has an extremely tender abdomen with "peritoneal signs" consisting of boardlike rigidity, rebound tenderness, and voluntary defense guarding. Surprisingly enough, less-experienced clinicians occasionally miss the diagnosis entirely. This is especially common in the geriatric patient, who may have weak abdominal musculature or may not exhibit the classical peritoneal signs. The most common error in the physical examination of a patient with acute abdominal pain is rough and "deep" palpation of the abdomen, which may elicit severe tenderness even in a patient without any abdominal pathology. Palpation of the abdomen should be very gentle and should not hurt the patient. The umbilicus is the shallowest part of the abdominal wall where the peritoneum almost touches the skin. Thus, one of the most effective maneuvers in the physical examination of a patient suspected of having peritonitis is gentle palpation in the umbilical groove, where tenderness is very obvious. We appreciate that at this stage of your surgical career you do not need a detailed lecture on the examination of the acute abdomen. Forgive us, however, for emphasizing that the absence of rebound tenderness means nothing, and that a good way to elicit peritoneal irritation is by asking the patient to cough, by shaking (gently) the patient's bed, or by very gentle percussion of the abdomen.

The three most common causes of generalized peritonitis in adults are a **perforated ulcer** (Chap. 18), **colonic perforation** (Chap. 25), and **perforated appendicitis** (Chap. 28). Classically, with exceptions listed here and in the individual chapters, the management of a patient with diffuse peritonitis is preoperative preparation and operation (*surgery tonight*). The patient should be taken to the operating room only after adequate preoperative preparation as outlined in Chap. 6.

An important exception to this management option is the patient with acute pancreatitis. Although most patients with acute pancreatitis present with mild epigastric tenderness, the occasional patient may present with a clinical picture mimicking diffuse peritonitis (Chap. 19). As a precaution against misdiagnosing these patients, it is essential practice always to measure the serum amylase (and lipase) in any patient presenting with significant abdominal symptoms (Chap. 4). An exploratory laparotomy in a patient suffering from acute severe pancreatitis may lead to disaster. Remember: He put the pancreas in the back because God did not want surgeons messing with it.

### Localized Peritonitis

In the patient with localized peritonitis, the clinical signs are confined to one quadrant of the abdomen. In the right lower quadrant (RLQ), the most common cause of localized peritonitis is **acute appendicitis** (Chap. 28). In the right upper

quadrant (RUQ), it is **acute cholecystitis** (Chap. 20.1), and in the left lower quadrant (LLQ), it is **acute diverticulitis** (Chap. 26). Peritonitis confined to the left upper quadrant (LUQ) is uncommon, making this quadrant the "silent one."

As a general rule, localized peritonitis is often not an indication for a surgery-tonight policy. Instead, when the diagnosis is uncertain, it may initially be treated conservatively. The patient is admitted to the surgical floor, given intravenous antibiotics (e.g., if the diagnosis of acute cholecystitis or diverticulitis is entertained) and hydration, and is actively observed by means of serial physical exams. **Time is a superb diagnostician;** when you return to the patient's bedside after a few hours, you may find all the previously missing clues. Of course, this is after you have consulted the relevant chapters in this book.

The exception to this rule is, of course, a tender RLQ, for which the working diagnosis is acute appendicitis, and appendectomy may therefore be indicated. However, if there is a palpable mass in the RLQ, the working diagnosis is an "appendiceal phlegmon," for which conservative management would be appropriate, at least initially (Chap. 28). In young women, RLQ signs may be gynecological in origin, and continued conservative management may also be appropriate in this situation (Chap. 33).

The management of **acute cholecystitis** varies among surgeons. While past experience taught us that most of these patients would respond to antibiotics, "modern" surgeons prefer to operate early on a "hot" gallbladder—usually the next morning or whenever the operating room schedule permits (Chap. 20.1).

### Intestinal Obstruction

The clinical pattern of intestinal obstruction consists of central, colicky abdominal pain, distension, constipation, and vomiting.

As a general rule, the earlier and more pronounced the vomiting, the more proximal the site of obstruction is likely to be. But, the more marked the distension, the more distal is the site of obstruction. Thus, vomiting and colicky pain are more characteristic of small bowel obstruction, whereas constipation and gross distension are typical of colonic obstruction. However, the distinction between these two kinds of obstruction usually hinges on the plain abdominal X-ray. There are two management options for these patients: conservative treatment or operative treatment after adequate preparation. The major problem with intestinal obstruction is not in making the diagnosis but in deciding on the appropriate course of action. If the patient has a history of previous abdominal surgery and presents with small bowel obstruction but without signs of peritonitis, the working diagnosis is "simple" adhesive small bowel obstruction. The initial management of these patients is conservative, with intravenous fluids and nasogastric tube decompression. If the obstruction is complete (e.g., no gas in the colon above the peritoneal reflection of the rectum), the chances of spontaneous

resolution are small, and some surgeons would opt for an operative intervention. In the presence of clinical peritonitis, fever, and elevated white blood cell count, the indication for laparotomy is clear-cut ( see Chap. 21).

**There are three classical pitfalls with small bowel obstruction:**

- The obese elderly lady with no previous surgical history who presents with small bowel obstruction, where an *incarcerated femoral hernia* can easily be missed if not specifically sought
- The elderly patient with a "simple" adhesive small bowel obstruction who improves on conservative treatment and is discharged only to come back later with a large *tumor mass in the right colon*
- The elderly lady whose "partial" small bowel obstruction "resolves and recurs" intermittently and is finally diagnosed as *gallstone ileus*
- The patient with a history of previous gastric surgery who presents with intermittent episodes of obstruction originating from a *bezoar* in the terminal ileum

Unlike small bowel obstruction, colon obstruction is always an indication for surgery—"tonight or tomorrow" but usually tomorrow. A plain abdominal X-ray cannot make the diagnosis since functional **colonic pseudo-obstruction** (Ogilvie's syndrome) or chronic megacolon cannot reliably be distinguished from a mechanical obstruction. Thus, these patients usually undergo either fiber-optic colonoscopy or a contrast enema (with or without CT) to clinch the diagnosis. The management option for these patients is operation after adequate preparation (Chap. 25).

## Important Medical Causes

While there is a large number of nonsurgical causes that may result in acute abdominal pain, two must be kept constantly in your mind: inferior wall myocardial infarction (MI) and diabetic ketoacidosis. A laparotomy for porphyria or even basal pneumonia is an unfortunate surgical (and medicolegal) occurrence, but inadvertently operating on a patient with an undiagnosed inferior wall MI or diabetic ketoacidosis may well be a lethal mistake. As a surgeon, you should strive to be a better physician than the internists, and wouldn't it be fun to show them a "medical" diagnosis they had missed.

Wherever you practice, you may be exposed to a growing number of HIV-positive patients suffering from AIDS, who are susceptible to a large number of abdominal conditions, which can produce or mimic an "acute abdomen." In (Chap. 36) we tell you how to deal with these patients, most of them being best treated without an operation.

## Conclusion

The multiple etiologies of the acute abdomen converge to five distinct and well-defined clinical patterns, each of which is associated with a specific management option. You should be familiar with these patterns and with the various management options. You should also keep in mind the classical pitfalls inherent in this common surgical condition to avoid gross errors in the surgical care of such patients. After all, you already have enough cases to present at the morbidity and mortality (M & M) meeting, don't you (› Chap. 59)?

**CT is the KING!**

Many of us were raised on the dictum that clinical peritonitis is an indication for abdominal exploration (be it by laparotomy or laparoscopy). The notions that "peritonitis is an indication for operation" and that "only skin separates us from the diagnosis" developed before the days of modern abdominal imaging, but is this still true today? We do not think so. We believe that modern abdominal imaging has revolutionized emergency abdominal surgery, and that if you have immediate access to abdominal CT or ultrasound, you have to use it. This—as is discussed in many of the following chapters—will avoid an operation in many patients or make operative treatment less invasive and more specific. Thanks to the abdominal CT, the abdomen is no longer a black box. Use abdominal imaging liberally for the benefit of your patients—especially when the diagnosis is not clearly evident. It is okay to operate on a young man with classical features of acute appendicitis without a preoperative CT, but a woman of childbearing age needs abdominal imaging (to exclude gynecological conditions) and so do elderly patients, in whom other pathologies are more likely. All this is just common sense really.

> "It is as much an intellectual exercise to tackle the problems of belly ache as to work on the human genome." (Hugh Dudley)
>
> Yes, what's common is common and what's rare is rare but rare things can be lethal—always keep them in mind!

## Who Should Look After the "Acute Abdomen" and Where?

> Everybody's business is nobody's business.

The majority of patients suspected of having an acute abdomen or other abdominal emergency do not require an operation. Nevertheless, it is you—the

surgeon—who should take, or be granted, the leadership in assessing, excluding, or treating this condition or at least play a major role in leading the managing team. To emphasize how crucial this issue is, we dedicate an entire section of this chapter to it—although its scope would fit into a paragraph.

Unfortunately, in "real life," surgeons are often denied the primary responsibility. Too often, we see patients with **mesenteric ischemia** (Chap. 23) rotting away in medical wards, the surgeon being consulted "to evaluate the abdomen" only when the bowel is dead, and the patient is soon to be. A characteristic scenario is a patient with an abdominal surgical emergency, admitted under the care of nonsurgeons who undertake a series of unnecessary, potentially harmful, and expensive diagnostic and therapeutic procedures. Typically, internists, gastroenterologists, infectious disease specialists, and radiologists are involved, each prescribing personal wisdom in isolation (Fig. 3.2). When, finally, called in, the surgeon finds the condition difficult to diagnose, partially treated, or maltreated. Eventually, the indicated operation is performed, but too late, thus carrying higher morbidity and mortality. The etiology of such chaos is not entirely clear. Motives of power, ego, and financial considerations are surely involved.

The team approach to the acutely ill surgical patient should not be discarded. The team, however, should be led and co-ordinated by a general surgeon. The surgeon is the one who knows the abdomen from within and without. The surgeon is the one qualified to call in consultants from other specialties, to order valuable tests and to veto those that are superfluous and wasteful. And, above all, the surgeon is the one who will eventually decide that enough is enough, and the patient needs to be taken to the operating room.

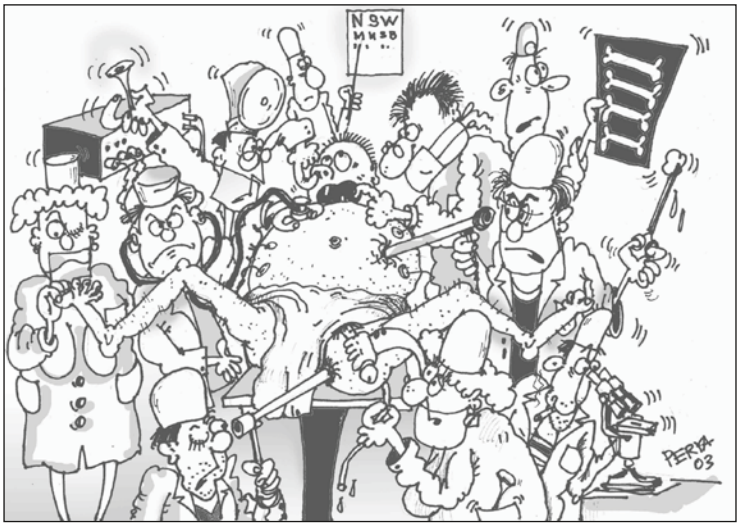


**Fig. 3.2.** "Who is responsible?"

**When you decided to become a general surgeon you became the captain of the ship, navigating the deep ocean of the abdomen. Do not abandon your ship while the storm rages on!**

Continuity of care is a *sine qua non* in the optimal care of the acute abdomen as the clinical picture, which may change rapidly, is a major determinant in the choice of therapy and its timing. Such patients need to be reassessed frequently by the same clinician, who should be a surgeon. Any deviation from this may be hazardous to the patient; this is our personal experience and that which is repeated ad nauseum in the literature. Why don't we learn? The place for the patient with an acute abdominal condition is on the surgical floor, in the surgical intensive care unit (ICU), or in the operating room and under the care of a surgeon—yourself. Don't duck your responsibilities!

Only 10 or 20 years ago, when we were residents, an acute abdomen and clinical evidence of peritonitis mandated an operation. Today, we are smarter. Judicious use of diagnostic modalities (see Chap. 4) and better understanding of the natural history of various disease processes allow us to decrease mortality and morbidity by being less invasive and more selective and, in general, to achieve more by doing less.

**The key for the "best" outcome of the acute abdomen is:**

- Operate only when necessary and do the minimum possible
- Do not delay a necessary operation and do the maximum when indicated

**Advice:** When you finish this book, go and buy yourself *Cope's Early Diagnosis of the Acute Abdomen*. Zachary Cope, who died in 1974, published the first edition of his book in 1921. The current edition is the 20th! You cannot be a real general surgeon without reading this book. Or can you?

# 4 Rational Diagnostic Procedures[1]

Moshe Schein

*Believe nobody—question everything.*

*"To open an abdomen and search for a lesion as lightly as one would open a bureau drawer to look for the laundry, may mean lack of mental overwork to the surgeon, but it means horror to the patient."* (J. Chalmers Da Costa, 1863–1933)

When treating a patient with acute abdominal pain, it is tempting to make extensive use of ancillary investigations. This leads to the emergence of "routines" in the emergency room (ER), by which every patient with acute abdominal pain undergoes a plain X-ray of the abdomen (AXR) and a series of blood tests, which typically include a complete blood count, routine blood chemistry, and serum amylase. These "routine" tests have a very low diagnostic yield and are not cost effective. However, they are also an unavoidable part of life in the ER and are often obtained before the surgical consultation.

For some patients who on examination have a clear-cut *diffuse peritonitis*, no imaging may be necessary because a laparotomy is indicated. But, what appears clear-cut to the experienced surgeon may be less so for you. Bear in mind the following caveats:

- Intestinal distension, associated with obstruction or inflammation (e.g., enteritis or colitis) may produce diffuse abdominal tenderness—mimicking "peritonitis." The "whole" clinical picture as well as the AXR will guide you toward the proper diagnosis (Chaps. 21 and 25).
- Acute pancreatitis may present with clinical acute peritonitis. You should always obtain a serum *amylase* or *lipase* level in every patient with significant abdominal pain to avoid falling into the not-so-uncommon trap of unnecessarily and dangerously operating on acute pancreatitis (Chap. 19).
- Clostridium difficile enterocolitis should be considered in any patient who receives or has recently received any quantity of antibiotics. This may present—from the beginning—as an acute abdomen without diarrhea. Here, the optimal initial management is medical and not a laparotomy; sigmoidoscopy or computed tomography (CT) may be diagnostic (Chap. 24).

[1]Asher Hirshberg, MD, contributed to this chapter in the first edition of the book.

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## Chest X-ray

A chest X-ray (CXR) is routinely obtained to search for free air under the diaphragms, which is demonstrated in the majority of patients with perforated peptic ulcer (› Chap. 18) but less frequently when colonic perforation is the underlying problem (› Chap. 25). Remember that free air is better seen on an erect CXR than AXR. Free intraperitoneal air is not always caused by a perforated viscus, and it is not always an indication for a laparotomy. There is a long list of "nonoperative" conditions that may produce free intraperitoneal air, such as a tension pneumothorax or even vigorous *cunnilingus* (oral sex). So, rather than being dogmatic, look at the whole clinical picture.

Any textbook tells you that lower lobe pneumonia may mimic an acute abdomen, so think about it. Obviously, findings such as lung metastases or pleural effusion may hint at the cause of the abdominal condition and influence treatment and prognosis. Pneumothorax, pnenumomediastinum, or pleural effusion may be associated with spontaneous esophageal perforation—Boerhaave's syndrome (› Chap. 15), which can present as an acute abdomen. The value of a CXR in blunt or penetrating abdominal injury is obvious. A pre-operative CXR may also be requested by the anesthesiologists, especially after you have inserted a central venous line or indeed for no reason at all.

In addition, rarely, what looks to you on chest radiography like free air under the right diaphragm is not free air but bowel (usually the hepatic flexure of the colon) interposed between the liver and diaphragm. This entity is named after the Austrian radiologist who described it: Dr. Chilaidiiti. If asymptomatic, it is termed the *Chilaidiiti sign*. When symptoms are attributed to it (subcostal pain, constipation, respiratory distress), it becomes the *Chilaidiiti syndrome*. We have never encountered this "syndrome," but others claim an occasional need for its operative treatment with "colopexy" or colectomy. In uncertain cases, abdominal CT shows the free air to be in the colon.

## Plain Abdominal X-ray

The plain AXR is the classical surgeon's X-ray as only surgeons know the true value of these simple and cheap radiographs. Radiologists can look at and talk about AXRs forever, searching for findings that could justify "additional" imaging studies. We surgeons need only a few seconds to decide whether the AXR is "nonspecific," namely, does not show any obvious abnormality, or shows an *abnormal gas pattern* or *abnormal "opacities."* Unfortunately, in many of today's "modern" ERs the humble AXR is bypassed in favor of the high-tech CT. In fact now, for many (but it is hoped not for you), the CT supplants the AXR as well as proper history

taking and physical examination. Do not forget that we operate on patients and not on CT abnormalities (see Chap. 5 for discussion of AXR in detail).

### Abdominal Ultrasound

Abdominal ultrasound (US) is a readily available diagnostic modality in most places. Its reliability is operator dependent; the ideal situation is when the US is performed and interpreted by an experienced clinician—a surgeon. US is very accurate in the diagnosis of acute cholecystitis (Chap. 20.1); it is also used by gynecologists to rule out acute pelvic pathology in female patients (Chap. 33) and to demonstrate an acutely obstructed kidney caused by a ureteric stone (Chap. 34). A noncompressible tubular structure (a "small sausage") in the right lower quadrant may be diagnostic of acute appendicitis, but as discussed in Chap. 28, you do not always need abdominal imaging to reach this diagnosis. US is useful in demonstrating intra-abdominal fluid—be it ascites, pus, or blood, localized or diffuse. In blunt abdominal trauma, FAST (focused abdominal sonography for trauma) has emerged as a serious rival to diagnostic peritoneal lavage (Chap. 39.1).

### Abdominal Computed Tomography

The use of the CT scan in the acute abdomen remains a subject of some controversy. While it is true that a CT scan should not be part of the management algorithm in many patients with acute abdominal pain, the new spiral CT technology is nevertheless immediately available, very powerful, and thus extremely tempting to use, especially by less-experienced clinicians.

A case in point is acute diverticulitis (Chap. 26). Once the clinical pattern of localized peritonitis in the lower left quadrant has been identified, initial management is conservative. A CT may show the inflammatory process and even a paracolic abscess but will not distinguish between diverticulitis and a localized perforation of a colonic tumor. In any case, this will not alter the approach because most surgeons would still opt for a trial of intravenous antibiotics as the initial treatment modality for this clinical pattern (Chap. 26).

The true role of the CT, where it can really make a critical difference, is with "clinical puzzles." Not infrequently, the surgeon encounters a patient with acute abdominal pain that does not fit any of the clinical patterns described in Chap. 3. The patient is obviously sick, but the diagnosis remains elusive. Occasionally, there may be a suspicion of acute intra-abdominal pathology in an unconscious patient. Under these circumstances, CT may be very helpful in identifying an intra-abdominal problem. It is even better in *excluding* the last by being absolutely

normal. Finally, CT is frequently indicated in patients with blunt abdominal trauma as discussed in ❯ Chap. 39.1.

Judicious and selective use of CT may help in avoiding surgery altogether—where previously "negative" or "exploratory" or "nontherapeutic" operations would have been performed. It may suggest that alternative percutaneous treatment is possible, and even if operation is still indicated, CT may dictate the optimal incision and approach (❯ Chap. 10). CT has a definite role in the post-laparotomy patient as discussed in ❯ Chaps. 48–52. For detailed discussion on the interpretation of abdominal CT, see ❯ Chap. 5.

## A Word of Caution

For most patients with acute abdominal pain, unnecessary ancillary investigations are merely a resource problem and a waste of time. But, for two types of surgical problems, unnecessary imaging is often lethal:

- Acute mesenteric ischemia is the only life-threatening abdominal condition that cannot be easily classified into one of the five clinical patterns described in ❯ Chap. 3. Because of this, and because the window of opportunity to salvage viable bowel is so narrow, you must have this diagnosis constantly embedded in the back of your mind. The best chance to salvage these patients is to identify the clinical picture of very severe abdominal pain with few objective findings in the appropriate clinical context (❯ Chap. 23) and to proceed directly to mesenteric *angiography*. Needless to say, if the patient has diffuse peritonitis, no imaging is necessary, and the next step is an urgent laparotomy. The tragedy in these patients is the inability of even an experienced clinician to make his or her mind up regarding the need for urgent angiography. As a result, the patient is sent for a long series of irrelevant imaging studies, and the opportunity to salvage viable bowel is lost.
- The second condition for which the abuse of imaging is often lethal is with a ruptured abdominal aortic aneurysm (AAA) (❯ Chap. 41). The first problem occurs in patients with a known aneurysm and a history of abdominal or back pain associated with hypotension who are subjected to an unnecessary CT that merely delays definitive treatment. The second problematic scenario arises as a result of the fact that a ruptured AAA may not present as abdominal pain and shock but merely as severe abdominal or back pain, and it may not be easily palpable in an obese patient. When the possibility of a contained rupture is raised in a hemodynamically stable patient, the one and only ancillary investigation that is required is an urgent CT scan of the abdomen. Unfortunately, too many times these patients spend several hours in the ER, waiting for the results of irrelevant blood tests and progressing slowly along the imaging path from AXRs, which are usually nondiagnostic, to US, which shows the aneurysm but usually cannot

diagnose a rupture, to a long wait for unnecessary contrast material to fill the bowel in preparation for a "technically perfect" CT scan. The tragic consequence of these delays is a dramatic hemodynamic collapse either before or during an abdominal CT scan.

### Contrast Studies: Barium Versus Water-Soluble Contrast

A caveat: in emergency situations do not use barium! Radiologists prefer barium because of its superior imaging qualities, but for us—surgeons—barium is an enemy. Bacteria love barium for it protects them from the peritoneal macrophages; a mixture of barium with feces is the best experimental recipe for the production of intractable peritonitis and multiple intra-abdominal abscesses. Once barium leaks into the peritoneal cavity, it is very difficult to remove. Barium administered to the gastrointestinal tract from above or below tends to stay there for days—distorting any subsequent CT or arteriography.

A gastrointestinal contrast study in the emergency situation has only two queries to answer:

- Is there a **leak**, and if so, where?
- Is there an **obstruction**, and if so, where?

For these purposes Gastrografin is adequate. Use Gastrografin in upper gastrointestinal studies to document or exclude gastric outlet obstruction or treat small bowel obstruction (Chap. 21) or use a Gastrografin enema to diagnose colonic obstruction or perforation. Unlike barium, Gastrografin is harmless should it leak into the peritoneal cavity. Try to operate on a colon full of barium: a clamp slides off, a stapler misfires, and you—not the radiologist—are the one left to clean the mess. Take some advice from our bitter experience: *ordering* a Gastrografin study is not enough; you must personally ensure that barium is not used.

A piece of general advice: do communicate with the radiologists and radiographers. As Leo Gordon said: "The quality of the X ray ordered is directly proportional to the specificity of the clinical information supplied to the radiologist."

### Blood Tests

As stated, "routine labs" are of minimal value. In addition to amylase level, the only "routines" that can be supported are white cell count and hematocrit. *Elevated white cell count* denotes an inflammatory response. Be aware, however, that acute cholecystitis or acute appendicitis can be present even when the white cell count is within normal range. Its elevation, however, supports the diagnosis. Low *hematocrit* in the emergency situation signifies a chronic or subacute anemia;

it does not reflect on the magnitude of any acute hemorrhage. *Liver function tests* are of some value in patients with right upper quadrant pain, diagnosed to have acute cholecystitis or cholangitis (› Chaps. 20.1 and 20.2). Serum *albumin* on admission is a useful marker of the severity of the acute or acute-on-chronic disease and is also of proven prognostic value. When operating, for example, on someone with albumin levels of 1.5 g%, you know that you have to do the minimum and to expect troubles after the operation.

Whichever tests are ordered, either by you or by someone else on your behalf (usually the ER doctor), be aware that the significance of the results should never be judged in isolation but considered as part of the whole clinical picture.

## Unnecessary Tests

Unnecessary testing is plaguing modern medical practice. Look around you and notice that the majority of investigations being ordered do not add much to the quality of care. These unnecessary tests are expensive and potentially harmful. In addition to the therapeutic delay they may cause, be familiar with the following paradigm: the more nonindicated tests you order, the more false-positive results are obtained, which in turn compel you to order more tests and lead to additional, potentially harmful, diagnostic and therapeutic interventions. Eventually, you lose control.

**What are the reasons for unnecessary tests?** The etiology is a combination of ignorance, lack of confidence, and laziness. When abdominal emergencies are initially assessed by nonsurgeons who do not "understand" the abdomen, unnecessary imaging is requested to compensate for ignorance. Junior clinicians who lack confidence tend to order tests "just to be sure—not to miss" a rare disorder. And, experienced clinicians occasionally ask for an abdominal CT over the phone to procrastinate. Isn't it easier to ask for a CT rather than to drive to the hospital in the middle of the night, or having to interrupt the golf game, and examine the patient? ("Let's do the CT and decide in the morning…")

An occasional surgical trainee finds it difficult to understand "what's wrong with excessive testing?" "Well," we explain, "Why do we need you at all? Let us all go home instead, and instruct our ER nurses to drive all patients with abdominal pain through a predetermined line of tests and imaging modalities." But, patients are not cars on a production line in Detroit. They are individuals who need your continuous judgment and selective use of tests.

Be careful before adopting an investigation claimed to be "effective" by others. You read, for example, that in a Boston ivory tower, routine CT of the abdomen has been proven cost effective in the diagnosis of acute appendicitis. Before succumbing to the temptation to order a CT for any suspected acute appendicitis,

check out whether the methods used in the original study can be duplicated in your environment. Do you have senior radiologists to read the CT at 3 a.m.—or would the CT be reported only in the morning—after the appendix is, or should be, in the formalin jar?

Perhaps the day is near when all patients on their way from the ambulance to the ER will be passed through a total body CT scanner—read by a computer. But, then luckily we will not be practicing surgery, and this book will be long out of print. We do not believe, however, that patients will fare better under such a system.

## Diagnostic Laparoscopy

Diagnostic laparoscopy is an invasive diagnostic tool (some call it "controlled penetrating abdominal trauma") to be used in the operating room after the decision to intervene already has been taken. It has a selective role as discussed in Chap. 57.

Before ending, we wish to cite yet again Leo Gordon: "The emergency room is the best place to evaluate an emergency." Think about what investigations you wish to order while the patient is still in the ER; logistically, in most hospitals, it will be more difficult to obtain all these tests after the patient has been admitted.

> The more the noise—the less the fact.
>
> "God gave you ears, eyes, and hands; use them on the patient in that order." (William Kelsey Fry, 1889–1963)

# 5 Abdominal Imaging

Moshe Schein · Sai Sajja · Hans Ulrich Elben

*"The diagnostic problem of to-day*
*Has greatly changed—the changes have come to stay;*
*We all have come to confess, though with a sigh*
*On complicated tests we much rely*
*And use too little hand and ear and eye."*
(The Acute Abdomen in Rhyme, Zachary Cope, 1881–1974)

There are fundamental differences in how physicians belonging to the different specialties involved in decision making concerning the "acute abdomen" look at abdominal imaging. The radiologists' sharp eyes see "everything," but they tend to see "too much" and do not always understand the clinical significance of what they see. Emergency room (ER) physicians do not see much and do not understand the meaning of the little they do see; all they care about is where to dump the patient. This leaves us with ourselves, the surgeons. Armed with a better understanding of the natural history of the disease processes and able to correlate radiological imaging with previous operative observations, we should be the finest interpreters of abdominal imaging. We have already discussed (Chap. 4) the role of abdominal imaging in the evaluation of the patient with an acute abdomen. In this chapter, we will try to provide you with practical tips on **how to look at the images and what to look for.**

## Plain Abdominal X-ray

Moshe Schein

Tragically, the simple, cheap, and safe abdominal X-ray (AXR) is increasingly bypassed in favor of an immediate computed tomographic (CT) scan, which delivers a much greater radiation dose. This is a pity because there is so much that you can learn from a quick glance at the AXR.

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### *Abnormal Gas Pattern*

#### Gas Outside the Bowel

**Free air** (pneumoperitoneum) is best seen on an erect chest X-ray but may also be seen on an AXR (Fig. 5.1). If the CXR is "normal" and you suspect perforation of a viscus, a left lateral decubitus abdominal film may show free gas in the peritoneal cavity.

Make a habit always to look for **atypical gas patterns**; occasionally, you may be rewarded with an eye-popping diagnosis: gas in the biliary tree (pneumobilia) implies a cholecystoenteric fistula (see gallstone ileus; Chap. 21), a previous enterobiliary bypass, or more commonly, a sphincterotomy of the sphincter of Oddi (via ERCP, endoscopic retrograde cholangiopancreatography) (Fig. 5.2). Note that gas in the intrahepatic biliary ducts is seen *centrally*, while gas in the *periphery* of the liver suggests portal vein gas. The gas finds its way into the portal venous system through a breach in the bowel wall, usually associated with *mesenteric ischemia* or *severe colitis* and rarely with *pyelophlebitis* (Fig. 5.3). Commonly, gas in the portal vein as a result of ischemic small or large bowel is associated with pneumatosis intestinalis, that is, the presence of intramural gas (Fig. 5.4).

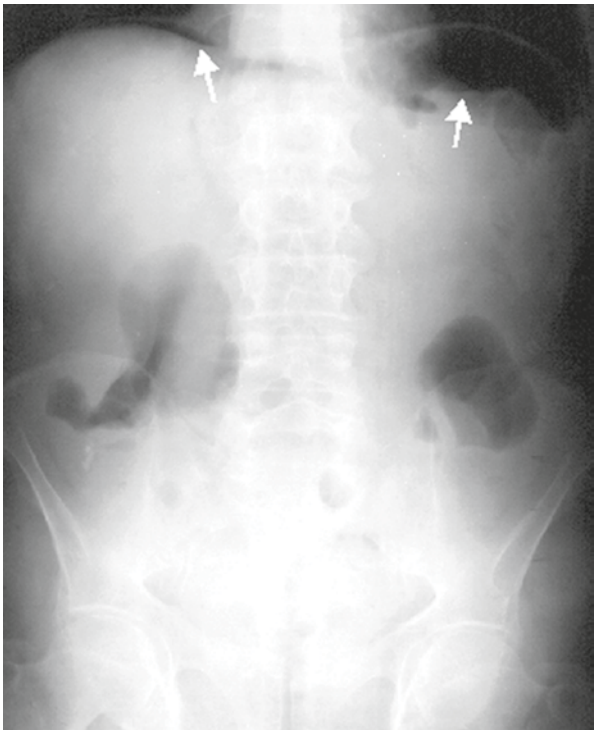

**Fig. 5.1.** Abdominal X-ray, upright position. Pneumoperitoneum. Air under both diaphragms (*arrows*)

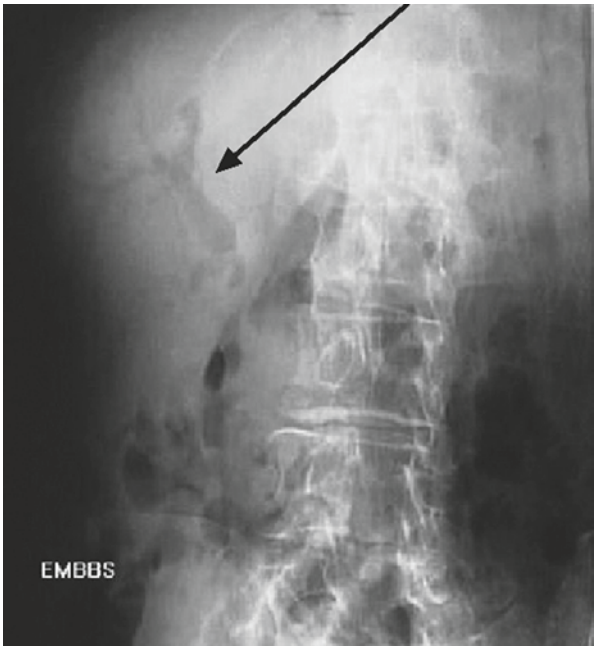


**Fig. 5.2.** Abdominal X-ray: air in biliary tract (*arrow*)

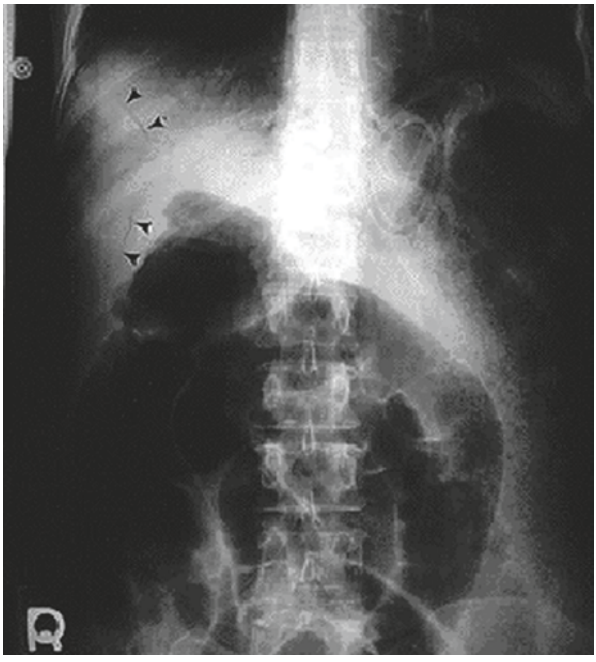

**Fig. 5.3.** Abdominal X-ray: air in portal veins (*arrowheads*)

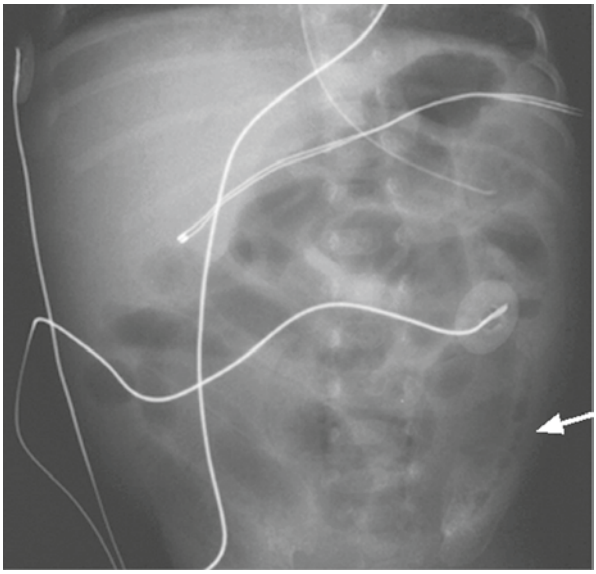

**Fig. 5.4.** Abdominal X-ray: pneumatosis intestinalis (*arrow*)

– Gas in the gallbladder (GB) wall signifies a necrotizing infection (› Chap. 20.1). Soap bubble appearance signifies free gas in the *retroperitoneum*; in the epigastrium, this is associated with *infected pancreatic necrosis* (› Chap. 19), in the right upper quadrant with a retroperitoneal *perforation of the duodenum*, and in either gutter with retroperitoneal perforation of the *colon* (› Fig. 5.5).

#### Gas Within the Bowel

– Abnormal gaseous distension or dilatation of **small bowel loops**, with or without fluid levels, implies a small bowel process—be it *obstructive* (small bowel obstruction; › Chap. 21), *paralytic* ileus (› Chap. 48), or *inflammatory* (Crohn's disease, › Chap. 24). Remember: *acute gastroenteritis* may produce small bowel fluid levels; the diarrhea hints at the diagnosis.

– Abnormal gaseous distention or dilatation of the **colon** denotes colonic *obstruction* or *volvulus* (› Chap. 25), colonic *inflammation* (inflammatory bowel disease; › Chap. 24), or *colonic ileus* (pseudo-obstruction; › Chap. 25).

Distinguishing small bowel from colon on an AXR is easy: the "transverse lines" go all the way across the diameter of the small bowel (the valvulae conniventes) and only partly across the colon (the haustra). In general, loops of small bowel are situated centrally, while large bowel occupies the periphery (› Fig. 5.6).

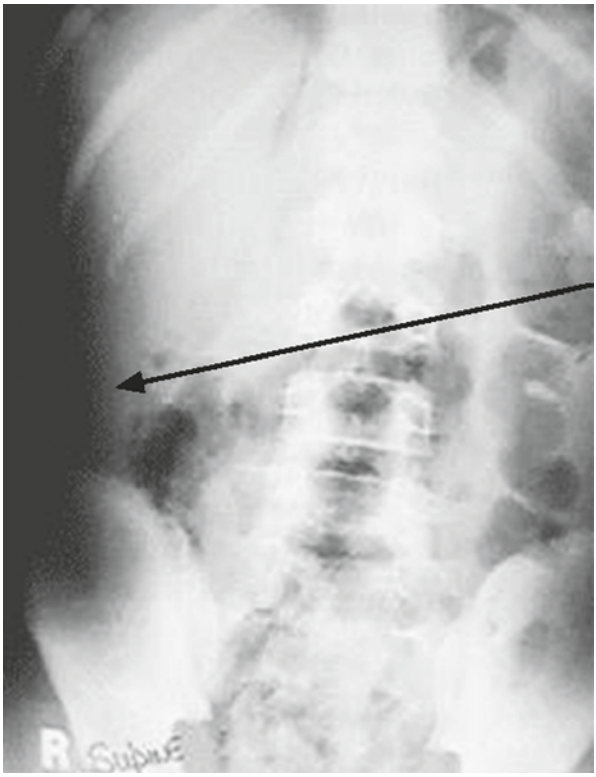

**Fig. 5.5.** Abdominal X-ray: free retroperitoneal air (*arrow*)

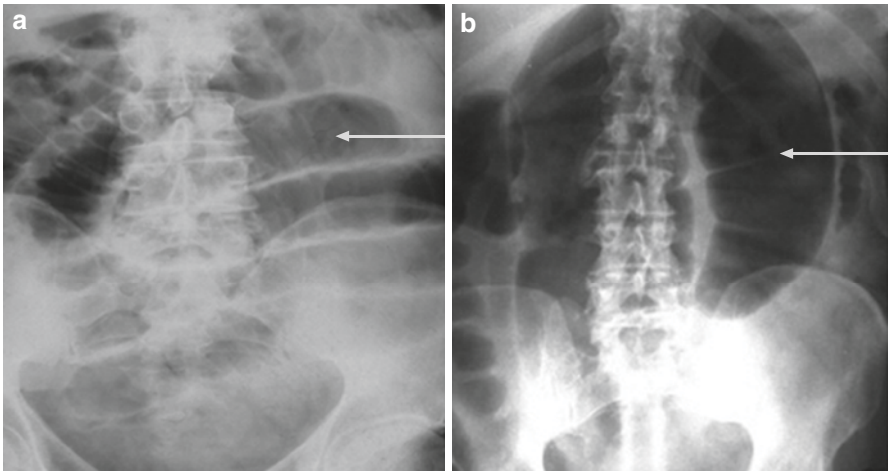


**Fig. 5.6.** Abdominal X-ray: small bowel versus large bowel. (**a**) Small bowel obstruction. Note the valvulae conniventes (*arrow*) crossing the whole width of bowel. (**b**) Volvulus of the sigmoid colon. Note the haustra crossing a portion of bowel width (*arrow*)

**Useful rules of thumb**

- Gaseous distension of small bowel + no gas in the colon = complete small bowel obstruction
- Gaseous distension of small bowel + minimal quantity of colonic gas = partial small bowel obstruction
- Significant gaseous distension of both the small bowel and the colon = paralyticileus
- Significant gaseous distension of the colon + minimal distention of the small bowel = colonic obstruction or pseudo-obstruction

### Abnormal Opacities

The opacities you are able to spot on the AXR are the calcified ones: *gallstones* in the GB (visible in about one-fifth of patients with cholelithiasis), *ureteric stones* (visible in some patients with ureteral colic), *pancreatic calcifications* (seen in some patients with chronic pancreatitis), and *appendicular fecaliths* (occasionally seen in patients with appendicitis) (› Fig. 5.7). Clinically irrelevant calcified

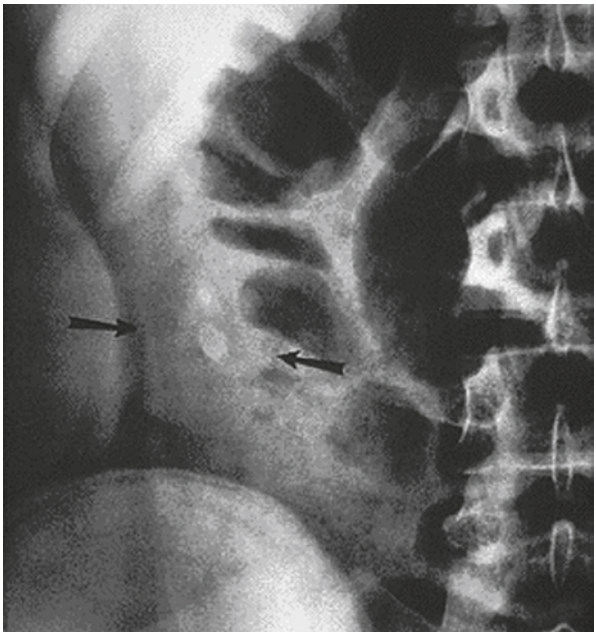

**Fig. 5.7.** Abdominal X-ray: appendicular fecalith (*arrows*; when visualized in a patient with symptoms and signs of acute appendicitis, it is highly diagnostic)

lesions that are common include phleboliths in the pelvis and calcified lymph nodes in the right iliac fossa, usually associated with previous tuberculosis. Fecal matter may opacify the rectum and colon to a variable degree—achieving extreme proportions in patients with fecal impaction. Note that a moderate amount of fecal material in the right colon is normal, while a column of feces on the left implies some abnormality, ranging in severity from simple constipation to early malignant obstruction. Another opacity, which may surprise you, is a forgotten surgical instrument or gauze swab (Fig. 5.8).

Also, **massive ascites** has a typical picture on AXR (Fig. 5.9).

The simple abdominal X-ray is an extension of your clinical evaluation and is not complete without it.

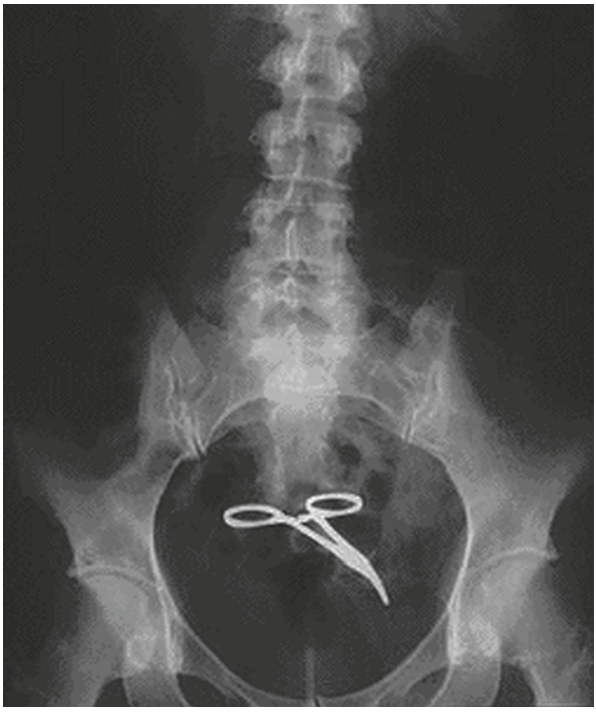

**Fig. 5.8.** Abdominal X-ray: retained surgical clamp

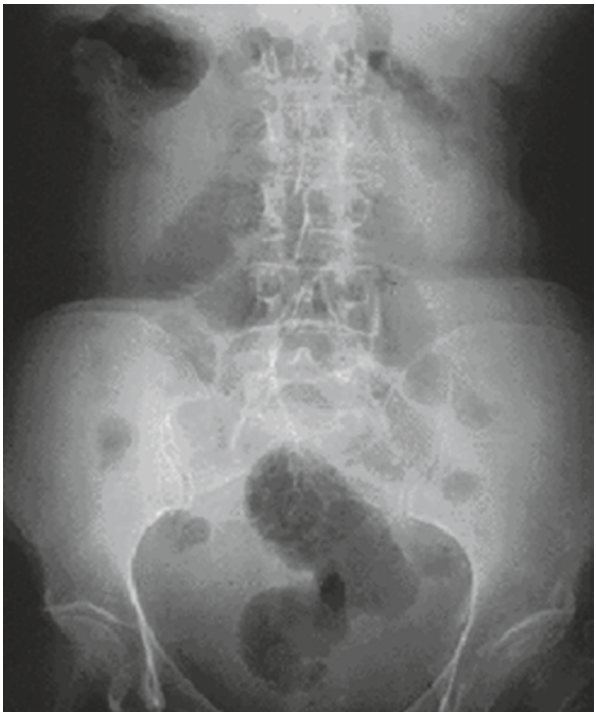

**Fig. 5.9.** Abdominal X-ray: massive ascites. In the supine position, the bowel gas lies centrally, and there is nothing peripherally. The lighter bowel loops are practically floating on a lake of ascites in the abdominal cavity

## Computed Tomography in Abdominal Emergencies

Sai Sajja and Moshe Schein

The road to the operating room does not always have to pass through the CT scanner but an appropriately indicated CT may obviate the need for a surgical route.

The supremacy of CT in the imaging of the abdomen is not in dispute. CT shows details that no other diagnostic method does: free gas, fluid, masses, tissue planes, inflammatory changes, opacities, blood vessels, and organ perfusion. So, why should we object to the indiscriminate use of CT as practiced today in many countries around the world?

We object for the simple reason that in most patients the diagnosis can be established without CT—the obtaining of which often only delays treatment and confuses the picture by showing nonsignificant findings (see Chap. 4). Typically, whenever

radiologists publish papers on the use of CT in various abdominal emergencies, they declare sensitivity and specificity rates approaching 100%. When surgeons, however, look objectively at the overall impact of CT on the diagnosis and treatment of specific conditions, the real impact of CT is often marginal (e.g., acute appendicitis).

In addition, remember that the radiation exposure of one abdominal CT examination can be several hundred times that of a CXR. According to the U.S. Food and Drug Administration, this amount of radiation exposure may be associated with a small increase in radiation-associated cancer in an individual. This is particularly relevant if people were to receive this examination *repeatedly*, starting at a young age—as in the young lady presenting with lower abdominal pain to an ER in Brooklyn, where CT showed an ovarian cyst. Two weeks later, she arrives up at another ER in the Bronx, where another CT documents (surprise!) the same cyst.

The key word in the effective use of abdominal CT is "selectivity." Rather than indicating a need for exploration, CT is more useful in deciding when *not* to operate—avoiding unnecessary "exploratory" laparotomies or "diagnostic" laparoscopies. Also, a "normal CT" can exclude surgical abdominal conditions, allowing the early discharge of patients without the need for admission for observation.

The recent introduction of fast scanners that image the abdomen from the diaphragm to the pubis in a single breath has greatly improved the image quality and reduced the time required to obtain the images. However, it does require that patients be transported to the CT suite and exposes them to the risks of aspiration of oral contrast media and adverse reactions to intravenous contrast media such as anaphylaxis and nephrotoxicity. Unenhanced (no intravenous contrast) helical or spiral CT scans are being increasingly used in suspected appendicitis, while CTs without oral contrast have been reported as accurate in patients suffering from blunt abdominal trauma. Whatever the CT methodology in your hospital, you—who know the abdomen inside out and understand the natural history of abdominal diseases—have to be able to analyze the CT images better than the radiologist.

As is the case with all imaging studies, interpretation of CT scan images requires a systematic approach, and it takes plenty of practice to become confident in one's ability. One also needs to spend time, and the more time you spend the more findings—both negative and positive—you pick up. We describe the way we look at a CT scan of the abdomen; it is not "ideal" or "perfect," but it works for us, especially in the middle of the night when all the radiologists are snoring in bed. (In the morning they will, with *latte* in hand, dictate detailed reports.)

**It is important to pay attention to a few technical aspects of the study before beginning to interpret it.** While there is a lot of literature to support the notion that there is no need for oral or intravenous contrast material, the use of the latter

improves *your* diagnostic yield. One exception to this is when *ureteric calculi* are at the top of the differential diagnosis list and a noncontrast study gives almost all the information required.

### *Contraindications to Intravenous Contrast Medium*

- Impaired renal function
- History of prior allergic reaction to iodinated contrast medium
- Severe asthma or congestive heart failure
- Diabetic patient on metformin (if renal function is normal, you can use intravenous contrast, but metformin should be stopped for 2 days thereafter)
- Multiple myeloma or sickle-cell anemia

### *Reviewing the Abdominal CT*

It is important to note the distance between two CT "slices." Usually, the technologists use 5-mm intervals between the slices, but it is sometimes helpful to request 3-mm cuts of the appendiceal area in a clinically challenging case. Also, it is essential to ensure that you have all the images by looking at the image numbers. Many hospitals have done away with hard copies and introduced instead picture archiving and communication systems (PACSs), which make access to images easier. In this last case, scrolling through the scan gives information that is much easier to interpret than if individual films are examined.

With individual films, we always begin with a good look at the scout film; it provides similar information to a flat plate of the abdomen and provides a "global view." The visualized portions of the lower lung fields should also be looked at in both *mediastinal* and *lung windows*. Pulmonary infiltrates and pleural effusions can be easily identified and at times are a reflection of an acute subdiaphragmatic process. An unsuspected pneumothorax in a trauma patient will also be obvious in the lung windows.

Although it is easier to concentrate on the area of interest (e.g., the right lower quadrant in a patient with suspected appendicitis) and look for findings to support or exclude the diagnosis, it is essential to look at the rest of the abdomen. One needs to look specifically for the presence of free gas and free fluid and to see all the solid organs (liver, spleen, kidneys), stomach, small and large bowel, the pancreas, and blood vessels. One key point is to follow the structure in question in serial images—stacking—to obtain as much information as possible.

Viewing the images with the PACS you can calculate the Hounsfield units (HU) for the various structures you see. To remind you:

| Structure | HU |
|---|---|
| Bone | 1,000 |
| Liver | 40–60 |
| Blood[a] | 40 |
| Muscle | 10–40 |
| Kidney | 30 |
| Water | 0 |
| Fat | −50 to −100 |
| Air | −1,000 |

[a] A fresh clot could measure over 70 HU. Fresh blood about 40 HU, but if you come back the next day or two, it is as little as 20 HU

### Pneumoperitoneum

While an erect chest film can identify a straightforward case of pneumoperitoneum, CT scan is the most sensitive means available for its detection. On a CT scan, gas collects beneath the two rectus muscles around the falciform ligament (Fig. 5.10). It also collects between the liver and anterior abdominal wall and within the "leaves" of the mesentery (Fig. 5.11). The findings are at times subtle, and only a few bubbles of extraluminal gas are all that is required to make the diagnosis of pneumoperitoneum. The key to the identification of extraluminal gas is inspection of all the scans of the abdomen in *lung windows*. It is easier with PACS as we can manipulate the window settings. Even if your hospital does not have PACS, the CT scan station will have the ability to do that.

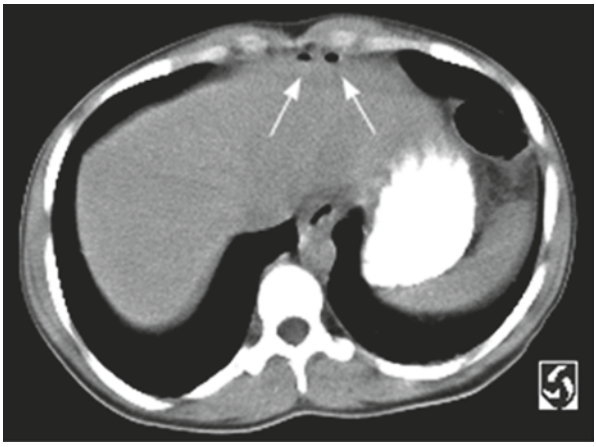

**Fig. 5.10.** CT: two pockets of extraluminal gas in the epigastric region (*arrows*)

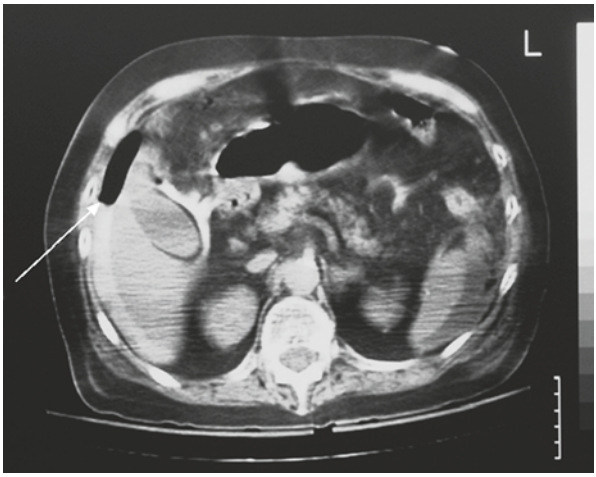


**Fig. 5.11.** CT in a patient with perforated duodenal ulcer: free gas between the liver and anterior abdominal wall (*arrow*). Gas is also seen around the gallbladder (GB) and leakage of orally administered contrast is seen around the liver

### Free Fluid

Free fluid from any source tends to accumulate in the most dependent parts of the peritoneal cavity, Morrison's hepatorenal pouch and the pelvis. When there is a large amount of fluid, the bowel loops float to the midline. In addition to identifying the presence of fluid, measurement of the fluid density offers some clues regarding its nature: less than 15 HU for transudative ascites and more than 30 HU for exudative ascites or blood.

### Solid Organs

While solid organ pathology is a rare cause of nontraumatic acute abdominal conditions, CT is the modality of choice in the investigation of the hemodynamically stable victim of blunt abdominal trauma. Lacerations of the solid organs appear as linear or branching low-attenuation areas. Subcapsular hematomas appear as crescentic low-attenuation areas at the periphery. Intraparenchymal hematomas appear as round or oval collections of blood within the parenchyma.

### Hollow Organs

The entire gastrointestinal tract from the stomach to rectum can be traced in serial sections, and abnormalities should be sought. In case of small bowel obstruction, the cause (e.g., tumor or inflammatory mass) and the site of obstruction

(the transition point) can be identified (Fig. 5.12). The presence of *pneumatosis* can be identified more readily with CT than a plain film and, if present, suggests intestinal ischemia. CT is also sensitive for identifying inflammation, which is suggested by the appearance of tissue infiltration or *stranding* (Figs. 5.13 and 5.14). If intravenous contrast has been administered, then reduced enhancement of loops of bowel may signal ischemia. Similarly, the origins of the mesenteric vessels may be inspected to get some idea about patency.

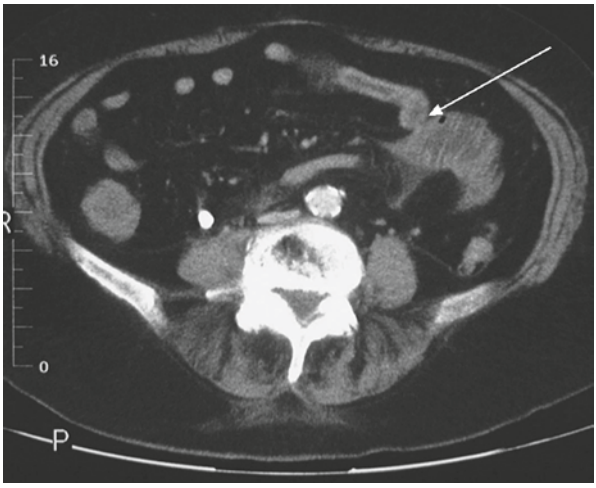


**Fig. 5.12.** CT in a patient with small bowel obstruction showing the transition point between the distended proximal and collapsed distal bowel (*arrow*)

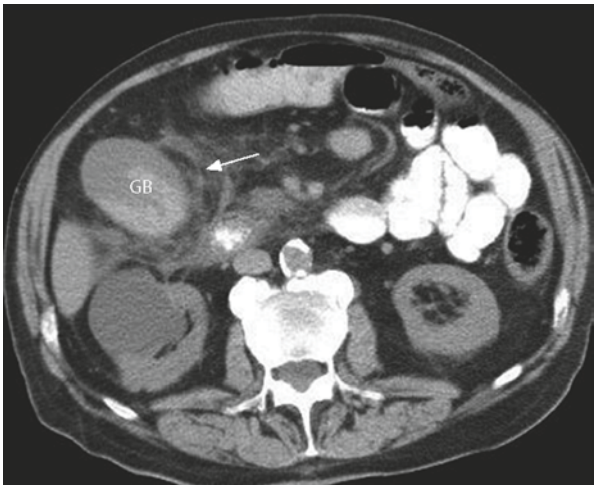


**Fig. 5.13.** CT scan through the upper abdomen shows a distended thick-walled *GB* with marked pericholecystic stranding (*arrow*) suggestive of acute cholecystitis

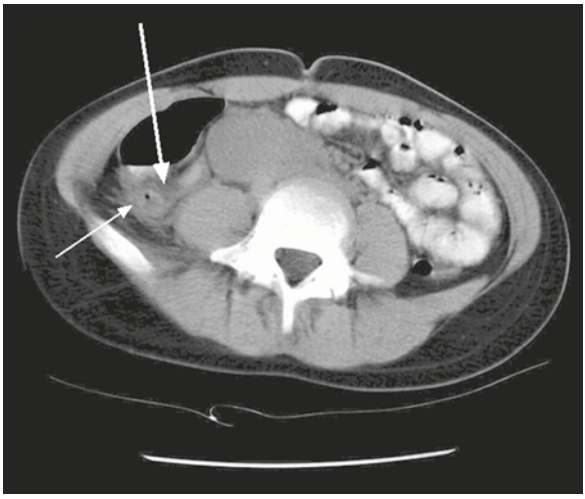

**Fig. 5.14.** CT scan through the right lower quadrant showing thickened appendix (*thin arrow*) with periappendiceal fat infiltration (*thick arrow*), confirming the diagnosis of acute appendicitis

The various CT scan findings that are associated with **acute appendicitis** are as follows:

**Appendiceal signs**

- Appendix >6 mm in diameter
- Failure of the appendix to fill with oral contrast or gas to its tip
- Enhancement of the appendix with intravenous contrast
- Appendicolith

**Periappendiceal signs**

- Increased fat attenuation (stranding) in the right lower quadrant
- Cecal wall thickening
- Phelgmon in the right lower quadrant
- Abscess or extraluminal gas
- Fluid in the right lower quadrant or pelvis

Similarly, stranding in the left lower quadrant or thickening of the sigmoid colon suggests diverticulitis (› Fig. 5.15). Diffuse thickening of the colon suggests an inflammatory process like colitis, whether infective or ischemic (› Fig. 5.16).

The retroperitoneum, including the pancreas, should then be looked at; the presence of stranding and fluid collections around the pancreas suggests pancreatitis. Retroperitoneal hematoma next to an abdominal aortic aneurysm suggests a leak.

It is also important to look at the pelvic organs in female patients. Particular attention should be paid to any large cystic masses in the adnexa, which may suggest a complicated cyst, ovarian torsion, or a tubo-ovarian abscess.

Your patient does not require a CT ticket to enter the OR (Fig. 5.17), but occasionally CT will change your operative plans or even cancel the need for the operation.

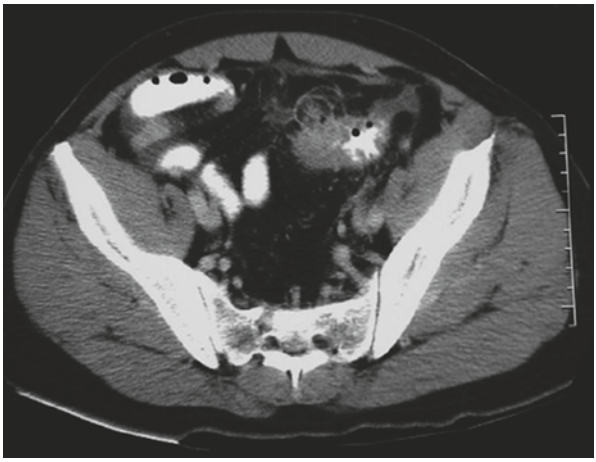

**Fig. 5.15.** Contrast-enhanced CT scan of the lower abdomen showing thickening of the sigmoid colon with diverticula and surrounding inflammation (acute diverticulitis)

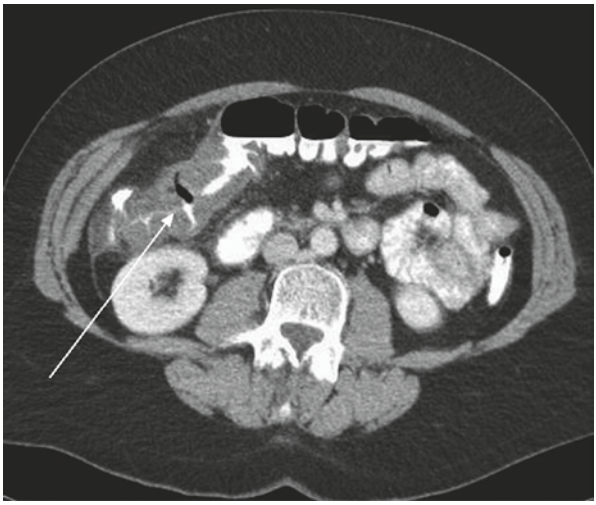

**Fig. 5.16.** Contrast-enhanced CT scan showing thickening of the hepatic flexure of the transverse colon (*arrow*) suggestive of colitis


**Fig. 5.17.** "Where is the CT?"

## Invited Commentary by a Radiologist: How to Read and Interpret the Abdominal CT for an Acute Abdomen

Hans Ulrich Elben

### *How to Order a CT Examination*

Contrary to what you may think, "*some* radiologists" understand something about medicine and surgery. And, a few of us know something about CT scans. We therefore respectfully request that you please provide us with an accurate clinical picture and your tentative diagnosis when requesting a scan. You should tell us also about any relevant previous operations or injuries (like cholecystectomy, appendectomy, hysterectomy).

### *Technically State-of-the-Art CT Examination*

A good CT examination is performed with a spiral CT after intravenous administration of a contrast medium. If possible, we also like to use an oral diluted Gastrografin medium. The latter can also be given rectally, especially when suspecting acute diverticulitis, an obstructing colonic lesion, or colonic trauma. In women with suspected gynecological pathology, you should mark the position of the vagina with a normal vaginal tampon. An important exception: in case of suspected ureteric colic, the use of oral contrast is not necessary.

### *Interpretation*

Start with a scout view, similar to a plain AXR in a supine patient. Look at the distribution of gas in the stomach and the small and large intestine. Are there signs of free gas outside the intestinal lumen? It is absolutely necessary to look at the CT images in a special window for chest examination (center –700 HU, window width 2,000 HU) as well as in a normal window (center 40 HU, window width 400 HU). Thus, you will recognize free gas outside the intestinal lumen much better.

### *Step-by-Step Interpretation of Images by Organs*

Try to examine every organ from cranial to caudal direction completely. Especially note the limits and the structures of the tissues.

#### Liver

Look at edges of the liver, homogeneous enhancement, and luminal contrast within the portal vein and its branches. **Important diagnoses** are blunt trauma with rupture of the liver, abscesses, portal vein thrombosis (› Fig. 5.18).

#### Gallbladder and Bile Ducts

The intrahepatic bile ducts accompany the branches of the portal vein. Normally, they are hardly recognized unless dilated. If there is cholangiectasis,

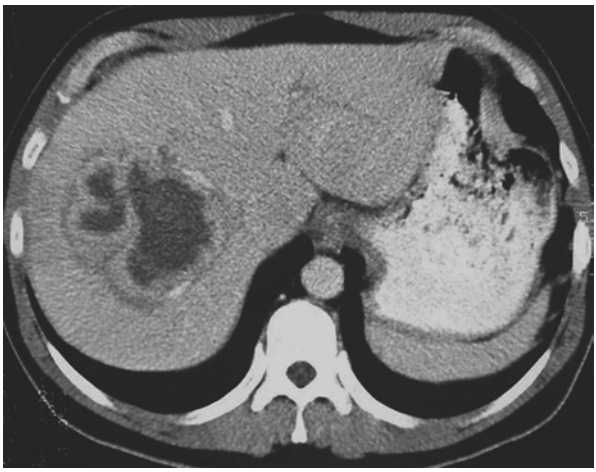

**Fig. 5.18.** Abdominal CT: intrahepatic abscess. Note the enhancement of the wall

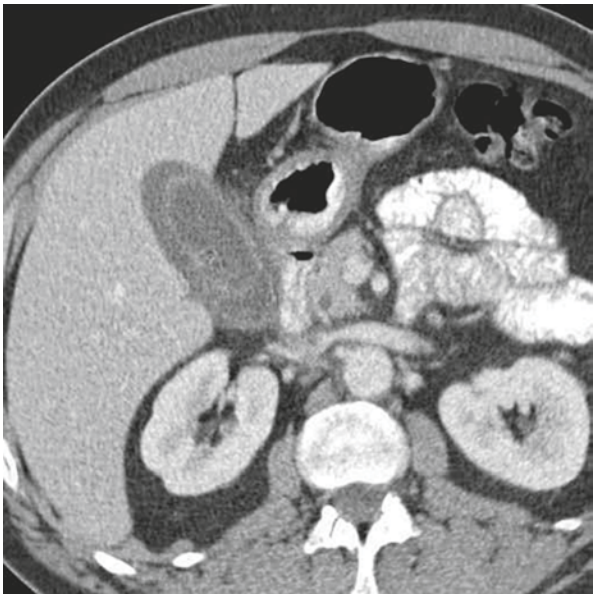

**Fig. 5.19.** Abdominal CT: acute cholecystitis

follow the common bile duct down to the duodenal papilla. Do you see any signs of tumor-associated obstruction or choledocholithiasis?

Normally, the wall of the GB is thin (about 2–3 mm). A distended GB, thickened wall, a pericholecystic layer of fluid, a "halo" sign, and intramural air are strong indications of cholecystitis (Fig. 5.19).

#### Spleen

Notice the size and form of the spleen. Is there homogeneous enhancement? Important diagnoses include traumatic or spontaneous rupture with lack of contrast and fluid around the spleen and infarct of the spleen with a hypoperfused wedgelike area.

#### Pancreas

The position of the pancreas is from the hilum of the spleen (cauda pancreatici), in front of the contrast-enhanced splenic artery and vein and superior mesenteric artery and vein to the duodenal loop (caput pancreatici). Normally, the pancreas shows uniform homogeneous enhancement. In pancreatitis, the organ is enlarged diffusely. In pancreatic necrosis, parts of the gland do not light up with

contrast. The surrounding fatty tissue is not dark and inconspicuous by comparison but shows bright streaks. Fluid around the pancreas signifies inflammatory exudate.

#### Kidneys, Ureters, Urinary Bladder, and Urethra

Kidney stones you will see best in a native (i.e., not contrasted) scan within the renal pelvis or one of the ureters. The ureters have to be examined along their entire course from the renal pelvis to the bladder. Is there any dilatation? Any tissue reaction surrounding calcification (rim sign)? Irregular spotty contrast of the renal tissue refers to nephritis, and wedge-shaped absence of contrast implies a renal infarct. In renal vein thrombosis, the renal vein does not enhance with contrast. Streaky changes in the perirenal fatty tissue suggest inflammation.

#### Organs of the Pelvis

**For women:** examine the uterus and the adnexa positioned laterally to it. Do you see cystic structures (ovarian cysts)? Do you recognize inflammatory signs in the surrounding fatty tissue, or is there fluid concentration with enhancement of its wall (tubo-ovarian abscess)? Are there signs of bleeding?

**For men:** identify bladder, prostate gland, and seminal vesicles.

#### Stomach, Gut, and Peritoneal Cavity

Examine the whole intestinal tract starting with the stomach and following the small bowel from duodenum to jejunum, ileum down to the ileocecal valve, the cecum and the ascending, transverse, descending, and pelvic colon to the rectum. CT features of obstruction and inflammation and other specific conditions are discussed elsewhere in this book. An inflamed Meckel's diverticulum can be identified by a diverticulation of the intestinal lumen with streaky reactions of the surrounding tissue (› Fig. 5.20). In the right lower quadrant, look for the cecum and the vermiform appendix; signs of acute appendicitis are well described in the previous section. In active Crohn's disease, you will often recognize a considerably thickened wall of the terminal ileum.

In the descending and pelvic colon, you should look for diverticula and signs of inflammation: thickened wall and streaky thickened structures in the pericolic fat. Complicated diverticulitis is suggested by extraluminal gas, leakage of contrast, and an abscess (› Fig. 5.21). *Appendagatis* is an inflammation of the appendix epiploica and needs no operation (› Fig. 5.22). Colonic diverticula tend to perforate in the high-pressure zone above an obstructing carcinoma. CT is not a good tool for distinguishing a colonic inflammatory mass from a malignant one.

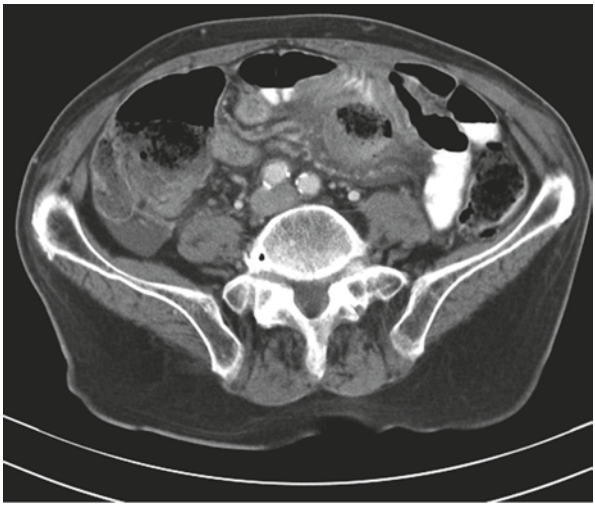

**Fig. 5.20.** Abdominal CT: perforation of a Meckel's diverticulum. Note the central structure, which lacks luminal contrast and is surrounded by tissue reaction

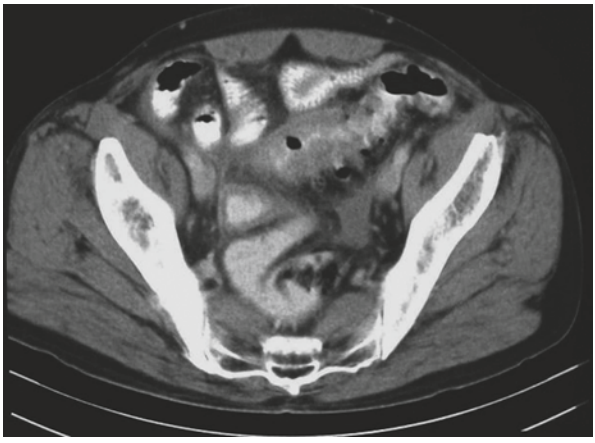

**Fig. 5.21.** Abdominal CT: acute sigmoid diverticulitis. Note the thickened loop of sigmoid with almost absent lumen and the tissue stranding around it, denoting inflammation

### Free Fluid

Watch for free fluid between the intestinal loops and elsewhere. The fluid density gives a clue to its nature: for ascites, it is like water, 0–20 HU; for pus, it is between 15 and 30 HU; and for blood, it is about 50 HU, but be aware that these specifications do not always allow an exact differentiation.

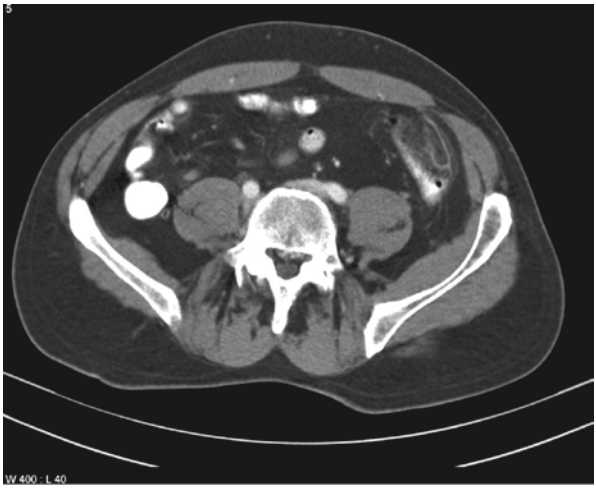


**Fig. 5.22.** Abdominal CT. Appendagatis: acute inflammation of the appendix epiploica of the sigmoid colon. See the inflammatory process compressing the lumen of the colon

An **abscess** shows an annular enhancement, and gas inclusions inside will prove it. Diffuse peritonitis is not easy to diagnose, but helpful signs include fluid collections between intestinal loops and in the pouch of Douglas and a thickened base of the small bowel mesentery.

#### Retroperitoneum, Big Vessels, and Abdominal Wall

Watch the lumen of the aorta and the pelvic vessels to find a ruptured aneurysm (Fig. 5.23). Look for free gas or a collection suggesting an abscess due to retroperitoneal perforation of a viscus such as the colon or duodenum.

Looking at the abdominal wall, try to find pathological changes like subcutaneous abscesses, rectus sheath hematomas, or abdominal wall hernias (Fig. 5.24).

And be nice to your radiologists … they can be your best friends.

## Final Words

Moshe Schein

Unfortunately or fortunately—depending on one's viewpoint—in the United States, where I practice, the decision about whom and when to scan is no longer in our surgical hands. The fact of the matter is that most (if not all) patients have already undergone a CT scan before surgeons are called on to assess them. Typically, such scans are ordered by ER physicians or other specialists before consulting the

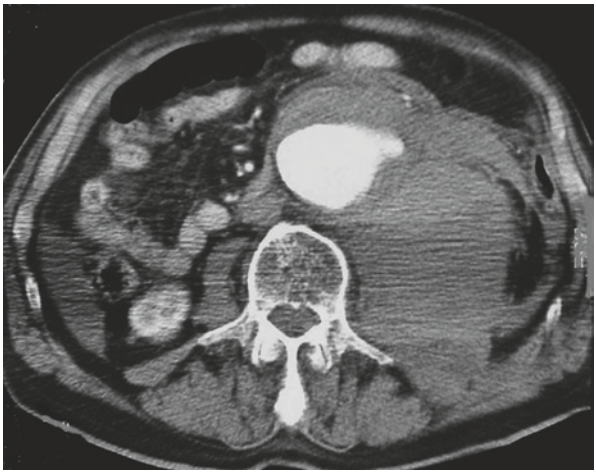

**Fig. 5.23.** Abdominal CT: leaking abdominal aortic aneurysm. See the aortic aneurysm and large retroperitoneal hematoma on the left

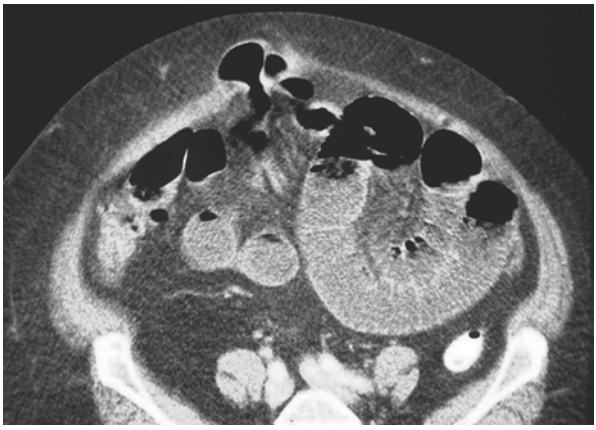

**Fig. 5.24.** Abdominal CT: incisional hernia. Note a loop of small bowel incarcerated within an incisional abdominal wall defect

surgeon. In most hospitals in the United States, even the tiny rural ones, high-tech CT images are much easier to obtain than a gourmet meal or even a cup of *real* coffee. And, radiologists are always readily available to interpret the images online. No wonder then that physicians confronted with the acute abdomen feel compelled to get a CT, which is as easily procured as junk food. Is this practice of (almost) routine CT scanning, imposed on us by others and impossible for us to modify or reverse, "good" or "bad" for our patients? It is very difficult, if not impossible, to prove scientifically that this increased use of CT scanning is beneficial overall. But, what about the individual patient?

Luckily, gone are the days when the acute abdomen represented a totally black box—days I remember well from my training—when peritoneal signs on examination mandated a laparotomy, which often proved to be "negative" or "nontherapeutic" and therefore unnecessary. The gradual introduction of CT imaging (and ultrasound) has made that abdominal black box much more penetrable and less mysterious. In the individual patient, it helps us to be more selective and more conservative; helps us to decide when *not* to operate, when to choose alternative modalities (e.g., percutaneous drainage); and guides us to the choice of incision. Equally important—for those of us who take emergency calls—CT lets us sleep better and longer at night.

So, from the individual patient's and surgeon's perspective, I believe that liberal use of abdominal CT in the setting of the acute abdomen reflects a positive trend, with two caveats: First, we have to try to prevent repeated exposures to CT radiation, particularly in younger patients; most importantly, an experienced abdominal surgeon must be the one interpreting the CT images (together with the radiologist) and deciding how to proceed. An abdominal image without an abdominal surgeon is only an image, but together, the surgeon and the CT, they represent the best modern surgical judgment—the human one supplemented and made more accurate.

# 6 Optimizing the Patient

James C. Rucinski

*When physiology is disrupted, attempts at restoring anatomy are futile.*

*The preparation of the patient for surgery may be as crucial as the operation itself.*

It's 4 a.m., and you assess your patient as having an "acute abdomen"—probably due to a perforated viscus. Clearly, your patient needs an emergency laparotomy; what is left to decide is what efforts, and how much time, should be invested in the patient's optimization before the operation.

Optimization is a double-edged sword: wasting time trying to "stabilize" an exsanguinating patient is an exercise in futility for the patient will die. Conversely, rushing to surgery with a hypovolemic patient suffering from intestinal obstruction is a recipe for disaster.

The issues to be discussed here are:

- Why preoperative optimization at all?
- What are the goals of optimization?
- Who needs optimization?
- How to do it?

## Why Is Preoperative Optimization Necessary?

Simply, preoperative optimization is necessary because volume-depleted patients do not tolerate anesthesia and operation. The induction of general anesthesia and muscle relaxation causes systemic vasodilatation, depressing the compensatory antishock physiologic mechanisms. On opening the abdomen, intraperitoneal pressure suddenly declines, allowing pooling of blood in the venous system, which in turn decreases venous return and thus depresses cardiac output. An emergency laparotomy in an underresuscitated patient may result in cardiac arrest even before the operation is started. In addition, the intraoperative fluid requirements are unpredictable: Do you want to start with a volume-depleted patient, having to chase your tail?

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## What Are the Goals of Optimization?

Patients awaiting an emergency laparotomy need optimization for two main reasons: *hypovolemia* or *"sepsis."* Both conditions cause underperfusion of the tissues and both are treated initially with volume expansion. The chief goal of preoperative optimization is to improve the delivery of oxygen to the cells. There is a direct relationship between cellular hypoxia and subsequent cellular dysfunction, systemic inflammatory response syndrome (SIRS), organ failure, and adverse outcome (Chap. 54).

In sick surgical patients, unlike the medical ones, optimization means volume and more volume—a lot of fluids. (This is, however, not true in actively bleeding patients; here, optimization means immediate control of the hemorrhage, and until this is achieved you should restrict fluids and keep the patient moderately hypotensive.)

## Who Needs Optimization?

Surgical patients often "look" sick. The appearance of the patient usually gives an important first impression even before factoring in tachycardia, tachypnea, hypotension, mental confusion, and poor peripheral perfusion.

Only basic laboratory studies are necessary. *Hemoconcentration*, reflected in an abnormally high hemoglobin and hematocrit, implies either severe dehydration or extracellular "third-space" fluid sequestration. *Urine analysis* with a high specific gravity (>1.039) provides similar information. *Electrolyte imbalance* and associated *prerenal azotemia* (with a >20:1 ratio of BUN [blood urea nitrogen] to creatinine), again imply volume depletion. *Arterial blood gas* measurement gives critical information regarding respiratory function and tissue perfusion. Note that in the emergency surgical patient metabolic acidosis almost always means lactic acidosis—associated with inadequate tissue oxygenation and anaerobic metabolism at the cellular level. Other causes of metabolic acidosis such as renal failure, diabetic ketoacidosis, or toxic poisoning are possible but extremely unlikely. A base deficit of more than 6 (base excess [BE] less than −6) is a marker of significant metabolic acidosis and adverse prognosis and indicates a need for aggressive resuscitation.

All patients with any degree of these physiological abnormalities need optimization. Naturally, the magnitude of your efforts should correlate with the severity of the disturbances.

### *Measurement of the Severity of Illness*

An experienced surgeon can "eyeball" his or her patient and estimate how sick the patient is by assessing "the glare in his eye and the strength of the grip."

| PHYSIOLOGIC VARIABLE | HIGH ABNORMAL RANGE +4 | +3 | +2 | +1 | 0 | +1 | LOW ABNORMAL RANGE +2 | +3 | +4 |
|---|---|---|---|---|---|---|---|---|---|
| 1. Temperature rectal (°C) | ≥ 41° ☐ | 39°-40.9° ☐ | | 38.5°-38.9° ☐ | 36°-38.4° ☐ | 34°-35.9° ☐ | 32°-33.9° ☐ | 30°-31.9° ☐ | ≤ 29.9° ☐ |
| 2. Mean arterial pressure | ≥ 160 ☐ | 130-159 ☐ | 110-129 ☐ | | 70-109 ☐ | | 50-69 ☐ | ☐ | ≤ 49 ☐ |
| 3. Heart rate (ventricular response) | ≥ 180 ☐ | 140-179 ☐ | 110-139 ☐ | | 70-109 ☐ | | 55-69 ☐ | 40-54 ☐ | ≤ 39 ☐ |
| 4. Respiratory rate (non-ventilated or ventilated) | ≥ 50 ☐ | 35-49 ☐ | | 2 5-34 ☐ | 12-24 ☐ | 10-11 ☐ | 6-9 ☐ | | ≤ 5 ☐ |
| 5. Oxygenation: A-$aDO_2$ or $PaO_2$ (mm Hg) a) $FiO_2$ > 0.5: record A-$aDO_2$ | ≥ 500 ☐ | 350-499 ☐ | 200-349 ☐ | | < 200 ☐ | | | | |
| b) $FiO_2$ < 0.5: record only $PaO_2$ | | | | | > 70 ☐ | 61-70 ☐ | | 55-60 ☐ | < 55 ☐ |
| 6. Arterial pH | ≥ 7.7 ☐ | 7.6-7.69 ☐ | | 7.5-7.59 ☐ | 7.33-7.49 ☐ | | 7.25-7.32 ☐ | 7.15-7.24 ☐ | < 7.15 ☐ |
| 7. Serum Sodium | ≥ 180 ☐ | 160-179 ☐ | 155-159 ☐ | 150-154 ☐ | 130-149 ☐ | | 120-129 ☐ | 111-119 ☐ | ≤ 110 ☐ |
| 8. Serum Potassium | ≥ 7 ☐ | 6-6.9 ☐ | | 5.5-5.9 ☐ | 3.5-5.4 ☐ | 3-3.4 ☐ | 2.5-2.9 ☐ | | > 2.5 ☐ |
| 9. Serum creatinine (mg/dl) | ≥ 3.5 ☐ | 2-3.4 ☐ | 1.5-1.9 ☐ | | 0.6-1.4 ☐ | | < 0.6 ☐ | | |
| 10. Hematocrit (%) | ≥ 60 ☐ | | 50-59.9 ☐ | 46-49.9 ☐ | 30-45.9 ☐ | | 20-29.9 ☐ | | < 20 ☐ |
| 11. White Blood Count | ≥ 40 ☐ | | 20-39.9 ☐ | 15-19.9 ☐ | 3-14.9 ☐ | | 1-2.9 ☐ | | < 1 ☐ |
| 12. Glasgow coma score | 15 - GCS = | | | | | | | | |
| A Total acute physiology score (APS) | Sum of the 12 individual variable points = ☐ | | | | | | | | |
| ● Serum $HCO_3$ (venous-mmol/l | ≥ 52 ☐ | 41-51.9 ☐ | | 32-40.9 ☐ | 22-31.9 ☐ | | 18-21.9 ☐ | 15-17.9 ☐ | < 15 ☐ |

**Glasgow Coma Scale** (Circle appropriate response)

Eyes open:
4-spontaneously
3-to verbal
2-to painful stimuli
1-no response

Motor response:
6-to verbal command
5-localizes to pain
4-with draws to pain
3-decorticate
2-decorabrate
1-no response

verbal-nonintubated
5-oriented and controverses
4-disoriented and talks
3-inappropriate words
2-incomprehensible sounds
1-no response

verbal-intubated
5-seems able to talk
3-questionable ability to talk
1-generally unresponsive

**B Age Points**

| Age | Points |
|---|---|
| ≤ 44 | 0 |
| 45-54 | 2 |
| 55-64 | 3 |
| 65-74 | 5 |
| ≥ 75 | 6 |

**Age Points =** ☐

**C Chronic Health Points**

If any of the 5 CHE estegories is answered with yes give +5 points for non-operative or emergency postoperative patients.

Liver • Cirrhosis with PHT or encephalopathy
Cardiovascular • Class IV angina or at rest or with minimal self-care activities
Pulmonary • Chronic hypoxemic or hypercapnoe or polycythemic of PHT > 40 mm Hg
Kidney • Chronic peritoneal or hemodialysis
Immune • Immune compromised host

**Chronic Health Points =** ☐

**Apache-II Score** (Sum of A+B+C)

A APS points
\+ B Age points
\+ C Chronic Health points

= ☐ Total Apache-II

**Fig. 6.1.** APACHE II (Acute Physiological and Chronic Health Evaluation II)

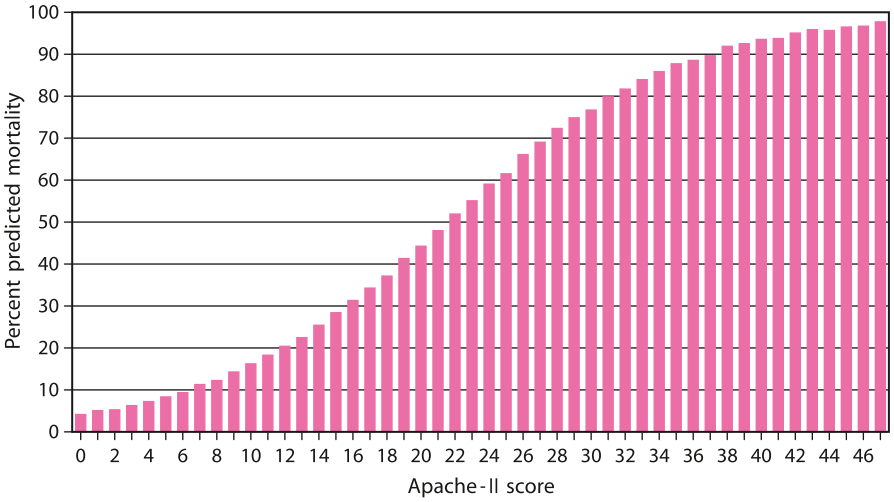


**Fig. 6.2.** Eventual morbidity and mortality in emergency abdominal surgery

But, terms such as "very sick," "critically ill," or "moribund" mean different things to different people. We recommend therefore that you become familiar with a universal physiological scoring system that gives an objective measure of "sickness." One scoring system, which has been validated in most emergency surgical situations, is the APACHE II (Acute Physiological and Chronic Health Evaluation II) (Fig. 6.1). It measures the physiological consequences of acute disease while taking into consideration the patient's premorbid state and age. The scores are easily measured from readily available basic clinical and laboratory variables and correlate with a prediction of morbidity and mortality (Fig. 6.2). A score of 10 or below represents a relatively mild disease; a score above 20 signals a critical illness. Instead of telling your chief resident that this patient is "really sick," you would say "his APACHE II is 29." Now, it is clear to everyone involved that the patient is moribund. [Next everyone will ask you: "What the **** is APACHE? A horse?" So now you will have the chance to teach them and appear smart! —The Editors]

### How to Do It? (Fig. 6.3)

Principles of optimization: air goes in and out; blood goes round and round; oxygen is good.

Despite the high-tech intensive care unit (ICU) environment, which may or may not be available to you, optimization of the surgical patient is simple. It can

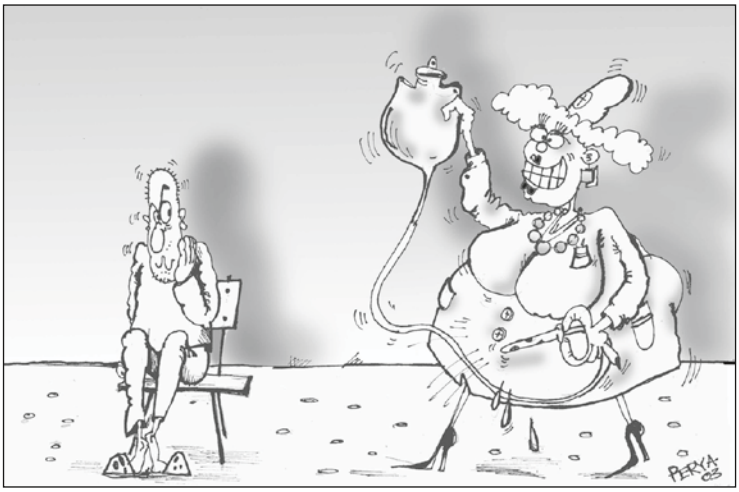

**Fig. 6.3.** "Let me optimize you…"

be accomplished anywhere and requires minimal facilities. All you want is better oxygen delivery, that is, increased oxygenation of arterial blood and enhanced tissue perfusion. You do not need a five-star ICU, but you do have to stick around with the patient. Writing orders and going to bed (until the operation) will unnecessarily prolong the optimization and delay the operation. So, stay with the patients, monitor their progress, and be there to decide when enough is enough.

### *Oxygenation*

Hypoxia not only stops the motor, it wrecks the engine.

Any patient who requires optimization should at least receive oxygen by mask. Look at the patient and the patient's pulse oximetry or arterial blood gases; evidence of severe hypoventilation or poor oxygenation may be an indication for endotracheal intubation and mechanical ventilation. Do not temporize; the patient will need intubation anyway, so why not now? Remember, pain and distension associated with any abdominal catastrophe impede ventilation. Effective analgesia impairs ventilation still further. If a nasogastric (NG) tube is not already in situ, this may be the time to insert one. The advantage of NG tube insertion before intubation is to decompress the distended stomach and reduce the risk of aspiration during the procedure. The disadvantage is that the presence of a tube through the cricopharyngeus may allow regurgitation during rapid sequence induction of anesthesia.

### *Restoration of Volume*

> The major cause of shock is decreased circulatory volume. Replace body fluids by the best means at hand. (Alfred Blalock, 1899–1964)

Now, after your patient is well oxygenated you must see to it that the oxygen arrives where it is needed by restoring blood volume. This is accomplished by intravenous infusion of crystalloids such as normal saline or Ringer's lactate. Forget about the much more expensive colloids such as fresh frozen plasma, albumin, or solutions containing synthetic organic macromolecules such as Hemastarch or low molecular weight dextran; their theoretical advantages have never been translated to better results. Hypertonic saline resuscitation may theoretically be advantageous, but it remains an investigational therapy at present. [It has been experimental since we finished medical school! —The Editors] Blood and blood products are given if necessary as discussed below.

How much crystalloid to infuse? An old rule of thumb was that the hypovolemic surgical patient needs more volume than you think he or she needs and much more than the nursing staff thinks he or she needs. [But, this rule seems to be outdated; see "editorial comment" below.] We assume that your patient already has a large-bore intravenous catheter in situ, so just hook it up to the solution, open the valve, and let it run. You run in a liter and hang up another. But, how much is enough? At this stage, you need to assess the *effectiveness of what you do*.

## Measurement of Effectiveness of Treatment

The principal goal of nonoperative treatment in the emergency surgical patient is the restoration of *adequate tissue oxygenation*. This endpoint is recognized by *physical examination* and measurement of *urinary output* in conjunction with the information provided by *selective invasive monitoring* and laboratory studies.

With fluid resuscitation, one hopes to see improvement of tissue oxygenation by normalization of vital signs and improvement in the visible peripheral circulation. Resolution of hypotension, mental confusion, tachypnea, and tachycardia may be seen either partially or fully. *Postural hypotension* reflects a significant deficit in the circulating blood volume. Remember that the usual response to a change in position from supine to upright is an increase in the systolic blood pressure, a widening of the pulse pressure. Consequently, if a narrowing of the pulse pressure is seen when the patient sits up, then postural hypotension is present. With fluid resuscitation, mottling of the skin and the palpable temperature of the fingers and

toes may improve. *Capillary refill* is a clinical test that observes the peripheral circulation in the nail bed. The nail bed blanches when pressed and should return to its normal pink color in less than 2 s. Fluid resuscitation aims to correct this subtle abnormality of the peripheral circulation as well.

### *Urine Output*

> Ventilate, perfuse, and piss is all that it is about! (Matt Oliver)

A Foley urinary bladder catheter is essential in any patient requiring optimization. It allows an accurate, if indirect, measurement of tissue perfusion and *adequacy of fluid resuscitation*, as reflected in the urine output.

Your aim each hour is at least 0.5–1 ml urine/kg patient's weight. This is the single best sign of adequate tissue perfusion associated with successful fluid resuscitation.

### *Invasive Monitoring*

The central venous catheter and the Swan-Ganz pulmonary arterial catheter are tools that permit "special studies" to be carried out rapidly and repeatedly. The downside of such devices is that they are invasive, expensive, often inaccurate, and associated with potentially life-threatening complications. Invasive hemodynamic monitoring provides endpoint measurements that, in conjunction with urinary output, indicate the adequacy of fluid resuscitation.

#### The Central Venous Catheter

The **central venous catheter** measures central venous pressure (CVP), which is a product of the venous return (i.e., blood volume) and right ventricular function. Low CVP always means hypovolemia, but a high CVP can signify either overexpansion of blood volume or cardiac failure. So, aim for an adequate urinary output with a CVP in the normal range, up to12 $cmH_2O$. When the CVP rises above the normal range and the urinary output is still not adequate, then either cardiac or renal function is impaired or the measurement is in error. False

elevations in CVP are caused by abnormally high intrathoracic or intra-abdominal pressure, which is directly transmitted to the great thoracic veins. The message is clear: as long as the urine output is not adequate and the CVP is low, pour in the fluids. But, remember that your patient may be far behind on fluid in the presence of a high or normal CVP. And another hint: the absolute CVP reading means less than its trend; it is when a low or normal CVP suddenly rises that you have to slow the fluids.

### The Swan-Ganz Pulmonary Artery Flotation Catheter

The Swan-Ganz pulmonary artery flotation catheter measures pulmonary capillary wedge pressure, which reflects the volume status and left cardiac function. Like the CVP catheter, the "Swan" is used in conjunction with the urinary output. We aim for a normal wedge pressure (around 14 mmHg) in conjunction with an adequate urinary output. As with the CVP, a low wedge always means hypovolemia; a high wedge, on the other hand, may indicate either volume overload or dysfunction of the left heart. With the Swan-Ganz in situ, you can calculate and derive information about cardiac function (cardiac output and cardiac index), adrenergic response to injury or illness (peripheral vascular resistance), or tissue perfusion (oxygen consumption and oxygen delivery). A normal cardiac index is a good confirmatory endpoint for resuscitation and, if pre-existing renal failure is present, is a good independent endpoint. When the wedge pressure is normal or high and the urinary output and cardiac index are still low, then pharmacological intervention with inotropic agents may be indicated.

We know that intensivists and junior doctors like to insert central lines, especially Swan-Ganz catheters. Being invasive and able to measure sophisticated data is fun and clinically attractive. But, invasive monitoring maybe a panacea or a Pandora's box. Wedge pressures are notoriously inaccurate in emergency surgical patients—prone to false high reading similar to the CVP. Swan-Ganz catheters are expensive, predisposed to complications, and—above all—they rarely add anything to the management of your patients. Consider this: when was the last time that your anesthesiologist *really* effectively used, intraoperatively, the Swan-Ganz you placed preoperatively? We cannot remember such a case.

### *Laboratory Studies*

The information provided by laboratory studies is easy to interpret. Aim for resolution of hemoconcentration; normalization of electrolyte, BUN, and creatinine levels; and resolution of metabolic acidosis. As mentioned, look at the BE—if persistently negative, the oxygen deficit at the tissue level has not resolved.

### *Blood and Blood Products*

Blood products, such as whole blood, packed red blood cells, fresh frozen plasma, cryoprecipitate, or platelet concentrate, are indicated selectively to restore oxygen-carrying capacity in actively bleeding or chronically anemic patients and to correct clotting abnormalities if present. Do not forget, however, that the blood bank blood is a double-edged sword. Beyond the usual and well-known complications of transfusion, blood is immunosuppressive and may be associated with an increased probability of postoperative infections. In addition, the more blood you give, the higher the risk of postoperative organ system dysfunction and mortality.

Do not forget that rehydration with crystalloids may unmask chronic anemia as the hematocrit falls with volume expansion.

## Suggested Steps in Volume Optimization

- Institute intravenous fluid therapy and if signs of intestinal dysfunction such as nausea, vomiting, or abdominal distension are present, then designate nil per mouth (NPO) and, in more severe cases, nasogastric suction. Intravenous crystalloid may be started at a basic rate of 100–200 ml/h with the addition of boluses of 250–500 ml given over intervals of 15–30 min. We advise you, however, to sit by your patient and completely open the valve of the transfusion set despite the "nurses' desire" to keep it on a pump.
- Institute procedures for monitoring the effectiveness of treatment, including serial physical exam, Foley catheter placement, and in more severe cases, central venous catheter placement. Swan-Ganz? Please, be very selective with this "gimmick."
- If the main underlying problem is hemorrhage, institute transfusion of packed red blood cells—typed and cross-matched if there is time, type specific only if there is not.
- Titrate the rate of fluid administration in light of the results of monitoring. Increase or decrease the basic rate of fluid flow and give additional bolus infusions as necessary.
- After the restoration of intravascular fluid volume, address any residual signs of physiologic dysfunction with inotropic agents to improve cardiac output and, possibly, an afterload reducing agent to improve myocardial oxygen supply and ease the workload of the heart. There is no shame in looking up the dosage and administration recommendations while the fluid is going in.
- Wheel the patient directly to the operating room yourself. Do not wait for the porter—aren't they usually late?
- If the basic problem is continuing hemorrhage, then forget this list and go directly to the operating room. The best resuscitation in actively bleeding patients is surgical control of the source. In addition, preoperative overresuscitation and transfusion increase the blood loss.

## When Is Enough Enough?

The steps discussed in optimization are done with the aim of correcting physiologic derangement as much as possible but without unnecessarily delaying operative intervention. There is no magic formula for achieving this balance. The disease process itself will determine the duration of preoperative optimization. At one end of the spectrum, uncontrolled hemorrhage will require immediate operative intervention after only partial fluid resuscitation or none at all. At the other end of the spectrum, intestinal obstruction that has been developing over several days will require a more complete resuscitation prior to operation. As in life in general, most cases will fall somewhere in between, which means around 3 hrs. Stubborn attempts to "improve" a "nonresponder" beyond 6 hrs are usually counterproductive. That you, or your boss, do not feel like leaving your warm beds at 3 a.m. is not an excuse to "continue aggressive resuscitation" until sunrise.

But stop: perhaps your patient does not need an operation? One of the cleverest aphorisms in surgery was coined by the late Francis D. Moore (1913–2001):

> Never operate on a patient who is getting rapidly better or rapidly worse.

## Conclusions

**The key to preoperative optimization in emergency surgery is oxygenation of the blood and intravenous fluid resuscitation with crystalloid solutions.** The only goal of resuscitation is the restoration of adequate tissue perfusion to supply oxygen to the suffocating mitochondria. Accomplish it aggressively to reduce intra- and postoperative complications.

> These old folks maintain a fragile system quite well … until it gets disturbed—like a house of cards.
>
> "Every operation is an experiment in physiology." (Tid Kommer)

## Editorial Comment

We agree that restoring blood volume is a crucial step before any emergency operation, but at the same time we have to warn you—as we'll do again and

again—not to drown your patients in too much fluid. Pre-op, intra-op, or post-op fluid administration can cut both ways. Equipped with huge-bore intravenous lines and fancy monitoring devices, enthusiastic surgeons and anesthetists commonly flood their patients with too much water and salt. We tend to ignore the "obligatory" postoperative weight gain caused by too aggressive resuscitation with a shrug: "Well," we say, "the patient is perfusing well, and his urine output is excellent—he'll diurese the excess fluids once he's well." But we are wrong!

Recent evidence shows that the deleterious effect of excess fluid is not limited to patients who are actively bleeding (by increasing the rate of hemorrhage and the risk of rebleeding) but can, in fact, be demonstrated in each and every one of our patients. Swollen, edematous cells are bad news in each and every system. Edema contributes to respiratory failure and cardiac dysfunction. It prevents tissue healing, adversely affecting intestinal anastomoses and fascial wounds. It swells abdominal contents, producing intra-abdominal hypertension.

So, do not go overboard. Give only as much fluid as is necessary and, above all, monitor what the anesthetist is doing on the other side of the screen. The old-fashioned formulas used to calculate how much fluid to administer during the operation are exaggerated and outdated. One has to replace blood loss and maintain hourly urine output at 0.5 ml/kg, which practically means 30 ml/h—nothing more. The more unnecessary fluid given before and during the operation—the more problems you will have with the patient in the ICU and on the floor (in the "ward" for non-U.S. surgeons).

## Resuscitation in the Traumatized or Bleeding Patient

We have to forget what the ATLS courses (and book) previously taught us—to flood the patient with crystalloids. Today, we know that overly aggressive fluid resuscitation "washes" out the clot, disturbs hemostasis, increases bleeding, and decreases survival. Hence, the new paradigm is "hypotensive resuscitation": keep the blood pressure just high enough to preserve vital organ perfusion. In practical terms, do not aim for "normal" blood pressure but keep systolic at around 90 mmHg.

In actively bleeding patients, start slowly with Ringer's lactate (do not "pump" it in) and then switch to blood. Recent studies suggested that whole fresh blood is better than component therapy. But, if you have to transfuse more than two units of packed red blood cells, growing evidence suggests that mortality and morbidity rates are improved by adding one unit of fresh frozen plasma and one unit of platelets for each unit of blood given.

This is as true for the injured patient, the one bleeding from an ulcer, and the one with a ruptured aortic aneurysm.

For a "classification" of the urgency of the case, see Table 6.1.

**Table 6.1.** How urgent is urgent

| Urgency | Examples | Meaning |
|---|---|---|
| Immediate | Uncontrolled internal hemorrhage, prolapse of cord | Run to the OR |
| Life-threatening conditions | Leaking abdominal aortic aneurysm | Walk to OR now |
| Potentially life threatening | Perforated viscus<br>Torsion of testis | Take to OR within 2–3 hrs |
| Should not be delayed | Acute appendicitis, intestinal obstruction | Can wait 6 hrs until the morning |
| Can be delayed | Acute cholecystitis | Can wait until the end of the weekend |

# 7 Preoperative Antibiotics

Moshe Schein

*Most men die of their remedies, not of their diseases.* (Molière, 1622–1673)

It is common practice to administer broad-spectrum antibiotics before a laparotomy for an acute surgical condition or trauma. In this situation, antibiotics are either **therapeutic** or **prophylactic**.

**Therapeutic antibiotics**: given for an already established, tissue invasive, infection (e.g., perforated appendicitis).

**Prophylactic antibiotics**: administered in the absence of infection, with the objective of reducing the anticipated incidence of infections due to existing (e.g., penetrating injury of the colon) or potential (e.g., gastrotomy to suture a bleeding ulcer) contamination during the operative procedure.

It is very important to distinguish between *contamination* and *infection* (Chap. 12) as only the latter requires postoperative antibiotic administration, a topic discussed in the postoperative section (Chap. 47). *Therapeutic* antibiotics assist the surgeon and the natural peritoneal defenses to eradicate an established infection. *Prophylactic* antibiotics prevent postoperative infections of the laparotomy wound; they do not prevent pulmonary or urinary infections or the occurrence of intra-abdominal abscesses and should not be administered in an attempt to do any of these things. The overprescription of antibiotics is a modern curse for which our patients pay the price in terms of morbidity and mortality from antibiotic-associated colitis and the emergence of resistant strains. All prescriptions should be provided with a clear purpose in mind and should be for as short a duration as possible. Finally, even dummies know that antibiotics are only an adjunct to the proper surgical management of contamination and infection (Chap. 12).

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## When Should You Start Antibiotics?

There are two schools of thought regarding when to start antibiotics. One says that if intra-abdominal contamination or infection is evident or strongly suspected preoperatively, administer antibiotics immediately—"the sooner the better." When there is delay in proceeding with the laparotomy, give a second dose of preincisional antibiotics in the operating room. Preincisional administration is best when contamination is expected to occur intraoperatively. Some surgeons believe differently and prefer to await the operative findings before giving antibiotics. Should, for example, the acute appendicitis prove to be "simple phlegmonous" (Chap. 28) or the blunt trauma not breach the lumen of a hollow viscus (Chap. 39), they would avoid antibiotics altogether. Alternatively, if contamination or infection were encountered, they would start antibiotic therapy a few minutes after abdominal entry, apparently with no disadvantage. Support for this second philosophy comes from the suggestion that antibiotics liberate endotoxin from the killed bacteria; this leads some surgeons to believe that evacuation of pus (containing the source of endotoxin) should be a prerequisite for commencing antimicrobial therapy.

We, among many others, believe that antibiotics should permeate the tissues at the time of the abdominal incision because immediate vasoconstriction at the incision site would prevent antibiotics—if given later—from reaching the operative wound. Thus, our position is to administer a dose of antibiotics prior to all emergency abdominal operations. When infection or contamination is present or when contamination is expected to occur, the prophylactic or therapeutic value of antibiotics is obvious. In view of the beneficial effects of prophylactic antibiotics in certain elective, clean procedures, we assume that the same may be true in the acutely ill patient who is subjected to laparotomy, even in the absence of contamination or infection. The clinical significance of any antibiotic-generated endotoxemia is presently unknown.

Not uncommonly, we observe surgeons who, in the perioperative chaos, forget to administer antibiotics. To compensate for their failure, they order antibiotics after the operation. This is utterly futile. Are dirty hands washed before or after the meal? The fate of the operative wound is sealed by intraoperative events, including timely administration of antibiotics. Nothing done after the operation can change the outcome of the wound (Chap. 55).

## Which Antibiotics to Use?

Contrary to what is preached by drug companies and their various beneficiaries or representatives—including certain clinicians who function as what we call "antibiotic whores" (others call them "medical advisers")—the choice of

drugs is straightforward. Many single-drug or combination regimens are available and equally effective; the most recent and expensive are not necessarily better. The bacterial flora of abdominal contamination or infection derives from the gastrointestinal tract and is predictable. When a drop of feces leaks into the peritoneal cavity, it contains more than 400 different species of bacteria; only a handful of these are involved in any ensuing infection. Thus, from the initial plethora of contaminating bacteria, the inoculum is spontaneously reduced and **simplified** to include only a few organisms that survive outside their natural environment. These are the endotoxin-generating facultative anaerobes such as *Escherichia coli* and obligate anaerobes such as *Bacteroides fragilis*, which act in synergy. Any agent or combination of agents that effectively kills these target bacteria can be used.

The once-popular "triple regimen" of the 1970s (ampicillin, an aminoglycoside, and metronidazole or clindamycin) has become obsolete. *Enterococcus*, frequently isolated in experimental and clinical peritonitis, is clinically almost nonsignificant as a pathogen in the peritoneal cavity and is not required to be "covered" with ampicillin. Aminoglycosides are markedly nephrotoxic (especially in critically ill patients), are inefficient in the low pH of the infected peritoneal environment, and are no longer the first choice of antibiotics in the initial treatment of intra-abdominal infection. Surgeons tend to be creatures of habit, desperately clinging to dogmas passed on by their mentors; the triple regimen is one such dogma that has been carried into the twenty-first century through ignorance.

You can choose from numerous agents on the market. You may use whichever agent, as monotherapy or in combination—as long as *E. coli* and *B. fragilis* are covered. In abdominal emergencies, the same agent should be used for prophylaxis and treatment. An initial dose of the appropriate drug is given preoperatively and, if indicated by the intraoperative findings, can be continued following the operation. The common (mal)practice of starting with a "weak" agent (e.g., cephazolin) before the operation and converting to the "strong" regimen is baseless.

In the course of the fluid resuscitation of hypovolemic patients, antimicrobials may be "diluted," reducing the availability of antimicrobial drugs at sites of contamination or infection. In these cases, especially in the trauma patient, higher initial doses should be used: "**Sooner and more is better than less and longer**."

## Conclusion

Start antibiotics prior to any emergency laparotomy; whether you continue administration after the operation depends on the operative findings (see Chap. 47). Know the target flora and use the cheapest and simplest regimen. The bacteria cannot be confused, nor should you be!

P.S.: Try to get a copy of Mazuski et al. paper on antimicrobial therapy for intra-abdominal infections. (2002). Or get a PDF of it online: http://www.escmid.org.

"Patients can get well without antibiotics." (Mark M. Ravitch, 1910–1989)

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# 8 Family, Ethics, Informed Consent and Medicolegal Issues

James C. Rucinski

*Doctor, my doctor, what do you say … ?* (Philip Roth)

*Stop lying! You know, and I know, that I am dying. So do at least stop lying about it!* (Leo Tolstoy, 1828–1910)

To understand the dying man you have to read Tolstoy's *The Death of Ivan Ilyich*. [The Editors]

The wind whistles through the cracks in your call room window when the emergency department (ED) calls, and suddenly you find yourself in the maelstrom of that environment, speaking to a small group of extremely anxious strangers—having to explain that an immediate operation will be required to save their beloved one. The operating room (OR) is ready.

Obtaining informed consent is a practical combination of salesmanship, ethical problem solving, and psychological nurturing. It involves the rapid marketing of one's own skills and plan for treatment. It requires the recruitment of the patient and the family as allies in the decision-making process. More than a legal requirement, however, informed consent requires an ethical commitment to the patient, your peers, and yourself.

## Salesmanship

Begin by explaining the problem and your proposed treatment using the same words and language that you might use in speaking to one of your nonmedical relatives. Describe the expected benefits of operation and what the consequences of alternative treatment approaches might be. (What happens if we do nothing.) Offer several scenarios. Take a case of obstructing carcinoma of the

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sigmoid colon, for example. At one end of the spectrum is nonoperative management, which almost certainly will result in a slow and difficult death. At the other end of the spectrum is rapid recovery from operation with long-term cure of the disease. In between lay the potential difficulties of perioperative complications or death, recovery with disability, or recurrent disease. It is crucial that you believe in the plan of treatment that you propose. If this is not the case, and the plan is not acceptable to you but dictated to you from above, then let the responsible surgeon (your boss) conduct his or her own preoperative "negotiations" with the patient and the patient's family.

"Sell" yourself to the patient and family as a scientific expert who recognizes the needs of another person and is participating with them in solving a difficult problem. Include a description, with approximate probabilities, of the most common "problems" (complications) for the proposed procedure in your particular patient. You will need to make an estimate based on general and specific information. For example, the risk of mortality for elective colon resection may be negligible, but in an elderly patient with acute colonic obstruction and hypoalbuminemia the odds of dying may be one in four (Chap. 6). Discuss general potential postoperative complications such as infection, hemorrhage (and risk of transfusion), poor healing, and death. Then, mention the unique complications specific to the procedure you are proposing to undertake, such as common bile duct injury or bile leak in laparoscopic cholecystectomy.

It is crucial that before any major emergency abdominal operation you emphasize that a reoperation may be necessary based on your operative finding or if a problem subsequently develops. This will drastically facilitate the "confrontation" with the family when a reoperation is indeed indicated (Chap. 52); they will understand that the reoperation represents a "continued management effort" rather than a "complication." Minor complications, such as phlebitis arising from perioperative intravenous therapy, may contribute to information overload and probably should be omitted. Try to conduct the above "script" in a relatively quiet setting—away from the usual chaos of the ER, surgical intensive care unit (SICU), or the OR. Use simple language and repeat yourself ad libitum; stressed family members may have difficulty grasping what you say. Offer the opportunity to ask questions and assess whether there is understanding of your discussion. The more they understand initially, the fewer "problems" you will have if complications subsequently develop. Be "human," friendly, empathetic, but professional. A good trick is to remind yourself from time to time that the family you are talking to could be yours. Finally, always leave open the possibility that what you think the problem to be is not correct. Similarly, if you are asked to provide a prognosis, always allow for the unexpected, both good and bad, so that if a disaster or a miracle should occur this will not be outside the bounds of the possibilities you outlined.

### Illustrate the Problem

When discussing the prospects of an operation with a patient or a family, we find that illustrating the problem and the planned procedure on a blank piece of paper greatly enhances the communication. Draw, schematically, the obstructed colon: "Here is the colon; this is the obstructing lesion, and here is the segment we want to remove. We hope to be able to join this piece of bowel to that one, a colostomy may, however, be needed; this is the place it will be brought out." Below the drawing, write the diagnosis and the name of the planned operation. At the end of the consultation, you will be surprised to see how carefully members of the family restudy the piece of paper you left with them, explaining to each other the diagnosis and planned operation. Very often, patients and their relatives are very enthusiastic about keeping any drawings you make for them.

## The Family

> When it comes to operation, you advise the patient and he and his family decide.

The patient's family is your greatest ally in promoting your plan of action. By involving them at an early point in the decision-making process, you may be able to make them partners in the relationship that you share with the patient. By avoiding the family, you may alienate potential allies or worsen an already "difficult" group. The *difficult family* is common. Long submerged conflicts and feelings of guilt tend to surface when a member of the group becomes ill. Recruit them as allies by offering them a chance to participate, by "reading" the nuances of their relationships, and by confidently and continuously selling yourself as a knowledgeable and compassionate advisor. Use your first meeting with the family to make a good impression and gain their trust so that you will continue to be trusted if a complication arises or if further therapy becomes necessary. Remember that if things turn out badly, it will be the surviving family members who will want to know "What went wrong?"

## Ethical Problem Solving

To sell a particular product or idea, one must believe in it. In other words, based on your knowledge and experience, the operation you offer should appear ethical to you. It is ethical if it is expected to save or prolong the patient's life or palliate the patient's symptoms and can achieve this goal with a reasonable risk-benefit ratio. At the same time, you must also be convinced that there are no

nonoperative treatment modalities that are safer or as effective as your proposed operation. The burden of proof is on you.

## Medicolegal Considerations

> Surgery is the most dangerous activity of legal society. (P. O. Nystrom)

The medicolegal dangers associated with emergency abdominal surgery greatly depend on where you practice. In some countries, surgeons can get away with almost anything; in other countries, emergency surgery is a legal minefield. There are a few simple but well-proven tactics to prevent lawsuits against you:

- Have the patient and family "on your side" (as mentioned) by being empathetic, caring, honest, open, informative, and at the same time professional. Young surgeons tend to be overoptimistic, trying to cheer up the family. A common scenario finds the surgeon emerging from the OR, assuming a "tired hero" pose, and announcing: "It was smooth and easy; I removed the cancer from the colon, relieving the obstruction. I was able to join the ends of the bowel together—avoiding a colostomy. Yes, your father is stable, he took the operation very well, let's hope he'll be home next week for Easter (or Passover or Ramadan)." Such a script is somewhat misguided in that it may raise high hopes and expectations, with subsequent anger and resentment if complications should develop. The better script might be: "The operation was difficult, but we managed to achieve our goals. The cancer is out, and we avoided a colostomy. Considering your father's age and other illnesses, he took it well. Let us hope for the best, but you must understand that the road to recovery is long, and as I mentioned before the operation, there are still many potential problems ahead."
- Detailed informed consent (Fig. 8.1).
- Documentation. This is crucial as "what has not been documented in writing did not actually take place." Your notes can be brief but must encompass the essentials. Prior to an emergency laparotomy for colonic obstruction, we would write: "78 YO male patient with hypertension, diabetes, and COPD [chronic obstructive pulmonary disease]. Three days of abdominal pain plus distension. Abdominal X-ray—suggesting a distal large bowel obstruction—confirmed on Gastrografin study. APACHE II score on admission 17—making him a high risk. Therapeutic options, risks, and potential complications, including anastomotic leak, wound infection, respiratory failure, explained in detail to the patient and family, who accept the need for an emergency laparotomy. They understand that a colostomy may be needed and that further operations may be necessary." A few years later—in court—this short note will prove invaluable to you.


**Fig. 8.1.** "Is he going to sign?"

## Avoid Selling Autopsies Under Anesthesia

We compared you above to an astute salesman, interacting with the patient and the patient's family. In this capacity, you, a respected clinician, can easily sell anything to the trusting clients. Be honest with yourself and consider as objectively as possible the risk-benefit ratio of the procedure you are trying to "sell." It may be easy to convince a worried family that a (futile) operation is indeed necessary and then at the inevitable M & M (morbidity and mortality) meeting (Chap. 59) to explain that the family forced the AUA (autopsy under anesthesia) on you. Easy and ethical do not always coexist!

> "One should advise surgery only if there is a reasonable chance of success. To operate without having a chance means to prostitute the beautiful art and science of surgery." (Theodor Billroth, 1829–1894)

## Concluding Remarks

Not only is what you say important but also how it is said. Introduce yourself and all members of your team who are present. Shake hands with *all* members of the family. Conduct the "session" in a sitting position—you sitting at eye

level, or lower, with the patient and the patient's family. Maintain constant eye contact with each of them—do not ignore the ugly daughter hiding in the corner of the room—she may be the one who becomes your enemy. Be "nice" but not "too nice"—this is not the time to smile or joke around. Just play the serious surgeon committed to the well-being of the patient. This surgeon is you, so play yourself!

Nothing is truer than the cliché that should be constantly replayed in your mind: would you recommend the same treatment to your father, mother, wife, or son? Studies show that surgeons are much less likely to recommend operations on themselves or their loved ones. Do unto others as you would have them do unto you—the golden rule.

> "The patient's family will never forgive a guarantee of cure that failed and the patient will not let the physician forget a pronouncement of incurability if he is so fortunate as to survive." (George T. Pack, 1898–1969)

# 9 Before the Flight: Pre-Op Checklist

Moshe Schein

*The pilot is by circumstances allowed only one serious mistake, while the surgeon may commit many and not even recognize his own errors as such.* (John S. Lockwood)

Like any military or commercial pilot, prior to any flight, you have to go over a "checklist" (Fig. 9.1). In fact, the need to check everything obsessively is more crucial to you than to the pilot. For while a team of dedicated and well-trained maintenance professionals surround the pilot, you are not uncommonly surrounded only by jerks. We do not want to be abusive or rude, but let us be realistic—at 2 a.m. your intern or junior resident is much more interested in his lost sleep than your prospective operation. And the anesthetist? Your emergency case is just a pain in the ass. The sooner he or she can administer the gases, the sooner he or she can dump your "case" in the recovery room or intensive care unit and the sooner they can crawl under the comfort of their warm duvet. The nursing staff? Forget them. Not in vain today are they called OR *technicians*. (Lest we be accused of painting with too wide a brush, there are always the wonderful exceptions in this scenario; let them know they are appreciated!)

So face it—you are alone; it is always a solo flight, and you can count only on yourself. You are responsible for the success, failure, morbidity, mortality, and potential lawsuit. His or her fate is in your hands. This patient, regardless of how many people are buzzing around him, is yours. So, wake up and go over the checklist.

## The Checklist

- **Does the patient really need the operation?** The cliché that it is more difficult to decide when not to operate than when to operate is mentioned elsewhere in this book. Variations of this aphorism are circulating around the world in many languages. But, even more difficult is to decide against the operation *after* the operation has been scheduled. So, you decided to book the patient for appendectomy based on what the chief resident told you over the phone—that "the CT [computed tomograph] is compatible with acute appendicitis"—and now, when you arrive in the OR, you find the patient smiling and sitting in bed with a soft and nontender abdomen. Do you want to operate on the CT or the patient? You

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**Fig. 9.1.** "Doctor, show me your pilot's license."

do not need big balls (or ovaries) to book a patient for operation, but you need large balls to *cancel* the operation and order the patient back to the floor (ward). You need huge balls to *remove* the patient from the operating table and massive balls to tell the anesthetist to *wake* him up … but if you palpate a large appendiceal mass (see Chap. 28) after the induction of anesthesia and abdominal wall relaxation—what is the point of continuing?

- **Examine the patient before the patient is put to sleep.** Never ever—we repeat—never, never, never, ever operate on a patient without having examined the patient yourself; if you do, then you are a butcher. That the endoscopist visualized a "bleeding ulcer" and the patient continues to vomit blood may be an indication for operation, but this is your chance to diagnose the large spleen and ascites, which were hitherto overlooked by the others. You do not want to operate on a Child's C portal hypertension patient, or do you? (See Chap. 17.)
- **Look at the X-rays and imaging studies.** Review all X-rays and imaging studies by yourself. Do not rely only on what the radiologist said or wrote. You may pick up findings, which may move you to cancel the operation or to decide on a different incision.
- **Position the patient.** Already before you start, you have to have a general idea what you are going to do or what you may have to do. This has an impact on your patient's position. For example, does the patient need a Lloyd-Davies position, offering access to the anus and rectum? This may be needed during colorectal procedures—to insert a scope, to decompress the colon, or to insert a stapler. You do not want to have to stop the operation and place the patient in the correct position or to send the intern crawling under soggy drapes to play peekaboo with the anus. In

whatever position your patient is to be, check that all limbs are protected and well padded at potential pressure sites. Poor positioning on the operating room (OR) table may result in damage to nerves, skin ulceration, and compartment syndrome of the extremities—and a lawsuit.

- **Warm your patient.** See that the patient is well covered and warmed. Hypothermia increases the likelihood of postoperative infections and contributes to intraoperative coagulopathy.
- **Think about preventing deep vein thrombosis (DVT).** Prevention of DVT should be initiated before the patient is put to sleep—not after the operation. Any abdominal procedure lasting longer than 30 min is associated with a moderate risk of DVT; you can add to this specific risk factors such as smoking, use of oral contraceptives, previous history of DVT, age, obesity, presence of a cancer, and so forth. But, instead of pondering too much—why don't you provide all your patients undergoing an emergency abdominal operation with DVT prophylaxis? Whether it is in the form of subcutaneous heparin or calf compression depends on what your OR can offer. Bear in mind that anticoagulation is not good for an exsanguinating patient. We have seen young patients dropping dead from pulmonary embolism a few days after appendectomy and young women developing intractable postphlebitic syndromes following appendectomy performed for pelvic inflammatory disease. Always think about this.
- **Is the bladder empty?** Most patients undergoing emergency operations arrive at the OR with a urinary catheter in place; for the rest, you will insert the catheter on the table. But, if contemplating a lower abdominal procedure on a noncatheterized patient, you have to check that the bladder is empty. When the bladder is full, it may look to you like the peritoneum. Bladder distension may also mimic a surgical abdominal condition, not rare in a mentally challenged patient.
- **Think antibiotic prophylaxis** (see Chap. 7).
- **Document everything** (see Chap. 8).

The formal "OR time-out"—the final review by the nursing team of the side, site, and nature of the procedure—cannot, and should not, come instead of your own checklist.

Now, you can go and scrub. While doing so, continue to think and contemplate about what you are going to do. Do not behave like Tolstoy's surgeon in *War and Peace*: "He … joked … and chatted carelessly, as a famous surgeon confident that he knows his job will often chat while he tucks up his sleeves and puts on his apron, and the patient is being strapped to the operating table. *'I have the whole business at my finger-tips, and it's all clear and definite in my head, When the time comes to set to work I shall do it as no one else could, but now I can jest, and the more I jest and the cooler I am the more hopeful and reassured you ought to feel, and the more you may wonder at my genius'*."

Remember:

> Many lives have been saved by a moment of reflection at the scrub sink. (Neal R. Reisman)

You are the captain of the ship—behave like one; the sight of a euphoric surgeon dramatically entering the room with scrubbed hands held high in the air is pitiful.

> "Poor judgment is responsible for much bad surgery, including the withholding of operations that are necessary or advisable, the performance of unnecessary and superfluous operations, and the performance of inefficient, imperfect, and wrongly chosen ones." (Charles F.M. Saint, 1886–1973)
>
> The surgeon, like the captain of the ship or a pilot of an aircraft, is responsible for everything that happened. His word is the only one that cannot be gainsaid. (Francis D. Moore, 1913–2001)

# The Operation

B

# 10 The Incision[1]

Moshe Schein

*Incisions heal from side to side, not from end to end, but length does matter.*

*When entering the abdomen, your finger is the best and safest instrument.*

The patient now lies on the table, anesthetized, and ready for your knife. Before you scrub, carefully examine the relaxed abdomen. Now, you can feel things that were impossible to feel in the tense and tender belly. You may feel a distended gallbladder in a patient diagnosed with an acute appendicitis or an appendiceal mass in a patient booked for a cholecystectomy. Yes, this may also occur in the era of ultrasound and computed tomography (CT).

Traditionally, abdominal entry in an emergency situation or for exploratory purposes has been through a generous and easily extensible vertical incision, especially a midline one. Generally, the trans linea alba midline incision is swiftly effected and relatively bloodless. On the other hand, transverse incisions are a little more time and blood consuming but are associated with a lower incidence of wound dehiscence and incisional hernia formation. In addition, transverse incisions are known to be "easier" on the patient and the patient's lung function in the postoperative period. Vertical paramedian incisions largely belong to history.

Keeping this in mind, we should **be pragmatic rather than dogmatic** and tailor the incision to the individual patient and his or her disease process. We should take into consideration the urgency of the situation, the site and nature of the condition, the confidence in (or uncertainty about) the preoperative diagnosis, and the build of the patient.

*Common sense dictates that the most direct access to the specific intra-abdominal pathology is preferable.* Thus, the biliary system is best approached through a transverse, right subcostal incision. Transverse incisions are easily lengthened to offer additional exposure; a right subcostal incision can be extended into the left side (as a "chevron"), offering an excellent view of the entire abdomen. When a normal appendix is uncovered through a limited, transverse, muscle-splitting, right lower quadrant incision, one can extend it by cutting the

[1] Asher Hirshberg, MD, contributed to this chapter in the first edition of the book.

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muscles across the midline to deal with any intestinal or pelvic condition. Alternatively, when an upper abdominal process is found, it is perfectly reasonable to close the small right iliac fossa incision and place a new, more appropriate, one. Two good incisions are better than one, poorly placed.

The midline incision—bloodless, rapid, and easily extended—affords superior exposure and versatility; it remains the classic "incision of indecision" when the site of the abdominal catastrophe is unknown and is the safest approach in trauma.

This is an occasion to mention that an emergency laparotomy without a diagnosis is not a sin. Do not surrender to the prevailing dogma that the patient cannot enter the operating theater without a ticket from the CT scanner. A clinical acute abdomen—when other diagnoses have been ruled out (see Chaps. 3 and 4)—remains an indication for laparotomy when the abdominal wall is the only structure separating the surgeon from an accurate diagnosis. Having said this, mainly to satisfy and pacify those of you who work under adverse circumstances, we have to admit that preoperative abdominal imaging (see Chap. 5) is of great help in choosing the correct incision. For example, in a patient needing splenectomy for a delayed rupture of the spleen, we would place a left subcostal incision rather than a midline one. The CT has shown us that this is an isolated splenic injury, and there is no need to explore the rest of the abdomen.

## At What Level Must the Midline Incision Start, and How Long Should It Be? (Fig. 10.1)

The macho surgeons of previous generations often screamed: "Make it long. It heals from side to side, not from end to end." Today, in the era of minimal-access

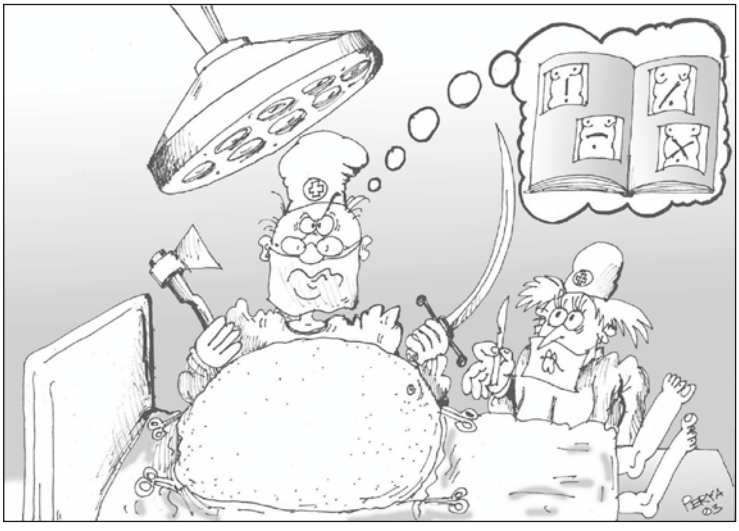

**Fig. 10.1.** "Which incision?"

surgery, we are familiar with the advantages of shorter incisions. In the absence of any obvious urgency, enter the abdomen through a short incision and then extend as necessary, but never accept less-than-adequate exposure or strive for keyhole surgery. Begin with an upper or lower midline incision, directed by your clinical assessment; when in doubt, start near the level of the umbilicus and "sniff" around from there, then extend toward the pathology. Just remember what the famous Swiss surgeon Theodor Kocher said more than 100 years ago: "The incision must be as long as necessary and as short as possible."

### Should You Extend Your Incision Into the Thorax?

Very rarely should the incision extend into the thorax. In the vast majority of cases, infradiaphragmatic pathology is approachable through abdominal incisions. The combination of a subcostal and upper midline incision offers an excellent exposure for almost all emergency hepatic procedures, with the exception of retrohepatic venous injuries, for which insertion of a transatrial vena cava shunt necessitates a median sternotomy—usually a futile exercise anyway. Thoracoabdominal incisions are mainly reserved for combined thoracoabdominal trauma.

### Knife or Diathermy?

A few studies suggest that the diathermy is a few minutes slower than the knife, while the knife sheds a few more drops of blood; otherwise, results are comparable. We use either. In extreme urgency, gain immediate entry with a few swift strokes of the knife; otherwise, diathermy is convenient, especially when performing transverse muscle-cutting incisions. Adequate hemostasis is a crucial surgical principle but do not go overboard chasing individual erythrocytes and avoid reducing the subcutaneous fat or skin to charcoal. The hypothesis that "You can tell how bad the surgeon is by the stink of the Bovie [electrocautery] in his OR" has not been proven by a double-blind randomized trial but makes sense nonetheless.

Subcutaneous hemostatic ligatures behave like a foreign body and are almost never necessary. In fact, most incisional "oozers" stop spontaneously, after a few minutes, under the pressure of a moist lap pad or a temporary hemostat. It is also unnecessary to "clean" the fascia by sweeping the fat laterally: the more you dissect and "burn," the more inflammation and infection-generating dead tissue you create!

### Keep in Mind Special Circumstances

If a stoma is anticipated, then place the incision away from its planned location. Abdominal re-entry into the "hostile abdomen" of a previously operated

patient can be problematic; you may spend more time, sweat, and blood, but the real danger is creating inadvertent enterotomies in intestine adherent to the previous incisional scar. This is a common cause of postoperative external bowel fistula (Chap. 50). The prevailing opinion is to use the previous incision for reentry, *if possible*. When doing so, however, start a few centimeters below or above the old incision and gain entry to the abdomen through virgin territory. Then, insert your finger into the peritoneal cavity and navigate your way safely in, taking down adhesions to the abdominal wall, which hamper the insertion of a self-retaining retractor. Essentially, you are finished "getting in" when you are able to place a self-retaining retractor to open the abdomen wide. In a dire emergency or when you expect the abdomen to be exceptionally scarred, it may be prudent to stay away from trouble and create an entirely fresh incision. In this situation, beware of parallel incisions in close proximity to one another because the intervening skin may be at risk of necrosis, particularly if the first incision is relatively recent.

## Pitfalls

- When in haste, do not forget that the *liver* lies in the upper extremity of the long midline incision and the *urinary bladder* at its lowermost. Be careful not to damage either.
- When approaching the upper abdomen, divide and ligate the *round* hepatic ligament. Leave it long; it could be used to elevate and retract on the liver. Take the opportunity to divide the bloodless *falciform* ligament, which runs from the anterior abdominal wall and the diaphragm to the liver. If left intact, it may "tear" off the liver, causing irritating bleeding.
- When performing any transverse incision across the midline, do not forget to ligate or transfix the *epigastric vessels* just behind the rectus abdominis muscles. They may retract and cause a delayed abdominal wall hematoma.
- **In the very obese patient**, in the upright position, the umbilicus commonly reaches the level of the pubis. After elevating the fat panniculus, you can place a lower midline incision between the pubis and umbilicus, but after the operation it will be macerated by the sweaty (and smelly) panniculus. Thus, in the superfat, a supraumbilical midline incision would provide better access into the lower abdomen.

> "Pray before surgery, but remember God will not alter a faulty incision." (Arthur H. Keeney)
>
> "When the doctor is in doubt and the patient in danger, make an exploratory incision and deal with what you find as best as you can." (Robert Lawson Tait, 1845–1899)

# 11 Abdominal Exploration: Finding What Is Wrong[1]

Moshe Schein

*Never let the skin stand between you and the diagnosis. (We understand that this is an old and venerated surgical aphorism but we don't wish to encourage reckless pursuit of diagnosis, do we?*—The Editors*)*

*"In surgery, eyes first and most; fingers next and little; tongue last and least."* (Humphrey George Murray, 1820–1896)

Not uncommonly—especially with the increased use of diagnostic imaging—when opening the abdomen, the surgeon knows what to expect inside; the clinical picture or ancillary tests direct the surgeon to the disease process. In some instances, however, the surgeon explores the unknown, led on only by the signs of peritoneal irritation, assuming that the peritoneal cavity is flooded with blood or pus. Usually, the surgeon speculates about the predicted diagnosis but always remains ready for the unexpected. This is what makes emergency abdominal surgery so exciting and demanding—the ever-looming catastrophe and the anxiety about whether you are able to tackle it competently. Yes, even in the days of computed tomography (CT) and magnetic resonance imaging (MRI), the abdomen can be full of surprises.

## Abdominal Exploration (› Fig. 11.1)

Although the specific sequence and extent of abdominal exploration are to be tailored to the clinical circumstances, the two principal stages of any exploration are:

- Identification of the specific pathology that prompted the laparotomy
- Routine exploration of the peritoneal cavity

Essentially, there is a sharp distinction between a laparotomy for nontraumatic conditions such as bowel obstruction, inflammation, or peritonitis and laparotomy for trauma with intra-abdominal hemorrhage, the latter rarely being due to spontaneous, nontraumatic intra-abdominal causes.

[1]Asher Hirshberg, MD, contributed to this chapter in the first edition of the book.

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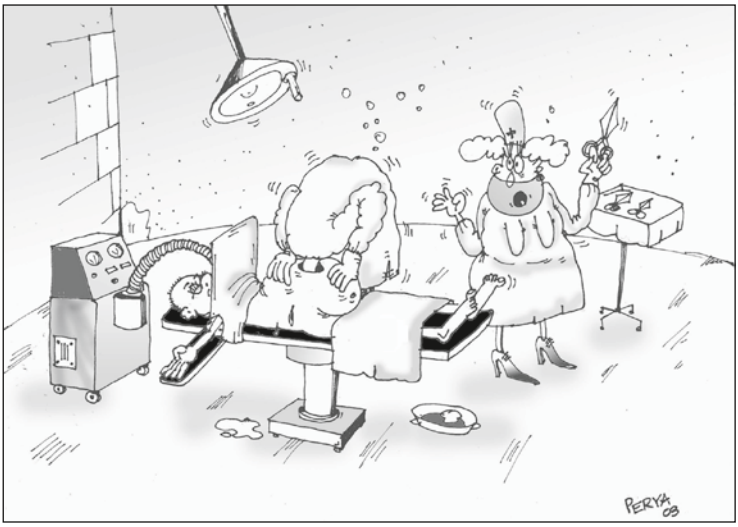


**Fig. 11.1.** "Hey Doc, did you find anything?"

So, you incise the peritoneum, what now? Your action depends on the urgency of the situation (condition of the patient), the mechanism of the abdominal pathology (spontaneous vs. trauma), and the initial findings (blood, contamination, or pus). Whatever you find, follow the main **priorities**:

- Identify and arrest active bleeding
- Identify and control continuing contamination

At the same time, **do not be distracted by trivia**. Do not chase isolated red blood cells or bacteria in a patient who is bleeding to death. For example, do not repair minor mesenteric tears in a patient who is busy exsanguinating from a torn inferior vena cava. This is not a joke—surgeons are easily distracted.

### *Intraperitoneal Blood*

The patient may have suffered a blunt or penetrating injury or no injury at all; in the latter case, the patient is suffering from spontaneous intra-abdominal hemorrhage (**abdominal apoplexy**), an uncommon entity. Its etiology is summarized in Table 11.1.

You may have been expecting the presence of free intraperitoneal blood from the clinical findings of hypovolemic shock or the results of CT, ultrasound, or peritoneal lavage. Your action depends on the magnitude of hemorrhage and the degree of resulting hemodynamic compromise. When the abdomen is full of blood and the patient unstable, you should act swiftly.

**Control the situation:**

- Enlarge your initial incision generously (avoid liver and bladder)
- Lift out the small bowel completely
- Suck out blood as fast as possible (always have two large suckers ready)
- Pack the four quadrants tightly with laparotomy pads

**Table 11.1.** Causes of spontaneous intra-abdominal hemorrhage ("abdominal apoplexy")

*Vascular*
Ruptured abdominal aortic aneurysm
Ruptured arterial visceral aneurysm (hepatic, gastroduodenal, splenic, pancreaticoduodenal, renal, gastroepiploic, middle colic, inferior mesenteric, left gastric, ileocolic [may be associated with Ehlers-Danlos syndrome])
Intraperitoneal rupture of varices associated with portal hypertension
Spontaneous rupture of the iliac vein

*Gynecological*
Ruptured ectopic pregnancy
Spontaneous rupture of the pregnant uterus with placenta percreta
Postpartum ovarian artery rupture
Spontaneous ovarian hemorrhage (idiopathic, ruptured follicular cyst or corpus luteum, ovarian cancer)

*Pancreatitis*
Erosion of adjacent vessels involved in the process of severe acute pancreatitis, chronic pancreatitis, or pancreatic pseudocyst

*Liver*
Rupture of benign (typically adenomas) or malignant hepatic tumors

*Spleen*
Spontaneous rupture

*Adrenal*
Spontaneous hemorrhage: normal gland or secondary to tumor

*Kidney*
Spontaneous rupture: normal kidney or secondary to tumor

*Anti-coagulation*
Patients on anticoagulation are prone to spontaneous retroperitoneal or intraperitoneal bleeding, often prompted by unrecognized minor trauma

*Unrecognized or denied trauma*
Patient "forgot" the kick to the left upper quadrant, which broke the spleen

*Miscellaneous*
Acute ruptured cholecystitis
Mediolytic arteritis of an omental artery
Periarteritis nodosa

**Evacuation of massive hemoperitoneum temporarily aggravates hypovolemia.** It releases the tamponade effect and relieves intra-abdominal hypertension (Chap. 40), resulting in sudden pooling of blood in the venous circulation. At this stage, compress the aorta at its diaphragmatic hiatus and let the anesthetist catch up with fluid and blood requirements.

Be patient, do not rush forward; with your fist on the aorta, the abdomen tightly packed, and the patient's vital organ perfusion improving, you have almost all the time in the world. Do not be tempted to continue with the operation, which can result in successful hemostasis in a dead patient. Relax and plan the next move, remembering that from now on you can afford to lose only a limited amount of blood before the vicious cycle of hypothermia, acidosis, and coagulopathy, "the triangle of death," will further frustrate efforts to achieve hemostasis.

### Primary Survey

Now, you are ready to identify and treat the life-threatening injuries. The initial direction of your search will be guided by the causative mechanisms. In penetrating injury, the bleeding source should be in the vicinity of the missile or knife tract; in blunt trauma, bleeding will probably originate from a ruptured solid organ—the liver or spleen—or the pelvic retroperitoneum.

Unpack, suck, and repack each quadrant consecutively, noting where there is blood reaccumulation (active bleeding) or hematoma. Having accurately identified the source (or sources) of bleeding, start definitive hemostasis, the rest of the abdomen being packed away. Simultaneously, if the situation permits, control contamination from injured bowel using clamps, staplers or tapes, or repacking in desperate situations.

**Stay tuned constantly to events behind the blood–brain barrier (BBB)**, which is the screen between you and the anesthetists. Wake them up from time to time and ask how the patient is doing. Take this opportunity also to explain how and what you are doing. Communication among members of the medical team in this situation is vital. While you are busy repairing the iliac vein, the patient may be developing a pericardial tamponade or pneumothorax.

### Secondary Survey

Now, the exsanguinating lesion is permanently or temporarily controlled, and the patient's hemodynamics have stabilized. With less adrenaline floating around you and the patient, you can divert your attention to all the rest and look around more precisely. With growing experience, your abdominal exploration will become more efficient but never less thorough as "missed" abdominal injuries

continue to be a common source of preventable morbidity. The praticalities of systemic abdominal exploration are described in a separate section.

### *Intraperitoneal Contamination or Infection*

First, you register the offensive fecal smell or fecal-looking fluid that denotes abundance of anaerobic bacteria and usually an infective source in the bowel. Note, however, that neglected infections from any source can be *pseudofeculant* due to the predominance of anaerobes. When, on opening the peritoneum, gas escapes with a hiss, be aware that a viscus has perforated. In the nontrauma situation, this usually implies perforated peptic ulcer or sigmoid diverticulitis. Bile staining of the exudate points to pathology in the biliary tract, gastroduodenum, or proximal small bowel. Dark, stout-beer fluid and fat necrosis hint at pancreatic necrosis or infection in the lesser sac. John Hunter (1728–1793) observed that "the gastric juice is a fluid somewhat transparent, and a little saltish or brackish to the taste," but we would not suggest you go that far. Whatever the nature of contamination or pus, suck and mop it away as soon as possible.

Generally, bile directs you proximally and feces distally, but "simple" pus can come from anywhere. When its source remains elusive, start a systematic search, keeping in mind all potential intra- and retroperitoneal sources "from the esophagus to the rectum." Be persistent with your search. We recall a case of spontaneous perforation of the rectum in a young male, twice explored by experienced surgeons who failed to appreciate the minute hole deep in the rectovesical pouch. It was found during a third operation.

Occasionally, however, the origin of contamination or secondary peritonitis is not found. A Gram stain disclosing a *solitary* bacterium—as opposed to a few—would support the diagnosis of **primary peritonitis** since *secondary peritonitis* (e.g., secondary to a visceral pathology) is always polymicrobial. More about this in Chap. 12.

## The Direction and Practicalities of Exploration

The direction and practicalities of exploration depend on the reason for the laparotomy; let us start with a general plan.

The peritoneal cavity comprises **two compartments: the supracolic and the infracolic.** The dividing line is the transverse (meso)colon, which in a xiphopubic midline incision is located approximately in the center of the incision. It is important to develop and adhere to a fixed routine of abdominal exploration, which

will include both compartments. Our preference is to begin with the infracolic compartment: The transverse colon is retracted upward, the small bowel eviscerated, and the rectosigmoid identified. Exploration begins with the pelvic reproductive organs in the female and then attention is turned to a systematic inspection and palpation of the rectosigmoid, progressing in a retrograde fashion to the left, transverse, and then right colon and cecum, including inspection of the mesocolon. The assistant follows the exploration with successive movements of a handheld retractor to retract the edge of the surgical incision and to enable good visualization of whichever abdominal structure is the focus of attention. Exploration then proceeds in a retrograde fashion from the ileocecal valve to the ligament of Treitz, with special care taken to inspect both "anterior" and "posterior" aspects of each loop of bowel as well as its mesentery.

Attention is then turned to the **supracolic compartment**. The transverse colon is pulled down, and the surgeon inspects and palpates the liver, gallbladder, stomach (including the proper placement of a nasogastric tube), and spleen. Special care should be taken to avoid iatrogenic damage to the spleen caused by pulling hard on the body of the stomach or the greater omentum. A complete abdominal exploration also includes entry into the lesser peritoneal sac, which is best undertaken through the gastrocolic omentum. This omentum is usually only a thin avascular membrane on the left side, and this should therefore be the preferred entry route into the lesser sac. Take care to avoid injury to the transverse mesocolon, which may be adherent to the gastrocolic omentum. A misdirected surgeon can be convinced that he or she is entering the lesser sac when in fact he or she is cutting a hole in the transverse mesocolon. If "vascular," the gastrocolic omentum is divided between ligatures, bringing the body and tail of the pancreas into full view.

**Exploration of retroperitoneal structures** involves two key mobilization maneuvers, which should be employed whenever access to the retroperitoneum is deemed necessary:

- **Kocher's maneuver** is mobilization of the duodenal loop and the head of the pancreas by incising the thin peritoneal membrane (posterior peritoneum) overlying the lateral aspect of the duodenum and gradually lifting the duodenum and pancreatic head medially. This maneuver is also the key to surgical exposure of the right kidney and its hilum and the right adrenal gland. Kocher's maneuver may be extended further caudad along the "white line" on the lateral aspect of the right colon all the way to the cecum. This extension allows medial rotation of the right colon and affords good exposure of the right-sided retroperitoneal, structures such as the inferior vena cava, iliac vessels, and the right ureter. Further extension of this incision angles around the cecum and continues in a superomedial direction along the line of fusion of the small bowel mesentery to the posterior abdominal wall. Thus, it is possible to mobilize and reflect the small bowel upward, the so-called Catell-Braasch maneuver. This affords optimal exposure of the entire inframesocolic retroperitoneum, including the aorta and its infrarenal branches.

The second key mobilization maneuver is called the *left-sided Kocher* or *medial visceral rotation* (also called by some the *Mattox maneuver*) and is used especially to gain access to the entire length of the abdominal aorta and to the left-sided retroperitoneal viscera. Depending on the structures to be exposed, this maneuver begins either lateral to the spleen (splenophrenic and splenorenal ligament), working caudally or in the white line of Toldt lateral to the junction of the descending and sigmoid colon, working upward. The peritoneum is incised, and the viscera, including the left colon, spleen, and tail of pancreas are gradually mobilized medially. The left kidney can either be mobilized or left in situ, depending on the surgical target of the exploration.

In cases of **spontaneous hemoperitoneum**, you will have to look for a ruptured aortic, iliac, or visceral arterial aneurysm, ectopic pregnancy, bleeding hepatic tumor, spontaneous rupture of an enlarged spleen, or any of the other causes listed in Table 11.1. In **penetrating trauma**, you will follow the entry–exit tract, taking into consideration the missile's energy, velocity, and potential to fragment. Wherever there is an entry wound in a viscus or blood vessel, look for the exit one. The latter may lie concealed on the lesser sac wall of the stomach, the retroperitoneal surface of the duodenum, or the mesenteric edge of the small bowel. Missing an exit wound is often a death sentence to your patient. It is the **blunt abdominal injury**, however, that requires the most extensive and less-directed search, from the surface of both hemidiaphragms to the pelvis, from gutter to gutter, on all solid organs, along the whole length of the gastrointestinal tract, and on the retroperitoneum (the retroperitoneum selectively, as discussed in Chap. 39). The exact sequence of exploration is less important than its thoroughness.

**Comment**: Because this book is aimed also at trainees, we had to be complete and describe the "classical abdominal exploration." Frankly, if the patient is bleeding from a ruptured liver, we would explore the upper abdomen, but if the infracolic compartment looks pristine and dry, we would leave it alone. So, use your common sense: do not look for ovarian cysts in a patient with a bleeding spleen. Like Dr. Leo Gordon said: "When common sense interferes with a protocol, follow common sense."

**What about retractors?** Use whatever is available at your institution. In most circumstances, we prefer one of the **handheld retractors** in the hand of the assistant. But, not all assistants are as passive or active as you wish them to be. As Arthur E. Hertzler (1870–1946) wrote: "If I ever deliberately commit murder I shall select an inattentive and awkward assistant as my victim. I shall select one who has assisted enough to delude himself into thinking he could himself do the work better than the surgeon who is operating. This usually reaches the high point at about the third week of the intern's experiences."

In some situations, a "passive" fixed "retractor" should be used—especially when operating in the pelvis or upper abdomen. The good old Balfour retractor is useful when doing a midline laparotomy. Of course, your hospital may have one of those fancy multiarm retractors (called omni or whatever) or the ingenious Bookwalter ring retractor; some surgeons like to use them, particularly those who do not have residents but have to rely on sleepy nurses. We try to avoid those types of mechanical retractors: often, the time needed to place them is longer than the operation, and we hate operating with a sharp metal frame piercing our paunch.

## Additional Points: Grading the Severity of Injury

Abdominal exploration for trauma ends with a strategic decision about the subsequent steps. Forget at this stage the many available organ injury scales, which are of only academic value; from the operating surgeon's point of view, there are essentially two patterns of visceral damage: minor and major trouble.

- **Minor trouble** involves easily fixable injuries, either because the injured organ is accessible or the surgical solution is straightforward (e.g., splenectomy, suture of mesenteric bleeders, or a colon perforation). There is no immediate danger of exsanguination or loss of surgical control. Under these circumstances, you can immediately proceed with definitive repair.
- **Major trouble** is when the spontaneous condition or injury is not easily rectified because of complexity or inaccessibility (e.g., a high-grade liver injury, a major retroperitoneal vascular injury in the supracolic compartment, or destruction of the pancreatoduodenal complex). Here, the secret of success is to **stop** the operation when temporary (usually digital or manual) control of bleeding is achieved. Take time to optimize the surgical attack on the injured organ. Update all members of the operating and anesthesia teams on the operative plan. Allow your anesthesiologist to use the time to stabilize the patient hemodynamically and to obtain more blood products. (Often, you have to think for your anesthetist; do not assume that the anesthetist is awake. However, bear in mind that just as you are a "modern" surgeon, there are now "modern" anesthetists, and they are an invaluable resource in the management of such patients. Take care not to alienate these excellent practitioners.) Order an autotransfusion device and a full range of vascular and thoracotomy instruments to be brought to the surgical suite. This is also the appropriate time to seek more competent help and to plan the operative attack, including additional exposure and mobilization. Such preparations are crucial for the survival of your patient.

**Remember**: very often the initial exploration of the abdomen in the trauma patient is incomplete because the patient's critical condition creates a situation in which every minute counts, and injuries are simply repaired as they are encountered.

Under these circumstances, you must complete the exploration before terminating the procedure.

Finally, **first do no harm**. This applies everywhere in medicine but is of paramount importance during abdominal exploration. The injured or infected contents of the peritoneal cavity may be inflamed, swollen, adherent, friable, and brittle. Careless and sloppy manipulation and separation of viscera during exploration commonly induce additional bleeding and may produce additional bowel defects or enlarge the existing ones. And as usual, new problems translate into additional therapies and morbidity.

This is what makes emergency abdominal surgery so exciting and demanding: the ever-looming catastrophe and the anxiety about whether you are able, or not, to tackle it competently.

12

# Peritonitis: Contamination and Infection—Principles of Treatment

Moshe Schein · Roger Saadia

*In peritonitis—source control is above all.*

*"The mechanical control of the source of infection, while itself nonbiologic, determines the extent of the host biologic response to the disease."* (Ronald V. Maier)

The finding of inflammation, bowel contents, or pus localized or dispersed throughout the peritoneal cavity is common at emergency laparotomy. How is this scenario best handled? This chapter discusses semantic distinctions and *general aspects* of the surgical treatment. For the management of individual causes of peritonitis, refer to the specific chapters.

## Nomenclature

Inflammation of the peritoneum is termed **peritonitis.** It is generally caused by a bacterial inoculum. This explains why peritonitis and **intra-abdominal infection (IAI)** are mistakenly used interchangeably. It is important to note though, that these two terms are not synonymous because peritonitis may also be sterile, as with the chemical peritonitis of early perforation of a peptic ulcer or inadvertent infusion of enteral feeding through a misplaced jejunostomy tube.

- **IAI.** For a condition to be labeled IAI, both the intraperitoneal presence of micro-organisms (or their toxins) and the inflammatory response of the peritoneum are required. At laparotomy, a purulent exudate is often found.
- **Peritoneal contamination is different**. It consists merely of the soiling of the peritoneal cavity by a fluid rich in micro-organisms, as in the immediate aftermath of a penetrating intestinal injury, before an inflammatory response has taken place. Peritoneal contamination occurs commonly in the course of routine elective surgery of the gastrointestinal tract.
- **IAI can be diffuse, as in generalized peritonitis, or localized**, as in intra-abdominal abscesses. Many surgical texts still erroneously use the term *intra-abdominal abscess* as a variant of peritonitis. This is not entirely correct since abscesses develop as a result of effective host defenses and represent a relatively successful outcome of peritonitis. *The mainstay of treatment is drainage*. For how and by which route, find out in Chap. 49.

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We like to refer to IAI as ***resectable*** when not only the source of infection is amenable to surgical removal but also when the remaining locoregional conditions of the peritoneal cavity are such that prolonged postoperative antibiotic therapy is not required (e.g., gangrenous appendicitis). ***Nonresectable*** IAI, in contrast, is an infection that has spread beyond the confines of the source organ; in perforated appendicitis, for instance, you may resect the appendix, but residual peritoneal infection persists, requiring extended antibiotic coverage (› Chap. 47).

**Abdominal sepsis** is still a term used very commonly, but we, semantic *nudniks*, do not like it. According to modern consensus, *sepsis* means the host's response to infection (systemic inflammatory response syndrome [SIRS] plus a source of infection) (› Chap. 54). Thus, the use of "sepsis," in the abdominal context, would not take into account the important initial *local* inflammation within the peritoneal cavity. This peritoneal response is analogous, at a local level, with SIRS at the systemic level because it represents, likewise, a nonspecific inflammatory response of the host to a variety of noxious stimuli, not necessarily infectious. Strictly speaking, therefore, *local contamination, infection, and sepsis refer to different processes*. Yet, they may coexist in the same patient, developing simultaneously or consecutively—a *continuum*. Untreated or neglected abdominal contamination progresses to IAI, which is invariably associated with a systemic inflammatory response. More significantly, abdominal inflammation or indeed the systemic response (fever, leukocytosis) may even persist after the intraperitoneal infection has been eradicated.

## Classification of Peritonitis

**Secondary peritonitis**. It is caused by the perforation or transmural necrosis of a hollow viscus. It is usually characterized by an aerobic and anaerobic polymicrobial inoculum, reflecting the flora of the gastrointestinal tract. This condition's management is the "bread and butter" of the general surgeon. Examples include perforated appendicitis, perforated diverticular disease of the colon, strangulation obstruction of the small bowel, and ruptured tubo-ovarian abscess.

**Primary peritonitis**, in contrast to secondary peritonitis, is not caused by a loss of gastrointestinal wall integrity and is not associated with leakage of intestinal contents into the peritoneal cavity. It is also referred to as *spontaneous bacterial peritonitis*. The responsible micro-organisms originate from a source outside the abdomen. In young girls, it is usually a *Streptococcus* gaining access via the genital tract. In cirrhotics, *Escherichia coli* is thought to be a blood-borne agent infecting the ascites. In patients receiving peritoneal dialysis, *Staphylococcus* migrates from the skin along the dialysis catheter (› Chap. 32). Primary peritonitis in patients without a predisposing factor, such as ascites or a dialysis catheter, is extremely rare. It is usually diagnosed when laparotomy performed for an "acute abdomen" reveals odorless fluid without an apparent source. The diagnosis is reached by exclusion after a thorough abdominal

exploration and is confirmed by a Gram stain and culture, which usually isolates a solitary aerobic organism: it is a "single-bug disease." In patients with a known predisposing factor (e.g., ascites associated with chronic liver disease), suspected primary peritonitis can be diagnosed by paracentesis (polymorphonuclear count in the ascitic fluid greater than 250 cells/mm$^3$); a positive culture confirms the diagnosis, but even with a negative culture, antibiotic treatment should be instituted. Whenever possible, a diagnostic exploratory laparotomy should be avoided because of its prohibitive mortality; in a patient with advanced cirrhosis, it often amounts to an *autopsy in vivo*. Initial antibiotic treatment is empiric until results of bacteriological sensitivities become available.

- **Tertiary peritonitis** is associated with multiple-organ failure and reflects the host's global immunodepression. The microbial inoculum consists mainly of organisms of low pathogenicity, such as *Staphylococcus epidermidis, Enterococcus,* and *Candida albicans* (Chap. 54). Some surgeons include "**postoperative peritonitis**" (e.g., due to a leaking anastomosis) in this category. Others, more correctly, consider this a more morbid variant of secondary peritonitis (Chap. 52).

## Management

The outcome of IAI depends on the virulence of the infection, the patient's premorbid reserves, and the patient's current physiological compromise. Your goal here is to assist the patient's own local and systemic defenses. **The philosophy of management** in a typical case of secondary peritonitis is simple, comprising two steps: source control followed by peritoneal toilet. More aggressive methods are also discussed.

### *Source Control*

The key to success is timely surgical intervention to interrupt the delivery of bacteria and adjuvants of inflammation (bile, blood, fecal matter, barium) into the peritoneal cavity. All other measures are of little use if the operation does not successfully eradicate the infective source and reduce the inoculum to an amount that can be handled effectively by the patient's defenses, supported by antibiotic therapy. This is not controversial—all the rest may be. Source control frequently involves a simple procedure such as appendectomy (Chap. 28) or patch closure of a perforated ulcer (Chap. 18). Occasionally, a major resection to remove the infective focus is indicated, such as gastrectomy for perforated gastric carcinoma (Chap. 18) or a colectomy for perforated diverticulitis (Chap. 26). Generally, the choice of the procedure, and whether the ends of resected bowel are anastomosed or exteriorized (creation of a stoma), depends on the anatomical source of infection, the degree of peritoneal inflammation and SIRS, and the patient's premorbid reserves, as discussed in the individual chapters.

Note that situations exist when the source cannot be eradicated or the expected price to pay for its removal is deemed too high. Less-radical options may then be used, such as *diversion* (e.g., colostomy proximal to a rectal injury) or *drainage* (of a leaking duodenum).

### *Peritoneal Toilet*

Once the source of infection is eradicated, cleaning the peritoneal cavity is aimed at minimizing the intraperitoneal bacterial load. Several maneuvers deserve discussion. Liquid contaminants and infected exudates should be aspirated and particulate matter removed by swabbing or mopping the peritoneal surfaces with moist laparotomy pads. Although cosmetically appealing and popular with surgeons, there is no scientific evidence that *intraoperative peritoneal lavage* reduces mortality or infectious complications in patients receiving adequate systemic antibiotics. Similarly, *peritoneal irrigation with antibiotics* is not advantageous, and the addition of antiseptics may produce local toxic effects. Irrigate copiously (to use a term popular among American surgeons) if you wish, but know that, beyond wetting your own underwear and shoes, you will not accomplish much. Should you choose to remain a dedicated irrigator, try to confine the irrigation to the contaminated area—to avoid spreading s**t all around—and do remember to suck out all the lavage fluid before you close; there is evidence that leaving irrigation fluids behind interferes with peritoneal defenses by "diluting the macrophages." Bacteria swim perhaps better than macrophages.

The concept of *radical debridement of the peritoneal cavity* is based on the premise that fibrin is a nidus for microbial implantation, hence the recommendation to peel off every bit of fibrin coating peritoneal surfaces and viscera. The procedure is tedious, results in excessive bleeding from the denuded peritoneal surfaces, and endangers the integrity of an already friable intestine. It did not withstand the test of a prospective randomized study comparing it to a more conservative approach.

Despite the dictum that it is impossible to effectively drain the free peritoneal cavity, drains are still used (and often misused). Their aim must be restricted to the evacuation of an "established" abscess (when the residual cavity will not collapse or cannot be filled with omentum or adjacent structures), to allowing escape of potential secretions (e.g., bile, pancreatic juice), or, rarely, to establishing a controlled intestinal fistula when exteriorization is not possible. To prevent intestinal erosion, soft drains should be left in place for the shortest duration possible and well away from bowel wall. In general, *active suction* drainage may be more effective than the *passive* kind, and infectious complications can be reduced by choosing "closed" systems. Drains provide a false sense of security and reassurance. We have all seen a moribund postoperative patient with an abdomen "crying" to be re-explored and a surgeon in denial because the


**Fig. 12.1.** "Which of the drains is draining?"

tiny four-quadrant drains are dry and nonproductive. This is particularly true of drains inserted to deal with postoperative hemorrhage (› Chap. 56); a tiny trickle of blood from a drain may hide a huge intra-abdominal clot. Drains inserted close to an anastomosis "just in case it leaks" are more likely to cause an anastomotic dehiscence than to establish a controlled fistula. For more on drains, refer to › Chap. 42.

The role of *postoperative peritoneal lavage* through tube drains left in place for this purpose is at best questionable. Is it really possible to irrigate the whole abdominal cavity? In our experience, such tubes are rapidly walled off by adhesions and adjacent tissues. At the end of the day, you will be irrigating nothing more than the drains' tracks (› Fig. 12.1).

### *Aggressive Modalities of Management*

"In doubtful cases do not wait too long
Before exploring, for it is quite wrong
To act upon the slogan Wait and See,
When looking may provide the remedy" (Zachary Cope, 1881–1974)

Most IAI patients respond to the combination of adequate source control, appropriate antibiotics, and competent supportive management. Might a few others need more? In the 1980s, it was believed that failure of the initial standard operation could be accounted for by either persisting or recurring infection diagnosed too late. Waiting for overt signs of infection or organ failure as the

indication for "on-demand" abdominal re-exploration was thought to be at best questionable. Hence, the emergence of an *aggressive management* approach in the form of planned relaparotomy and open management of the abdomen (laparostomy). These two modalities were often combined.

- **Planned re-laparotomy** pushes the process of source control to its limit. By staging repeated operative interventions to follow the first "index" procedure for peritonitis, the surgeon makes a commitment to return to the abdominal cavity again and again to re-explore, evacuate, wash out, debride, or resect as needed until the disease process is definitively controlled (Chap. 52.1). This dogged pursuit is justified by local intra-abdominal conditions rather than the patient's overall status.
- **Open management (laparostomy)** is, in concept, an extension to peritoneal toilet by providing maximal drainage for the purulent abdomen. It facilitates frequent re-explorations. We now know also that it serves as a prevention of the **abdominal compartment syndrome** (Chaps. 40 and 52).

Early results of these methods seemed promising, particularly in the management of infected pancreatic necrosis but were less favorable in cases of postoperative peritonitis, perhaps because the sickest patients were included. Intestinal fistulas plagued simple open management, but this problem was somewhat minimized by the introduction of modern ***temporary abdominal closure*** (TAC) techniques (Chap. 52.2).

Recent prospective randomized studies, imperfect as they were given the difficulties in patient enrollment and stratification, failed to show an advantage for the planned re-laparotomy policy and pointed to a higher complication rate (in surgery, there is no free lunch). The possibility was raised that relaparotomies constitute a "second hit" in patients in whom the inflammatory response was already "switched on," escalating the SIRS (Chap. 54). It is more likely, though, that sick patients were subjected to a double iatrogenic insult: frequent trips out of the intensive care unit (ICU) and intempestive surgical manipulations. Is there still a place for the "aggressive management"? We believe there is one, in a very small minority of carefully selected patients at the hands of expert teams. Wholesale application or relegating the relaparotomy performance by junior staff to the end of the operating room (OR) slate are recipes for disaster (Chap. 52).

Our indications for these modalities are summarized in Table 12.1.

## Need for Peritoneal Cultures

At age 70, Average Citizen undergoes a laparotomy for a perforated sigmoid diverticular disease with generalized fecal peritonitis. Are you one of those sur-

**Table 12.1.** Indications for laparostomy/planned relaparotomy (Chap. 52)

- Critical patient condition (hemodynamic instability) precluding appropriate source control at the first operation, thus calling for "abbreviated laparotomy" or "damage control" strategy
- Excessive peritoneal (visceral) swelling preventing tension-free abdominal closure (abdominal compartment syndrome; > Chap. 40)
- Massive abdominal wall loss
- Inability to eliminate or to control the source of infection
- Incomplete debridement of necrotic tissue
- Uncertain viability of remaining bowel (> Chap. 23)
- Uncontrolled bleeding (the need for "packing")

geons who would send a specimen of peritoneal fluid for culture? If so, how often do you follow up the results and modify accordingly your antibiotic regimen? What indeed can be gained by culturing Mr. Average Citizen's feces?

This is a typical example of secondary peritonitis that is community acquired (i.e., Mr. Average Citizen perforated his colon at home and then walked in through the emergency room doors). The microbiology of this type of IAI is predictable and responds readily to an empiric broad-spectrum antibiotic regimen, initiated preoperatively, that includes antianaerobic cover (Chap. 7). In any case, culture results would become available well after the completion of the antibiotic course (Chap. 47).

Peritoneal cultures are useful in the following scenarios:

- Primary peritonitis for which there is no intra-abdominal source of infection; the fluid contains an organism that has migrated from somewhere else
- Secondary peritonitis when it is nosocomial (acquired in an already hospitalized patient), the best example being postoperative peritonitis
- Tertiary peritonitis, which is commonly associated with a peculiar microbiology (Chap. 54)
- Peritonitis in the immunocompromised patients (AIDS) and those already on antibiotics

(If you want to read much more about this topic, then find a copy of *Source Control,* edited by M. Schein and J. Marshall, Springer, Berlin, 2002.)

> "Shakiness of the hand may be some bar to the successful performance of an operation, but he of a shaky mind is hopeless." (Sir William MacEwen, 1848–1924)

# 13 The Intestinal Anastomosis

Moshe Schein

*Best is the enemy of good ... the first layer is the best—why spoil it?*

## The Ideal Anastomosis

The ideal intestinal anastomosis is the one that does not leak, for leaks, although relatively rare, represent a dreaded and potentially deadly disaster (› Chap. 50). In addition, the anastomosis should not obstruct, allowing normal function of the gastrointestinal tract within a few days of construction.

Any experienced surgeon thinks that his or her anastomotic technique, adopted from mentors and with a touch of personal virtuosity, is the "best." Many methods are practiced: end to end, end to side, or side to side; single versus double layered, interrupted versus continuous, using absorbable versus nonabsorbable and braided versus monofilament suture materials. We even know some obsessive-compulsive surgeons (do you know any?) who carefully construct a three-layered anastomosis in an interrupted fashion. Now, add staplers to the mix. So, where do we stand; what is preferable (› Fig. 13.1)?

## Pros and Cons

Numerous experimental and clinical studies support the following:

- **Leakage**: the incidence of anastomotic dehiscence is identical—irrespective of the method used—provided the anastomosis is technically sound; constructed with well-perfused bowel without tension; and water—and airtight.
- **Stricture**: the single-layer anastomosis is associated with a lower risk of stricture formation than the multilayered one. Strictures are also more common following end-to-end anastomosis performed with the circular stapler (especially when the smaller sizes are used).
- **Misadventure:** intraoperative technical failures with staplers are more frequent due to "misfires."

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**Fig. 13.1.** "Give it to me, nurse. … This will be a perfect anastomosis!"

- **Speed**: stapled anastomoses, on the average, are slightly faster than those sutured by hand. The fewer the layers, the faster the anastomosis is, and the continuous method is swifter than the interrupted one. In practice, the time consumed in placing two hand-fashioned "purse-string" sutures for a stapled circular anastomosis is identical to that required to complete a hand-sutured, single-layered, continuous anastomosis.
- **Suture material**: braided sutures (e.g., silk or Vicryl) "saw" through tissues and, experimentally at least, are associated with greater inflammation and activation of collagenases than monofilament material (e.g., PDS, Prolene). "Chromic catgut" is too rapidly absorbed to support (alone) an anastomosis. Monofilament slides better through the tissues and, when used in a continuous fashion, is self-adjustable, allowing equal distribution of the tension around the entire circumference of the anastomosis.
- **Cost**: staplers are much more expensive than sutures and thus generally not cost-effective. The single-layer continuous technique requires less suture material and is therefore more economical than the interrupted method.

## The Choice of Anastomotic Technique

Since all methods, if correctly performed, are safe, nobody can fault you for using the anastomotic method with which you are most familiar and

comfortable. We maintain, however, and we may be biased, that the one-layer, continuous method, using a monofilament suture material, is the one that a "modern surgeon" should adopt because it is fast, cheap, and safe. What is good for the high-pressure vascular anastomosis should be as good for the low-pressure intestinal one. If the first layer suffices, why narrow and injure it with inverted and strangulated tissue? Would you replace a well-done hamburger on the grill? As with any beautiful piece of art, less is more.

We acknowledge that staplers are elegant, admired by the nursing operating room staff, "fun" to use, and of great financial benefit to the manufacturers. Certainly, staples may be advantageous in selected "problematic," rectal, or esophageal anastomoses, deep in the pelvis or high under the diaphragm. But, those types of anastomoses are seldom performed in emergency situations. Furthermore, as a surgical trainee you should start using the staplers only after achieving maximal proficiency in manual techniques and in difficult circumstances. Even the stapler aficionado has to use hands when the instrument misfires or cannot be used because of specific anatomic constraints, such as the retroperitoneal duodenum. The modern surgeon, and the trainee, need to be equally proficient in hand-sewn and stapled anastomotic techniques; we suggest, however, that before driving a truck you should be able to manage a car.

### *The Edematous Bowel*

There is some evidence (not level I) that, in trauma patients, stapled intestinal anastomoses are more prone to leak than the hand-sewn ones. This has been attributed to the postresuscitation bowel edema that develops after severe injury. (The staplers cannot "adjust" to the swelling of the bowel; the surgeon's hands can.) It is also our experience that a continuous, monolayer anastomosis occasionally fails when performed in edematous bowel (e.g., after massive fluid resuscitation or severe peritonitis). From findings at reoperation, we have learned that subsequently, as the bowel edema subsides, the suture becomes loose, leading to anastomotic dehiscence. Therefore, when anastomosing swollen, edematous bowel, we prefer not to use staplers or the continuous hand-sutured method. Instead, we use a closely placed single layer of interrupted sutures—individually tied "not too tight, not too loose"—not only to avoid cutting through the bowel edges but also to obviate the risk of loosening after the edema subsides. A similar interrupted technique may be preferred in colo-colo anastomoses where the avoidance of the hemostatic effects of continuous sutures may have theoretical advantages. Furthermore, in this situation the ability of the colon to change dramatically in diameter under normal physiological conditions may be impaired if a continuous suture with its fixed length is utilized. We admit, however, that scientific data to back these hypotheses are lacking.

### *Technique*

Our preferred continuous, monolayered anastomosis uses one double-armed, or two regular, 3–0 or 4–0 monofilament sutures (PDS or Maxon). No bowel clamps are used as we like to assess the adequacy of blood supply to the bowel edges. It is not necessary to devascularize the bowel edges by "cleaning off" the fat at the mesenteric side or removing appendices epiploica. The suture line begins at the posterior/mesenteric wall, running "over and over" toward both sides to meet, and be tied, anteriorly (at the antimesenteric border). The secret is to take generous bites through the submucosa, muscularis, and serosa and avoid the mucosa ("big bites outside, small bites inside"), thus *inverting* it. This suturing technique is known variously as extramucosal or serosubmucosal. The needle exit or entry site on the serosal side is 5–7 mm from the bowel edge (even 1 cm or more when the bowel is thin or edematous—"too big bites can't harm but too small can leak"), while the distance between the bites should be such as not to allow access to the tips of a Debakey forceps (3–4 mm). The assistant who "follows" the suture should use just enough tension to maintain approximation and avoid strangulation of the tissue (a reliable assistant is crucial). In addition to the end-to-end situation, this technique suits also both the end-to-side and side-to-side versions, and in essence, it is the intestinal version of a routine vascular anastomosis except that the vascular one is "*everted*." We use this technique throughout the entire gastrointestinal tract, from the esophagus to the rectum. Essentially, you create an inverted and safe anastomosis, with a wide lumen, using only a suture or two, in less than 15 min.

In "difficult" situations, when the anastomotic site is relatively inaccessible or the bowel edematous, we prefer a one-layer interrupted technique, which allows more accurate placement of sutures and the theoretical advantage mentioned. For this purpose, we use Vicryl sutures, 3–0 or 4–0, which are easier to tie than monofilament. Again, we start with the posterior wall and progress, alternately, on each side to meet at the front. Like with the continuous method, we take big bites on the outside and tiny on the inside, inverting the mucosa. All sutures, except the last few at the front, are tied inside the lumen.

## A Few More Words on Staplers

Correct use of staplers is something you will learn from your mentors. In fact, unlike what you may think, we are not rigidly "staplerophobic." We use staplers generously in emergency situations to *occlude*, rather than *anastomose*; a classical example would be closure of the rectum after a Hartmann's procedure or small bowel transection in an abbreviated laparotomy for trauma or ischemia. Doing a *functional end-to-end* small bowel or ileocolic anastomosis after,

respectively, small bowel resection or right hemicolectomy—using a linear cutting (GIA) and linear occluding (TA) staplers makes sense to us. But, a side-to-side gastrojejunostomy, when you insert the GIA into the stomach and small bowel through two holes, which you then have to close with sutures, makes no sense as the combined size of the gastric and jejunal enterotomies is almost that of the gastrojejunostomy you could have created and sutured by hand. Moreover, these enterotomies, which are used to insert the jaws of the linear cutting staplers and are then closed by hand, seem to be the Achilles' heel of the anastomosis: they, rather the stapler line, are often the site of a leak.

### Testing the Anastomosis

A correctly performed anastomosis should not leak. There is little point in routinely testing your simple intra-abdominal intestinal anastomosis; the common practice of pinching-masturbating the anastomosis to confirm an adequate lumen is laughable if you used a one-layer technique as described. Problematic anastomoses, such as those performed in the lower rectum, should be tested: simply clamp the bowel above the anastomosis, fill the pelvis with saline, and inject air into the rectum. Instead of air, you may wish to use dye. If air bubbles (or dye) are observed leaking, an attempt to identify and correct the defect is indicated; if unsuccessful or doubtful, a proximal diverting stoma is necessary.

### When Not to Perform an Anastomosis

We wish we had an exact answer regarding when to perform an anastomosis. In broad terms, whenever the probability of a leak is high, avoid an anastomosis since any anastomotic leak portends disastrous consequences (Chap. 50). But, how do you accurately predict anastomotic failure?

Traditionally, the avoidance of colonic suture lines during emergency operations for trauma, obstruction, or perforation was the standard practice. But, times are changing. During World War II, a colostomy was mandatory for any colonic injury; now we successfully repair most of these wounds (Chap. 39.2). Furthermore, three- or two-stage procedures for colonic obstruction have been replaced by the one-stage resection with anastomosis (Chap. 25). And, as you will read in Chaps. 25 and 26, the issue of whether the large bowel is "prepared" or not has become a nonissue. Multiple prospective randomized trials have shown that safe colorectal suture lines can be effected in unprepared bowel.

It is difficult to lay down precise guidelines regarding when an intestinal anastomosis is not to be made. You should make a careful decision after considering **the condition of the patient, the intestine, and the peritoneal cavity.**

**Table 13.1.** Factors that may influence us not to anastomose

- Diffuse established peritonitis
- Postoperative peritonitis (Chap. 52)
- Leaking anastomosis (Chap. 50)
- Mesenteric ischemia (Chap. 23)
- Extreme bowel edema/distension
- Extreme malnutrition with low serum albumin (Chap. 46)
- Chronic steroid intake
- Unstable patient (damage control situation) (Chap. 39)

Generally, we would avoid a colonic anastomosis in the presence of established and diffuse intra-abdominal infection (as opposed to contamination) (Chap. 26) and under the conditions listed in Table 13.1. Regarding the *small bowel*, anastomosis is indicated in most instances; however, when more than one of the factors listed in the table are present, we would tend to err on the conservative side and exteriorize or divert, depending on technical circumstances.

No formula or algorithm is available, so use your judgment and try not to be too obsessive in always attempting an anastomosis. Yes, we know that you wish the patient well by wanting to spare the patient a stoma, but he or she will not be impressed if dead. You should not be fearful of creating a high small bowel stoma. Previously, these were considered to be unmanageable, but with total parenteral nutrition, techniques of distal enteric feeding and reinfusion, somatostatin, and stoma care, these temporary proximal intestinal "vents" can be lifesaving (see also Chaps. 46 and 50). On the other hand, do not be a wuss by avoiding an anastomosis when it is indicated and possible.

Whatever you do, some people will be unhappy. If you do a colostomy, there will be always someone to ask you, why not primary anastomosis? If you do a primary anastomosis, there will be always someone to say, why not colostomy? Only being a football coach is worse is this regard.

## Conclusions

The intestinal anastomosis is the "elective" part of the emergency operation you are going to perform. Remember—your aim is to save life and minimize morbidity; create an anastomosis when its chances of success are at least reasonable. There are many ways to skin a cat and to fashion an anastomosis. Master a few methods and use them selectively.

# 14 Intestinal Stomas

Luis Carriquiry

Throughout surgical history, surgeons have viewed the creation of an intestinal stoma with distaste but, at the same time understood its potential lifesaving value—as reflected by these two quotations from master surgeons:

> Of all the diseases to which man is liable, there is no one so inconvenient and disgusting as the artificial anus. How wretched is the patient from whom, despite his will, the alimentary, bilious and fecal matter contained in his intestines are constantly escaping. (Guillaume Dupuytren, 1777–1835)
>
> About colostomy: But it is surely far better to part with one of the conveniences of Life, than to part with Life itself. Beside, the excrements that are voided by this passage, are not altogether so offensive, as those that are voided per anum. (Lorenz Heister, 1683–1758)

Nobody likes an operation that results in an intestinal stoma, neither the patients, who habitually hate the mere idea of them, nor the surgeons, who may look at stomas as a mark of failure. But, the creation of a stoma may be a lifesaving procedure, so they deserve particular attention. A large part of the bad reputation of stomas results from poor stoma creation technique. In contrast, properly created stomas are easier to manage in the postoperative period (sometimes for the rest of the patient's life) and lighter to bear as an incapacity. So, even after a long, difficult, and tiresome emergency operation, you have to pay due attention to technical details in the performance of the stoma and not "pass the buck" to an insufficiently trained assistant.

Intestinal stomas are created when the large or small bowel is exteriorized through the abdominal wall. The only exception to this is the *tube cecostomy*, in

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which communication to the exterior is indirect via a tube (e.g., Pezzer, Malecot, or Foley). The current indications for this are few and may include Ogilvie's syndrome or cecal volvulus (Chap. 25).

Intestinal stomas can be either:

- *Terminal* or "end" stomas—when the surgeon exteriorizes the proximal limb of the transected bowel
- *Lateral* or "loop" stomas—when the surgeon exteriorizes a loop of noninterrupted bowel and makes a hole in its apex

Both types of stomas fully divert the bowel content to the exterior. This is obvious in the case of end stomas but has been debated in the case of loop stomas; now the debate is settled, and everyone accepts that feces are incapable of finding their way to the distal bowel in a correctly fashioned loop stoma.

Turnbull's (Rupert Beach Turnbull Jr., 1913–1981) criteria for optimal stoma construction have withstood the test of time:

- Proper location
- Adequate abdominal wall aperture
- Tension-free intestinal exteriorization
- Adequate blood supply to the exteriorized segment
- Immediate maturation of the mucosa of the stoma

### The Site of the Stoma

In the emergency setting, the surgeon does not have the help of a stoma therapist or the possibility of experimenting with the appliance in different positions before the operation. But, this is not an excuse for not trying to choose the *best site,* which is:

- Lateral, half the way along a line drawn from the umbilicus to the superior iliac spine
- Not very near to the umbilicus, the costal margin, or the iliac spine
- Preferably through the rectus abdominis muscle
- Away from creases in the skin, especially in obese patients
- Away from scars

But, of course, unusual sites of stomas in the bowel (e.g., proximal small bowel, transverse colon) could force us to compromise with an unconventional and nonperfect abdominal wall site.

In addition, it has been said: "Bringing a colostomy out through a laparotomy incision is like putting a toilet in the kitchen."

### The Hole in the Abdominal Wall

The hole in the abdominal wall is made after completing the abdominal part of the operation and before closing the abdomen. A disk of skin (average diameter of 2 cm), including some subcutaneous fat, is resected at the chosen location; through a cruciate incision in the anterior rectus sheath, the fibers of the rectus muscle are split; the posterior fascia (if present) and peritoneum are generously incised, allowing the passage of two fingers. Beware of any bleeding that may result from an injury to the epigastric artery.

### Exteriorization of the Bowel

Tension-free exteriorization of a well-perfused bowel segment is essential. If necessary, you have to return your attention to the abdominal cavity to liberate or mobilize the bowel further to prevent an ischemic and retracted stoma, which otherwise will be a nightmare for the patient, the surgeon, and the stoma therapist.

### Primary Maturation

Primary maturation of the stoma, suturing the bowel wall to the surrounding skin or to the subcuticular layer with absorbable sutures, is performed only *after* closing the abdominal incision, including the skin, and covering it to prevent contamination from the stoma. Leaving the closed exteriorized bowel hanging out and maturing it a day or two later ("delayed maturation") is still practiced by some "antique" surgeons but no longer has a role.

Respect for these principles is fundamental in achieving a satisfactory stoma, whatever its type and location. But, the various types and locations have different indications and require technical refinements, which are addressed next.

### Small Bowel Stomas

The small bowel stomas are almost always **ileostomies**. Proximal small bowel stomas (jejunostomies) are indicated only in desperate cases and most are end stomas after resections for massive mesenteric ischemia (Chap. 23) or for proximal "diversion" for complex leaking small bowel anastomoses (Chap. 50). Although difficult to manage because of their high output, they are preferred to an anastomosis that is doomed to fail or intractable intra-abdominal infection.

**End ileostomies** may be indicated in the following situations:

- After right hemicolectomy or subtotal colectomy, when the surgeon decides not to anastomose the ileum to the colon or rectum (Chaps. 24, 25, and 27)
- After dismantling a leaking ileocolic or ileorectal anastomosis (Chap. 51)

The **end ileostomy** has to be located in the right side, exiting through the rectus abdominis muscle. If you have liberal access to mechanical suturing devices, you can use them to cut the bowel, sealing its open end to prevent contamination; otherwise, occlude the cut end with a clamp. When the operation is "primary" on a virginal abdomen, the ileum is easily exteriorized; however, in the postoperative setting, with an engorged bowel and an edematous, shortened mesentery, exteriorization is more difficult and sometimes requires further resection or construction of an **end-loop ileostomy**, in which the apex of the loop reaches the skin with less tension than the end because of mesenteric retraction (the end of the loop is sutured or stapled, and its side is opened and matured).

There is no need to fix the loop of bowel to the peritoneum or the fascia; simple proper exteriorization and primary maturation are enough to prevent retraction. But, in the case of the end ileostomy, exteriorization should be generous enough (5 cm) to allow eversion of the stoma, thus creating a spout that will be fundamental for future management of the effluent. Eversion is achieved by a few sutures, placed in the antemesenteric border and both sides of the mesentery, which go from the skin (or subcuticula) to the intestinal serosa and then to the cut end of the bowel.

**What do you do with the distal end of divided small bowel (or colon—after right hemicolectomy)?** To facilitate future closure of the stoma (surgeons tend to be chronic optimists) through the actual site of the stoma, the distal end should be situated near the stoma itself. Simply attach the sutured, or stapled, distal end with a few sutures (end to side) to the serosa of the proximal loop at the point where it exits the abdominal wall. Obviously, when the bowel or mesentery are distended, inflamed, and shortened, this is impossible. In such situations, try to bring out the distal end of bowel elsewhere as mucous fistula; otherwise, close it the best you can and drop it back into the peritoneal cavity. Naturally, such patients will need a formal laparotomy to reverse their stomas.

**Loop ileostomies** are seldom done emergently. We have used them quite rarely:

- To divert—"protect" risky distal colonic anastomoses after emergency resections—sort of a compromise measure between primary anastomosis and Hartmann's procedure, having the advantage of an easier and less-risky reconstruction if everything goes well.
- To divert early recognized leaks in ileocolic anastomoses, when there is no advanced peritonitis. This is frequently combined with resuturing of the leak.

The location of the stoma is as mentioned, and the most distal segment of the ileum must be chosen to afford easy exteriorization. We prefer to pass

a vessel loop through the mesentery without damaging the marginal vascular arcades and pull the bowel delicately through the (generous) hole in the abdominal wall. After closing the main abdominal incision, a transverse incision is placed on the antimesenteric surface of exteriorized loop (incise about half of the circumference), and the distal end is sutured flush to the skin; the proximal end is everted (see discussion for end ileostomy) to achieve a 2- to 3-cm spout. Be careful to identify properly what is proximal and distal; I have seen some cases of proximal flush limbs and distal everted ones. We do not use any type of rod to maintain the stoma in place, and we have never seen retraction of a well-mobilized and matured loop ileostomy.

> If you think about a colostomy, you should (probably) do a colostomy. (Leo A. Gordon)

## Colostomies

**End colostomy has a few variants:**

- When only the proximal limb of the bowel is exteriorized and the distal one is closed and left in the abdominal cavity—the Hartmann procedure.
- When both limbs are exteriorized through the same hole: "double-barrel colostomy" (the distal end could be closed and attached to the serosa of the emerging proximal end).
- When both limbs are exteriorized through separate abdominal wall holes, the proximal one as an end colostomy, the distal one as a "mucous fistula."

All these procedures usually follow a transverse or a left-sided colectomy when an anastomosis is not performed. Bringing out both limbs of the remaining colon through one hole in the abdominal wall (as is usually feasible after segmental resections of the splenic flexure, descending or sigmoid colon for obstruction, perforation, or sigmoid volvulus) allows easier reconstruction with no need to reopen the general abdominal cavity. When the two limbs cannot be brought together in one abdominal wall opening, it is necessary to bring out the two loops through separate holes. Hartmann's procedure (end left colostomy + closure of the rectal stump) should be the last choice but sadly is still frequently used because of the high incidence of low tumors and diverticular complications affecting the sigmoid, in which resection includes the rectosigmoid junction with no possibility of bringing out the remaining rectum as a mucous fistula. And, I say the "last choice" because "take down" of colostomy after the Hartmann procedure is a major operation that cannot always be accomplished, often more due to adhesions than to difficulties with the new anastomosis.

**Some technical advice:**

- Make a bigger hole than for ileostomy (a future paracolostomy hernia is better than a colostomy that is too tight).
- Always be sure you are exteriorizing a well-perfused colon (watch for arterial bleeding from the divided end of the bowel and the marginal artery); necrosis of the colostomy is a serious complication even if it does not reach the peritoneal cavity because it will lead to stenosis.
- If necessary, free more proximal colon: free the bowel from its lateral peritoneal attachments; when more length of left colon is needed, do not hesitate to divide the inferior mesenteric artery or vein.
- If you do a divided colostomy, closing the end of the distal bowel, make sure—again and again—that the end colostomy is created in the proximal loop and not the distal one. (It can happen to anyone.)
- There is no need for any fixation of the bowel loop to the peritoneum or the fascia.
- Some surgeons close—but many believe this is unnecessary—with a few sutures, the lateral space between the emerging loop and the abdominal wall to prevent internal hernia leading to postoperative small bowel obstruction (Chap. 48).
- Primary maturation of the stoma, after abdominal closure, should be done flush to the skin, with no need for eversion.
- In the case of double-barrel colostomy, suture the exteriorized limbs where they are in contact with one another as well as suture the remaining edges to the skin.

## Loop Colostomies

Loop colostomies are used to divert the fecal stream after anorectal trauma or necrotizing infection of the perineum (Chaps. 29 and 39.2). The most common locations are the transverse colon and the sigmoid, but they can be done in any segment of the large bowel that can be exteriorized without tension (e.g., cecum). A loop of colon is pulled outward through the abdominal wall hole using the same technique described for loop ileostomies, but here we prefer using a rod to prevent retraction and ensure proper diversion of feces. Many types of rods have been described. We prefer a segment of latex tubing longitudinally cut and everted (with the aid of sutures) at both ends, a maneuver that prevents sliding of the rod and allows proper placement of the colostomy bag. Primary maturation should be the rule; the practice of delayed opening of the colostomy has no current role. A longitudinal incision is done on the surface of the loop, and absorbable sutures are passed between the edges of the colostomy and the skin or its subcuticular layer.

## Postoperative Care

Before leaving the operating room, the surgeon should supervise proper placement of the stoma appliance. The bag should be transparent to allow

inspection of the stoma in the first postoperative days and properly fitted to the base to allow distension with gases—a finding that makes the surgeon happy.

**Early postoperative complications** of stomas are not rare and have the potential of wrecking the result of the operation.

**Ileostomies**: with necrosis and retraction being rare, the main complication is the high output of the stoma, which could lead to dehydration and electrolyte imbalance, more frequently in older patients. Careful measurement of the output is important, especially when the ileostomy has been done after intestinal obstruction or is situated in the proximal ileum. If high output develops, intravenous rehydration, dietary adjustments, and even medications to slow intestinal transit may be indicated.

**Colostomies**: here, high output is never a problem, but the surgeon should be aware of the risk of **early necrosis**. Starting on the first operative day, inspection of the bowel through the transparent bag, or directly, is a must. If a dark or black mucosa is seen, it is important to evaluate, under good light, or even an anoscope, the depth of the ischemia. That is, how far does the "blackness" extend down the lumen of the stoma? If it is superficial (involving only the "end of the stoma"), one does not have to worry too much, although it may lead to some retraction and subsequent stenosis. But, if it is "deep" and goes beyond the level of the fascia, intervention is mandatory and implies a re-laparotomy and better mobilization of the exteriorized colon to achieve a new, well-perfused colostomy. Trying simply to pull the colon outward through the hole can be dangerous.

Another infrequent early complication is the paracolostomy abscess (one wonders why such a complication is so rare), which should be drained through the mucocutaneous suture line, leaving the new hole within the colostomy bag. Some of these infections may be resistant to local care, occasionally requiring change of the site of the colostomy.

The patient should be educated about stoma function and its management with the invaluable aid of the stoma therapist.

This book is about *emergency* treatment, so I will not bore you with late complications or the "take down" of the stoma. But here are warnings by some wise men:

> Failure of a colostomy closure is more due to the youth of the colostomy than the youth of the surgeon. (Ivor Lewis, 1895–1982)
>
> There is no law that says that a colostomy must be closed. (Leo A. Gordon)

# 15 Esophageal Emergencies

Thomas Anthony Horan

*"If thou examinest a man having a gaping wound piercing through to his gullet; if he drinks water he chokes (and) it come out of the mouth of his wound; it is greatly inflamed, so that he develops fever from it; thou shouldst draw together that wound with stitching. Thou shouldst bind it with fresh meat the first day. Thou shouldst treat it afterward with grease, honey, (and) lint every day, until he recovers. If, however, thou findst him continuing to have fever from that wound thou shouldst apply for him dry lint in the mouth of his wound, (and) moor (him) at his mooring stakes until he recovers."* (From the Edwin Smith papyrus, written in Egypt roughly 3,000 years ago)

The esophagus gives no pleasure, but senses all forms of pain. Its job is simple: relax and let the bolus in, push it with gravity assistance, relax again to let it into the stomach. Despite this simplicity, it is prone to obstructive problems. As there is no serosa, the submucosa is its one layer of strength; thus, it is relatively weak. The stomach can generate two to three times the force necessary to rupture it during vomiting. Almost all neoplastic or inflammatory lesions of the esophagus rapidly affect this one layer of strength, as may vigorous endoscopic manipulation. When something disrupts its integrity, the esophagus delivers a cocktail of mouth anaerobes directly into the mediastinum, which is one of the body's least-resistant areas.

As a general surgeon, the two esophageal emergencies you are most likely to be called to solve are **obstruction and perforation**.

## Foreign Body Ingestion: Obstruction

The night nursing matron ate her tuna sandwich as always at 2 a.m. but felt something sharp in her throat. The X-rays in the morning were normal. Discomfort persisted for the next 3 weeks; barium swallow suggested cervical esophageal cancer. On September 10, I slipped out her dentures and passed the flexible scope for biopsy. The bread bag clip, deeply embedded in the esophageal wall, read "best before August 13." How true.

Swallowed foreign bodies (FBs) are the most frequent cause of acute dysphagia. Food with bones and other embedded sharp objects take the unaware—children,

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patients with dentures, the intoxicated—and their doctors by surprise. Children will swallow just about anything that fits in the mouth, coins and safety pins predominating. Psychiatric patients swallow the most interesting things. Most of the rest of FB-related esophageal obstruction is superimposed on underlying esophageal diseases such as motility disorder, hiatus hernia, stricture, diverticula, and cancer. Even the notorious *steak house syndrome* is more common in patients with underlying esophageal pathology. Therefore, after the FB is evacuated, all patients deserve evaluation of their esophagus. Delay in treatment vies with ill-conceived efforts at retrieval as the cause for most perforations from FBs.

### *How to Manage Esophageal Foreign Bodies?*

The average transit time from cricopharyngeus to stomach is between 3 and 5 s. Thus, if the FB is still in the esophagus when the patient gets to the hospital, it is by definition **stuck**. A stuck FB may cause the patient to gag, cough, drool, hurt, bleed, and aspirate while it tries to move down, up, or through the esophagus. So, you have to assist it out through an anatomical passage before it describes a nonanatomical one by itself.

- **First, find it.** Frequently, the patient knows right where it is, what it is, and why and how it got there. So ask the patient. Insistence on anteroposterior and lateral neck, chest, and abdomen X-rays seems a bit old fashioned, but these are cheap and efficient and may quickly define the problem. If they do not, there is always contrast or computed tomography (CT) to help. Sometimes a wisp of contrast-soaked cotton baton will hang up on it. The X-rays help you plan, choose your equipment, and warn you about possible risks of perforation. Because of false-negative radiology and associated conditions, *every symptomatic patient should get endoscopy*.
- **Methods of FB removal** are based on visualization for safe removal (see near disaster). No matter how sophisticated your snares and flexible equipment, you must know how to use the good-old rigid scope for the removal of sharp objects. Blind methods or pushing impacted boluses into the stomach are risky and can lead to iatrogenic perforation. Judging when the risk of endoscopic removal outweighs risk of open surgery is aided by a history of bleeding, pus, and buried sharp objects next to the aortic indentation. A large number of exculpatory reports testify to the role for operative removal in selected high-risk cases. We note, for example, a recent report of poor planning, which led to pneumomediastinum and bilateral pneumothorax while attempting to remove a knitting needle incarcerated in a hiatus hernia.
- There is controversy about asymptomatic patients with *smooth small* FBs, like coins, since left alone most pass into the stomach and then go down the whole distance without further trouble. Some caution needs to be exercised, but if you are sure the FB is not chemically active (button batteries and zinc-containing coins), how long should you wait? Many suggest "up to 3 days," but this sounds suspiciously

like it is based on the length of the weekend. In the absence of a fail-safe rule, any symptom or failure to reach the stomach by the time of the X-ray in the morning should urge you do endoscopic removal.

***Summary***

- Investigate all symptomatic patients
- Remove all FBs *not* in the stomach within 24 hrs
- Get help from someone who can use a rigid scope
- Consider surgery in selected cases

## Esophageal Perforation

The admiral had eaten a heavy meal. During the next few hours, he had taken small cups of a mild emetic, as was usual when he was feeling heavy. Four times he had about 28 g of olive oil and later drank about 180 g of beer. When this did not have the desired effect, he took another four cups. He tried to throw up but suddenly screamed because of an excruciating pain in the chest. He immediately declared himself dying and started praying. It was a very sick patient, though free of fever, who met Boerhaave. The house physician, Dr. de Bye, had tried bleeding. There were no symptoms of any known disease or poisoning, and the two physicians ordered another bleeding, something nonalcoholic to drink and warm compresses. But, in vain, the baron succumbed the next day. Herman Boerhaave conducted an autopsy that revealed the rent in the esophagus and the contents of a previous meal, gas, and fluid in the chest.

Esophageal perforations continue to increase in number, with the "traditional" FB and vomiting etiologies remaining constant but instrumental or iatrogenic perforations having a fourfold increase. These are likely to increase even further with the current enthusiasm for laparoscopic Nissen fundoplication and the plethora of new gimmicks for the endoluminal treatment of gastroesophageal reflux. Prior to World War II, the mortality of esophageal perforations was horrendous. Today, the emphasis on early diagnosis and treatment, and perhaps effective antibiotic treatment of the associated mediastinitis, have greatly improved results. The improvement in survival seems correlated with the large number of endoscopic instrumental perforations, which make early diagnosis correspondingly easier.

In general, there are four major groups of esophageal perforations, each with differing therapy.

### *The Kind You Think You Can Get Away With*

The catheter slipped beyond the impacted 4-cm beach rock. The 30-ml balloon got a good hold. Increasing withdrawal tension released with a "thwack." Nothing. Reinspection revealed blood and esophageal muscle but no rock. Gastrografin swallow suggested a contained rupture and a rock packed in the posterior nose. Nasal disimpaction, NPO (nothing by mouth [nil per os]), nasogastric suction, antibiotics, parenteral nutrition, and discharge to the patient's mental hospital followed over the next 2 weeks.

The nonoperative treatment of a small, contained, intramuscular, incomplete tear of the esophagus is employed when it is recognized right away and only if there is no adverse systemic response, tachycardia, fever, or pain. The trick is being sure that there is no residual material retained outside the esophageal wall and no dependent pockets (by which I mean what goes in comes right back out). The mucosal defect closes on the follow-up esophagram. If anything is not right, treat like all ruptures (see below). Optimistic overdiagnosis of this entity leads to delay and possible disaster.

### *The Kind a Surgeon Likes to Treat (If Someone Else Caused It)*

This is the classic early-diagnosed perforation treated in the first 24 hrs. But, it is surprising how frequently patients get sent home despite the obvious—"*Worst case of esophagitis I ever saw*" (Fig. 15.1). Reticence to accept the obvious costs lives and reputations. Pain after endoscopy means a high risk of perforation,

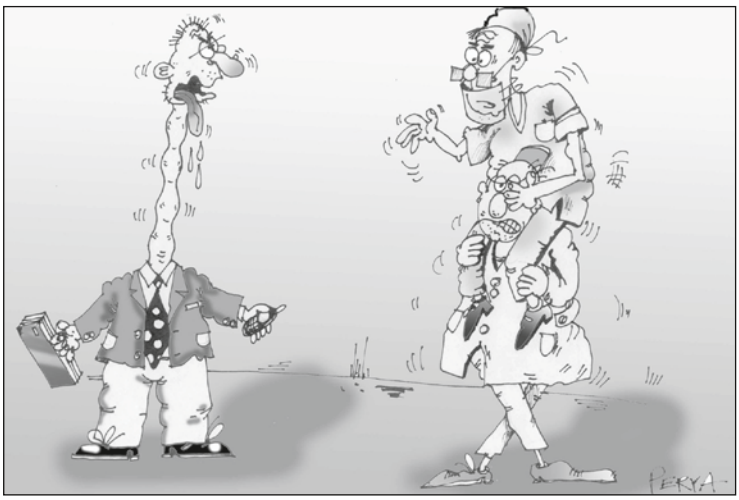

**Fig. 15.1.** "Gee, this is severe esophagitis!"

subcutaneous emphysema indicates the same, and fever means mediastinitis. History almost always gives the cause and diagnosis and frequently the level of injury. Positive physical findings of emphysema, pericardial crunch, pneumothorax, or hydrothorax used to be indicators of late diagnosis. Now, with the air pumped in by endoscopes, they may be the first noted abnormality. The level of emphysema on X-ray corresponds to the level of injury: low perforations typically give left hydropneumothorax, midesophageal perforations are suspected in right hydrothorax. Diagnosis is confirmed with contrast studies, with or without CT. Do not waste time; treat with NPO, resuscitation, antibiotics, and surgery.

- **Perforations in the neck** and upper mediastinum are repaired and drained through the neck. Use the anterior border of the sternomastoid approach because it is so easy to extend and lends itself to the rotation of buttressing strap muscles. See the whole defect and close the mucosa/submucosa. Close the muscle over it, buttress with local muscles, and use a big floppy soft drain. Make a two-finger hole for the drain. If they leak, they get better anyway as long as there is good drainage.
- **For perforations in the chest**, follow exactly the same principles. Go to the left side for low leaks or right side for higher leaks and get wide mediastinal exposure for good visualization and drainage. See and close the whole mucosal defect. Then, close the esophageal muscle over it. Cover all with good tissue, like pleural flaps or intercostal muscle flap wrapped around the esophagus and sewn on the esophageal closure, not just laid on it. Every book written in the last 40 years shows this technique; use it even if you do not think it is necessary because you only get one good chance. Finish with big dependent chest drains, provisions for nutrition, and antibiotics.
- **Spontaneous perforation** results from vomiting against a conscious attempt to prevent it (Boerhaave syndrome). The pressure generated in the esophagus blows out the weakest point, usually located just above the lower esophageal sphincter. It is treated just as all the other perforations, with surgery at the earliest opportunity. However, the risk of spontaneous rupture is higher in patients with an already sick esophagus due to hiatus hernia, congenital or acquired connective tissue disorders, use of steroids, malnutrition, and old age. Because of a particularly high mortality and morbidity, these special cases need to be considered in the same category as late-diagnosed perforations (see below).
- **Associated lesions**. Closing a perforation above an obstructing lesion does not work. In *achalasia*, epiphrenic diverticula, and diffuse esophageal spasm, **do the myotomy** on the opposite side of the esophagus from the perforation. Make the myotomy from the gastroesophageal junction to 8 cm above the level of the perforation. Then, close the perforation just like above. Strictures distal to a perforation have to be dealt with one way or another. Perforation at or just above a stricture associated with gastroesophageal reflux may pose a special problem with full-thickness fibrosis and shortening of the esophagus. The antireflux wrap will greatly assist the closure in these cases provided that the fundus is able to be sutured to the perforation. Do not simply wrap like usual but be sure to secure in place over the perforation. You

may need to add a *Collis gastroplasty* to lengthen the esophagus or to close the perforation using the gastric fundus as a serosal patch, combined with a floppy, nonconstricting antireflux procedure. This is the one time I do not insist on the wrap being in the abdomen; it is most important not to put any tension on the repair of the perforation. If it does not easily go back into the abdomen, then leave it in the chest.

- **Isolated external penetrating and blunt injuries** to the esophagus are rare. Thoracic gunshot injury will hit the esophagus about 1 in 20 cases. Associated injuries to heart, lung, blood vessels, spine, and airway always take precedence. Signs of esophageal injury such as hemothorax or mediastinal emphysema are similarly attributable to concomitant injuries. Therefore, always examine the esophagus prior to closure after all the other more dramatic injuries to lung, heart, and blood vessels have been controlled. Complete exploration of the hematoma and tracts of penetrating objects usually prevents missing the esophageal defect. There is nothing worse than operating for trauma and 2 days later finding the patient moribund from mediastinitis from overlooked esophageal perforation. *Blunt disruption* of the esophagus implies violent deceleration, difficult diagnosis, and a terrible prognosis. However, blunt trauma may occasionally cause rupture of the esophagus from intrusion of osteophytes at a lower degree of violence.

### *The Kind Nobody Wants*

The kind nobody wants is the killer. The patient presents late, is septic, and has mediastinitis and empyema, whether from spontaneous vomiting-induced rupture, FB, neglect, or missed iatrogenic perforation—it doesn't matter; the patient is in big trouble, and you know it. Perforations occurring in cancer, manipulation of extensive caustic strictures, third-degree caustic burns, congenital connective tissue disorders, and congenital epidermolysis bullosa are all in the same category. Rapid concerted effort from a dedicated group is critical. You have to stabilize and then operate. You have to control the source of the infection - often this requires an esophagectomy—and provide wide drainage. You can restore continuity at a time of your choice; after all, you can always reoperate on an alive patient.

The toughest decision to make in perforation of the esophagus is what to do when the diagnosis has been *delayed* in a previously well esophagus. The inclination is to preserve the esophagus if possible. The mediastinal tissues and the state of the esophagus will tell you. If the patient is lucky, the perforation has passed directly into the pleural cavity, and the mediastinum and esophagus are relatively intact. The basis of surgical treatment is surgical closure as described above, but you must debride any necrotic tissue prior to any attempt at closure. Even so, the sutures you place will be at a high risk of leakage. Thus, along with wide mediastinal drainage, large-bore chest tubes, antibiotics, and nutrition you

must consider proximal diversion and gastrostomy to protect the closure, especially if the mucosa is edematous, stiff, and friable.

When the perforation is mostly contained within the mediastinum, severe mediastinitis is invariable. None of the choices is good but do not give up; remember, the patient survived with no treatment until now. With good treatment, the patient can still pull through. The inflamed esophagus will not hold sutures, so it is useless to think of primary closure. Wide drainage alone will lead to a long, debilitating, and perilous course; therefore, isolation-exclusion of the esophagus by cervical diversion proximally and gastrostomy distally is a safer option. Emergency resection is my preferred option, especially when the esophagus is partially necrotic, and wide debridement will make stricture a near certainty. The benefits of this approach are rapid effective control of the mediastinum contamination and avoidance of complications such as vascular or tracheal fistula.

Perforations from dilatation of strictures secondary to accidental caustic ingestion outnumber acute perforations from liquefaction of the esophagus secondary to suicide attempts. Both need emergency resection. I have never understood the reticence in resection of severe caustic injuries. Should patients survive without resection, they are condemned to a life of stricture dilatation, with one in five suffering instrumental perforations. Anyway, if not resected at the acute stage many of these patients will come to resection and replacement at a later date because of difficulties with nutrition and the high risk of malignancy developing in the burned esophagus—perhaps as high as 1,000 times the general risk.

### *Perforations That Cannot Be Fixed*

"Doctor, the lymphoma patient you "mediastinoscoped" yesterday wants to go home."

"Sure, but how is he?"

"Just fine, but he feels a little cold coming on and would feel better at home. Oh! By the way, his neck is kind of sore and crinkly. Do you think he needs a prescription for antibiotics before he goes?"

Inaudible response.

Never underestimate the risk in this type of patient. They need a little operation for diagnostic biopsy or palliative therapy, but their disease cannot be cured surgically. Do not give up too easily. Thoracostomy, drainage, antibiotics, nutrition, and proximal diversion and gastrostomy may salvage the inoperable or unfixable patient. This is worth the effort, especially if there is other effective ancillary therapy for the underlying disease, like for my patient involved in the lymphoma disaster.

Palliative attempts to dilate, stent, or laser ablate inoperable or nonresectable carcinomas should have been discussed with the patient and family prior to the procedure. The instrumental perforation rate is in the region of 10%, and it will not be long before the surgeon is confronted with this problem. If you have already dilated it and accessed the distal esophagus, put in a **stent** and count yourself lucky. The patient still has a chance for palliation if the antibiotics, nasogastric suction, and nothing-by-mouth regimen is successful. Otherwise, morphine may be your only choice.

***Summary***

- Always suspect perforation, especially following instrumentation
- Investigate and manage aggressively
- Be aware of esophageal comorbidity
- Preserve the normal esophagus
- Close and patch perforations
- Drain widely
- Resect if the gullet is very diseased (cancer, long strictures, burns)

> When it [occurs] it can be recognized but it cannot be remedied by the medical profession. (Herman Boerhaave, 1668–1738)

## Invited Commentary

Philip T. Peverada

As Dr. Horan so ably discusses, the two most common surgical esophageal emergencies are foreign body (FB) obstruction and perforation. The former outnumbers the latter by a significant amount.

**Foreign body obstruction.** In the modern hospital, the obstructed patient frequently presents first to the medical gastroenterologist, who will pursue the obstructing agent with the flexible gastroscope. The surgeon will be called when that attempt is unsuccessful in clearing the obstruction or when it has cleared the obstruction but caused the graver problem of esophageal perforation. In the case of the retained FB, facility with the rigid Jackson esophagoscope is required. The esophagus is by now raw, inflamed, and prone to perforation. It is folly to attempt removal with the rigid scope in this situation if one's skills are rudimentary or long unused. If transfer to another facility with skilled endoscopy is not an option,

thoracotomy and esophagotomy are preferable and less morbid than a perforation with subsequent need for drainage and repair. If a trip to the operating room is deemed necessary, it is always prudent to repeat the endoscopy just after induction as general anesthesia and relaxation can result in FB migration and militate the need for thoracotomy.

**Esophageal perforation and postoperative anastomotic leaks.** Although many new minimally invasive and endoscopic techniques are now available for managing esophageal pathology, the disaster of esophageal perforation requires adherence to basic surgical principles and judicious use of the newer technologies. Since many perforations are iatrogenic, the tendency of the clinician is to push for the less-invasive approach, perhaps as a way of lessening the self-guilt surrounding the complication. The key is prompt recognition and treatment. No matter how stable and comfortable the patient may seem, oral flora in the mediastinum are not tolerated for long, and untreated the problem is uniformly fatal. Whatever approach is used in treating an esophageal perforation, one must adhere to the time-honored principles of dealing with this problem:

- Elimination of the septic process
- Provision of adequate drainage
- Augmentation of host defenses by antibiotics
- Maintenance of adequate nutrition

How this is accomplished is less important than that it is accomplished expeditiously.

Dr. Horan discussed the criteria for nonoperative management, namely, a contained perforation, "self-drainage" back into the esophagus, and minimal signs of sepsis. This selective approach can be used with up to 80% success in avoiding surgery.

### Endoscopic Treatment

Although thoracotomy with debridement, drainage, and repair remains the gold standard for the perforated esophagus, the introduction of video-assisted thoracic surgery (VATS) and endoscopic approaches has provided alternative methods of achieving the same goal. Initially, there were case reports of success in managing selected patients without a thoracotomy. With time, larger experience is beginning to emerge reporting VATS and stenting with varying degrees of success. However, I believe that meanwhile the "gold standard" remains thoracotomy.

Drainage and debridement are easily accomplished via the thoracoscope, but the closure of the defect with a secure buttress is problematic. It appears that small defects with minimal contamination can be primarily repaired with a degree of success. Larger defects have been repaired over a T tube with reported

good results. Endoscopic stenting of the disruption combined with VATS debridement and drainage have also been described. Sealing in around 70% of patients with ultimate stent extraction in two-thirds of the long-term survivors has been reported (Tuebergen et al. 2008). However, others reported high reoperation rates and leak rates in the perforation groups (Pennathur et al. 2008; Zisis et al. 2008;), so the absolute utility of these techniques is not currently established. Further technological improvements may result in fewer leaks and less stent migration.

In approaching esophageal perforation, attention to long-established surgical principles is required. As techniques evolve, a VATS/endoscopic approach will probably become the norm. This will be delivered best in specialty centers with expertise in VATS procedures and surgical endoscopy. The question then becomes how to best serve the patient.

I would concur with Dr. Horan that time matters in these patients, and that operative intervention should not be delayed in the hopes of avoiding a thoracotomy. Perform the operation needed using the techniques available and best fitting the surgeon's training and experience. In the end, it is the outcome and not the size of the scar that is the indicator of success.

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## Editorial Comment

Some authorities believe that the time factor is unimportant. This is not necessarily to disagree with Dr. Horan since he observed that the important issue is to tailor the procedure to the state of the gullet at the time of surgery. The time interval may obviously have some indirect bearing on this. We wish to quote another expert in this field: Dr. David J. Richardson of Louisville as published in the book *Source Control* (2002).

— I really do not think that the time of perforation should enter into treatment decisions to any significant extent. Generally, we try to treat the patients

in a similar fashion regardless of the time that they are seen after esophageal perforation.

- If the esophagus is presumably normal prior to the insult (such as with Boerhaave's syndrome), preservation of the esophagus should be the primary goal with attempted closure of the perforation.
- If the underlying disease is cancer and an iatrogenic perforation has occurred during its diagnosis or treatment, then the patient is best treated by esophagectomy and immediate reconstruction in my experience.
- I believe nonoperative treatment must be reserved for relatively few patients and should only be done in a very narrow set of circumstances. If contrast goes beyond the wall of the esophagus, it is my opinion that the patient should have operation and treatment of that problem—even if the contrast drains back into the esophagus from a cavity. Granted, some patients may be able to heal such a perforation nonoperatively, but I am aware of several disasters with patient deaths where this strategy was tried and subsequently failed. I have also observed significant scarring at the area of the healed perforation. Therefore, I reserve nonoperative treatment only for "micro-perforations" in which there may be some small tear observed in the muscle itself, but the contrast does not actually escape the confines of the esophagus.
- I believe that an attempt should be made to close every perforation.
- In my opinion, esophageal diversion should be reserved only for those patients who are almost certainly going to die unless diversion is done.

## Reference

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# Diaphragmatic Emergencies

16

Ulrich Schoeffel · Moshe Schein

The only diaphragmatic pathology of interest to the emergency abdominal surgeon is the diaphragmatic hernia through which one or more abdominal structures may migrate into the thorax and become incarcerated or strangulated. This may occur in different settings, each of which however shares many clinical features.

## Diaphragmatic Hernia

### *Congenital Diaphragmatic Hernia*

Congenital diaphragmatic hernias are very rare entities that include the *Bochdalek* hernia (posterolateral defect in the trigonum lumbocostale) or *Morgagni* hernia (just posterior to the xyphoid at the foramen Morgagni or the trigonum sternocostale), a defect of the tendinous center of the left diaphragm, or the paraesophageal hernia. Left-sided defects more commonly become symptomatic because the liver seals those of the right side. Most congenital hernias are symptomatic and require operative treatment during the first hours of life due to compression of the lungs and mediastinal structures. This can easily be accomplished from an abdominal incision by manual reposition (there are no adhesions between thoracic structures and abdominal viscera in these cases) and direct closure of the defect. If several organs are transposed intrathoracically, the reposition should follow the order stomach first, small bowel next, then large bowel, and finally parenchymal organs such as the spleen.

If there is no overt herniation during the fetal development, the newborn may appear normal but may develop herniation anytime later in life. It then would present like any acquired diaphragmatic hernia, with the final diagnosis established at operation when the exact localization of the defect is revealed.

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### *Rupture of the Diaphragm Due to Blunt or Penetrating Trauma*

When the diaphragm is ruptured due to blunt or penetrating trauma, the hernia may become symptomatic acutely, immediately after the injury, or may present many years later—in a patient who has almost forgotten the trivial car accident 14 years ago. Read about the diagnosis and treatment of acute traumatic hernia in Chaps. 38 and 39. Late complications are diagnosed and managed along the lines described next for nontraumatic diaphragmatic hernia.

### *Acquired Diaphragmatic Hernia*

In clinical practice, only two entities have to be considered in the context of acquired diaphragmatic hernia: the herniation through a traumatic or congenital defect of the tendinous part of the left diaphragm and paraesophageal hernia.

- **Left diaphragmatic hernia**. It has been stated commonly that older posttraumatic hernia should be approached via a thoracotomy and nontraumatic hernia by laparotomy. Indeed, the presence of a peritoneal envelope in a nontraumatic hernia generally simplifies repositioning by gentle traction from below, whereas perforation or rupture of the diaphragm often includes a tear in the peritoneal lining, thus leading to dense adhesions between the thoracic and herniated abdominal structures. However, in the individual case, the etiology is often not clear, and the presence or absence of a peritoneal hernia sac is difficult to predict preoperatively. Therefore, the operative strategy is influenced by probability and personal experience. It has to be stressed, however, that a posterolateral thoracotomy in the seventh intracostal space always permits careful dissection of herniated organs and exploration of the subdiaphragmatic space, whereas an abdominal approach may prove more difficult and hazardous. If, irrespective of the approach, a small hernia ring has to be enlarged, the radial ramifications of the phrenic nerve have to be respected.
- **Paraesophageal hernia**. Here, the gastroesophageal junction lies inside the abdomen, anchored by the phrenoesophageal membrane (nonsliding hernia) and the herniation—most commonly of the stomach—develops through the enlarged esophageal hiatus and a defect in the phrenoesophageal membrane alongside the esophagus. The fundus of the stomach may roll up and down intermittently, producing no or only "subacute" symptoms but occasionally a larger portion or even the whole of the stomach may herniate into the chest, producing the so-called intrathoracic gastric volvulus (the "upside-down stomach," or giant type II hiatal hernia). Common complications include gastric strangulation with infarction, necrosis, and perforation, mucosal bleeding, or acute intrathoracic dilatation causing compression of other intrathoracic structures.

## Gastric Volvulus

Gastric volvulus is defined as an abnormal rotation of the stomach of at least 180°, creating a closed-loop obstruction. According to the axis around which the stomach rotates, volvulus of the stomach may be **organoaxial, mesenteroaxial**, or a combination of both.

In organoaxial volvulus, which is the more common variant, the stomach rotates around an axis that connects the gastroesophageal junction and the pylorus. In this situation, the stomach flips up into the chest with the greater curvature—dragging the omentum with it—coming to lie at the top; this kinks the esophagogastric junction as well as the distal stomach, producing a closed-loop gastric obstruction. The less-common variant, mesenteroaxial volvulus, occurs around the axis that runs from the center of the greater curvature of the stomach to the gastric angulus. Gastric volvulus can occur at any age and with equal frequency in both men and women and has been reported in neonates and infants.

### *Clinical Features*

Acute gastric volvulus may develop against a background of intermittent nonspecific dyspepsia attributed to the known presence of a paraesophageal hernia, but usually it presents acutely "out of the blue." Precipitating events may be a heavy meal or any event that increases intra-abdominal pressure, such as postoperative ileus, pregnancy, or parturition.

The abdomen is relatively innocent, with little epigastric pain and no abdominal findings on examination. There is more pain substernally or in the chest, and the compression of the left lung by the herniated stomach (or other viscera) may result in acute respiratory distress. A shift of mediastinal structures to the right may result in cardiovascular instability, while kinking of the gastroesophageal junction may produce retching. The diagnostic triad described by Moritz **Borchardt** (1868–1948) includes epigastric/substernal pain, retching without vomiting, and the inability to pass a nasogastric tube.

Traditionally, acute gastric volvulus was diagnosed on a chest X-ray showing a retrocardiac air bubble or a large fluid level in the chest (Fig. 16.1). An oral contrast study, showing obstruction of the stomach at the site of the volvulus, would have then confirmed the diagnosis. Currently, however, a computed tomographic (CT) scan can offer an immediate diagnosis with all the anatomical details (see Figs. 16.2 and 16.3).

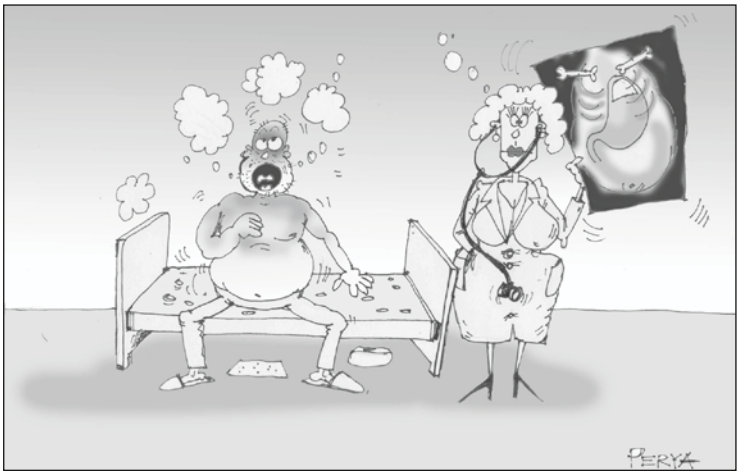

**Fig. 16.1.** "What is your stomach doing in the chest?"

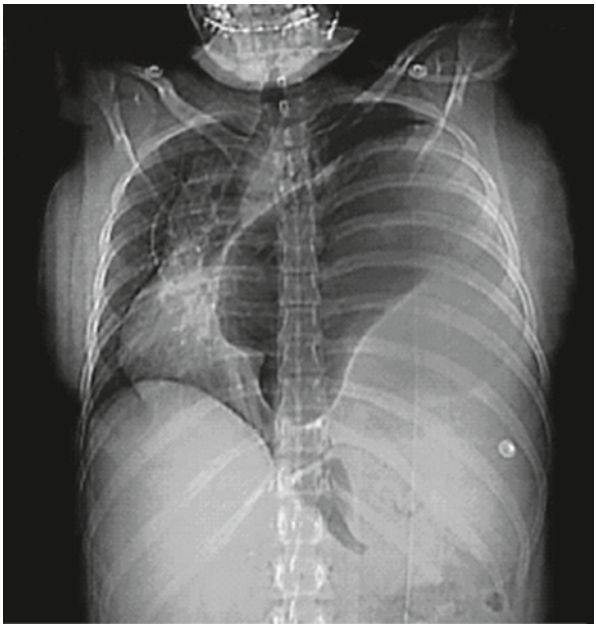

**Fig. 16.2.** Scout film of the CT scan showing a distended stomach occupying the left hemithorax with gross shift of mediastinum to the right

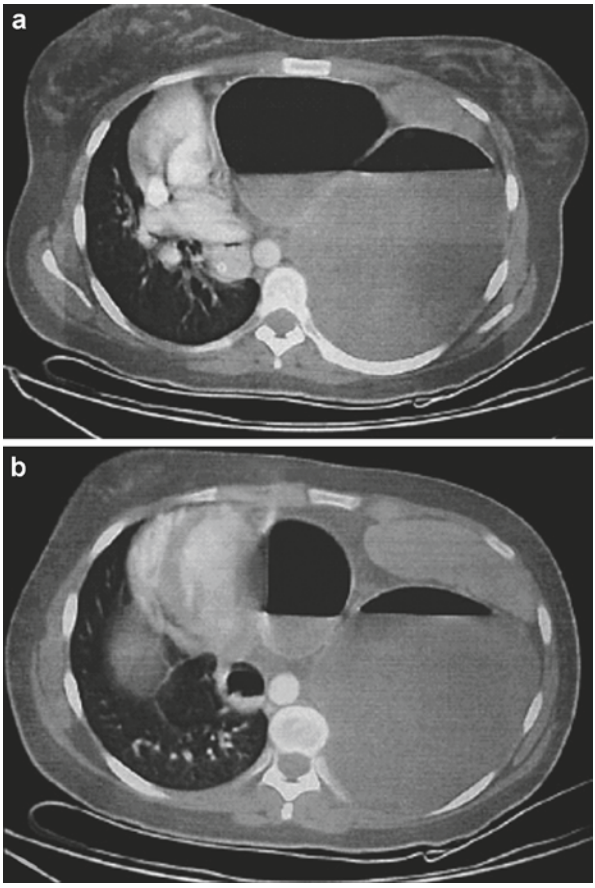


**Fig. 16.3.** Axial CT scan through the lower thorax. (**a**) Air fluid level in a distended stomach with shift of the heart to the right. The "bubble" on the *right* represents the gastric antrum. Note the nasogastric tube in the distal esophagus to the right of the aorta. (**b**) A lower cut with the spleen visible on the *left*. The *left bubble* represents the gastric fundus. Note the "transition line" between the two bubbles representing the site of the volvulus

### *Management*

Although emergency room staff tend initially to eyeball these patients and label them as suffering from "respiratory failure" or myocardial infarction, a prompt chest X-ray will suggest the diagnosis and promote further aggressive imaging studies. The presence of the stomach (or other viscera) in the chest in this clinical scenario is a dire surgical emergency because of the unpredictability

of the situation; the patient may appear well, but the stomach may be rapidly becoming necrotic. On the other hand, of course, an asymptomatic upside-down stomach may coexist with other intrathoracic emergencies.

The treatment of acute gastric volvulus is surgical, consisting of a laparotomy, reduction of the volvulus, and assessment of gastric viability. The vast majority of acute diaphragmatic hernias can be reduced through the abdomen and very rarely is there a need to add a thoracotomy. Reduction of the herniated viscera may be aided by two maneuvers: inserting a wide tube through the diaphragmatic defect abolishes the negative, "sucking" thoracic pressure, and a nasogastric tube may be manipulated into the distended stomach to reduce its size. When the latter is not successful, a decompressive gastrotomy is necessary before the stomach can be reduced into the abdomen. This should be performed carefully in order not to contaminate the thoracic cavity—an event that often leads to postoperative empyema.

After the hernia is reduced, the hernial sac is excised, and the diaphragmatic defect is closed with interrupted sutures. A very large defect may need to be patched with a synthetic prosthesis, although this is not advised in the presence of contamination. Finally, some experts would recommend a tube gastrostomy—well sutured to the anterior abdominal wall—to decompress the stomach and prevent recurrence of the volvulus. Others have recommended gastropexy—suturing of the stomach to the abdominal wall or even to a window created in the avascular area of the transverse mesocolon. The addition of an antireflux procedure, such as fundoplication, is controversial—and most probably inadvisable—in such emergency situations when it is unknown whether the patient has also a sliding hernia and gastroesophageal reflux.

When the stomach is found to be nonviable, gangrenous portions are resected by partial or total gastrectomy as required. In the moribund patient who needs total gastrectomy, it may be safer to postpone the reconstruction: insert a tube to drain the distal esophagus, close the duodenal stump, and place a tube jejunostomy distal to the level of the eventual planned entero-entero component of the Roux-en-Y loop jejunoesophagostomy, which will be performed once the patient is stabilized and ready for such a reintervention.

In selected hemodynamically stable patients, laparoscopic reduction and detorsion of the stomach followed by endoscopic gastropexy or fundoplication have been reported possible. The finding of gastric necrosis would call for immediate conversion to an open approach.

17

# Upper Gastrointestinal Hemorrhage (and Portal Hypertension)

Moshe Schein

*"If anyone should consider removing half of my good stomach to cure a small ulcer in my duodenum, I would run faster than he."* (Charles H. Mayo, 1861–1939)

*"About gastrectomy for duodenal ulcer: in this operation … a segment of an essentially normal stomach is removed to treat the disease next door in the duodenum. It is like taking out the engine to decrease noise in the gear box."* (Francis D. Moore, 1913–2001)

During my residency in the 1980s, not a week passed without a few operations for bleeding duodenal (DU) or gastric (GU) ulcers. Emergency gastrectomies, antrectomies, truncal vagotomies, and highly selective vagotomies were our daily bread and butter. But, gradually things started to change. First appeared the $H_2$ antagonists, followed by proton pump inhibitors (PPIs), and then anti-*Helicobacter* therapy. In addition, novel methods of achieving transendoscopic hemostasis of bleeding ulcers emerged. As a result, at least where we practice, operations for upper gastrointestinal hemorrhage (UGI-H) have become a rarity, and our approach to them has been modified. However, if you work somewhere in the so-called developing world, where modern antiulcer medications are not freely available, you may be exposed still to the old pattern of peptic ulcer disease and the traditional methods of dealing with it.

In all likelihood, you are becoming less and less familiar and skilled in the operative management of UGI-H. Therefore, you need to listen to us—☺ …

## The Problem

Upper gastrointestinal hemorrhage implies a source of bleeding **proximal to the ligament of Treitz**. Although textbooks list multiple causes, the vast majority of patients bleed from a chronic DU or GU, complications of portal hypertension (*esophageal varices* or *hypertensive gastropathy*; acute complications of

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portal hypertension), or *acute gastric mucosal lesions* (e.g., stress ulcers, erosive gastritis, and other terms that mean more or less the same). The last are usually due to ingestion of analgesics or alcohol ("aspirin for the hangover"). With the routine use of antiulcer prophylaxis in hospitalized "stressed" patients, significant UGI-H from mucosal lesions is now rare. In fact, hemorrhage in stressed patients often originates from reactivated chronic peptic ulcers. Other potential sources of bleeding are the *Mallory-Weiss tears* in the mucosa at the gastroesophageal junction, usually caused by severe retching, coughing, or vomiting, and the *Dieulafoy lesion*, a manifestation of a gastric submucosal vascular abnormality.

The exact mixture of etiologies in your hospital depends on local social habits and the sort of population with which you work.

## Presentation

Patients present either with hematemesis (vomiting fresh blood), melenemesis (vomiting altered "coffee-ground" material), or melena (passage of black stool per rectum). Hematochezia (passage of fresh or altered nonblack blood per rectum) usually originates from a source below the ligament of Treitz. Nevertheless, with massive UGI-H and rapid intestinal transit, unaltered blood may appear in the rectum.

***Remember:***

- Melena is black, sticky, and very smelly
- Maroon feces are not melena
- Red feces are not UGI bleeding
- Black blood per rectum always means UGI bleeding
- Fresh, red blood per rectum in a hemodynamically stable patient means that the source is *not* in the UGI tract
- Any type of blood—fresh or old, vomited, or retrieved through the nasogastric tube—means that the source is in the UGI tract

You do not need panendoscopy to diagnose UGI-H—contrary to the gastroenterologists' credo. A finger, a nasogastric tube, and a pair of eyes are as good.

## Key Issue: Is the Hemorrhage "Serious"?

Whether the hemorrhage is "serious" is a key issue because the seriousness of hemorrhage determines your diagnostic-therapeutic steps and the patient's outcome. In general, the larger the bleeding vessel, the more serious the hemorrhage.

The more serious the hemorrhage, the less likely it is to stop without an intervention, and the more likely it is to recur after it has stopped. As with almost any acute medical or surgical condition, the affected patients can be classified into three groups: the obviously serious and obviously not serious at both extremes and the potentially serious group in the middle. This intermediate group is always the most problematic in terms of diagnosis and selection of therapy but, at the same time, includes those patients for whom your correct management can improve outcome. Whatever the condition, the mildly ill patient should do well, and the very sick one may die in spite your efforts. It is the moderately ill who will most benefit from your ministrations.

## Stratification

Massive bleeding from a large vessel requires your immediate attention and intervention. A small ooze from a tiny vessel is usually self-limiting and of minor significance at least for the moment; you can investigate it electively. For most patients, however, the emergence of any quantity of blood from the mouth, anus, or any bodily orifice is alarming.

### *When Should You Be Alarmed?*

The literature contains various formulas, usually based on hemodynamic parameters and the volume of blood transfusions required, to distinguish between "massive" versus "nonmassive" UGI-H. We suggest, however, that you use your common sense and consider the following clinical paradigm for when to be alarmed:

- Is the vomited blood (or the aspirate in the nasogastric tube) fresh or coffee ground material?
- Are the rectal contents fresh, juicy melena or old dry melena?
- Is, or was, the patient hemodynamically compromised?
- Is there laboratory evidence (hemoglobin/hematocrit) of severe bleeding?
- Is the patient over 60 years of age? *Bleeding in elderly patients merits greater concern* because they are less likely to withstand a prolonged hemorrhage. (We find the APACHE II [Acute Physiological and Chronic Health Evaluation II] scoring system discussed in Chap. 6 useful in this situation as it reflects the acute physiological compromise inflicted on the patient by the bleeding, while taking account of age and comorbitidies.)

These considerations should place your patients somewhere on the large spectrum of UGI-H seriousness. At one extreme, the patient presenting in shock, with fresh blood pouring from the stomach, belongs to the serious group (group I);

at the other, the stable patient, with a little coffee ground material and old, hard melena is definitively not serious (group III). Many patients, however, belong to the potentially serious group (group II); the problem here is to distinguish between those who continue to ooze, or will rebleed, and those who have stopped bleeding and whose chance of rebleeding is low. This distinction requires active observation and endoscopy.

## Approach

In many parts of the world, patients presenting with UGI-H are initially seen by internists or gastroenterologists. We surgeons are usually called to take part in the management only when these specialists believe that they cannot stop the bleeding without our help—and this may be too late—which means that they may call you "to operate" when the patient is already unsalvageable. Yes, even in today's era of fancy endoscopic hemostasis and intensive care units (ICUs), patients can die from bleeding ulcers—I recall a young man admitted to a teaching New York hospital and undergoing two attempts at endoscopic control of his bleeding DU. He continued bleeding in the ICU; when I was called, he had bled out. I operated but too late—his life was lost because a simple hemostatic stitch to block the pumping gastroduodenal artery was not inserted in time. Thus, we have to know better how to manage these patients and encourage early referral to the surgeon.

*Check vital signs. Aggressive management of hypovolemic shock is the first priority.* Do not overtransfuse as there is evidence that excessive blood product administration exacerbates bleeding and results in a higher incidence of rebleeding.

- **With resuscitation under way, take a history.** Previous peptic ulceration? Dyspepsia? Antiulcer medications? (Remember, bleeding patients do not have pain because blood is alkaline and serves as an antacid.) Recent consumption of analgesics or alcohol? Severe vomiting or retching (Mallory-Weiss)? Chronic liver disease or varices? Nosebleed (swallowed blood)? Coagulopathy? Amount of blood vomited or passed per rectum (extremely inaccurate)? Full medical history (operative risk factors)?
- **Pass a large-bore nasogastric tube.** Flush the stomach with 50 ml water, and aspirate; fresh blood indicates active or a very recent hemorrhage; coffee ground material denotes recent bleeding that has stopped, while clean aspirate or bile means no recent hemorrhage. **Note:** very rarely, a bleeding DU is associated with pyloric spasm with no blood refluxing into the stomach; bile-stained aspirate excludes such a possibility.
- **Perform a rectal examination.** Fresh blood or juicy soft melena indicates active or very recent bleeding, while dry and solid melena signifies a nonrecent UGI-H (Fig. 17.1).

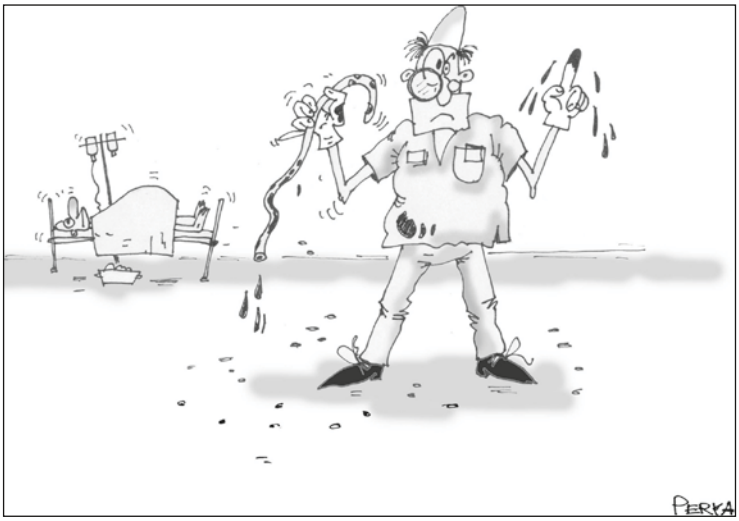

**Fig. 17.1.** "This is a 'serious' UGI hemorrhage"

## How to Proceed?

Now, with all this information in mind, you can classify the patients into one of the three groups (Table 17.1).

The "nonserious bleeder" (group III). These patients have suffered a minor hemorrhage, which has stopped. Do not rush to endoscopy in the middle of the

**Table 17.1.** Stratification and management of patients with upper gastrointestinal hemorrhage (UGI-H)

| | Group I, serious | Group II, potentially serious | Group III, not serious |
|---|---|---|---|
| Vomiting | Fresh blood | Coffee ground or fresh | Nothing/coffee ground |
| Per rectum | Fresh melena/blood | Fresh melena | Old melena |
| Hemodynamically | Compromised | Stable | Stable |
| Hemoglobin/ hematocrit | <9/27 | | >9/27 |
| Approach | Endoscopy now | Endoscopy soon | Endoscopy tomorrow |
| Prognosis | Requires hemostasis | Variable | Self-limiting |

night. Semi-elective investigation suffices and is more accurate and safer. Note that a very low hematocrit/hemoglobin in patients belonging to this group results from a chronic or intermittent ooze. The very anemic patient will tolerate endoscopy better after his or general condition is improved. These patients do not require an emergency operation, and therefore they are not discussed further.

The serious bleeders (group I). In a minority of patients belonging to this group, fresh blood is pouring torrentially from the stomach; they are virtually exsanguinating. You have to move fast. Esophageal or gastric varices often bleed this way—like an *open tap*. In such cases, a previous history of portal hypertension or clinical stigmata of chronic liver disease often coexist, suggesting the diagnosis. Remember: **you do not want to operate on varices** (see the end of this chapter).

In any event, you should transfer the exsanguinating patient to a critical care facility or the operating room. Intubate and sedate him or her to facilitate gastric lavage and subsequent endoscopy and, most importantly, to reduce the risk of aspiration of the gastric contents in the shocked, obtunded, bleeding patient. You should attempt endoscopy because, even if gastroduodenal visualization is totally obscured by blood, fresh bleeding from esophageal varices (usually at 40 cm from the teeth, at the gastroesophageal junction) always can be detected, mandating a subsequent nonoperative approach. In the absence of varices, proceed to surgery. The serious patients who are not exsanguinating should undergo an emergency endoscopy (as discussed for group II individuals).

The potentially serious bleeder (group II). Perform an emergency endoscopy.

### *Emergency Endoscopy for UGI-H*

Emergency endoscopy for UGI-H should be done only after you have resuscitated the patient and are in a controlled environment. Endoscopy induces hypoxemia and vagal stimulation; we have seen it cause cardiac arrest in unstable and poorly oxygenated patients. (In addition, closed cardiac massage on a patient with a stomach ballooned with blood may lead to gastric rupture). *Ideally, you—the surgeon—should be the one who performs the procedure*. Unfortunately, because of political and fiscal considerations, in many hospitals you are denied this access to endoscopy. If this is the case, at least be present at the endoscopy to visualize the findings first hand. Do not entirely trust the gastroenterologist, who will be going home soon, leaving you with the patient and any problems resulting from a poorly identified bleeding site.

To improve the diagnostic yield, the stomach should be prepared for endoscopy. Pass the largest nasogastric tube you can find and flush the stomach rapidly and repeatedly, aspirating as many clots as possible. A common ritual is to use ice-cold saline (with or without a vasoconstricting agent) for this purpose. None of these methods has been proven to be *therapeutic*. Tap water is just as good and much cheaper and does not aggravate hypothermia.

**Table 17.2.** Suggested classification of UGI-H

| No evidence of recent bleeding | Evidence of recent bleeding | Active bleeding |
|---|---|---|
| Clean base | Flat spot<br>Adherent clot<br>Visible vessel | Oozing<br>Spurting |

At endoscopy, you attempt to visualize the source of bleeding, which may be esophageal (varices, Mallory-Weiss, esophagitis); gastric (chronic GU or superficial lesions); duodenal (DU); solitary (chronic ulcer); or multiple (erosive gastritis). Look also for the following prognostic stigmata:

- Active bleeding from lesion
- A "visible vessel" standing up in the ulcer's base, indicating that the bleeding originated from a large vessel and that there is a high chance of further hemorrhage
- A clot adherent to the ulcer's base, signifying a recent hemorrhage

You may like to classify the findings as presented in Table 17.2.

### *Endoscopic Management*

Having visualized the lesion you should now treat it endoscopically to achieve hemostasis and to reduce the risk of further hemorrhage. In broad terms, endoscopic therapy has a better chance of success in shallow lesions, which contain small vessels. However, you should also attempt endoscopic hemostasis in deeper, large-vessel-containing lesions, with the aim of achieving at least temporary cessation of bleeding. This will permit a safer, elective, definitive operation to be performed in a better-prepared patient. The specific method of endoscopic hemostasis, be it a "hot" probe or injection with adrenaline or a sclerosant, depends on local skills and facilities. As in most places it will be performed by the gastroenterologists, the technique is not discussed here.

### *Postendoscopy Decision Making*

At the end of endoscopy, you are left with the following categories of patients:

- **Actively bleeding:** failed endoscopic hemostasis. The source is usually a chronic ulcer and emergency operation is indicated.
- **Bleeding (apparently) stopped:** chronic ulcer with a "visible vessel" or adherent clot visualized. The chances of further hemorrhage, usually within 48–72 hrs, are substantial. Treat conservatively but *observe closely*.
- **Bleeding stopped:** acute shallow lesion or chronic ulcer without the aforementioned stigmata. In these patients, further hemorrhage is unlikely; treat conservatively and *relax*.

## Conservative Treatment

*The mainstay of conservative treatment comprises completion and maintenance of resuscitative measures and observation for further hemorrhage.* In earlier editions of this book, we decried the use of PPIs as a useless intervention in acute UGI-H. We have changed our opinion. ("When the facts change, I change my mind! What do you do, sir?" John Maynard Keynes) There is now good evidence that administration of high-dose PPIs in patients with peptic ulceration reduces the incidence of rebleeding and the need for surgery following endoscopic hemostasis. Obviously, correct coagulopathies if present. All you need to do is to sustain the patient's organ systems and watch for rebleeding, which usually occurs within 48–72 hrs and can be massive and lethal. Careful monitoring of vital signs, observation of the number and character of melena stools, and serial hematocrit measurements will detect episodes of further hemorrhage. A nasogastric tube on suction is often advocated to provide early warning. In our experience, however, it is often blocked by clots, is of great discomfort to the patient, and therefore worse than useless. If, nonetheless, you choose to use it, flush it frequently.

## Indications for Operation

I do not suggest that you use cookbook recipes or formulas as they are of little help in the individual patient. Instead, use clinical judgment. That the exsanguinating patient and the one who continues to bleed after endoscopic hemostasis fails need an emergency operation is clear and has been discussed. Regarding those in whom the hemorrhage has stopped, with or without endoscopic hemostasis, the main indication for operation is *recurrent hemorrhage*. Factors that may or may not modify your decision to operate include the magnitude of recurrent hemorrhage, its source, and the age and general condition of the patient.

In general terms, recurrent hemorrhage is an ominous sign, meaning that bleeding will continue or, if stopped again, may well recur.

- If hemodynamically significant or originating from a chronic ulcer, *you have to operate*.
- If rebleeding seems of mild or moderate magnitude and stems from a superficial lesion, you may elect to continue conservative treatment or retreat endoscopically.

Gastroenterologists are now keen to repeat endoscopic therapy in rebleeding patients and even to do so a few times. Commonly, those patients "belong" to them, and you cannot interfere but watch them carefully and be ready to act.

After repeated endoscopic maneuvers, not much will be left of the first part of the duodenum when you eventually operate. A colleague said this:

> "Why are gastroenterologists more imaginative and courageous than we surgeons in employing new and bizarre invasive therapeutic modalities? Because they have somebody (us) to bail them out!" (Eli Mavor)

But, whatever you do, remember that old and chronically ill patients poorly tolerate repeated episodes of bleeding; do not mess around with them. As a rough guide, when the transfusion requirement exceeds four units of blood in a patient over 65 years of age, consider surgery.

## Operative Management

### *Repeat Endoscopy*

It is crucial that you know the exact location in the UGI tract from which the patient is bleeding. If the initial endoscopy was not done by you, or in your presence, *do it again*. In an anesthetized patient, it will not take you more than 5 min to insert and remove the endoscope. Do not trust the scribbled, 2-day-old endoscopy report that the "source of hemorrhage appeared to be in the duodenum." This could lead you to start with an unnecessary duodenotomy while the source lies high in the stomach.

### *Exploration*

An upper midline incision, supplemented with a paraxyphoid extension and forceful upward sternal retraction, lets you deal with anything in the foregut. In obese patients with a wide costal angle, however, a transverse, chevron-type incision may take a few more minutes but affords a more comfortable exposure. In addition, *a generous reverse-Trendelenburg tilt of the patient will bring the upper stomach almost to your nose*.

Start by searching for external visual or palpable features of chronic ulceration. The latter are invariably associated with serosal inflammatory changes. Look for evidence of chronic ulcers from the duodenum to the gastric cardia. Duodenal "Kocherization" (Theodor Kocher is perhaps the only surgeon in history to have his name used as a verb) will be necessary to reveal the sporadic

postbulbar ulcer in the second portion of the duodenum. Occasionally, a posterior or lesser-curvature GU will become palpable only through the lesser sac. Acute superficial mucosal lesions are unfortunately not identifiable from the outside, although a Mallory-Weiss lesion may be *tattooed* by bluish serosal staining at the gastroesophageal junction.

The finding of a chronic ulcer in accordance with the preoperative endoscopic finding tells you where the trouble is, but what is to be done in the absence of any external evidence of pathology? You have a few options:

- Proceed according to the endoscopist's findings, if you trust them, but they will not always be correct
- Surgical exploration
- Intraoperative endoscopy

### *Intraoperative Endoscopy*

Having endoscopically visualized, with your own eyes, an actively bleeding DU, you should not have any doubts. A doubtful endoscopic report, however, may promote a negative duodenotomy, extending it—piecemeal—proximally until the high gastric lesion is found. All that was needed was a small gastrotomy and suture ligation of the lesion; instead, you are left with a very long, messy, and unnecessary duodenogastrotomy to repair. To obviate such a mini-disaster, we would unscrub for a moment and shove in an endoscope. Sometimes, when the stomach is distended with huge clots, we would place a purse-string suture at the anterior wall of the antrum, perform a small gastrotomy, and with a large sucker remove and irrigate all clots. An endoscope is then inserted through the gastrotomy with the purse string tightened to allow gastric insufflation; this offers an excellent and controlled view of the stomach and duodenum. We call it "intraoperative retrograde gastroscopy."

### *Philosophy of Surgical Management*

A friend of ours, Asher Hirshberg, aptly stated that, "In the era of Helicobacter pylori, doing a gastrectomy for peptic ulcer is like doing a lobectomy for pneumonia." Clearly, where potent antiulcer drugs are available elective ulcer surgery has disappeared, and definitive antiulcer procedures during emergency surgery for complications of ulcer are disappearing as well. Why do a *surgical vagotomy* when PPIs offer a "medical vagotomy"?

The general philosophy is that saving lives, that is, stopping the bleeding, comes first. This is the main consideration in severely ill patients. In the less-compromised subjects, *the secondary issue of long-term cure of disease may be considered*. But now, when such a goal can be achieved by medical means, the role of definitive antiulcer procedures is limited and should be considered only

in well-selected patients: those expected to be noncompliant with medication and in situations where such medication is not readily available. This applies also to the management of perforated ulcers (Chap. 18).

Our current operative approach in the vast majority of cases is thus limited to *hemostasis only*. In a few selected and good-risk (e.g., APACHE II < 10) patients, we may consider a definitive antiulcer procedure *tailored* to the patient and the type of ulcer.

## Specific Sources of Bleeding

### *Duodenal Ulcer*

For a duodenal ulcer, the source of bleeding is always the *gastroduodenal artery* at the base of a posterior ulcer. Hemostasis is accomplished through an anterior, *longitudinal*, duodenotomy, underrunning the base (and bleeding vessel) with two or three (2–0 monofilament) deeply placed sutures—each placed on a different axis. When bleeding is active, successful ligation of the vessel will be evident; in its absence, you may want to abrade the ulcer's base, dislodging the clot and inducing bleeding. Otherwise, just underrun the base, deeply, and in a few directions. *The theoretical danger of underrunning a nearby common bile duct has been mentioned, but we are unaware of even a single report of such a case*. Others have described ligating the gastroduodenal artery from the outside, above, and behind the duodenum. However, we have no experience with this and would be anxious fishing for the artery at the base of the gastrohepatic omentum, which would be inflamed by the adjacent ulcerating process.

After achieving hemostasis, you are left with a few options. In the compromised patient—and most such patients are compromised—all you want is to stop the bleeding, close the duodenotomy without constricting the lumen, and get out. The eventual cure of the ulcer is left to acid—or *Helicobacter*-reducing drugs.

If the patient is in good shape and requires a definitive procedure (an extremely rare situation in our current practice), you may choose to prolong the operation by 30 min, adding a truncal vagotomy (TV), extending the duodenotomy across the pylorus, and closing it to form a Heinke–Mikulicz pyloroplasty. In a fit and stable patient, only 10 years ago we would close the duodenotomy and perform a highly selective vagotomy (HSV), adding an hour to the procedure. But today, we do not find suitable candidates for this procedure, and I bet that you have never learned how to do it.

Local hemostasis can be achieved even in the base of *giant ulcers* or when the duodenum is extremely inflamed or scarred. When simple closure of the duodenotomy appears to compromise the lumen or pyloroplasty is deemed otherwise unsatisfactory, just close the duodenum and do a posterior gastroenterostomy

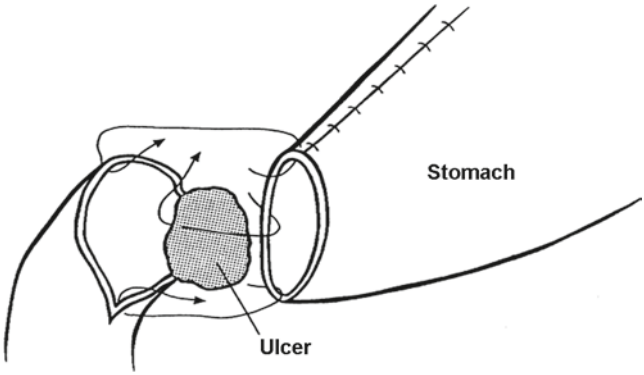


**Fig. 17.2.** Gastroduodenostomy: note that the posterior aspect of the anastomosis is performed with interrupted sutures, taking "big bites" of the duodenum (which is adherent to the pancreas)—well into the scar tissue at the base of the (now-excluded) ulcer

(GE), alone or added to the TV. The proponents of antrectomy plus vagotomy for bleeding DUs claim an increased incidence of rehemorrhage when gastric resection is avoided. In over 100 emergency operations for bleeding DUs, this has not been our experience, and we believe that there is no sense in removing a healthy stomach, producing a gastric cripple, for benign duodenal disease, which in any case can be subsequently cured with medications.

When, however, the duodenum is virtually replaced by a huge ulcer involving the anterior and posterior wall of the duodenal cap ("kissing ulcer"), one essentially is forced to perform an **antrectomy** (with a TV). In this situation, to avoid creating a duodenal stump that can be difficult to close and can leak, we prefer a Billroth I gastroduodenostomy (Fig. 17.2).

### *Postbulbar DU*

For unknown reasons, the postbulbar DU has almost disappeared from the Western world. Although extensive resective procedures (including an emergency Whipple) are mentioned in the old literature, all you need to do is to mobilize the duodenum, underrun the ulcer through a duodenotomy, and—perhaps—add a GE, with or without TV (in the previous editions, we mentioned HSV as another option, but again, this has become a procedure known only to the old surgical farts).

### *Gastric Ulcer*

Traditionally, for most surgeons a bleeding GU mandated a partial gastrectomy. Gastric resection is indeed effective in controlling the hemorrhage but in most

instances represents a superfluous ritual. For acute superficial ulcers, all that is required is simple underrunning of the lesion through a small gastrotomy. Even in patients who bleed from a chronic GU, simple underrunning of the ulcer from within, through a gastrotomy, usually suffices. In large chronic ulcers, we first underrun the bleeding point with an absorbable suture; with a heavy absorbable suture, we then obliterate the ulcer's base. UGI-H from a malignant ulcer very rarely requires an emergency operation. We would, however, take tissue from the ulcer's edges for histology. Partial gastrectomy becomes necessary only in cases of a giant GU on the lesser curvature with direct involvement of the left gastric or splenic arteries.

#### Definitive Procedure?

After hemostasis, in selected patients, as discussed, a definitive ulcer procedure may be considered. Chronic GU is not "one disease" to be managed by a ritual gastrectomy; instead, it is comprised of different types, which should be managed selectively. But frankly, in our part of the world this has become useless information; perhaps it is still valuable in yours?

- **Type I** is the classical lesser-curvature GU. Billroth I partial gastrectomy is the textbook recommendation. An HSV (from the ulcer proximally) plus the excision of the ulcer (from inside the stomach) is the alternative that we would recommend instead.
- **Type II** is a prepyloric ulcer. Although antrectomy plus vagotomy are popular for this "hybrid"—between DU and GU—ulcer, excellent results are achieved with HSV plus pyloroplasty. This is what we would do.
- **Type III** is a combination of a GU and a DU; it should be treated as type II.
- **Type IV** implies a high, juxtacardial, lesser-curvature GU. Prior to the days of effective antiulcer medication, partial gastrectomy—distal to the ulcer—was the procedure of choice. Since the entire lesser curvature may be obliterated, HSV is usually impossible, making TV plus a drainage procedure a reasonable alternative.
- **"Riding" GU** is a variant of a high GU associated with sliding hiatal hernia, produced by injury to the herniated stomach "riding" against the diaphragm. Surgical therapy involves reduction of the stomach by pinching the ulcer away from the adherent diaphragm, local hemostasis, and crural repair. This may be easier said than done since occasionally the huge riding ulcer adheres to mediastinal structures and may require major resective surgery.

### *Stomal Ulcer*

The stomal ulcer develops on the jejunal side of the gastrojejunal anastomosis following a previous vagotomy and GE or Billroth II gastrectomy. Because stomal

ulcers almost never involve a large blood vessel, hemorrhage is usually self-limiting or amenable to endoscopic therapy. Remember also that all stomal ulcers will heal on modern acid-suppressing medications. Persisting or recurrent hemorrhage, however, will force you, rarely, to operate. In the high-risk patient, do the minimum: through a small gastrotomy, perpendicular to the anastomosis, examine the stoma and ulcer; underrun the latter with a few deeply placed absorbable sutures; close the gastrotomy and put the patient on $H_2$ antagonists or PPIs for life. In selected patients, you can opt for a more definitive procedure. If the previous operation was a vagotomy plus GE, look for a missed vagal nerve or add an antrectomy. In the case of a previous Billroth II gastrectomy, add TV or consider a higher gastrectomy (do not forget to rule out Zollinger-Ellison syndrome later). Remember: hemorrhage from a stomal ulcer can be arrested with a simple surgical maneuver (underrunning); try to stay out of trouble by not escalating the emergency procedure into complicated reconstructive gastric surgery, which may kill your bleeding patient.

### *Dieulafoy's Lesion*

The small, solitary, and difficult to diagnose gastric vascular malformation termed a Dieulafoy's lesion typically causes a recurrent "obscure" massive UGI-H. It is best managed by transgastric local excision or underrunning.

### *Acute Superficial Mucosal Lesions*

With effective antiulcer prophylaxis in critically ill patients, you will be called to operate on acute superficial mucosal lesions only a few times in your surgical life. When massive hemorrhage necessitating an operation occurs, however, the involved stomach may look and behave like a blood-soaked and dripping sponge. Surgical options mentioned by the standard textbooks include TV and drainage or total gastrectomy. The former is associated with a very high rate of rebleeding and the latter with a prohibitive mortality rate. In this situation, we advocate gastric devascularization by ligating the two gastroepiploic and left and right gastric arteries near the stomach's wall. In our experience, this relatively simple and well-tolerated procedure results in an immediate drying of the gastric sponge.

### *UGI-H from an Unknown Source*

You will not encounter many UGI-H from an unknown source if the management plan has been followed, including—if necessary—the resort to intraoperative endoscopy. Angiography is an option and an excuse exercised by those looking

for a pretext to delay surgery. It is useless if performed when bleeding is not active. When talking about angiography, we have to mention another management option: *angiographic embolization* of the bleeding vessel. We would consider this option as an alternative to an operation in special circumstances, for example, bleeding DU when the risk of operative intervention would be prohibitive (e.g., after myocardial infarction) or an UGI-H from a **pseudoaneurysm** of the splenic artery associated with chronic or acute pancreatitis. Obviously, you have to have immediate access to a skilled invasive radiologist.

#### *Conclusions*

Admit patients with UGI-H to your surgical service. Do not leave them to the internists, who will call you when the patient is almost dead. After resuscitation, diagnose the source of hemorrhage and stage it. Give endoscopic treatment a chance but do not delay an indicated operation. At surgery, the goal is to stop the bleeding—remembering that most ulcers can be cured later by medication. Life comes first. Perhaps this rhyme will help you to remember:

> When the blood is fresh and pink and the patient is old
> It is time to be active and bold.
> When the patient is young and the blood is dark and old
> You can relax and put your knife on hold.

### Esophageal Varices, Portal Hypertension, and Cirrhosis

Luckily, abdominal surgery plays almost no role in the modern management of bleeding from esophageal or gastric varices. Luckily because some of us still remember the old days when we spent the night inflicting on these patients all sorts of emergency portocaval shunts or devascularization procedures, which were effective in arresting the hemorrhage but led to tremendous mortality from postoperative liver failure and its complications. (The operation was successful, but the patient died.) In this section, I briefly touch on the nonsurgical approach to variceal bleeding and the cirrhotic patient in general.

#### *Stratification*

Remember that *anything* you plan to do in a cirrhotic patient, with or without varices, depends on his or her hepatic reserves, which are best assessed by the modified Child-Pugh classification presented in Table 17.3.

**Table 17.3.** The Child-Pugh classification[a]

| | Score[b] | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| Bilirubin (mg%) | <2 | 2–3 | >3 |
| Albumin (g%) | >3.5 | 2.8–3..5 | <2.8 |
| INR | <1.7 | 1.7–2.3 | >2.3 |
| Encephalopathy | None | Mild | Marked |
| Ascites | None | Mild | Marked |

[a] Charles Gardner Child III (1908–1991) was a professor of surgery at the University of Michigan. Pugh published his classification in 1973 (Pugh et al. 1973)

[b] The individual scores are summed and then grouped as: <7 = Child A; 7–9 = Child B; >9 = Child C (a Child C classification forecasts a survival of less than 12 months)

**Child A patients** have good hepatic reserves. They will tolerate variceal bleeding and its management fairly well. They are also reasonable candidates for any indicated emergency abdominal procedures. Essentially, you can treat them as you treat noncirrhotic patients. But, bear in mind that the chronically diseased liver may decompensate when burdened with the metabolic consequences of severe surgical complications.

**Child C patients** (some call them "yellow balloons") have no hepatic reserves whatsoever, and in the absence of successful hepatic transplantation they are doomed to die within a year or so. Child C patients tolerate surgical procedures and their complications poorly. Consequently, operate on them only for lifesaving indications, in the absence of non-operative alternatives, and expect very high mortality and morbidity, depending of course on the specific problem and the magnitude of the operation.

**Child B patients** fall in between groups A and C; do the minimum necessary and be very careful.

### *Bleeding Varices*

Patients presenting with UGI-H from varices will usually provide a history of chronic liver disease or cirrhosis (alcoholic, viral) or previous episodes of bleeding. On examination, most of them will have features of portal hypertension and liver dysfunction listed in Fig. 17.3. The variceal source of the hemorrhage will be diagnosed or confirmed during the emergency endoscopy—not forgetting the cliché that one-third of UGI-H's in portal hypertension patients are not

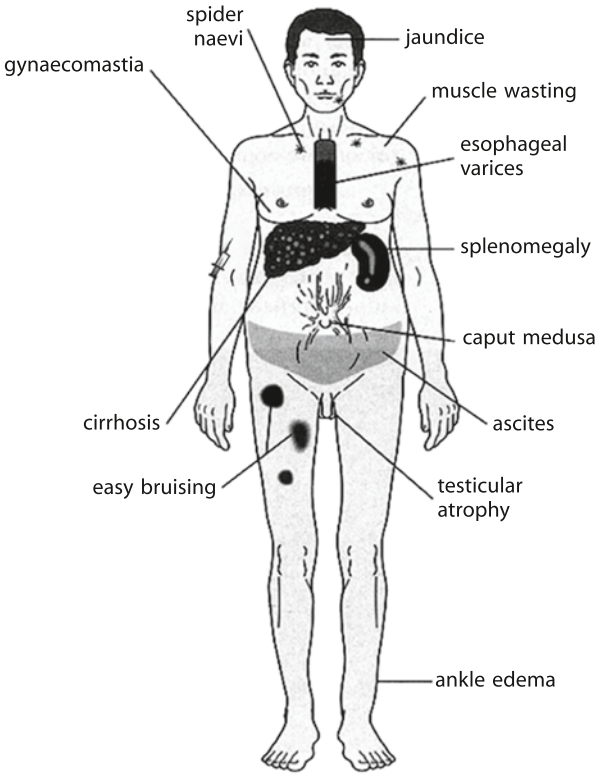


**Fig. 17.3.** Clinical features of cirrhosis

variceal but from other sources such as peptic ulcers. While *portal hypertensive gastropathy* can be a source for minor and chronic blood loss, it is probably not a cause for severe UGI-H. It is a classic and unforgivable error to attribute bleeding in a cirrhotic patient to varices while overlooking the responsible DU.

How best to manage an episode of esophageal variceal hemorrhage depends on the local facilities and expertise in your hospital and the tertiary care available in your environment. The essential options of management are outlined in Fig. 17.4).

### *Summary*

The surgeon's role in variceal hemorrhage is limited. Resuscitate, exclude nonvariceal causes of hemorrhage, tamponade bleeding with a balloon tube, and then send for help from the gastrointestinal specialists. For once, you get to go home at night.

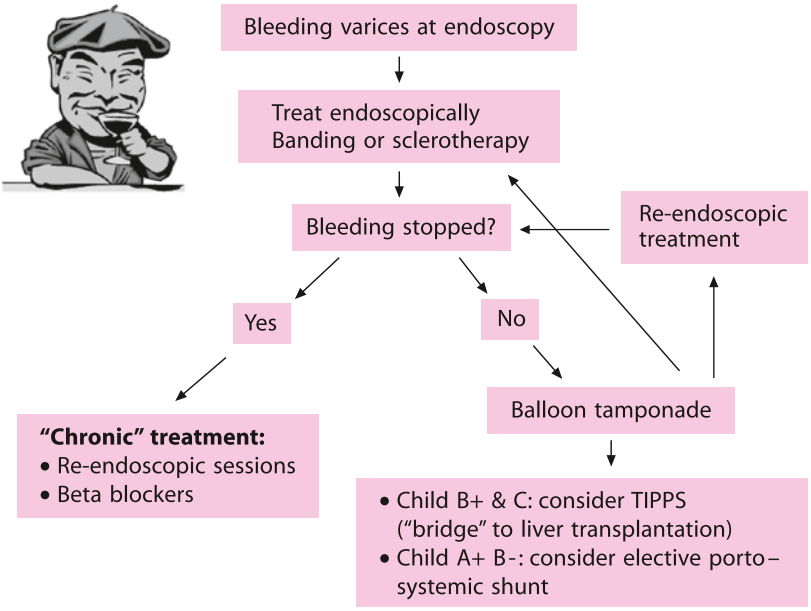


**Fig. 17.4.** Variceal bleeding: options in management. *TIPPS* Transjugular intrahepatic portal systemic shunt

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# 18 Perforated Peptic Ulcer

Moshe Schein

*There's a hole in my bucket. … How should I mend it?*
*Just patch it!* (A folk song)

*"Every doctor, faced with a perforated ulcer of the stomach or intestine, must consider opening the abdomen, sewing up the hole, and averting a possible or actual inflammation by careful cleansing of the abdominal cavity."* (Johan Mikulicz-Radecki, 1850–1905)

Thanks to effective, modern antiulcer drug management, the incidence of perforated peptic ulcers has decreased drastically (some say that the incidence and "virulence" of peptic ulcers started to decline even before such drugs became available and *Helicobacter pylori* was discovered as a cause)—but not everywhere. Perforated ulcers are still common in socioeconomically disadvantaged or stressed populations worldwide. Usually, perforations develop against a background of chronic symptomatic ulceration, but for a patient to present with a perforation "out of the blue," without previous history of peptic ulcer disease whatsoever, is not uncommon. In the Western world, perforated duodenal ulcers (DUs) are much more common than perforated gastric ulcers (GUs), which are seen more in lower socioeconomic groups.

## Natural History

In perforated peptic ulcers: "It must be remembered that the exudate in the early cases is sterile or nearly so, and the peritoneal reaction is a response to chemical irritation by the gastric and duodenal contents rather than the result of bacterial invasion" (John Blair Deaver, 1855–1931).

Classically, the abdominal pain caused by a peptic perforation develops very suddenly in the upper abdomen. Most patients can accurately time the dramatic onset of symptoms. The natural history of such an episode can be divided into three phases:

- **Chemical peritonitis/contamination**. Initially, the perforation leads to chemical peritonitis, with or without contamination with microorganisms. (Note that the presence of acid sterilizes gastroduodenal contents; it is when gastric acid

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is reduced by acid-reducing treatment or disease [e.g., gastric cancer] that bacteria and fungi are present in the stomach and duodenum). Spillage of gastroduodenal contents is usually diffuse but may be localized in the upper abdomen by adhesions or the omentum. Spillage along the right gutter into the right lower quadrant, mimicking acute appendicitis, is mentioned in every textbook but almost never seen in clinical practice (Chap. 28, about the "Valentino" appendix).

- **Intermediate stage**. After 6–12 hrs, many patients obtain some spontaneous relief of the pain. This is probably due to the dilution of the irritating gastroduodenal contents by the ensuing peritoneal exudate.
- **Intra-abdominal infection**. Should the patient escape the scalpel initially, after 12–24 hrs intra-abdominal infection supervenes. The exact point in time in the individual patient when contaminating microorganisms become invasive-infective is unknown. Therefore, you should consider any perforation operated on with a delay of more than 12 hrs as infection rather than contamination. This bears on your postoperative antibiotic therapy as discussed in a separate section here. Neglected patients may present a few days after the perforation in septic shock. Shock in the earlier stages is very rare although mentioned commonly by medical students, but when confronted with a combination of *shock* and *abdominal pain*, think about ruptured aortic aneurysm, mesenteric ischemia, or severe acute pancreatitis. Untreated perforation can lead eventually to an early "septic" death from peritonitis or the development of an intra-abdominal abscess.

## Diagnosis

The vast majority of patients present with signs of diffuse or localized peritoneal irritation; most lie still, groaning, and have a boardlike abdomen as described in textbooks (some call it "textbook peritonitis"). Spontaneous "sealing off" of the perforation, or localization of the spill or leakage into the lesser sac, causes atypical and delayed presentation. We had a patient who reperforated his DU a few years after receiving an omental patch. The second perforation was thus diverted backward into the retroperitoneum—behind the pancreas, the left colon, and into the scrotum—while the abdomen remained soft.

In a patient with an abrupt onset of upper abdominal pain and diffuse peritonitis, the diagnosis is simple. It can be summarized in the following *formulas*:

**Sudden onset peritonitis + free gas = perforated viscus**
**Sudden onset peritonitis + no free gas + normal amylase = perforated viscus**

There is free gas under the diaphragm in about two-thirds of perforated patients. Remember, free gas is visualized better on an upright chest X-ray than

on plain abdominal radiographs (Chaps. 4 and 5). If your patient cannot stand or sit up, order a left lateral decubitus abdominal film. Free gas is diagnostic, although it is not always due to a perforated peptic ulcer. But, so what? It signifies a perforated viscus, and a laparotomy is almost always indicated. "Almost always" means "not always": free gas without clinical peritonitis is not an indication for an emergency laparotomy. As mentioned in Chap. 4, there is a long list of "nonoperative" conditions that may produce free intraperitoneal gas. Free gas in a soft abdomen may also mean that the perforation has been spontaneously sealed and is thus amenable to nonoperative therapy, as discussed in a separate section.

**In the absence of free air, acute pancreatitis—the "great simulator"—should be considered and excluded** (Chap. 19). Normal serum amylase levels would support a diagnosis of a perforation, while very elevated amylase levels in a "susceptible" patient (e.g., alcoholic, with gallstones) would suggest acute pancreatitis. The "borderline" patient with atypical presentation and marginal elevation of amylase remains a problem because perforated ulcer may cause hyperamylasemia. In the good old days, before imaging techniques replaced clinical skills, our decision to operate or observe would have depended on the whole clinical picture. Rarely, a Gastrografin contrast study was performed to demonstrate or exclude leakage. Faced with such a patient today, we would advise you to obtain a computed tomographic (CT) scan of the abdomen, looking for free gas, extraluminal Gastrografin, and free peritoneal fluid. CT is excellent at picking up minute amounts of free intraperitoneal gas and is thus a valuable tool in clarifying the diagnosis in patients with an ambiguous clinical picture. Those of you who are lucky to practice in the United States know that in most such patients a CT is obtained before you have a chance to see them.

## Philosophy of Treatment

The **primary goal** of treatment is **to save the patient's life by eliminating the source of infection and cleaning the abdominal cavity. The secondary goal is to cure, if possible, the ulcer diathesis.** The former goal is achieved by simple closure of the ulcer; the latter requires a definitive ulcer operation, in other words, to know **when to do what**.

This is what we wrote in the previous editions of this book, but is it still relevant today? Not only are these perforations less frequent and (perhaps) less nasty, but such ulcers now can be healed in the long term (abolishing the ulcer diathesis) in most patients by modern antacid drugs and the eradication of the causative *Helicobacter pylori* bacteria. This being so, is there any place today for definitive antiulcer operations?

To find out about the current (2008) practice concerning perforated ulcers worldwide, we polled the international membership of SURGINET (an online general surgical discussion group). And, here is what we found:

- **How common is the problem?** It is very rare in developed countries. Seven U.K. surgeons, for example, reported two cases per year, while a surgeon in a small Australian town had seen only one case in 8 years. One respondent from a large urban American hospital estimated that he saw three cases per year. When Western lifestyle and availability of medications reaches a previously developing region, perforated ulcer becomes rare (e.g., Odessa, Ukraine). On the other hand, perforated ulcers are still common in indigent populations, such as in South Africa or India, where some surgeons report as many as 25 cases per month.
- **What is the site of perforated ulcers?** The vast majority are situated in the duodenum. A few are prepyloric or gastric (associated with nonsteroidal anti-inflammatory drug [NSAID] use). Because gastric resections for benign disease are so rarely performed, perforated *stomal ulcers* have almost disappeared.
- **Which operation?** All responders would use a *simple closure* as the preferred operative treatment. Some would add, infrequently, and in special circumstances, a definitive antiulcer procedure, as described below.
- **Open procedure versus laparoscopy?** Unless they are dedicated "advanced laparoscopists," most surgeons seem to prefer an open laparotomy.

## Who Are the Patients Who May Require a Definitive Procedure?

The patient who may require a definitive procedure is, according to Dr. Alex Berzoy of Ukraine: "The patient who would buy vodka instead of a protein pump inhibitor." And, he is right. The very patients who are susceptible to perforation also suffer from substandard access to medical care and reduced compliance, both adversely affecting successful medical antiulcer therapies. This is obviously much more common in the developing world. Consequently, if the operation for a perforated ulcer can kill two birds with one stone (especially if the environment around you cannot ensure optimal medical management and follow-up of your patient), why not add a definitive procedure (that is, if you know how to do it)? While "intractability" as a real issue seems to be limited to the developing world, other special problems that could indicate a definitive procedure may be present anywhere (see below).

### *For Which Patients Is a Definitive Procedure Safe?*

Surely, you do not want to embark on a lengthy definitive procedure in a critically ill and septic patient. Over the years, we encountered surgeons who omitted a definitive procedure because of "severe contamination," often citing a myth that vagotomy in a perforated patient may "spread the infection into the

mediastinum." The Hong Kong group showed that when the following three factors are present, an antiulcer procedure can be safely performed: blood pressure >90 mmHg, operation within 48 hrs of perforation, and lack of associated medical illnesses. We found the APACHE II (Acute Physiological and Chronic Health Evaluation II) scoring system (Chap. 6) useful in this situation as patients with perforated ulcers with scores less than 11 can tolerate a definitive procedure of any magnitude. Conversely, in patients with higher APACHE II scores, the simplest operation should be performed.

## Operative Treatment: Simple Closure (Fig. 18.1)

Classically, simple closure of the ulcer is best achieved by an omental Graham's patch, also called *omentopexy*. A few "through-all-layers" interrupted sutures are placed through both edges of the perforation (transversely, not vertically, so the lumen is not narrowed) and are left untied; a pedicle of the greater omentum is created and flipped over the perforation; the sutures are then *gently* tied over the omentum in order *not to strangulate* it (Fig. 18.2). At this stage, the anesthetist may be asked to inject saline, with or without dye, through the nasogastric tube to ascertain that the patch is waterproof.

More than a few surgeons misunderstand this operation; they initially suture close the perforation and only then *cover* the suture line with the omentum. However, the approximation of the edematous, friable edges of perforation can be troublesome. Some surgeons omit omental patching altogether and simply suture the hole. This may be successful in small, fresh perforations when the edges of the defect are pliable, but in all cases of **postoperative duodenal fistula** witnessed by


**Fig. 18.1.** "How should we mend it?"

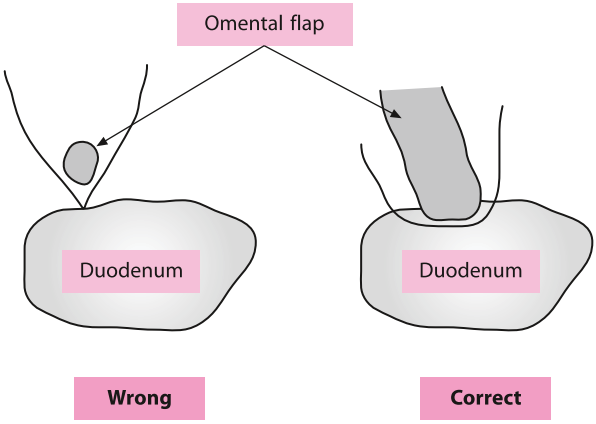


**Fig. 18.2.** Simple closure. Note the omental patch should "plug" the hole with the sutures tied over it. First suturing the hole and then sticking omentum over the repair is wrong

us, simple suture closure of a perforated DU was the causative mechanism. Be smarter, do not stitch the perforation but plug it with viable omentum.

**Should you leave a drain behind?** Only do this if you think that your omental patch is likely to leak, which should not be the case; thus, the answer is "no" (Chap. 42).

Do not forget a course of anti-*Helicobacter* antibiotics after the operation, combined with appropriate acid-reducing agents. High-risk elderly patients may need acid suppression for the rest of their lives. The duration of such management in younger patients, and whether they will be reinfected with the ulcerogenic bacteria, is controverisal.

### *Special Problems*

Special problems are the situations that may require more than simple closure:

- **"Kissing" ulcers.** Any evidence of a preceding or coexisting upper gastrointestinal (UGI) hemorrhage (e.g., finding "coffee ground" or fresh blood in the nasogastric tube or at the perforation site or in the peritoneal cavity) suggests the possibility of *kissing ulcers*—the anterior perforated, the posterior bleeding. Simple closure of the former without hemostasis for the latter could lead to a severe postoperative hemorrhage. In such circumstances, enlarge the duodenal perforation into a duodenotomy and explore the inside of the duodenum. If a bleeding posterior ulcer is found, suture transfix its base as described in Chap. 17.

- **Perforated giant ulcers.** Omentopexy can be easily performed for most perforated DUs. Rarely, a giant perforated DU creates a huge anterior bulbar-pyloric defect, which is not amenable to safe closure and thus mandates partial gastrectomy (Fig 17.2 in the Chap. 17).
- **Perforated GUs.** These are usually larger than the duodenal ones. For those positioned on the greater curvature of the stomach, a wedge resection of the ulcer, hand sutured or stapled, may be easier and safer than omentopexy. For chronic and large lesser-curvature ulcers, omentopexy is notoriously difficult and unsafe; partial gastrectomy may serve the patient better. Perforations of *malignant* GUs are very rare in the West (but in India, for example, many gastric perforations are malignant). Be that as it may, if we decide to patch a gastric perforation, we would take a few biopsies from its edges before closing it. If positive for cancer, an elective reoperation for "oncological" gastrectomy may be necessary.
- **Pyloric obstruction.** Perforated DUs are rarely associated with chronic narrowing of the gastric outlet. But, if the patient gives a history of prolonged postprandial vomiting or at operation the patient's stomach appears dilated and thickened, consider that possibility. Insert your index finger through the duodenal perforation and up the pylorus or feed in a Foley catheter and check whether the inflated balloon (5 cc) passes easily through the pylorus. Documented pyloric stenosis would demand the addition of some form of drainage procedure (pyloroplasty or gastrojejunostomy).
- **Intractable cases.** Selected patients with a clear history of chronicity or intractability (e.g., a recurrent perforation) with no easy access to health care and medications may benefit from a definitive antiulcer procedure. That surgeons in London or Houston no longer find indications for acid-reducing procedures does not mean that there are no patients in Calcutta who would benefit from it.

## Operative Treatment: Which Definitive Procedure?

Ideally, in an emergency you should choose the antiulcer procedure with which you are most familiar in the elective situation. The problem is that today you and other young surgeons are deprived of experience with elective antiulcer operations. Based on our philosophy to avoid, if possible, a gastric resection for a benign process and on results of elective ulcer operations (good old history!), we recommend an operative policy that tailors the definitive procedure to the specific ulcer (Table 18.1). Whatever you do, please remember that if your patient is *sick* and you are not a skilled gastroduodenal surgeon, forget about the definitive procedure. Just patch the hole and get out. Anyway, it seems that in a few years no surgeon able to decipher Table 18.1 will still be practicing. So, perhaps in future editions of this book there will be no chapter dedicated to perforated peptic ulcer or the only operative option mentioned will be omentopexy.

**Table 18.1.** Selection of procedures in perforated ulcers

| Ulcer type | Textbook options | | We recommend | |
|---|---|---|---|---|
| | Good risk | Poor risk | Good risk | Poor risk |
| Duodenal | Omentopexy ± TV + D or HSV or TV + A | Omentopexy | Omentopexy plus HSV | Omentopexy |
| Prepyloric | Omentopexy ± TV + D or TV + A | Omentopexy | Omentopexy plus HSV + D | Omentopexy |
| Gastric | Omentopexy or wedge excision or partial gastrectomy | Omentopexy or partial gastrectomy | Omentopexy with HSV + D or partial gastrectomy | Omentopexy or partial gastrectomy |

*TV + D* truncal vagotomy and drainage procedure; *TV + A* truncal vagotomy and antrectomy; *HSV* highly selective vagotomy; *HSV + D* highly selective vagotomy and drainage

### *Laparoscopic Management of Perforated Ulcers*

Omentopexy and peritoneal toilet can be executed laparoscopically (Chap. 57). A large amount of experience with conflicting results has been accumulated in the treatment of perforated DUs. We suggest that a laparoscopic procedure is a reasonable option in stable and well-resuscitated patients and when the perforation can be promptly and securely closed. Conversely, a prolonged pneumoperitoneum will be poorly tolerated in the high-risk or severely septic patients. And of course, you must be a skilled laparoscopist (do you know any surgeon who does not consider him—or herself "skilled in everything"?) to be able to perform a safe, watertight omentopexy.

By the way, any laparotomy for omentopexy need not always be a *macrolaparotomy*. Instead, with accurate preoperative diagnosis on CT, you can repair the perforation and suck out the free peritoneal fluid through a limited transverse right subcostal incision or a short midline epigastric incision, either of which is easier on the patient than the traditional *long* midline approach (Chap. 10).

### *Nonoperative Management of Perforated Ulcers*

A nonoperative approach consisting of nothing by mouth (NPO, nil per os), nasogastric suction, systemic antibiotics, and acid secretion inhibitors has been proven effective by a few enthusiastic groups. The sine qua non for success

is the spontaneous sealing of the perforation by the omentum or other adjacent structures; if this occurs, a nonoperative approach would be successful in the majority of cases.

Nonoperative treatment may be of particular value for two types of patients: the "late presenter" and the "extremely sick." The **late presenter** comes to you a day or more after the perforation occurred, with an already improving clinical picture and minimal abdominal findings. This, together with radiographic evidence of free air, hints at a localized and spontaneously sealed perforation. Nonoperative treatment, following a Gastrografin UGI study, or contrast CT, to document that the perforation is sealed should be successful in most instances. The **extremely sick** are the other candidates for conservative therapy: those for whom the risk of any operation could be prohibitive, such as the early post-massive myocardial infarct patient, the patient with grade IV COPD (chronic obstructive pulmonary disease), or the patient with an APACHE II score over 25. Also in this group, however, conservative treatment may be successful only if the perforation is sealed and radiographically proven to be so. Of course, localized collections or abscesses developing at the site of the sealed perforation can be drained percutaneously under CT guidance (Chap. 49).

#### Simple Surgical Drainage (in Difficult Circumstances)

In the absence of basic anesthetic facilities (e.g., somewhere in the bush or remote India) and when confronted with a patient who clearly needs an operation, there is a viable (and well-described) option. Under local anesthesia, the upper abdomen may be entered through a limited incision, and after aspirating as much muck and pus as possible, a large drain can be left under the liver in the region of the duodenum. If the perforation seals spontaneously, the drain helps to control the associated abscess; if not, then it forms a lifesaving controlled duodenal fistula to be dealt with—conservatively or operatively—later and elsewhere (not in the bush).

### Antibiotics

As soon as the diagnosis of perforation is made and the patient is booked for a laparotomy, administer a dose of wide-spectrum antibiotics. The vast majority of patients present for treatment within 12 hrs of perforation and suffer, therefore, from peritoneal **contamination** rather than **infection**. In many of them, in fact, the peritonitis is chemical and does not contain any micro-organisms. Antibiotics in this group will serve for prophylaxis. Postoperative therapeutic antibiotics are not needed in these patients. Those who present later than 12 hrs may suffer from

intra-abdominal infection; here, antibiotics should be continued in the postoperative phase (Chap. 47). The antibiotics given, in the form of either monotherapy or combination therapy, should "cover," empirically, Gram negatives and anaerobes. Routine culturing of the peritoneal fluid in perforated patients is not indicated (Chap. 12). *Candida*, which is a contaminant, does not need specific therapy.

## Conclusions

Patch a perforated ulcer if you can; in most patients this is possible, but if not, then you must resect. Consider adding a definitive antiulcer procedure on an extremely selective basis and do not forget that a nonoperative approach is possible, beneficial, and indicated in selected patients. Whatever you do, large studies show that one-third of these patients will be dead within 5 years; the same factors that led to the perforation shorten their life.

> "We have no responsibility to such patients but to save their lives. Any procedure, which aims to do more than this, can quite significantly be considered meddlesome surgery. We have no responsibility during the surgery to carry out any procedure to cure the patient of his duodenal ulcer." (Roscoe R. Graham, 1890–1948)

# Acute Pancreatitis

19

Joshua G. Barton · Michael G. Sarr · Moshe Schein

*"Acute pancreatitis is the most terrible of all the calamities that occur in connection with the abdominal viscera."* (Berkeley Moynihan, 1865–1936)

*God put the pancreas in the back because He did not want surgeons messing with it.*

## Editorial Introduction

In this edition, we decided to invite Drs. Sarr and Barton from the Mayo Clinic to write this chapter on the surgical management of acute pancreatitis. As the treatment of this condition has become less aggressive, increasingly non-invasive, and tailored to the individual patient, it requires a dedicated multispecialty team, which includes surgeons, radiologists, endoscopists, and intensivists. If possible, patients suffering from complications of severe pancreatitis should not be treated in community or rural hospitals but transferred to centers that can offer the best care—based on the multiple modalities of treatment mentioned by Drs. Sarr and Barton.

But, in the real world where many of you readers practice, not all such patients can be transferred to the Mayo Clinic or your local university hospital. It is important, therefore, that general surgeons, wherever they practice, develop understanding of this complex and multifaceted disease. The following text is aimed to provide an introduction to emphasize basic concepts before turning to Drs. Sarr and Barton's contribution to the chapter.

## The Basics

Moshe Schein

## Natural History

Uncomplicated acute pancreatitis (AP) is "a 1-week disease." Failure to recover or the persistence of local and systemic signs of pancreatic inflammation beyond the seventh day are signs that a complication may be brewing. You will best understand this complicated disease, and develop a rational clinical approach to its treatment, when you consider its evolution week by week (Fig. 19.1).

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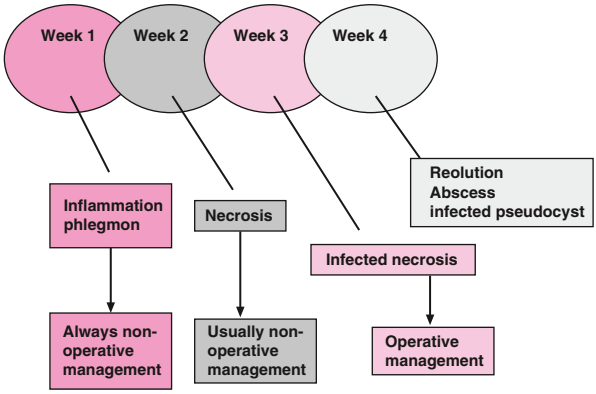


**Fig. 19.1.** Natural history of complicated acute pancreatitis (AP) and its management

### *First Week: Inflammation*

Inflammation in the first week is the phase of acute inflammation resulting in an inflammatory mass, which consists of the pancreas and adjacent structures—the so-called pancreatic phlegmon. Proinflammatory mediators (e.g., cytokines) are present in the beerlike (we refer here to real European dark beer, not the insipid pilsner consumed in the United States) hemorrhagic exudate of severe AP and are responsible for producing the characteristic local and systemic clinical inflammation (SIRS, systemic inflammatory response syndrome). The systemic repercussions of AP (e.g., respiratory or renal failure) depend on the intensity of the process and the quantity of mediators entering the retroperitoneum, the peritoneal cavity, and the circulation. In most patients, the inflammation is mild and will resolve in a few days. Patients with a severe inflammatory process tend to progress into the second week.

### *Second Week: Necrosis*

The phase of necrosis starts toward the end of the first week. The necrotizing process may involve the pancreas and its surroundings; retroperitoneal spread is hastened by activated proteolytic pancreatic enzymes. The severity of disease and therefore the prognosis depend on the quantity and extent of necrotic tissue (sometimes involving the entire retroperitoneum) and whether secondary infection supervenes. Pooling of the exudate in the lesser sac and beyond forms the so-called **acute peripancreatic fluid collections**, which may resolve spontaneously or gradually develop an *inflammatory wall* to become a **pancreatic pseudocyst**. The necrotic process may resolve spontaneously over a period of weeks. It may, however, become secondarily infected, a process that may occur as early as the second week but usually later.

### *Third Week: Infection*

The third week is the phase of infection. The diagnostic modalities described in this chapter may point to infection of the necrotic tissue by the middle of the second week, but its peak incidence is the third week. The causative organisms probably originate from the nearby colon by translocation, but superinfection with *Candida* species is not uncommon. The resulting infection of necrotic tissue produces infected pancreatic or peripancreatic necrosis, whereas secondary infection of a pseudocyst results in an *infected pseudocyst* (a late, rarer, and more benign process). The combined effects of necrosis and infection give rise to the clinical manifestations of local and systemic inflammatory syndromes.

Sterile and infected pancreatic necrosis (IPN) are clinically indistinguishable. IPN may occasionally produce a relatively mild systemic illness, while widespread sterile necrosis may cause the patient's demise, the outcome probably depending on the intensity of the inflammatory response in the individual patient.

### *Fourth Week and Beyond*

Patients with noninfected pancreatic or peripancreatic necrosis whose hitherto relatively benign clinical course did not mandate an operation (and only very rarely should an operation be performed at such an early phase) enter this "late" phase. We do not know what quantity of necrotic pancreatic parenchyma is capable of spontaneous resolution. We know, however, that large necrotic zones may be reabsorbed and thus resolve or, alternatively, undergo secondary infection, to present weeks later as a pancreatic abscess. This is an infective localized process developing after the resolution of the acute pancreatic inflammatory process. Therefore, its presentation, management, and prognosis differ drastically from those of IPN. Pseudocysts may also develop at this stage.

## Estimation of the Severity of Illness

Severe AP will eventually declare itself either by failing to resolve or by its dramatic systemic effects. It is important for you to recognize early that the attack is severe to optimize patient care, prevent infective complications, and estimate the prognosis.

Early attempts to estimate severity of disease revolved around measurement of levels of specific pancreatic enzymes or acute phase reactants, but it became obvious that one or two biochemical tests would not suffice. Beerlike, murky peritoneal fluid is diagnostic of necrotizing-hemorrhagic pancreatitis (i.e., severe AP), but this observation requires peritoneal aspiration, which is an invasive procedure and is unacceptable as a routine in the early phase of AP.

A number of scoring systems have been developed to estimate the severity of AP. Most are based on the estimation of clinical and laboratory variables that reflect the intensity of the inflammatory process. Imrie's (Clement Imrie, contemporary, Glasgow) method is popular in the United Kingdom, whereas elsewhere most medical students and enthusiastic medical residents can recite the lengthy list of early and late Ranson's criteria (John C. Ranson, 1938–1995). The APACHE II (Acute Physiological and Chronic Health Evaluation II) scoring system is useful in measuring the severity of *any acute disease* and has been shown to prognosticate the outcome of AP better than any other system. We advise you to use this uniform and user-friendly scoring system (Chap. 6). A patient with an APACHE II score of more than 8 has severe AP.

**Contrast-enhanced dynamic computed tomography** (CT) has been reported to be useful in diagnosing AP and grading its severity. The clinical diagnosis of AP is, however, straightforward, and scoring can assess the severity of disease better. Not uncommonly, we see patients with CT images of "horrendous pancreatitis" who feel well and go home after a few days without any complications. Moreover, contrast-enhanced CT examination has been implicated in the aggravation of microvascular damage in the pancreatic parenchyma. In addition, CT findings during the first week of AP will very rarely influence management decisions. We suggest that you avoid (as much as possible) CT scanning the AP patient in the early phase of the disease and reserve this examination for patients in whom the diagnosis of AP is uncertain. Please do not treat severe AP with daily CT scans. Ultrasound should, however, be performed early to confirm or exclude cholelithiasis as a possible cause of AP.

## Diagnostic and Therapeutic Approach

### *Inflammation: The First Week*

Generally, the approach to early severe AP is *conservative* and the treatment *supportive*. Historically, many different approaches have been tried in attempts to limit the effects of this disease. For example, since proinflammatory mediators cause the clinical manifestations, there were attempts to prevent or diminish such responses with *early pancreatectomy* or *peritoneal lavage*, respectively. Pancreatic resection in early severe AP is associated with a horrendous mortality rate and anyway does not prevent the development of intra-abdominal infection. Although continuous peritoneal lavage, if started within a day or two, may improve systemic manifestations, it is clear that it does not prevent the late major complications (and mortality) we are talking about. "Hemofiltration" of the blood of the noxious mediators liberated by AP has been tried but remains experimental.

**It appears, therefore, that you should offer these patients nothing more (and nothing less) than supportive care, preferably in the surgical intensive care**

**unit.** You should remember that severe AP represents a major abdominal "chemical burn," with many liters of fluid sequestrated in the retroperitoneum and peritoneal cavity. Optimal fluid balance and replacement are mandatory to protect the kidneys and provide an adequate venous return to the heart, which may be adversely affected by the pancreatitis-related myocardial-depressing factor. Overhydration, on the other hand, should be prevented especially in the presence of an associated ARDS (acute respiratory distress syndrome).

Note that the swollen pancreas, together with the edematous SIRS-affected viscera, may easily produce intra-abdominal hypertension. You will not know about it unless you measure the intra-abdominal pressure. When **abdominal compartment syndrome** complicates severe AP, the abdomen should be decompressed (Chap. 40). To us, this is the only indication for early laparotomy in AP.

We have always been told that "resting the pancreas," by gastric decompression and an NPO (nil per os, nothing by mouth) regimen is beneficial. This remains unproven. Gastric decompression with a nasogastric tube should be employed only in the presence of gastric ileus or outlet obstruction due to the swollen pancreas. Classically, the parenteral route was used for nutritional support, but recent evidence suggests that enteral nutrition via a transduodenal tube is well tolerated and results in fewer local and systemic complications and better outcome (Chap. 46). Early enteral feeding may indeed be beneficial.

What about antibiotics? Some evidence suggests that intravenous antibiotics are to be started in any AP patient assessed as "severe." This serves to prevent superinfection of the necrotic tissue, thus reducing the incidence of IPN. *Imipenem*, a wide-spectrum agent that achieves high levels within the pancreatic parenchyma appears to be the drug of choice. Some authorities recommend the addition of an antifungal agent (e.g., fluconazole) to prevent fungal superinfection of the necrotic pancreas. Others would administer antibiotics in all cases of *biliary pancreatitis;* this, of course, would be the logical thing to do when there are associated features of *ascending cholangitis*.

As mentioned, there is no indication at this stage to obtain a CT scan unless you are insecure about your diagnosis. Laparotomy is almost contraindicated during early AP and should be allowed only when a life-threatening surgical catastrophe cannot be otherwise excluded or to decompress an *abdominal compartment syndrome*. Indeed, *exploratory laparotomy in AP is not innocuous*; it adversely affects the natural history of the disease by increasing the incidence of infective complications. For this reason, no laparotomy for unexplained peritonitis should be undertaken unless the diagnosis of AP has been excluded.

**Endoscopic sphincterotomy is the only invasive therapeutic modality that should be considered early, during the first week, in the course of severe biliary**

**AP, especially if features of ascending cholangitis are present and the presence of common bile duct (CBD) stones is suspected** (see Chap. 20.3).

Your dedicated supportive care will result in the survival of most of these patients until their disease process enters the second week.

#### *Second Week and Beyond*

The second week and beyond is the time of necrosis, infection, and other complications. We are grateful to Drs. Sarr and Barton for the following section that details the modern expert approach to the complex problems of AP.

## Surgical Management of Acute Pancreatitis: State of the Art

Joshua G. Barton and Michael G. Sarr

*It is fascinating to conjecture how an inflammatory process in a retroperitoneal gland can produce abnormalities in so many organs.* (Reginald Fritz, 1843–1913)

Although surgeons are often involved in the care of patients with AP, surgery is only rarely required unless the etiology is related to gallstones. When operative intervention is required, the patients are usually extremely ill, and surgical intervention carries a substantial mortality (10–20%). Therefore, it is essential that practicing surgeons have a clear understanding of AP and the decision-making processes used in determining appropriate treatment.

### Classification

The Atlanta classification of AP was developed in 1992 to clarify and unify the nomenclature used in describing AP. Unfortunately, it has not been adopted consistently or universally. The Working Group Classification, devised in 2006, describes AP in terms that are more in keeping with the pathophysiology of the disease (Table 19.1).

### Clinical Course

#### *Mild Acute Pancreatitis*

Acute pancreatitis has a mild clinical course in the vast majority of patients. In patients with mild pancreatitis, the disease process is self-limiting and usually

**Table 19.1.** Working Group Classification, 2006

| |
|---|
| *Acute pancreatitis* |
| Interstitial edematous pancreatitis |
| Necrotizing pancreatitis (pancreatic necrosis and/or peripancreatic necrosis) |
| • Sterile necrosis |
| • Infected necrosis |
| *Fluid collections during acute pancreatitis* |
| **<4 weeks after onset of pancreatitis** |
| Acute peripancreatic fluid collection |
| • Sterile |
| • Infected |
| Postnecrotic pancreatic/peripancreatic fluid collection (PNPFC) |
| • Sterile |
| • Infected |
| **>4 weeks after onset of pancreatitis** |
| Pancreatic pseudocyst (usually has increased amylase/lipase activity) |
| • Sterile |
| • Infected |
| Walled-off pancreatic necrosis (WOPN) (PNPFC with defined wall) |
| • Sterile |
| • Infected |

subsides in 3–5 days with a mortality of less than 1%. Surgery or critical care management is rarely required in these patients. A surgeon's participation in the care of patients with mild AP is usually limited to performing a cholecystectomy during the same admission for cholelithiasis if present; a laparoscopic approach is usually possible (see Chap. 20.3).

### *Severe Acute Pancreatitis*

In contrast, AP should be classified as **severe** when the disease process is associated with organ failure for three consecutive days. A clinically relevant

SIRS and multiple organ dysfunction (MOD) often complicate severe AP. Local complications are frequent and may include sterile or infected pancreatic or peripancreatic necrosis, fluid collections, and pancreatic, enteric, or colonic fistulae. Operative intervention, when required, is directed at one of these complications.

#### Severe Acute Pancreatitis Manifests Two Different Temporal Phases

The *first phase* is characterized by the presence of distant organ dysfunction primarily and not by morphologic changes within the pancreas *per se*. Hence, this phase is described best by clinical parameters, the hallmark of which is organ dysfunction:

- Occurs within the first week of disease onset
- Serum amylase or lipase activity more than three times the upper limit of normal
- Characteristic findings of AP on CT, ultrasonography, or magnetic resonance imaging
- Organ dysfunction for three consecutive days

The *second phase*, which occurs 1–2 weeks after the onset of symptoms, is described more appropriately by morphologic changes within the pancreas itself. Typically, CT will reveal changes within the pancreatic parenchyma consistent with necrosis, marked interstitial edema of the pancreas, or fluid collections (fluid or solid content) outside the pancreas (› Fig. 19.2).

**Pathology that may require operation:**

- Gallstone pancreatitis (see › Chap. 20.3)
- Pancreatic necrosis (sterile and infected)
- Pancreatic pseudocyst
- Pancreatic fistula

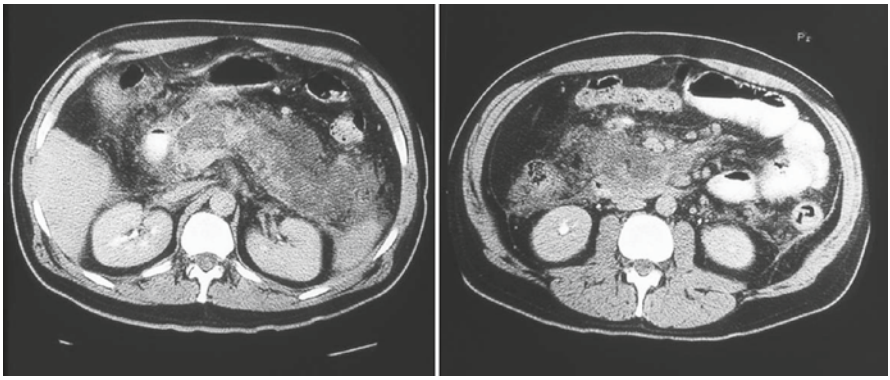

**Fig. 19.2.** Computed tomography of the abdomen revealing changes of nonenhancement consistent with necrosis and marked interstitial edema of the pancreas

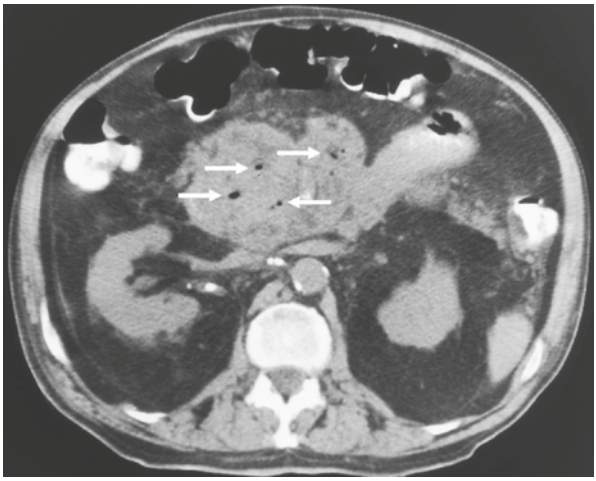

**Fig. 19.3.** Computed tomography of the abdomen showing areas of extraluminal gas (*arrows*) in the setting of AP

### *Pancreatic Necrosis*

The issue of primary importance to determine when pancreatic necrosis develops is if the necrotic tissue is sterile or infected. On contrast-enhanced CT, *extraluminal gas* should be considered pathognomonic of infection (Fig. 19.3). In the absence of extraluminal gas, a CT-guided fine-needle aspiration can be used to search for infection of the area of necrosis or fluid collection when clinical suspicion exists. The aspirated material should then be analyzed by both Gram stain and culture.

The culture results in particular should guide the use of antibiotic suppression therapy in an attempt to delay operative intervention for **at least** 4 weeks after the onset of pancreatitis. By so delaying the need for early operative intervention, mortality and morbidity can be decreased.

#### Sterile Necrosis

Over the last two decades, there has been a shift in viewpoints on the need for operative treatment of sterile pancreatic necrosis:

- Operating early in the course of sterile pancreatic necrosis with the goal of removing the devitalized tissue to improve the SIRS and thereby prevent MODS is no longer recommended.

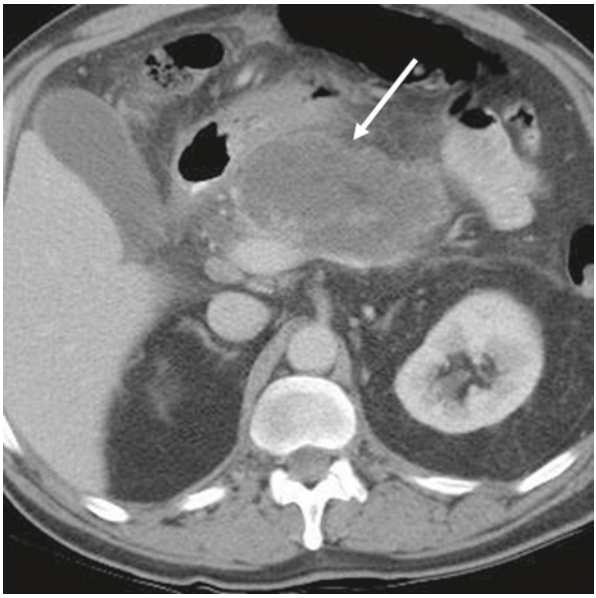

**Fig. 19.4.** Computed tomography of the abdomen revealing walled-off pancreatic necrosis (*arrow*)

- The aggressive use of prophylactic, broad-spectrum antibiotics appears to decrease the rate of infected necrosis but does not alter overall mortality.
- Advances in critical care have allowed conservative management of sterile pancreatic necrosis even when organ dysfunction is present.

These developments represent an important advance in the treatment of severe AP because surgery in the first 3 weeks is associated with a risk of substantial blood loss and poor outcomes.

Therefore, every attempt should be made to delay laparotomy until 4 weeks from the onset of AP and even then only in patients with persistent symptoms (or the persistently "unwell" patient). These symptoms may include anorexia, early satiety, vomiting, or fever. Waiting more than 4 weeks allows sterile necrosis, and any accompanying acute peripancreatic fluid collection (APFC) or postnecrotic pancreatic fluid collection (PNPFC), to either resolve or mature into walled-off pancreatic necrosis (WOPN) (› Fig. 19.4) or a pseudocyst (detailed in a separate section).

#### *Debridement for Sterile Necrosis*

Removal or debridement of sterile necrosis (better termed "necrosectomy") has been approached traditionally by an open laparotomy through a midline or

subcostal incision. The area of necrosis can be accessed through the transverse mesocolon, but to avoid injury to mesenteric vessels, entering the lesser sac through the gastrocolic ligament allows a wider exposure of the lesser sac and is usually more prudent.

A newer approach to necrosectomy in selected patients is by **minimally invasive techniques**, such as **peroral endoscopic interventional** techniques or **percutaneous and/or laparoscopic techniques**. Using the former method, a skilled interventional endoscopist accesses the area of necrosis via a transluminal, endoscopically created duodenotomy or gastrotomy, in the area of the duodenum or stomach where the pancreatic necrosis is adjacent and adherent; typically, this area bulges into the lumen and is relatively obvious. The necrotic material is removed and debrided using a combination of endoscopic graspers and irrigation. The resultant cavity is then drained by a series of transnasal or internal pigtail catheters. Good outcomes require substantial experience on the part of the endoscopist. In the future, this approach will likely be the procedure of choice for treating patients with isolated necrosis and WOPN accessible via the gut lumen. Similarly, a **laparoscopic approach** can be employed for accessible localized areas of necrosis. Currently, however, the standard therapy remains open debridement. Practicing surgeons should be comfortable and well versed with open debridement as their primary approach.

#### Debridement for Infected Necrosis

Once infected necrosis is diagnosed, some form of intervention is indicated. Although **minimally invasive techniques** for treating infected necrosis have been described, such techniques should be reserved for those centers experienced with these techniques. Open debridement via laparotomy remains, however, the standard of care in most centers. **The goals of operative treatment are:**

- As complete a necrosectomy as possible.
- Allow egress of resultant exudative fluid or extravasated pancreatic exocrine secretions.
- Treat the cause of biliary pancreatitis if safe and possible (e.g., cholelithiasis, choledocholithiasis).
- Provide a means for enteral nutrition (feeding jejunostomy) or gastric decompression (tube gastrostomy)—necrosis of the head and uncinate process often leads to a prolonged mechanical or functional outlet obstruction.

**There are four main approaches to the operative treatment of infected necrosis.** The mortality rates for each approach are generally felt to be similar, but complication types and rates differ.

1. **Necrosectomy and closed drainage:** one operation with fascial closure, multiple drains placed within necrosectomy site.

| Advantages | Disadvantages |
|---|---|
| One-stage necrosectomy | Potential for overaggressive debridement with subsequent hemorrhage |
| Low mortality | High recurrent abscess rate requiring reoperation or repeated percutaneous drainage (20–40%) |

2. **Necrosectomy and open drainage**: marsupialization of lesser sac by suturing omentum to fascial edges with open packing (peritoneostomy). This allows the area of necrosectomy to be re-dressed repeatedly, allegedly at the bedside.

| Advantages | Disadvantages |
|---|---|
| Open drainage | Increased risk of hemorrhage and fistula formation |
| Few recurrent abscesses | Open wound—loss of abdominal domain, fluid loss |

3. **Necrosectomy and closed lavage:** operative debridement followed by continuous, high-volume lavage of pancreatic bed via operatively placed drains.

| Advantages | Disadvantages |
|---|---|
| One-stage necrosectomy | Difficult to treat extensive necrosis extending inferiorly in the retroperitoneum |
| One operation (intended) | Actual or reintervention rate to treat recurrence or complications as high as 20–40% |

4. **Planned repeated necrosectomy**
   (a) Debridement followed by temporary abdominal closure
   (b) Return to the operating room in 48 hrs and as often as needed for further debridement

| Advantages | Disadvantages |
|---|---|
| By returning to the OR, subsequent issues can be addressed (persistent areas of necrosis, enteral access, bile duct exploration, etc.) | Repeated anesthetics and reoperation(s) |
| Very low unplanned reoperative rate | Loss of domain—abdominal wall defect |

## Surgical Wisdom

The key advance in the last decade that has allowed surgeons to avoid early operation for pancreatic necrosis is the aggressive use of intravenous antibiotics. Broad-spectrum antibiotics should be initiated prophylactically in all patients with severe pancreatitis early on, especially patients with SIRS, MOD, and sepsis syndrome.

During operations for pancreatic necrosis, blood loss can be minimized by avoiding overly aggressive sharp debridement. One should focus on bluntly removing only the tissue that gives way easily. Sponge forceps or the fingertips are good instruments for debriding necrotic pancreatic and peripancreatic tissue. If the completeness of debridement is in doubt, temporary closure of the abdomen and return to the operating room 48 hrs later can be advantageous.

## Pseudocysts

Of those with AP, fluid collections occur in 5–10% of patients. Over 80% of these collections resolve spontaneously. A pseudocyst usually follows extravasation of pancreatic ductal secretions secondary to duct disruption. The ductal communication persists, and the fluid collection organizes into discrete areas surrounded by a fibrous capsule. At the time of diagnosis, pseudocysts often but not always communicate with the pancreatic ductal system. Nevertheless, one must assume that communication with the ductal system exists; therefore, the type of operative intervention must be designed to manage this presumed communication.

Some form of active intervention is reserved typically for symptomatic pseudocysts. Symptoms are most often related to pain. In most patients, it is prudent to allow the pseudocyst to mature beyond 6 weeks; this approach allows sufficient time not only for potential resolution but also for the fibrous capsule of granulation tissue to mature enough to hold sutures to allow internal enteric drainage.

Prior to any procedure for a pseudocyst, an endoscopic retrograde cholangiopancreatography (ERCP) is usually advisable to assess for the site of connection between the pseudocyst and pancreatic duct as well as to look for associated ductal strictures proximal to the connection to the duct (and possibly for biliary obstruction that would require treatment as well either before or at the time of operative intervention). Pancreatic ductal strictures should be addressed prior to intervention, either via endoscopic treatment at the time of the initial ERCP or by operative treatment at the time of operative internal drainage. The ERCP can show etiologic causes of pancreatitis from the biliary tree (stones, duodenal diverticula).

### *Approaches to Pseudocyst Intervention*

**Percutaneous drainage**. This approach is less desirable due to the possibility of formation of a pancreatico-cutaneous fistula. This technique should be reserved for when the cyst wall has not matured and intervention is required. The usual indication in this setting would be infection of the pseudocyst (per the Atlanta classification, this would technically be a "pancreatic abscess"). In any situation, minimizing the risk of cutaneous fistulas and complete resolution of the cyst requires absence of any proximal pancreatic ductal obstruction.

**Open internal drainage**. Internal drainage by either transgastric cyst-gastrostomy, transduodenal cyst-duodenostomy, or cyst-jejunostomy to a Roux-en-Y limb is the gold standard for internal drainage of pancreatic pseudocysts. Several factors make internal drainage superior to other means. Internal drainage is the most flexible means of drainage in terms of accessing cysts in myriad locations. Perhaps the biggest advantage is that biopsy of the cyst wall is attained easily; this biopsy allows excluding cystic neoplasms in patients with a less-classic history of pancreatitis.

- Of the various forms of operative internal drainage, enteric drainage into the stomach is the most common route. To ascertain whether a cyst-gastrostomy is possible, preoperative imaging should ensure the presence of a pseudocyst adherent closely to the posterior wall of the stomach (Fig. 19.5a). The procedure can be performed through a small upper midline or left subcostal incision. A gastrotomy is first made in the anterior wall of the stomach, which should reveal the extraluminal pseudocyst bulging into the posterior gastric wall. Needle aspiration of pancreatic fluid will confirm the location of the cyst. An oval-shaped, full-thickness incision of the fused stomach and cyst walls is then made. A full-thickness biopsy of the cyst wall should be sent for pathologic analysis to rule out a cystic neoplasm. A formal cyst-gastrostomy anastomosis is then accomplished by "reefing" the fused stomach/pseudocyst with permanent suture material or with long-lasting absorbable suture to prevent bleeding.
- If the pseudocyst is located in areas not adjacent to the posterior stomach, two other options exist. Pseudocysts arising from the pancreatic head may be amenable to transduodenal drainage via cyst-duodenostomy (Fig. 19.5b) in a fashion similar to transgastric cyst-gastrostomy. In other locations not adherent to stomach or duodenum, the pseudocyst may be drained into a Roux-en-Y limb of jejunum.

**Minimal access drainage**. The mentioned open procedures can all be accomplished via laparoscopic or laparoendoscopic means depending on the experience of the surgeon. The most common and the least technically complex operation is a laparoscopic cyst-gastrostomy. As with an open cyst-gastrostomy, an anterior gastrotomy is performed into the stomach, and a square or triangular shaped cyst-gastrostomy is made in the posterior gastric wall with endoscopic stapling devices.

**Peroral endoscopic drainage**. In a procedure similar to endoscopic treatment of pancreatic necrosis, cysts adjacent to the stomach or duodenum can be

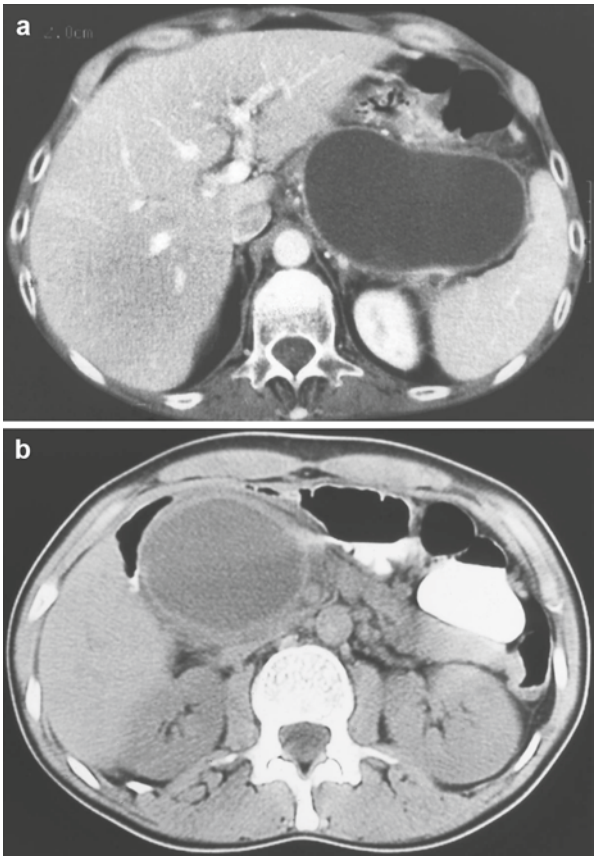


**Fig. 19.5.** (**a**) Computed tomography revealing a pseudocyst closely adherent to the posterior wall of the stomach suitable for cyst-gastrostomy drainage. (**b**) Computed tomography revealing a pseudocyst arising from the head of the pancreas in close apposition to the duodenum suitable for transduodenal drainage into the duodenum

drained endoscopically. A cystotomy is made through the wall of the duodenum or stomach into the adjacent pseudocyst. This entry into the pseudocyst is then kept open via multiple, internal, pigtail catheters. This procedure has produced excellent outcomes. Currently, endoscopic drainage of pseudocysts is eclipsing open internal drainage as the procedure of choice in many centers.

### Pancreatic Fistula

Pancreaticocutaneous fistulas may complicate the operative treatment of pancreatic necrosis. Fortunately, most fistulas can be managed nonoperatively. Once a fistula is diagnosed, initiation of bowel rest and parenteral nutrition are traditionally recommended. Tube feeding directly into the jejunum, thus

"bypassing" the duodenum and pancreas, does the same job. ERCP can be performed to define the pancreatic ductal anatomy and both to stent any ductal obstruction proximal to the fistula and to perform a pancreatic ductal sphincterotomy. For fistulas that persist after a trial of bowel rest, a regular diet can be reintroduced provided there is no increase in fistula output.

For persistent fistulas, it is reasonable to continue conservative therapy for 6 months. Thereafter, the likelihood of successful closure diminishes, and operative treatment should be entertained. Waiting 6 months not only provides adequate time for spontaneous closure of the fistula but also allows the abdominal cavity as a whole to recover such that operating becomes easier and the fistula tract matures. The ductal anatomy and, specifically, the point at which the fistula communicates with the pancreatic duct dictate which operation is most appropriate. For fistulas in the body and tail of the pancreas, a distal pancreatectomy would be most prudent. For more proximal fistulas, anastomosing a Roux-en-Y limb of jejunum to the fistula opening as an onlay pancreatojejunostomy is the best means of treatment.

## Conclusion

Acute pancreatitis, while typically a mild disease, can be devastating in its severe form. The last two decades have witnessed major advances not only in our understanding of the etiology and pathogenesis of the disease but also in its treatment—necrosectomy, antibiotic suppression/prophylaxis, minimal-access approaches, delayed necrosectomy, and more. Knowing the indications for operative intervention will have an impact not only on the morbidity but also on the mortality of the disease.

> "The most common errors in the surgical treatment of acute pancreatitis are to operate too early in the course of the disease, and to do too much, or in the secondary or septic phase of the disease to operate too late and to do too little." (Kenneth W. Warren, 1911–2001)

## Editorial Commentary

Indications for pancreatic necrosectomy are suggested in Table 19.2.

Drs. Sarr and Barton described the various operative strategies but which is the "best"?

It appears that the more aggressive approach (planned reoperations and laparostomy) is associated with increased morbidity, including hemorrhage, fistulization of the transverse colon, and abdominal wall defects. Each of the methods described may succeed in a certain patient and should be used selectively depending on the

**Table 19.2.** Indication for pancreatic necrosectomy (Uhl et al. 2002)

| |
|---|
| Presence of infected pancreatic necrosis (IPN) on CT (extraintestinal air) or fine-needle aspiration biopsy culture |
| Irreversible clinical deterioration despite maximum supportive care for at least 2 weeks from onset of symptoms |
| Suspicion of IPN in the absence of above features (item 1) in patients with more than 50% of their pancreas assessed as necrotic on CT |
| Extensive (>50%) necrosis and a prolonged ileus or continuing symptoms (pain, vomiting, inability to eat) despite resolution of distant organ dysfunction |

extent of IPN and severity of the illness in the individual patient. The less-aggressive approach may suffice in a patient with a localized process and small quantity of necrosis. Extensive IPN, however, may require the most aggressive treatments.

### *Practical Operative Points for the Tyro*

When operating on pancreatic necrosis or IPN, you must understand that it is often impossible to be performing a *definitive* debridement. Leave the rest for tomorrow (i.e., reoperation). Overenthusiastic debridement will debride the bowel (which will leak) or adjacent vessels (which will bleed). Follow the necrotizing process down the retroperitoneum; it may extend behind the left and right colon into the pelvis. Only the soft necrotic black/gray Camembert cheese-like material should be removed. By using your fingers or blunt sponge forceps to pick up the material, you will avoid the hard, nonnecrotic pancreas and other structures.

Enter the lesser sac from whichever direction is easiest, but expose it completely. *Try not to add insult to injury*. This is easier said than done while burrowing within inflamed and friable tissues. Safeguard the vessels in the transverse mesocolon; these are commonly injured during transmesocolon entry into the lesser sac or by drains placed through this route. It is tempting to remove the spleen, which may take part in an inflammatory mass in the pancreatic tail. This is not necessary; try not to injure the spleen during reoperations. The adherent duodenum and loops of small bowel are frequently injured during reoperations; this, together with the corrosive action of activated pancreatic enzymes, causes intestinal leaks. Be extremely gentle with the bowel and avoid rigid drains near the duodenum for they will erode. Often after *necrosectomy,* there is diffuse ooze from the resulting cavity. Pack it. Try not to place packs directly on exposed veins; they will be eroded and bleed. Safeguard the omentum and place it between the packs and exposed vessels. For more on the conduct of laparostomy, see Chap. 52.2.

### *Final Words*

The proper management of severe AP requires that you understand its natural history and be armed with lots of patience. During the early phases of the disease *"our patience will achieve more than our force" (Edmund Burke)*; later, when called to operate on necrotic and infected complications, remember that *"patience and diligence, like faith, remove mountains" (William Penn)*. If you can, refer these patients to centers of excellence (occasionally of arrogance), which have the expertise to do the job better, and less invasively, than you.

"Everything in surgery is complicated until one learns to do it well, then it is easy." (Robert E. Condon)

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# 20

# Gallbladder and Biliary Emergencies

Moshe Schein · Ahmad Assalia · Gary Gecelter · B. Ramana

*In dropsy of the gallbladder … and in gallstones we should not wait 'til the patient's strength is exhausted, or 'til the blood becomes poisoned with bile, producing hemorrhage; we should make an early abdominal incision, ascertain the true nature of the disease, and then carry out the surgical treatment that necessities of the case demand.* (James Marion Sims, 1813–1883)

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# 20.1 Acute Cholecystitis

Moshe Schein · Ahmad Assalia

*Acute cholecystitis (AC) is either calculous or, less commonly, acalculous. Since the clinical picture of these two entities differs they are discussed separately.*

## Calculous Acute Cholecystitis

Acute cholecystitis is initiated by a gallstone, which obstructs the gallbladder's outlet. Its spontaneous dislodgement results in so-called biliary colic, while persisting impaction of the stone produces gallbladder distension and inflammation, namely, AC. The latter is initially chemical, but gradually, as gut bacteria invade the inflamed organ, infection supervenes. The combination of distension, ischemia, and infection may result in a gallbladder empyema, necrosis, perforation, pericholecystic abscess, or bile peritonitis. Doubtless you must have heard or read numerous times about the classical symptoms and signs of AC. Let us therefore concentrate on problem areas.

### *How to Differentiate Between Biliary Colic and AC*

Time is the best discriminator between biliary colic and AC as the pain and epigastric/right upper quadrant (RUQ) symptoms of biliary colic are self-limited, disappearing within a few hours. Conversely, in AC the symptoms and signs persist. Furthermore, AC is accompanied by local (e.g., local peritonitis or tender mass) and systemic (e.g., fever, leukocytosis) evidence of inflammation, while biliary colic is not.

**The clinical picture**, which you know so well (we do not need to mention Murphy's sign again) is very suggestive. Laboratory findings of leukocytosis and (slight) elevation of bilirubin or liver enzymes may back it up. But, note that a lack of some or all features of inflammation or infection does not rule out AC—as is true also for acute appendicitis.

Luckily, you can (and should) confirm your diagnosis of AC with ultrasound (US) or a *radionuclide HIDA (hepatic iminodiacetic acid) scan*, which are readily

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available. Which of the two you should ask for first depends on its availability and the expertise in your hospital. We prefer ultrasound as, in addition to documenting the gallstones, it may provide incidental information concerning the liver, bile ducts, pancreas, kidneys, and peritoneal fluid, possibly suggesting alternative diagnoses. The *ultrasonographic findings in AC* include a distended, stone—or sludge-containing gallbladder; thickened wall; mucosal separation; pericholecystic fluid collection; or intramural gas. Not all of these findings are necessary to make a diagnosis. Positive *radionuclide scan* in AC means **nonfilling** of the gallbladder by the isotope. The specificity of the test is increased (i.e., fewer false positives) if morphine is administered, causing spasm of the sphincter of Oddi and reflux of isotope into the cystic duct. A false-positive result may occur in patients with significant hyperbilirubinemia. There are other (chronic) causes of nonfilling of the gallbladder (e.g., mucocele), but a negative scan with the isotope entering the gallbladder excludes AC. Of course, not a few of these patients will come your way after having undergone a computed tomographic (CT) examination in the emergency room. This can show the same features of AC as US.

**Whatever test you use, remember the following: you cannot diagnose AC when the gallbladder is nondistended.**

**Associated jaundice** ▶ mild-to-moderate elevation of bilirubin and hepatic enzymes is a relatively common feature of advanced AC, caused by reactive inflammation of the hepatic pedicle and the surrounding liver parenchyma. Thus, you need not attribute the jaundice to choledocholithiasis unless there are also clinical and ultrasonographic features of ascending cholangitis or bile duct stones (see below).

**Associated hyperamylasemia** ▶ similarly, mild elevation of the serum amylase does not mean that the patient is suffering from biliary pancreatitis. Commonly, hyperamylasemia is produced by AC with no signs of acute pancreatitis detected at operation.

In my [MS] environment, where there are no waiting lists for any operation and the operating room (OR) is always available, distinguishing between biliary colic and AC is purely an *academic exercise* because both conditions suggest the need for an early cholecystectomy, usually within 24 hrs.

### *Management*

In the vast majority of patients, the treatment of AC could be summarized in a few words: **proceed to laparoscopic cholecystectomy (LC)**. We know, however, that you would like a little more detail.

### Nonoperative Management

The natural history of AC is such that in more than two-thirds of patients treated nonoperatively the increased intra-gallbladder pressure will be relieved by dislodgment of the obstructing stone and resolution of the process. Conservative therapy, which should be started in all AC patients after the diagnosis is established, includes nil per os (NPO, nothing by mouth; nasogastric tube only if the patient is vomiting); analgesia (use a nonopioid if you believe in the hypothetical importance of avoiding constriction of the sphincter of Oddi); and antibiotics (active against enteric Gram-negative bacteria).

In the "old days," patients were discharged home after responding to a few days of conservative treatment to return for a *delayed, "interval" cholecystectomy* a few weeks later. This approach has been discontinued because of unpredictable failure to respond and recurrences of AC prior to the planned operation. Today, we reserve *delayed cholecystectomy* for patients who are medically unfit to undergo an operation in the acute stage, provided they respond to conservative management. There is abundant evidence showing that the earlier the operation, the easier it is. The acute inflammatory edema provides tissue planes, which facilitate cholecystectomy. Conversely, the more one delays the operation, the more "organized" is the inflammatory response, the more fibrosis and scar tissue forms—and the more difficult and traumatic the LC and hence the need to convert it to an open procedure.

### Surgical Management

Cholecystectomy is the optimal procedure; it eradicates the inflammation and infection and prevents their recurrence. Based on your clinical impression, it will be performed either as an "emergency" (rarely needed) or, usually, "early" procedure.

#### *Emergency Cholecystectomy*

An immediate, emergency procedure should be performed following resuscitation in patients with clinical evidence of *diffuse peritonitis and systemic toxicity or presence of gas within the gallbladder wall*—features suggesting perforation, necrosis, or empyema of the gallbladder. Most surgeons today would attempt a trial LC in this situation, converting to "open" in the presence of technical difficulties. We would warn, however, against prolonged peritoneal insufflation in the critically ill patient and would avoid prolonged attempts with laparoscopic dissection of the necrotic, perforated and difficult-to-grasp gallbladder. Emergency cholecystectomy for complicated AC in the critically ill or compromised patient

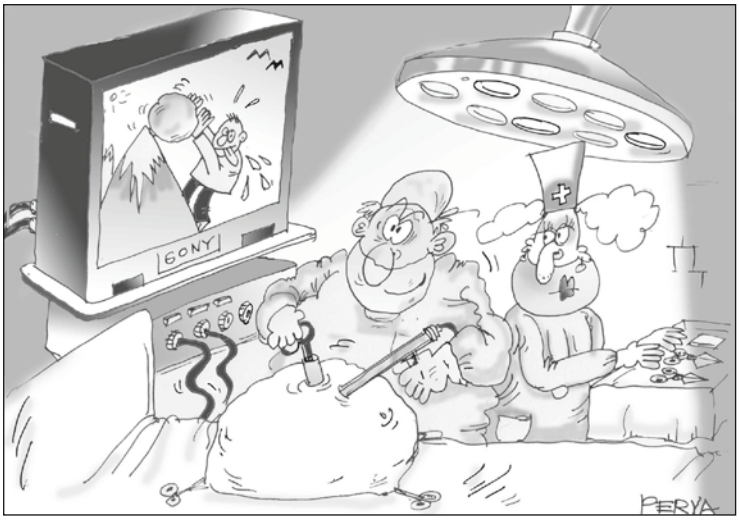


**Fig. 20.1.1.** "I never convert…"

could be open as described below. Obviously, a brief open cholecystectomy is easier on your patient than an open cholecystectomy following 2 hrs of futile laparoscopic excavation! (Fig. 20.1.1).

#### *Early Cholecystectomy*

Patients in whom emergency cholecystectomy is not clinically indicated should undergo an early cholecystectomy. But what is "early"? For some, it means that you do not need to rush to the OR in the middle of the night but operate in the daylight hours under favorable elective conditions. For others, it means to operate on the "first elective list." Depending on the surgeon's schedule and the availability of the OR, patients are often left "to cool down" for days awaiting their "semielective" cholecystectomy, which is often performed at the end of the elective lists. Occasionally, a waiting period as short as 48 hrs results in deterioration of the patient, but as observed already, the majority of patients with AC will settle down without an early operation.

Clinical appraisal of the severity of AC is notoriously unreliable; patients with gallbladder empyema or necrosis may be initially clinically silent only to deteriorate suddenly, while those with impressive RUQ signs may harbor just a simple AC. A mandatory operation within 24 hrs will prevent any problems arising from a delay in operation. Furthermore, it should be pointed out again that the operative dissection (laparoscopic or open) is easier and less bloody during the early phase of inflammation, with tissue planes becoming progressively more difficult as the process progresses. **Thus, the definition here of early cholecystectomy is an operation within 24 hrs of admission.**

**Note:** there is a subgroup of patients who will benefit from a delayed approach to prepare them better for surgery. For example, decompensated cardiac failure should be treated and coagulation disturbances corrected. Do not brandish your knife at unprepared patients!

#### *The High-Risk Patient Who Needs an Emergency Procedure*

With today's advanced anesthetic techniques and intensive care unit (ICU) support, it is rare to encounter a patient who cannot be subjected to an emergency procedure under general anesthesia. But, what are we to do with the occasional extremely sick patient who is "not even fit for a haircut under local," as they used to say? The best option is a *tube cholecystostomy* under local anesthesia. This can be done by you in the OR or—even better and less traumatic—by the radiologist, inserting the tube into the gallbladder percutaneously, and transhepatic, under CT guidance. Failure of the patient to improve within 24–48 hrs, particularly after the percutaneous procedure, should suggest the presence of undrained pus or necrotic gallbladder wall and the need to operate. Saying this, we have to admit that this last statement may be unnecessary because we have never experienced a patient whose AC—however severe it is—cannot be alleviated with tube cholecystostomy and antibiotics.

### *Acute Cholecystitis in Cirrhotic Patients*

An emergency cholecystectomy in cirrhotic patients with portal hypertension not uncommonly culminates in a *bloody disaster* due to an intra—or postoperative hemorrhage from the congested gallbladder's hepatic bed or large venous collaterals at the duodenohepatic ligament. Although *elective* conventional LC has been judged safe in "Child A" portal hypertension patients (Chap. 17), we believe that the secret here is to stay away from trouble. This means avoiding dissection near engorged and rigid hepatic parenchyma and staying away from the excessively vascular triangle of Calot in the emergency situation, particularly in patients with advanced cirrhosis. Subtotal or partial cholecystectomy is the procedure of choice in this situation (see below).

### *Technical Points*

#### Cholecystectomy

As mentioned, emergency procedures may be open unless you like to play around with the laparoscope in desperately ill patients. In early cholecystectomy, you—like most of us—may start laparoscopically, accepting a need to convert to

open—depending on your laparoscopic skills, patience, and courage—in up to one-third of the patients. It is important, however, not to be carried away, persisting with laparoscopic dissection in the face of hostile anatomy. A practical rule of thumb is to convert to laparotomy if after 45–60 min of laparoscopy you feel like you are "going nowhere." In many patients, a decision to convert can be made even much earlier than this, and you should not be afraid to abandon the laparoscopic approach at any stage if the circumstances are obviously unfavorable. Inappropriate persistence with the laparoscopic approach may well end in disaster with a bile duct injury. Having reviewed many litigated cases of post-LC disasters, there is no doubt in our mind that timely decision to convert could have prevented most such catastrophes. For an excellent list of rules of thumb to prevent this calamity, look at the article by Way et al. (2003) and pay attention to the commentary by co-editor AA in this chapter.

You may need some advice on the open procedure, which is becoming rare in elective practice and is increasingly being reserved for the "difficult" cases—the routine, "maxi," full-size gallbladder abdominal incision belongs to history. In the acute situation, start with a "midi" (5–10 cm) transverse RUQ incision, extending "piecemeal" as necessary. When converting from LC, simply extend the epigastric trocar site laterally—very rarely will you need a larger incision than one that connects the epigastric trocar site to the lateral one in the RUQ. Be aware that the results with open cholecystectomy through a midi- (<8 cm) or "mini-" (<5 cm) incisions are as good as those with LC.

The wise man's rule is to go fundus first (dome down) and stay near the gallbladder. After needle decompression (connect a wide-bore needle to the suction) of the distended gallbladder, hold the fundus up and away from the liver with an instrument and dissect down toward the cystic duct and artery, which are the last attachments to be secured and divided. By observing this rule, it is virtually impossible to damage anything significant such as the bile duct. When you are done, fold the omentum into the empty gallbladder bed; it helps in hemostasis, avoids formation of collections, and prevents the duodenum or colon from adhering to the liver, which will make your life much easier should you need in the future to reoperate, say to explore the common bile duct (CBD) for retained stones. Should you leave a drain? Sometimes and selectively (see Chap. 42).

### Subtotal (Partial) Cholecystectomy

Asher Hirshberg MD, summarized subtotal cholecystectomy aptly: **"It is better to remove 95% of the gallbladder [i.e., subtotal cholecystectomy] than 101% [i.e., together with a piece of the bile duct]."**

And yes, yes, yes—any weathered surgeon will tell you that this is the procedure to use to avoid misery in problematic situations such as those involving scarring—the "impossible" triangle of Calot—portal hypertension, or coagulopathy. Partial or subtotal cholecystectomy was popularized in the United States by Max Thorek (1880–1960); thus, some call it the Thorek procedure. Thorek, by the way, was a keen aphorist, and also said: **"How old is our newest knowledge, how painfully and proudly we struggle to discoveries, which, instead of being new truth, are only rediscoveries of lost knowledge."**

The gallbladder is resected starting at the fundus; the posterior wall (or what remains of it when a necrotizing attack has occurred) is left attached to the hepatic bed, and its rim is diathermized or oversewn for hemostasis with a running suture. At the level of Hartmann's pouch, after all stones have been evacuated, the cystic duct opening is identified from within. The accurate placement of a purse-string suture around this opening, as described by others, is not satisfactory because the suture tends to tear out of the inflamed and friable tissues. A better option is to leave a 1-cm rim of Hartmann's pouch tissue and suture-buttress it over the opening of the cystic duct. When no healthy gallbladder wall remains to close the cystic duct, it is absolutely safe just to leave a suction drain and bail out. In the absence of distal CBD obstruction, you will rarely see even a drop of bile in the drain because in such cases the cystic duct is obstructed due the inflammatory process. The exposed and often necrotic mucosa of the posterior gallbladder wall is "painted" with diathermy (some say until you smell fried liver), and the omentum is brought into the area. In this operation, the structures in Calot's triangle are not dissected out, and bleeding from the hepatic bed is avoided; it is a fast and safe procedure having the advantages of both cholecystectomy and cholecystostomy.

An extremely rare (so rare it deserves being published as an isolated case report) complication of subtotal cholecystectomy is the late **enlargement of the gallbladder remnant**—if left too large—presenting as symptomatic cholelithiasis, with US reporting "stones within the gallbladder." Differential diagnosis would include late enlargement of the **cystic duct remnant** and a **duplication of the gallbladder** (one of which was missed during the initial operation). This complication has been also described following conventional LC, in which the surgeon divided and occluded Hartmann's pouch instead of the cystic duct. Whatever the specific cause, the treatment is a "re-cholecystectomy" (preferably open) with a preoperative magnetic resonance cholangiopancreatography (MRCP) or endoscopic retrograde cholangiopancreatography (ERCP) providing a road map for the biliary anatomy. When doing a subtotal cholecystectomy, always make a detailed operative report, including the indications, and explain to the patient what was done and why, by this pre-emptively suppressing any future lawsuit.

**Laparoscopic subtotal cholecystectomy** is addressed below.

### Cholecystostomy

In our hands, subtotal cholecystectomy has almost replaced open tube cholecystostomy for the difficult gallbladder. Cholecystostomy is indicated in the very rare patient who must be done under local anesthesia and then only when percutaneous cholecystostomy is not available or is not successful.

After infiltration of local anesthetic, place a mini-incision over the point of maximum tenderness or the palpable gallbladder mass. You can mark the position of the fundus on the skin at the preoperative US as it is rather unpleasant for both you and the patient to enter the abdomen, under local anesthesia, and find that the gallbladder is far away. Visualization of gallbladder wall necrosis at this stage mandates a subtotal cholecystectomy; otherwise, open the fundus and remove all stones from the gallbladder and Hartmann's pouch. For improved inspection of the gallbladder lumen, and complete extraction of stones and sludge, a *sterile* proctoscope may be useful. Thereafter, insert into the fundus a tube of your choice (we prefer a large Foley), securing it in place with a purse-string suture. Fix the fundus to the abdominal wall, as you would do with a gastrostomy, and if possible place some omentum around. A tube cholangiogram performed a week later will tell you whether the cystic duct is patent and if so whether the bile duct is free of stones. The tube can be safely removed a few weeks later if all is well. Whether an interval cholecystectomy is subsequently indicated is controversial. Cystic duct obstruction, on the other hand (according to the prevailing dogma), would mandate interval cholecystectomy.

### Choledocholithiasis Associated with Acute Cholecystitis

About a tenth of patients who suffer from AC also have stones in the bile ducts. Remember, however, that AC may produce jaundice and liver enzyme disturbances in the absence of any ductal pathology. AC is very rarely associated with active complications of choledocholithiasis. In other words, AC combined with acute pancreatitis, ascending cholangitis, or jaundice is unusual. The emphasis, therefore, should be on the treatment of AC, which is the potentially life-threatening condition; ductal stones, if present, are of secondary importance.

Our management of patients with diagnosed AC and suspected choledocholithiasis would be tailored to the severity of the AC, the US appearances of the bile ducts, and the condition of the patient. Add to the decision tree your local facilities. As you know, there are many ways to skin this particular cat:

- **Severe AC, mildly elevated bilirubin and enzymes, bile ducts not dilated on US.** We would start with LC combined with intraoperative cholangiography. Should the latter be positive, we would proceed with an open CBD exploration or—if the stones are small—leave them to be dealt with by ERCP after the operation. Of course, if you are skilled at laparoscopic transcystic CBD exploration, go for it.

- **If the bile ducts are dilated on US,** there are liver function disturbances, and the AC is clinically not "severe," we would treat it conservatively and evaluate the duct with MRCP or ERCP. Any ductal stones would be dealt with by endoscopic sphincterotomy prior to LC.
- **In the critically ill patient** with or without gallbladder empyema or perforation, we would even "waive" the cholangiogram, leaving the symptomatic ductal stones to endoscopic retrieval after the life-saving cholecystectomy or cholecystostomy.

**A realistic perspective:** CBD exploration should be very rarely necessary in an environment providing modern imaging and ERCP service.

### Acalculous Cholecystitis

Acalculous cholecystitis is a manifestation of the disturbed microcirculation in critically ill patients. Although of *multifactorial etiology* (e.g., prolonged fasting, administration of total parenteral nutrition, etc.), the common pathogenic pathway is probably gallbladder ischemia, mucosal injury, and secondary bacterial invasion. Acalculous cholecystitis is a *life-threatening condition* developing during a serious illness, such as following major surgery or after severe injury. Stones may occasionally be present in the acutely inflamed gallbladders in these circumstances but are probably etiologically irrelevant.

*Clinical diagnosis is extremely difficult* in the postoperative, critically ill, or traumatized patient as abdominal complaints are masked. Fever, jaundice, leukocytosis, and disturbed liver function tests are commonly present but are entirely nonspecific. Early diagnosis requires a high degree of suspicion on your part: suspect and exclude cholecystitis as the cause of an otherwise unexplained "septic state" or SIRS (systemic inflammatory response syndrome).

*Ultrasonography* performed at the bedside is the diagnostic modality of choice. Gallbladder *wall thickness* (>3.0–3.5 mm), *intramural gas*, the *"halo" sign*, and *pericholecystic fluid* are very suggestive. Similar findings on CT examination would confirm the diagnosis. False-positive and -negative studies have been reported with both imaging modalities. *Hepatobiliary radioisotope* scanning is associated with a high incidence of false-positive studies. However, *filling of the gallbladder with the radioisotope (morphine assisted, if necessary) excludes cholecystitis*. A highly suggestive clinical scenario and diagnostic uncertainty together are an indication for active treatment.

**Management** should be promptly instituted as acalculous cholecystitis progresses rapidly to necrosis and perforation. *Select* the best treatment modality based on the condition of your patient and the expertise available in your hospital. In patients stable enough to undergo general anesthesia, *cholecystectomy* is indicated. When coagulopathy, portal hypertension, or severe inflammatory

obliteration of the triangle of Calot are present, *subtotal cholecystectomy* appears to be safer. *LC* may be performed in well-selected and stable patients. **Note:** insufflation pressure during laparoscopy should be kept as low as possible so the flimsy cardiorespiratory balance and hemodynamics in such patients are not upset.

**Open tube cholecystostomy** under local anesthesia may be indicated in the moribund patient when expertise for **percutaneous transhepatic cholecystostomy** is not locally available. The latter is the procedure of choice in the severely ill patient when diagnostic certainty is strong.

**Remember:** a few of these patients will have a totally necrotic or perforated gallbladder. In these, cholecystostomy may not suffice. *Percutaneous cholecystostomy* is a *blind* procedure; when rapid resolution of "sepsis" does not follow, suspect residual pus or necrosis or an alternative intra-abdominal or systemic diagnosis.

## Antibiotics in Acute Cholecystitis

Although routinely administered, the role of antibiotics is only adjunctive to the operative treatment as outlined in this chapter. In its early phase, AC represents a sterile inflammation, while later in most instances it represents a "resectable infection," that is, infection contained within the gallbladder that is to be removed (Chap. 12). Therefore, cases with simple AC need only perioperative antibiotic "coverage," which is discontinued postoperatively. In gangrene or contained empyema of the gallbladder, we recommend a day or two of postcholecystectomy antibiotic administration. In cases of perforation with a peri-cholecystic abscess or bile peritonitis, we suggest that you administer the maximal postoperative course of 5 days (Chap. 47). Which drug? Any drug effective against *E. coli* can be used. Antianaerobic drugs are not necessary, and monotherapy suffices.

When the gallbladder is difficult, go fundus first and stay near the wall.

## Technical Tips for Laparoscopic Cholecystectomy

Ahmad Assalia

Mastering the procedure for LC is considered basic and a "must" in modern surgical practice. Usually, this is considered the first laparoscopic procedure (sometimes together with laparoscopic appendectomy) every trainee should learn. Adhering to simple guidelines prevents catastrophes. As in every laparoscopic procedure, correct positioning of the patient and trocars is essential for successful performance.

**Positioning of patient:** make sure you are in the right position—between the legs in the lithotomy (the "French") position or on the left side of the patient in the supine position (the "American" position). A reverse Trendelenburg position and a slight rotation of the table to the left side would be helpful. I personally like the French position as it offers a more ergonomic way of working in any operation in the upper abdomen.

**Positioning of trocars:** the first should be at the umbilicus (upper or lower aspect, depending on the distance between the umbilicus and the costal margin). The two working trocars are located at the epigastrium and the RUQ. The trocar for retraction of the gallbladder should be located at the anterior axillary line away from the gallbladder. Try to triangulate your trocar positions and keep a distance of 8–10 cm between different trocars. Position of the trocars may vary according to the habitus of the patient.

In **obese patients** and often in difficult cases, you may need to insert an additional (fifth) trocar at the LUQ (left upper quadrant; some prefer to insert it at the right midabdomen) to help retract the colon, with the attached omentum, downward or even the duodenum. Do not hesitate to insert additional 5-mm trocars to improve your exposure. They do not need closure and are not painful afterward. Do not limit yourself to a certain number of trocars because it is "the common practice." Instead, use as many trocars as you need to optimize the procedure.

**For the cholecystectomy:**

1. As a beginner, use a high-quality 0°, 10-mm scope. This is the best scope that provides you with the optimal vision and is easy to drive by the inexperienced assistant. After you gain experience you may find that the 30° scope is more versatile in obtaining a complete view.
2. As in open surgery, work with both hands. This will improve your performance and dexterity.
3. Deal with the adhesions first (if they are present) to gain an initial anatomical orientation.
4. In cases of acute inflammation with marked distension of the gallbladder, it may be difficult to grasp and manipulate it. The edematous tense and friable wall may even render it a risky task because of the possibility of tearing off the wall and causing an unnecessary spillage of potentially infected bile, stones, or even pus. Therefore, as a rule always first decompress the distended gallbladder by aspirating its liquid contents. Do it under vision with a long, large-bore needle inserted through the anterior abdominal wall (a Veress needle could be ideal) or by using a designated endoscopic needle connected to a suction tube or large syringe through one of the trocars in the right abdomen.
5. By grasping the fundus, retract the gallbladder toward the right shoulder and the infundibulum laterally so that Calot's triangle opens up. Do not apply excessive

traction because of the risk of "tenting" of the CBD, which may result in injury; excessive traction may also tear the gallbladder—it does not look nice…

6. What to do with the "impossible-to-grasp" infundibulum? This is usually due to a large stone impacted in the distal portion of the gallbladder or extreme thickness of its wall due to severe inflammation. If Hartmann's pouch is packed with stones or with one big stone, try to move—"milk"—the stone upward; if this fails, just retract the infundibulum upward with an open blunt grasper or the jaws of the endoclinch grasper, without even trying to grasp the wall (retract it en bloc). This will do the job of opening up Calot's triangle. Normally, one of these two tricks works very well. I personally have never had to open the gallbladder (unless it happened "spontaneously" during dissection) to remove impacted stones, but it may be a logical option if the above maneuvers fail; then, you have to try to minimize the risks of contamination and "lost stones."
7. Start dissecting the triangle as close as possible to the gallbladder. Stay on the gallbladder wall. You have nothing to look for far away from the gallbladder. If you stick to it, the chances for biliary injury are minimized.
8. Minimize the use of cautery in this area. Usually, a simple hook cautery will suffice. Set it up to the minimal required coagulation degree (around 30) . Elegant blunt or sharp dissection or even gentle use of the suction device will help identify the important structures.
9. Always open the anterior and the posterior peritoneal layer at Calot's triangle and extend it to both sides of the gallbladder. By doing this, you will be able to improve the retraction and take the gallbladder away from the porta hepatis. Clear up Calot's triangle and create a space there where you can see just the cystic duct and artery entering the gallbladder and the liver clearly visible behind—the "critical view."
10. Do not cut what you think are the cystic duct and the cystic artery before making sure that these are the only structures entering the gallbladder. The gallbladder should be seen to funnel down and terminate in the cystic duct. Do not hesitate to continue the dissection to be sure. It is better to spend another 5–10 min with your patient than 5-10 years with your lawyer! Take all the time that you need. It is better to "lose" an extra moment than to lose the patient in a moment.
11. Try to define the cystic duct but do not overskeletonize it. This may cause an injury. While disconnecting the cystic duct and artery, go close to the gallbladder and as high as possible. Leave room for mistakes. Never place clips on a nonidentified structure.
12. In cases of thickened or widened cystic duct, make sure your clips securely close the whole width of the cystic duct without cutting it. This may occur with friable ducts in cases of acute severe inflammation. When in doubt, use an endoloop or even a stapler (white vascular load). But, before using a stapler, try to be sure again that you will staple off the cystic duct. In this case, you will have to replace the epigastric trocar with a 12-mm one.

13. For dissection of the gallbladder from the liver, simple traction, countertraction, and diathermy will do the job.
14. In cases of bleeding, do not blindly apply any clips or cautery. This is dangerous. Do not panic; first apply direct pressure with a blunt instrument or gauze, reorganize your setup, and then control the bleeding source under direct vision. Take a suction device in one hand and fine forceps in the other and precisely find and control the bleeding even if this necessitates the introduction of another trocar.
15. If you are unable to control the bleeding safely, then convert. Blind attempts to control bleeding in the porta hepatis may culminate in more bleeding, ligation of the right hepatic artery, or biliary injury. In contrast, bleeding from the liver (gallbladder bed) is almost always controllable with pressure, patience, and the use of electrocoagulation. It is helpful to increase the coagulation intensity for this purpose together with switching to the "spray" mode.
16. In cases of bleeding from the porta hepatis or the gallbladder bed, and if the bleeding is not profuse or originating from a "pumper," you may apply a piece of Surgicel, followed by local pressure and patience. Usually, this will prevent unnecessary application of clips with its potential risk. You may leave the Surgicel there as it is absorbable and even has some antibacterial potential.
17. What do you do if you face difficulty in identifying the anatomy in Calot's triangle? The options are as follows: try the (a) fundus first ("fundus down") approach; (b) subtotal cholecystectomy performed with the fundus down approach; or (3) convert. When choosing the first two options the most lateral trocar will serve to retract the liver, while the mid-upper right trocar provides countertraction on the gallbladder. If you have decided on partial cholecystectomy, then you can do it as described for the open procedure. Proceed downward to Hartman's pouch and stop there. Make sure there are no stones left in the pouch. Then, either oversew the infundibulum or use the endostapler.
18. In cases of unclear anatomy, be liberal in performing intraoperative cholangiography (IOC) before committing yourself. This is done easily using different available kits. You should also perform this in cases of suspected CBD stones.
19. If according to preoperative imaging there is a suspicion of gallbladder malignancy or if you suspect it intraoperatively, it might be wise to start with an open procedure or to convert because of the possibility of disseminating malignant cells. This is the only proven malignancy for which laparoscopy may be detrimental.
20. Irrigate and suction out any blood or spilled bile and stones. Make every effort to clean up any spilled stones. They may cause late abscesses; the large ones have been implicated in a long list of bizarre complications. You should irrigate the operative field, the subhepatis space, and the subphrenic space. Aspirate gently in the porta hepatis area so that the clips previously applied on the duct and artery will not be dislodged. Take a final look at the operative field and gallbladder fossa before leaving the abdomen. Take out the trocars under direct vision to identify and control any trocar site bleeding.

21. Use a retrieval bag if the gallbladder is inflamed, enlarged, or damaged during resection. You may retrieve it through the umbilical or the epigastric trocar openings, enlarging them slightly as necessary. You may aspirate any bile and crush the gallbladder stones before attempting to remove the GB from the abdomen.
22. Usually, there is no need to place a drain. If you decide for some reason (difficult case or difficult cystic stump, "wet" operative field, suspected accessory ducts that were identified or not), you may take it out through one of the lateral 5-mm trocars in the right abdomen (see Chap. 42).

**When should you convert?**

1. Vascular or bowel injury
2. Difficult or unclear anatomy
3. Uncontrollable bleeding
4. Suspected gallbladder malignancy
5. Identified biliary injury (see also the next section)

In general, be liberal with conversion. This reflects mature judgment. Safety of the patient is your goal and not your ego (we hope). In the difficult cholecystectomy, a trial of up to 1 hr or so is reasonable. Failure to progress is an indication for conversion.

> Intra-operative cholangiogram is a religion—not science. (Nathaniel J. Soper)

## Complications of Laparoscopic Cholecystectomy

*Ahmad Assalia and Moshe Schein*

Although relatively rare—but more common than after open cholecystectomy—complications of LC can cause severe morbidity and be life threatening. They are also a major source of litigation. Only surgeons who do not operate do not "produce" complications. But, we surgeons tend to minimize the importance of adverse events after operations that we have performed and are reluctant to consider them; this is our human nature and the fault of the overdeveloped "surgical ego." But, in all of the post-LC disasters that we have personally produced (fortunately few) or post-LC legal cases we have reviewed (not so rare), we have been able, looking through the retrospectoscope, variously to identify operative technical errors, serious faults in postoperative judgment, or negligent procrastination (the "surgical ostrich syndrome"; Chap. 45).

We have already discussed how to avoid complications during LC by proper technique and optimal judgment. Next, we dwell on the management of postoperative complications.

**The key is to be suspicious:** the normal course following LC is characterized by minimal pain, mainly at trocar sites, and some nausea and even vomiting, which should resolve in the first 24 hrs. *Anything beyond that* should raise the suspicion that something wrong is happening. Hemodynamic instability, continuing pain or nausea/vomiting, abdominal distension, fever, and jaundice are all signs of untoward complications. The abdominal pain could be localized (excluding trocar sites) or diffuse. If you have left a drain behind, any significant blood or anything bilious or resembling succus entericus might suggest disaster.

These are the common scenarios that you may encounter following LC:

**Biliary leakage**

Biliary leakage could originate from the cystic duct, "accessory" ducts (of Lushka), or the major bile ducts. Leakage from the cystic duct is usually secondary to dislodgement of clips, inadvertent injury during dissection, or inadequate closure of a large, friable, and edematous duct. In general, leakage from the cystic or accessory ducts does not require operative intervention unless the leak is uncontrollable and causes sepsis or diffuse peritonitis (if a drain has been left in situ during the operation, and is adequately controlling the leak, you can sit and relax as most such leaks will close sponataneouly within a week or so).

The patient with a bile leak may present with abdominal pain, malaise, fever, jaundice, and abdominal distension. The jaundice is the result of bile absorption from the peritoneal surface or due to an associated injury to the bile duct. These signs following LC warrant urgent intervention. We repeat: as a rule, if the patient is not septic and there is no generalized peritonitis, there is no need for immediate surgical intervention. Put your patient on NPO, intravenous fluids, and antibiotics and obtain a full lab workup. Abdominal US is the next step. If this is not diagnostic (i.e., no dilated bile ducts, no significant sub- or perihepatic fluid collection), then CT may be more helpful. Remember that immediately after LC there is almost always a small local fluid collection; this is normal. Only if this is larger than expected, associated with diffuse intra-abdominal fluid or dilatation of the bile ducts (intrahepatic), should it raise the suspicion that there is a bile leak and possible CBD injury.

Early imaging might be falsely negative. Biliary scintigraphy for the diagnosis of bile leak is probably useless. If there is a significant fluid collection without bile duct dilatation, you should perform percutaneous drainage guided by either US or CT scan to rule out or confirm bile leakage. After you have drained the biloma, the next step is endoscopic retrograde cholangiography (ERC) to identify any associated bile duct injury and, if necessary, to control leakages from cystic or accessory ducts by sphincterotomy and placement of a biliary stent.

**Common bile duct injury**

Injury to the CBD is the most serious complication of LC. Its incidence may be as high as 0.5%. Even experienced surgeons may encounter such a complication and not just the "freshmen." If the ERC reveals such an injury, then transfer your patient to a tertiary care facility to the care of a solid hepatobiliary surgeon. The best results for bile duct reconstructions are obtained in the right hands. Remember, the patient has a "one-shot chance" with this kind of surgery. Preferably, this shot should be done by an experienced hepatobiliary surgeon to ensure optimal outcome. Obviously, you, or your seniors, may know how to hook up the cut end of the CBD to a loop of jejunum, but here small details make a big difference. Sometimes, the injury is complicated and has an associated vascular injury. No urgency exists for immediate reconstruction once the patient is not septic and has been adequately drained.

**When to re-operate.**

This is necessary only if the patient is grossly septic or has generalized peritonitis. In all other cases of "pure" bile leakage, percutaneous drainage and ERC would suffice.

**How to re-explore?**

Whether you wish to attempt your re-exploration laparoscopically (see Chap. 52.3) or by open surgery is up to you.

**When to pursue biliary reconstruction?**

The following variables might help you to determine the appropriate timing for definitive reconstruction (in most cases with Roux-en-Y hepaticojejunostomy):

- Pattern of injury
- Timing of diagnosis
- Degree of inflammation and peritonitis
- Condition of local tissues
- Size of bile ducts
- The general status of the patient
- Surgeon's preference and experience

In general, all you want to do if forced to re-operate is to drain the bile collections, divert the bile leak by intubating the proximal bile duct or point of leakage. Your primary goal in the acute phase is to control SIRS or sepsis and not biliary reconstruction.

Early reconstruction could be accomplished if ideal conditions are present: patient is stable and not septic, no significant biloma or peritonitis, within the first 48 hrs from surgery, and preferably, a sharp and not diathermy mechanism of injury. In all other cases, allow for the patient to recuperate adequately, the sepsis or inflammation to resolve, and perhaps the bile ducts to dilate to some degree to, it is hoped, make the late definitive reconstruction easier—by an expert in this field.

Regarding what to do if duct injury is diagnosed during the cholecystectomy, see Chap 20.3.

**Bleeding** *(see also* Chap. 58*):*

Bleeding during or following LC can range from nonsignificant to life threatening. Potential sources are:

- Mesentery or major blood vessels during initial access. (One has to be truly negligent to produce such complications.) An immediate conversion and control of the bleeding is mandatory.
- Trocar sites. Normally, this is identified and dealt with intraoperatively but occasionally is missed. Usually, it does not require operative re-intervention, but it could be troublesome, causing blood loss and abdominal wall hematomas. This is especially true in patients with portal hypertension and patients on antiplatelet aggregation agents (aspirin/Plavix). In cases of intraoperative bleeding from the trocar site, you may angulate the trocar to the direction that applies pressure to the bleeding point. If bleeding continues, insert a Foley catheter through your trocar, inflate the balloon inside the abdomen (under vision), and pull out the trocar and the catheter so that it acts as a balloon tamponade for at least 5–6 min. If this does not work well, then you can pass a transfacial suture with a straight needle or a suture passer. Routinely visualize your trocar sites from inside the abdomen after you pull out the trocars so that you can identify bleeding before exiting the abdomen.
- Cystic artery or right hepatic artery. This was previously described (in technical aspects of LC).
- Liver bed. Also as above.

If bleeding occurs postoperatively, the patient may present with tachycardia, hypotension, and pallor. Blood count confirms the clinical suspicion. A drain in place may indicate bleeding but do not count on this; they usually become clotted and obstructed. As a rule, any bleeding causing hemodynamic instability warrants immediate surgical reintervention. We may be biased, but we personally prefer an open re-intervention rather than a laparoscopic one. The reasons are:

- Laparoscopy requires some extra time that patients may not have.
- Laparoscopy is not appropriate in the setting of hemodynamic instability.
- With laparoscopy, identifying the bleeding source could be difficult as the bloody field absorbs light and affects adequate visualization. In addition, evacuation of large clots could be a difficult task in laparoscopy.
- Anything that affects visualization may cause CBD injury or additional vascular injury from misguided clips or diathermy.

In all other cases of postoperative bleeding, selected patients might be managed by nonoperative means: optimizing the coagulation profile, blood

transfusion, and again patience. In cases of continued bleeding and if the patient is warm, is nonacidotic, and has normalized coagulation status, re-operation is indicated. In this situation laparoscopy may be appropriate. For more about postoperative bleeding, see Chaps. 56 and 57.

**Visceral injury** *(see also* Chap. 58*):*

Visceral injuries, if not recognized immediately, are commonly lethal. They have to be prevented, and if inflicted, they must be diagnosed immediately and corrected promptly.

Most commonly, such injuries occur not only during initial peritoneal access from Veress needle or trocar insertion (yes, they can occur also during open peritoneal access under vision) but also during subsequent port placement, adhesiolysis, or dissection of the duodenum or colon adherent to the gallbladder.

When you recognize visceral injuries during the operation, they can be safely repaired laparoscopically in experienced hands. If you do not have that experience, consider mini-incision or even a formal laparotomy for proper visualization and repair. With bowel injury caused by Veress needle or trocar, always look for a second enterotomy on the contralateral bowel wall.

**Unrecognized bowel injury** will present itself postoperatively. More on this is found in Chap. 57. But, we wish to repeat and emphasize again, and again, that most patients with such injuries die because of the surgeon's blasé attitude. Patients with a hole in their duodenum after LC do not present with classical peritonitis, and most will die before the leaking duodenal contents reach the trocar site or drains. Thus, if you ignore the soft, warning signs of excessive pain and need for analgesia, tachycardia, and oliguria, you will not be able to save the patient for most will die within 24 hrs. And the plaintiff's expert witness, reading through the dead patient's chart, will find the typical scenario: the nurse reports to the surgeon that "the patient is not doing too well"; the surgeon prescribes more morphine over the phone and goes back to sleep. So does the patient—permanently.

Timely recognition of complications and re-intervention, if necessary, will save lives and prevent significant long-term disabilities.

## Reference

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# 20.2 Acute Cholangitis

Gary Gecelter

*Thus with stone obstruction of the duct … dilation of the gallbladder is rarely observed; the organ has already undergone contraction; with obstruction from other causes, dilation is to be expected; atrophy exists only in 1/12 cases.*
(Ludwig Courvoisier, 1843–1918)

## What Is the Mechanism?

Acute ascending cholangitis is an infectious inflammatory consequence of biliary obstruction. Increased intrabiliary pressure above 30 cm$H_2O$ (normal 10–15) is associated with complete bile stasis and induces cholangiovenous reflux. This results in translocation of organisms and an inflammatory response that can result in death if not properly treated.

Cholangitis may "ascend" from an obstruction arising in the extrahepatic biliary tree, the two common causes being common bile duct (CBD) stones and pancreatic (or periampullary) carcinoma. Choledocholithiasis is more common as a primary cause of cholangitis, whereas the endoscopic treatment of periampullary carcinomas is the most common cause of iatrogenic cholangitis. Also, patients who have undergone a previous hepaticojejunostomy or endoscopic retrograde cholangiopancreatography (ERCP) stenting for iatrogenic biliary injury or stricture often present with recurrent attacks of cholangitis. Typical of cholangitis arising from choledocholithiasis is the prior history of "fluctuant" jaundice—an awareness of having been jaundiced at various times in the past. This is in contrast to patients who present with progressive (or crescendo) jaundice typical of periampullary tumors. The patient may also admit to having had gallstones diagnosed in the past or may have had a prior cholecystectomy. However, this classic presentation is by no means a rule, so do not presume a diagnosis when the patient presents for the first time with obstructive jaundice.

## What Are the Risks?

As with any acute illness, age and comorbidity are major determinants of the risk of dying from acute cholangitis. It is useful to run an APACHE II (Acute Physiological and Chronic Health Evaluation II) baseline in the emergency room

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(ER) and to keep a mental note of the changes as you monitor your patient to ensure that your interventions, or lack thereof, are not causing a rise in your patient's score (Chap. 6). If an elderly patient with high levels of serum bilirubin has leukocytosis (or worse, leukopenia), decreased urine output, a bad chest, and is drowsy, you should immediately consider him or her as a high-risk case. Renal failure, liver abscesses, and malignancies are associated with higher mortality. As a rule in this condition, the direct bilirubin decreases as the treatment takes effect.

### How to Make the Diagnosis (Fig. 20.2.1)

**Charcot's triad** (Jean Martin Charcot of Paris, 1825–1893) characterizes acute ascending cholangitis:

- Right upper quadrant (RUQ) pain
- Fever
- Jaundice

The fever and jaundice are easy to determine. Disproportionate pain may be due to coexisting acute cholecystitis. RUQ tenderness may be due to either cholecystitis (**Murphy's sign**) or tenderness in the liver itself. The distinction is probably unimportant as the treatment is the same. The feel of the liver, however, may lend itself to a bedside provisional diagnosis: a hard nodular liver is likely to be due to metastasis from a gallbladder cancer (especially in countries like Chile, India, and other endemic zones for gallbladder cancer) or a pancreatic or other gastrointestinal (GI) malignancy.

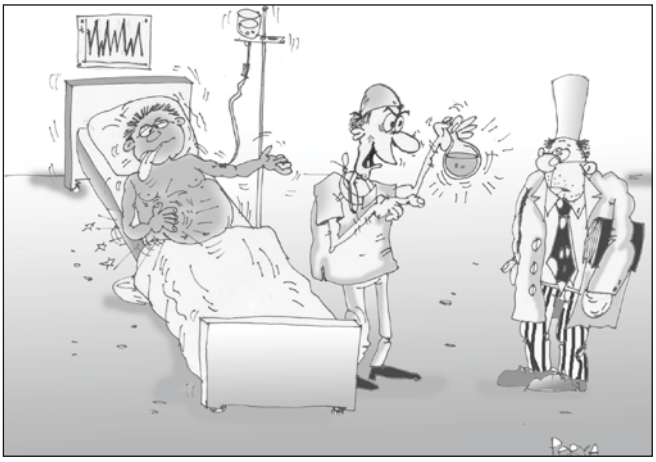

**Fig. 20.2.1.** "Oh, the urine is dark … what do you call that, triad? Charcoal triad?"

### *What Are the Signs of Complications?*

In the elderly patient, or when medical intervention is delayed, the syndrome can progress to include two further clinical features:

- Confusion (do not assume that any elderly confused patient has senile dementia; ask about the patient's baseline mental status)
- Septic shock

These two, when added to the *Charcot's triad*, become the *Reynold's pentad* (B. M. Reynolds, United States), which is associated with a fourfold mortality risk increase; consequently, clinical decision intervals must be very diligent and hourly.

### *Special Investigations*

Ascending cholangitis is diagnosed on the aforementioned clinical grounds. With early presentation, the jaundice may only be biochemical and must be substantiated by a liver panel. A typical panel has mildly elevated transaminases, variably elevated total bilirubin with a direct preponderance, and a disproportionately elevated alkaline phosphatase and glutamyl transferase; white cells are usually elevated. Amylase may be mildly elevated (less than fivefold elevation), representing, perhaps, "chemical pancreatitis." Note, however, that patients with gallstone pancreatitis (Chap. 20.3) may have an associated element of ascending cholangitis. Other laboratory data will be appropriate for the patient's degree of hydration and respiratory status, which can deteriorate rapidly if the patient presents late or the diagnosis is delayed.

The RUQ sonogram is the simplest test to confirm the diagnosis. Most of the time, gallstones are seen in the gallbladder (unless the patient has had a prior cholecystectomy). Mild intrahepatic ductal dilatation may be demonstrated, and the common hepatic duct/CBD axis will be variably dilated above a normal level of 7 mm. Ultrasound is notorious for missing around half of bile duct stones, so do not be fooled if you do not see any. If gallstones are not seen in the gallbladder, then the diagnosis of malignant periampullary biliary obstruction must be suspected to justify the performance of a computed tomographic (CT) scan, MRCP, or both or even an endoscopic ultrasound (EUS), depending on the facilities available in your center.

## Treatment

### *Initial Management*

- **Antibiotics.** Initial management comprises appropriate empiric antibiotics with bowel rest and rehydration. Although it has always been felt that antibiotic

selection should be based on the drug's ability to concentrate in the biliary system, recent re-evaluation of this concept has concluded that no antibiotics are able to reach obstructed bile, and that the spectrum of suspected pathogens is a better target for antimicrobial selection. Coverage must be directed against gram-negative, gut-derived organisms (typically *Escherichia coli* and *Klebsiella*). Up to a fifth of bile cultures will grow anaerobic organisms such as *Bacteroides* or *Clostridia* sp., so it is a good idea to include a drug like metronidazole.

- **ERCP.** It is important to recognize that most patients will defervesce within 24 hrs on the above treatment, allowing interventional therapies to be scheduled electively and selectively. A minority of patients will have persistent fever and pain, and their bilirubin may rise, implying a persistent complete obstruction. It is at this time that urgent ERCP is indicated with sphincterotomy and stone extraction. It is the gastroenterologist's task to ensure biliary decompression at the first attempt. This does not mean complete duct clearance as stones may be difficult to extract at one session, but it may mean that the placement of a plastic biliary stent or nasobiliary tube is necessary. The latter's advantage is that it can be removed without re-endoscopy after cholecystectomy. If ERCP fails in the critically ill cholangitis patient, there is another nonoperative alternative: ultrasound-guided percutaneous drainage of the obstructed ductal system by the radiologist.

### *Surgical Strategies*

If the patient is one of the majority who settle with initial conservative measures, then one can elect to perform one of the following semielective procedures, based on local expertise:

- Preoperative ERCP with common duct clearance, followed by laparoscopic cholecystectomy.
- ERCP with common duct clearance alone, leaving the gallbladder in situ. This is indicated in the very high-risk patient; on follow-up, most patients so treated never require a cholecystectomy.
- Laparoscopic cholecystectomy with laparoscopic CBD exploration (with or without a choledochoduodenostomy).
- Open cholecystectomy with CBD exploration.

In most hospitals, preoperative ERCP is selected if it is available. It is diagnostic if periampullary carcinoma is suspected. If it is unsuccessful and the papilla cannot be cannulated, then the surgeon knows preoperatively that clearance of the biliary tree at operation must be ensured (or the duct bypassed). In most large centers, biliary and pancreatic anatomical imaging is mostly noninvasive these days. The role for diagnostic ERCP is shrinking, and fast.

### *Primary Emergency Surgical Treatment*

We have encountered another subset of patients who present with rapid clinical deterioration and may even develop diffuse signs suggesting gallbladder perforation. It is this group who probably benefits from expeditious surgery following resuscitation. The case is made more compelling if they have had a prior gastrectomy that prevents rapid cannulation for ERCP.

Another option is percutaneous decompression of the biliary system. Percutaneous US or CT-guided cholecystostomy may do the job if the gallbladder is in situ, dilated, and communicating with the CBD through a patent cystic duct. [Percutaneous transhepatic drainage is another option, which may be safer, in our opinion, than emergency definitive surgery in these severely sick patients—The Editors.]

## Conclusions

Acute cholangitis is best managed by a concordant multidisciplinary team that understands when appropriate interventions are needed. Since the introduction of endoscopic management of bile duct stones, surgery is seldom required as an emergency. Removal of the gallbladder and clearance of the bile duct of all stones are the two goals of treatment. In the absence of stones, suspect periampullary carcinoma. When the patient is toxic and ERCP fails or is not immediately available, do not procrastinate, waiting for "re-ERCP tomorrow"; rather, operate and drain the obstructed biliary system.

In ascending cholangitis consider the CBD an abscess.

20.3

# Management of CBD Stones in Acute Biliary Pancreatitis

B. Ramana

You can read about acute pancreatitis in general in Chap. 19. Here, the focus is on the approach to patients with **gallstone pancreatitis**.

You should suspect gallstone pancreatitis in patients who present with acute pancreatitis (Chap. 19) and are found (on ultrasound) to harbor stones in the gallbladder. Suspect it also in nonalcoholic patients even if stones are not visualized as occasionally "idiopathic acute pancreatitis" is caused by tiny gallbladder stones or sludge (microlithiasis).

Commonly, in addition to the elevated pancreatic enzymes, there is some degree of chemical liver dysfunction (similar to that described for patients with ascending cholangitis). It is believed that biliary pancreatitis is caused by small stones dropping into the common bile duct (CBD) from the gallbladder and migrating distally through the papilla. More than 30 years ago, Dr. John Acosta established his name in the hall of fame of surgery by sifting through the feces of patients with suspected gallstone pancreatitis, finding small stones in their feces within 10 days of their admission. In those patients who underwent a laparotomy within 48 hrs, impacted stones in the papilla were found in more than two-thirds of individuals (and the morbidity and mortality [M & M] was high); in those who underwent a delayed operation, no impacted stones were found, and the M & M was minimal. From Acosta (and the other stool strainers who duplicated his findings and added more information), we learned:

- The vast majority of the CBD stones responsible for pancreatitis pass spontaneously.
- Most of the so-called impacted stones will pass into the duodenum if you wait long enough.
- In most such patients, preoperative ERCP is negative for bile duct stones.
- In most such patients, the (intraoperative) cholangiogram during laparoscopic cholecystectomy (LC) is normal.
- Sifting through patients' feces may change your life and make you famous.

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This has taught us how to **manage** these patients:

Start conservative treatment as described in Chap. 19. In most patients, resolution of the clinical features of pancreatitis occurs within a few days and is marked by normalization of white cell count and pancreatic and liver enzymes. It is then—within a week or so—that you want to go ahead with cholecystectomy—preventing recurrent biliary pancreatitis by removing the source of the problem. There is no need to wait longer; once signs of pancreatic inflammation have subsided and chemical cholestasis is improving, you can safely go ahead with surgery. The aim should be to perform cholecystectomy during the same hospital admission as the episode of acute pancreatitis.

**What about "suspected" CBD stones?** How can you be sure that they have indeed migrated into the duodenum?

- **If the CBD is not dilated on ultrasound (US) and liver enzymes are back to normal**, there is no need for any preoperative imaging of the CBD. Adding routine intraoperative cholangiogram in this situation is controversial. Cholangiography may indeed demonstrate small stones, but stones that would pass spontaneously in most instances.
- **If the CBD is dilated and liver function is deteriorating**, you have to suspect impacted CBD stones (often associated with cholangitis). An urgent therapeutic ERCP may be needed and if successful followed a day or so later by LC. Whether an early endoscopic sphincterotomy to remove an impacted stone is beneficial in aborting the episode of acute pancreatitis is controversial. Some claim it is—if performed early enough (within a few hours…)—but try to find a center where patients undergo ERCP within a few hours after their pains have started.
- **Nowadays** magnetic resonance cholangiopancreatography (MRCP) is a good option to select which patients need to undergo invasive ERCP before cholecystectomy. If normal, you can proceed with LC.

**What do you do with patients with complicated acute pancreatitis?** You surely do not want to operate on them. Treat conservatively as described in Chap. 19. Delay the cholecystectomy until pancreatitis and its complications are resolved.

**What do you do with patients who are not fit for LC?** Clearly, you do not have to rush with LC in medically unfit patients. Let them recuperate from the acute disease and try to improve their general condition before proceeding with cholecystectomy. Do note, however, that some patients may suffer recurrent acute pancreatitis during the waiting period. Another option (as in high-risk patients with cholangitis) is ERCP with endoscopic sphincterotomy, leaving the gallbladder in situ. Now, the stones can enter the CBD and rapidly fall into the duodenum without producing pancreatitis. This is a viable option on the very old, frail, and medically unfit, and it has been shown to reduce the risk of recurrent AP.

**In conclusion**, in most patients let the pancreatic inflammation subside, wait for the CBD stones to pass spontaneously, and then remove the gallbladder. Some patients need bile duct imaging and possibly ERCP and sphincterotomy. In a few patients, you will have to wait longer for the acute pancreatitis to resolve.

## Common Bile Duct Injury Diagnosed During Laparoscopic Cholecystectomy

It is well said that a bile duct injury is usually the result of a three-part combo (like a Mac, Coke, and fries): an easy case, abnormal anatomy, and an overconfident surgeon—often showing off his or her speed or in a hurry. Do remember this all your life and spare everyone the rigors of a bile duct injury. In case you are unfortunate enough to have actually done it, and you have realized it, what then?

- Take a deep breath, relax, and accept it. You have done the deed, so do your best to redeem yourself. The best thing is to avoid denial, something that leads to a missed diagnosis and kills many. You may find the theater staff or the anesthetist looking at you like it is all your fault. Whatever happens, never panic: it is bad for your heart—and for the patient's hepatic duct or whatever is left of it.
- Call for help if you do not have experience in managing such injuries. We hope of course that you do not have too much experience.
- Discuss and decide with your buddies about the next step: leave a drain, close, and ship a patient to the ivory tower institution or *convert* to laparotomy and address the damage.
- Assess whether you have a partial or total injury. This would mean the difference between doing a hepaticojejunostomy versus a simple suture repair of the bile duct, often over a T-tube. Also assess if you have injured anything else, including the hepatic artery, right duct, and so on.
- A full-blown excision-transection injury needs a hepaticojejunostomy, an operation that is not very forgiving. Restenosis and biliary cirrhosis are not uncommon when repair is done by untrained or unskilled hands. Moreover, there is the issue of ischemia of a duct that has been ruthlessly uprooted from its bed. Therefore, if you cannot, do not even try.
- We repeat: if you are going to ship the patient out, you should drain the right subhepatic space. You can ligate the duct off or intubate it (to prevent biliary peritonitis before the resurgery), but there is a risk of reducing bile duct length, which may adversely affect prognosis, converting a not-so-high "Bismuth 2" injury into a higher "Bismuth 3" one. (Henri Bismuth is a contemporary French surgeon.)

- It has been assumed that you would have opened up the patient by now: it would be very thrilling and dangerous indeed if someone tries to do a hepaticojejunostomy laparoscopically, and there are people who have done just about everything laparoscopically. Do not be tempted.
- Counsel the patient and the relatives extensively, showing sympathy and concern. Do not appear to have lost your confidence but never be cocky, saying things like, "Oh, you will be cured of your problems in no time."
- If you have shipped the patient to an ivory tower institution, keep in touch with the treating surgical team. You never look good when running away from your own complications.
- Keep the number of your lawyer in your mobile speed dial list; you may need it sooner rather than later.

# 21 Small Bowel Obstruction

Moshe Schein

*It is less dangerous to leap from the Clifton Suspension Bridge than to suffer from acute intestinal obstruction and decline operation.* (Fredrick Treves, 1853–1923)

By far, the most common causes of small bowel obstruction (SBO) are postoperative adhesions and hernias. Other less-common mechanical etiologies are bolus obstruction (e.g., bezoar), malignant or inflammatory (e.g., Crohn's disease) causes, or intussusception. Hernias causing SBO are discussed in Chap. 22; early postoperative small bowel obstruction (EPSBO) and paralytic ileus are discussed in Chap. 48. SBO developing in the aftermath of bariatric abdominal surgery is discussed in Chap. 31. Mention is made here of SBO in the virgin abdomen, intussusception, the cancer patient, radiation enteritis, and gallstone ileus. Peritoneal tuberculosis as a cause of SBO is mentioned in Chap. 37.1. The bulk of this chapter is, however, devoted to **adhesive SBO**.

Sir William Osler (1849–1919) used to say that "intestinal adhesions are the refuge of the diagnostically destitute," but the truth of the matter is that iatrogenic—surgeon-made—adhesions are responsible for more than two-thirds of episodes of obstruction, whatever the exact mechanisms are. Please note that in this era of laparoscopic surgery some patients may not volunteer a history of previous surgery, and the abdominal scar is often almost invisible when the previous operation has been, say, something as banal as laparoscopic tubal ligation. Banal—yes, but it could have left a single "band" adhesion causing complete SBO. Remember also that upper abdominal, supracolic, procedures are much less likely to be associated with small bowel adhesions than the infracolic ones. Finally, as you are not an internist, we scarcely need remind you that adhesions almost never (never say never in surgery) cause colonic obstruction.

## The Dilemma

The majority of patients with adhesive SBO (at least half of them, if not more) respond to conservative (nonoperative) treatment. But, persevering with conservative management in SBO may delay the recognition of compromised

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(strangulated) bowel, leading to a poor outcome. Clearly, your challenge is to resolve the following issues:

- Which patients need an urgent laparotomy for impending or established bowel strangulation? And when is initial, conservative treatment appropriate and safe?
- Once instituted, how long should conservative treatment be continued before an operation is deemed necessary? In other words, how do you avoid an operation without risking intestinal compromise?

All surgeons acknowledge that symptoms and signs suggesting that the bowel may be compromised call for an immediate operation. However, surgeons across the world tend to offer a wide range of opinions regarding the duration of nonoperative therapy before declaring that it failed. Some still preach the outdated dictum "Never let the sun set or rise over intestinal obstruction," while others persist in avoiding an operation seemingly forever.

We aim to provide you with guidelines to answer these questions and help you develop a commonsense approach. First, we need to clarify some terminology.

## Definitions

- **"Simple" obstruction:** the bowel is blocked, compressed, or kinked, but its vascular supply is not threatened.
- **Strangulation-obstruction:** the vascular supply to the segment of obstructed bowel is compromised.
- **Closed-loop obstruction:** a segment of bowel is obstructed at a proximal and distal point. Commonly, the involved bowel is strangulated.

Understanding the terms **partial** versus **complete obstruction** is crucial to the planning of treatment. Some surgeons offer definitions based on symptoms, which are notoriously inaccurate. To us, the best way to distinguish between partial and complete SBO is radiology, starting with the humble *plain abdominal X-ray* (see Chap. 5)

- **Partial obstruction:** there is gas seen in the colon in addition to the small bowel distension with fluid levels.
- **Complete obstruction:** no gas is seen in the colon.

Most episodes of partial SBO will resolve without an operation, while the majority of patients presenting with a complete obstruction will require one.

**Fig. 21.1.** "I suspect this is intestinal obstruction…"

## Clinical Features (› Fig. 21.1)

The three important clinical manifestations of SBO are colicky abdominal pain, vomiting, and abdominal distension. Constipation and absence of flatus are relatively late symptoms of SBO. The pattern of these features depends on the site, cause, and duration of the obstruction. For example, in high obstruction, vomiting is prominent, while pain and distension are absent or mild; as the level of obstruction descends, the crampy pain becomes more marked. In distal SBO, distension is the outstanding symptom, with vomiting appearing later. **Feculent vomiting** is the hallmark of long-standing, distal, complete SBO and is characteristic of massive bacterial overgrowth proximal to the obstruction (*remember:* the main bulk of feces is made of bacteria). It is a poor prognostic sign: the more thick and smelly the nasogastric (NG) aspirate, the less chance there is that the obstruction will resolve spontaneously. **When we see s**t, sorry, feces coming from the NG tube, we start preparing the patient for surgery!**

The essential radiographic features seen on supine and erect abdominal X-rays are gaseous distension of the bowel proximal to the obstruction, presence of fluid levels, and in complete SBO, absence of gas distal to the obstruction. The presence of parallel striations (caused by the valvulae conniventes) running transversely, right across the lumen, is characteristic of distended small bowel. Colonic gas shadows lack this pattern. (See also › Chaps. 4 and 5.)

### *Is There a Strangulation?*

Whether there is a strangulation is crucial. If the answer is "yes," not only is an operation compulsory, but it also needs to be performed promptly. The most important feature of strangulation is *continuous* pain. Signs of peritoneal irritation (guarding, rebound tenderness) may be present, but remember that:

- Dead bowel can be present in a relatively "innocent" abdomen.
- Signs of peritoneal irritation are rarely useful in differentiating "simple" obstruction from strangulation because they may also be found in "simple" SBO when the distension is severe. Dilated loops of intestine are tender; you must surely have seen internists poking aggressively into distended abdomens and diagnosing "peritonitis" in patients suffering from gastroenteritis?

Closed-loop obstruction always equals strangulation. Here, a loop of bowel is twisted (volvulus), and its blood supply is compromised. Plain abdominal X-ray is commonly misleading in this situation. The intestine above the twisted loop may be full of fluid and thus appears opaque; all one sees is a single dilated loop of bowel (but CT would be diagnostic; see below). Patients with this type of obstruction tend to cry out in pain—like a siren.

**Remember**: no isolated clinical feature or laboratory finding can tell you if the intestine is strangulating or dead. Only fools let themselves be guided by isolated lactic acid levels. Do not wait for fever, leukocytosis, or acidosis to diagnose ischemic bowel because when all these systemic signs are present, the intestine is already dead!

Having diagnosed strangulation, you will be congratulated for having expeditiously resuscitated and wheeled your patient to the operating room. Save yourself the embarrassment of explaining, the next day, the presence of the long midline incision to deal with a knuckle of ischemic gut trapped in the groin. Never forget that a common cause of strangulated bowel is an external hernia! The suspicion of strangulation must make you examine, or rather re-examine more carefully, the five external hernial orifices: two inguinal, two femoral, and one umbilical (Chap. 22).

By now, you understand that nothing, nothing can accurately distinguish between simple and strangulating SBO. So, how do you play it safe?

## Management

### *Fluid and Electrolytes*

There is hardly a need to remind you that SBO results in significant losses, or sequestration, of extracellular fluid and electrolytes (into the lumen of the

bowel, within its edematous wall, and as the obstruction progresses, into the peritoneal cavity), which have to be replaced intravenously. The aggressiveness of fluid management and hemodynamic monitoring depends on the condition of the individual patient. The fluid of choice is Ringer's lactate. The charting of urine output in a catheterized patient is the minimal monitoring necessary. Even patients scheduled for urgent laparotomy for strangulation require adequate preoperative resuscitation (Chap. 6). Patients with SBO sometimes have intra-abdominal hypertension (we have seen patients with distal SBO presenting with a full-blown abdominal compartment syndrome), which may falsely raise their cardiac filling pressures (central venous pressure [CVP], wedge). These patients require all the more aggressive fluid administration to maintain adequate cardiac output (Chap. 40).

### *Nasogastric Aspiration*

"My work essentially has been that of plumber of the alimentary canal. I have worked on both ends, but largely in between," wrote Owen H. Wangensteen (1898–1981) of Minneapolis. And indeed, already in the 1930s he had introduced the NG tube as a crucial and indispensable aid in the management of SBO. So, how sad and pathetic it is to find—70 years later—patients admitted from the emergency room with the diagnosis of SBO, with their abdomens distended, their pajamas stained green, and no tube sticking from the nose.

A large NG tube (at least 18F diameter) is needed. The NG tube has both therapeutic and diagnostic functions. It controls vomiting, but its main aim is to decompress the dilated stomach and consequently the gut proximal to the obstruction. In a simple obstruction, decompression of the bowel results in rapid pain relief and alleviates the distension. Essentially, the segment of intestine *proximal* to the obstruction and *distal* to the gastroesophageal junction behaves like a closed loop; decompression of the stomach with an NG tube converts it to a simple obstruction. In strangulation or closed-loop obstruction, the pain persists despite NG aspiration.

Insertion of an NG tube is extremely unpleasant. Many patients remember it as the most horrendous experience of their hospital stay (and would certainly resist fiercely any attempt at reinsertion). The procedure can, however, be made much "kinder": soften the rigid tube by immersion for a minute or two in very hot water, spray the nostril of the patient with a local anesthetic, and lubricate the tube. There is no advantage in connecting the NG tube to a suction apparatus; drainage by gravity is as effective. Long nasointestinal tubes (Cantor, Linton, Moss, whatever some of the names) are a gimmick with unproven benefits—requiring cumbersome manipulations and causing delay when operation is necessary.

### *When to Operate?*

An hour or two of fluid replenishment is compulsory in the management of every patient. Reassess your resuscitated and NG-decompressed patient. What is the pattern of pain now? Is there improvement on abdominal re-examination?

**Immediate operation** is required in a minority of patients: those who did not improve, those who experience continuous pain, or those with significant abdominal tenderness combined with the features stated (e.g., fecal NG aspirate, systemic inflammatory response syndrome [SIRS]). Here, abdominal X-rays usually show a complete obstruction. The probability of strangulation is high. Book these patients for an emergency operation.

**An initial nonoperative approach** is often possible because most patients improve at first on the "drip-and-suck" regimen. It would be safe to bet, at this stage, that patients with radiological partial obstruction will eventually escape surgery, whereas those with complete obstruction will eventually visit the operating room. But, how long is it safe to continue with conservative management? Some surgeons would abort the conservative trial at 24 hrs if the patient fails to "open up" because of the nagging concern about strangulation even in a benign-looking abdomen. Others are prepared to persevere, up to 5 days in a carefully monitored patient—especially in patients who give a history of repeated episodes of adhesive SBO.

In the absence of an immediate indication for operation, we favor the use of an oral water-soluble contrast medium (e.g., Gastrografin) as soon as the diagnosis of SBO is made. Gastrografin, a hyperosmolar agent that promotes intestinal "hurry," plays two roles: *diagnostic-prognostic* and *therapeutic*.

### *The Gastrografin "Challenge"*

After the initial gastric decompression, instill 100 ml Gastrografin via the NG tube, which is then clamped. After 4–6 hrs, a simple plain abdominal X-ray is obtained. This is not a formal radiological study under fluoroscopy. Make sure that your patient does not get barium (Chap. 4).

- Presence of contrast in the large bowel proves that the obstruction is partial. In most of these instances, the Gastrografin is very soon passed per rectum as well. In partial SBO, Gastrografin is often therapeutic as it expedites resolution of the obstructing episode. On the other hand, failure of Gastrografin to reach the colon within 6 hrs indicates a complete obstruction. The probability of spontaneous resolution after a failed Gastrografin "challenge" is very low; most of these patients will require surgery anyway, so why not operate on them now!
- Another sign of failed Gastrografin challenge is the failure of Gastrografin to leave the stomach and enter the small bowel. It signifies significant back-pressure in the obstructed bowel and the need for an immediate operation.

So, if we admit a patient during evening hours with suspected adhesive SBO and without features mandating an immediate operation, we perform the Gastrografin challenge, and if by the morning the contrast has not reached the colon, we would operate. Of course, the results of the Gastrografin challenge test should be correlated with the whole clinical picture. Note that Gastrografin may pass across a chronic small bowel narrowing. Thus, for the obstructive episode to be considered "resolved," the abdominal symptoms and signs should disappear as well.

This approach has led us to modify that old fashioned aphorism ("never let the sun rise over an intestinal obstruction"); the new version should read: "Never let a patient with a **complete** intestinal obstruction escape an operation for more than 24 hrs."

### *Additional Investigations (Computed Tomography)*

Clinical examination and plain abdominal radiographs complemented by a Gastrografin challenge are sufficient to allow us to reach the correct decision in the majority of patients. Is additional imaging necessary or useful? *Ultrasonography* has been reported by enthusiasts to define accurately the site of obstruction and establish whether strangulation is present. It requires access to an expert, which most institutions lack. *Oral and intravenous contrast-enhanced computed tomography* (CT) has been shown accurately to define the level of obstruction (the "transition point") and identify a strangulated bowel segment (see Chap. 5). This, however, does not mean that CT is usually necessary and, if obtained, that it has much impact on the decision to wait or to operate in patients with adhesive SBO; you do not need to *see* the transition point to know that it is there. But, should you find yourself working in one of these places where the abdominal CT has replaced the plain abdominal X-ray, see to it that the "oral" contrast used is water soluble—the result being a more detailed and more expensive Gastrografin challenge.

We would, however, obtain CT, selectively, when suspecting a nonadhesive etiology of obstruction, as in the following scenarios:

- History of abdominal malignancy. A CT finding of diffuse carcinomatosis indicates that symptomatic management is the correct option.
- "Virgin" abdomen (discussed in a separate section).
- Clinical picture not consistent with the usual partial adhesive SBO. *Paralytic ileus* may be easily confused with a partial SBO (Chap. 48). There is air in the large bowel, and the Gastrografin may go through, but the patient remains symptomatic; fever or leukocytosis may be present. CT will document the underlying responsible cause for the paralytic ileus, such as acute appendicitis or acute diverticulitis.
- Suspected Crohn's Disease (Chap. 24).
- Early postoperative SBO (Chap. 48).
- Post-laparoscopic SBO (Chap. 48).

But, whether you want it or not, many of your SBO patients would have already passed through the scanner by the time you are summoned to see them. So look for a "transition point" which signifies what the radiologists call "high grade obstruction"—a finding which however does not rule out successful conservative treatment. Search also for the classical CT features of intestinal compromise such as pneumatosis intestinalis and portal venous gas; look for features of "fixed" obstruction (e.g. intussusception, torsion of mesentery); and observe for less specific features associated with intestinal compromise (e.g. free intraperitoneal fluid, mesenteric edema)—which if present make the decision to go to the OR easier. And obviously, a plain abdominal X ray taken a few hours after the CT scanning would show you whether the contrast has progressed into the colon, or whether it has left the stomach at all. Obviously, all these CT features of SBO should be incorporated into the whole clinical picture and decision making.

### *Antibiotics*

In animal models of SBO, systemic antibiotics delay intestinal compromise and decrease mortality. In clinical practice, there is no need for antibiotics in patients treated conservatively, and we operate whenever the suspicion of intestinal compromise is entertained. A single preoperative dose of antibiotics is administered prophylactically; no postoperative antibiotics are necessary even if bowel resection has been performed (› Chaps. 7 and 47). The only indication for postoperative antibiotic administration would be long-standing bowel gangrene with established intra-abdominal infection.

## The Conduct of the Operation

- The incision for abdominal re-entry is discussed in › Chap. 10, but we need to remind you to carefully avoid iatrogenic enterotomies with their associated postoperative morbidity. Finding your way into the peritoneal cavity may take time but be patient for this is the longest part of the procedure. The rest is usually simpler. In this scenario, the gentle hand of the "slow" surgeon is much preferred over that of the macho cowboy.
- Find a loop of collapsed small bowel and follow it proximally. It will lead you to the point of obstruction just distal to the dilated obstructed intestine. Now, deal with the cause of obstruction, be it a simple band or a bowel kink. Mobilize the involved bowel segment using sharp and blunt dissection with traction applied on the two structures to be separated.
- Resect only nonviable bowel or when the obstructed segment is impossible to be freed. Frequently, an ischemic-looking loop of bowel is dusky after being released.

Do not rush to resect; cover the bowel with a warm, wet laparotomy pad and wait patiently; it will usually pink up within 10 min. If not, it requires resection.

- Concentrate on the loop that is responsible for the obstruction; there is no need to free the whole intestine by dividing all the remaining innocent adhesions. This maneuver may be cosmetically appealing, but adhesions lysed today will re-form tomorrow. As aptly stated by Timothy Fabian: "Lysis of all small bowel adhesions is not required because I believe that the bowel is 'locked in the open position' by these chronic adhesions."
- Occasionally, multiple points of obstruction appear to be present with no clear area of demarcation between dilated and collapsed bowel. This is more common in patients after multiple operations for SBO or those with early postoperative SBO. In this situation, the whole length of the "frozen" gut has to be unraveled—again, very carefully and patiently in order not to damage the bowel. This is tedious surgery indeed.

### *How Is an Iatrogenic Intestinal Injury Managed During Adhesiolysis?*

To manage an iatrogenic intestinal injury during adhesiolysis, transmural enterotomies should be repaired transversely. We recommend a running, one-layered, absorbable, monofilament technique (Chap. 13). Superficial serosal tears should be left alone. Areas where the mucosa pouts through the defect should be repaired with a running monofilament seromuscular suture.

### *Decompress or Not?*

Ah yes. The proverbial double-edged sword! On the one hand, excessive bowel distension impedes abdominal closure and contributes to postoperative intra-abdominal hypertension with its well-known deleterious physiological consequences (Chap. 40). On the other hand, bowel decompression may contribute to postoperative ileus and even cause peritoneal contamination. We, like most others, would decompress the distended bowel if abdominal closure seems to need excessive tension. Gently milk its contents toward the stomach, from where it is sucked, through the NG tube, by the anesthetist. Milk the bowel very gently by successively squeezing the loops in a sequential manner as the obstructed bowel is thin walled and very easily injured. The practice of "stripping" the gut between your fingers is brutal and potentially damaging. Do not pull too hard on the mesentery; it may tear as well (remember that injury to the peritoneal surfaces promotes formation of adhesions). Palpate the stomach from time to time; if full, gently squeeze and shake it to restore patency of the NG tube. For a distal SBO, you may also milk the small bowel contents toward the collapsed colon. Open decompression through an enterotomy is unwise given the risk of gross bacterial contamination. Needle decompression is not effective with the thick bowel contents.

Obviously, open decompression should be performed if bowel is being resected; insert a *Poole* sucker or a large sump drain connected to the suction through the proximal line of bowel transection and gently "accordion" the bowel onto your suction device.

Before closing, run the bowel again for missed enterotomies. Check for hemostasis as extensive adhesiolysis leaves large, oozing, raw areas; intraperitoneal blood promotes ileus, infection, and more adhesion formation. Close the abdomen safely (Chap. 43). SBO is a setup for wound dehiscence and a ticket to the M & M conference (Chap. 59).

## Laparoscopic Approach

Wouldn't it be nice to relieve the SBO laparoscopically? Indeed, laparoscopic lysis of the obstructing adhesions seems attractive because in many cases the cause of SBO is a single fibrous band. This is easier said than done. The collective published experience (and that which is not published, which is more realistic) points to a higher risk of injury to the distended and friable obstructed intestine during the laparoscopic operation. This, of course, translates to a higher rate of septic complications and postoperative morbidity.

Should you wish to attempt laparoscopic approach, do it *selectively* on the easier cases:

- First episode of SBO
- Abdomen not excessively distended (e.g., more proximal SBO)
- Patient stable and able to endure a prolonged pneumoperitoneum—superimposed on an already distended abdomen

The first port should be placed through an open approach and away from the old incision. Most important, do not be obstinate; know when to abort—before you create too many holes.

## Special Circumstances

### *The "Virgin" Abdomen*

Patients presenting with SBO but without a previous history of abdominal surgery need special attention; it is here that you have to suspect nonadhesive causes of SBO, including rare "zebras" like, for example, the one and only obstructing *obturator hernia* you are likely to diagnose and treat during your entire glorious surgical career.

So, the patient presents with clinical and radiological features of SBO but with no abdominal wall scar of previous surgery. What do you do? (First, ask again

about all past procedures, including that laparoscopic ovarian cystectomy and a tiny scar hidden in the umbilicus; while you are at it, why not re-examine the groin for incarcerated hernias.) Evidence of a complete obstruction is of course an indication for a laparotomy, but what about partial SBO? As with the adhesive partial obstruction, we recommend a Gastrografin challenge. In an obstruction caused by an intraluminal bolus, whether from parasites or dry fruits, Gastrografin may disimpact the bowel. In these cases, we would recommend abdominal imaging to exclude an underlying cause. Non-resolving partial obstruction despite the Gastrografin challenge suggests a mechanical cause, such as a congenital band, an internal hernia, malignancy, inflammation, or even an impacted bezoar. Laparotomy usually uncovers a treatable cause of obstruction. A preoperative CT scan "just to find out what we are dealing with" is not mandatory and may only delay the operation without changing its indication. But when in doubt, if readily available, and in the absence of clinical strangulation, it may be helpful. *Cecal carcinoma* is a typical cause of distal "SBO" in the virgin (or non-virgin) abdomen. The clinical presentation is commonly gradual and "smoldering." Gastrografin may pass through into the cecum. In this case, CT would be diagnostic. SBO due to previously undiagnosed but suspected *Crohn's disease* is an exception; here, a CT may be very suggestive, indicating continued conservative therapy (Chap. 24).

### *Intussusception*

Although common in pediatric patients (Chap. 35), intussusception is a very rare cause of SBO in adults. In adults, the "leading point" is usually organic (e.g., neoplasm, inflammatory lesions) and seldom *idiopathic* as in children. Patients with small bowel or ileocolic intussusception present with nonspecific features of SBO (in a virgin abdomen), necessitating operative treatment. A specific preoperative diagnosis can be obtained with ultrasound or CT, showing the *multiple concentric ring sign* (bowel within bowel) but will not change what you need to do—operate and resect the involved segment of bowel. Although controversial, some would attempt reduction of intussusception when there are no external signs of ischemia or malignancy, and if after reduction no leading point is found (i.e., idiopathic intussusception), one could leave the bowel alone.

### *The Known Cancer Patient*

A patient is admitted with SBO a year or two following an operation for gastric or colonic cancer. You should first attempt to obtain information about the findings of the previous laparotomy. The more advanced the cancer, the higher the probability that the current obstruction is malignant. Clinically, cachexia, ascites, or an abdominal mass suggests diffuse carcinomatosis. These cases present a medical

and ethical dilemma. On the one hand, one wishes to relieve the obstruction and offer the patient a further spell of quality life. On the other hand, one tries to spare a terminal patient an unnecessary operation. Each case should be assessed on merit. In the absence of stigmata of advanced disease, surgery for complete obstruction is justifiable. In many instances, adhesions may be found; in others, a bowel segment obstructed by local spread or metastases can be bypassed. When diffuse carcinomatosis is suspected clinically or on CT scan, a reasonable option would be to insert a palliative, venting percutaneous gastrostomy, allowing the patient to drink and to die peacefully at home or in a hospice environment.

### *Radiation Enteritis*

Radiation treatment of abdominal or pelvic malignancies is not an uncommon cause of SBO; this usually develops months or even years after irradiation. A relentless course of multiple episodes of partial SBO, initially responding to conservative treatment but eventually culminating in a complete obstruction, is characteristic. There is also the uncertainty about the obstruction being malignant or adhesive in nature. One always hopes that it is adhesive because SBO due to radiation injury is "bad news" indeed. When forced to operate for complete obstruction, one finds irradiated loops of bowel glued or welded together and onto adjacent structures. The paper-thin bowel tears easily. Accidental enterotomies are frequent, difficult to repair, and commonly result in postoperative fistulas. Short involved segments of bowel are best resected, but when longer segments are encountered, usually stuck in the pelvis, it is safest to bail out with an enteroenteric or enterocolic bypass, using nonirradiated bowel for this purpose. Postoperative short-bowel syndrome is common whatever the procedure. Long-term prognosis is poor; radiation enteritis is almost as bad as the malignancy the radiation had attempted to control (see also Chap. 48).

### *Recurrent Multiple Episodes of SBO*

In recurrent multiple episodes of SBO, the patient is typically re-admitted every second month for SBO and has undergone, in the past, multiple operations for this condition. How should this patient be managed? We would treat this patient as any other patient presenting with adhesive SBO. Fortunately, most such episodes are "partial" and responsive to conservative treatment. When complete obstruction develops, operative management is obviously necessary. Attempts at preventing subsequent episodes with plication of bowel or mesentery or long-tube stenting are recommended by some. The evidence in favor of such maneuvers is

anecdotal at best. We do not practice them. Occasionally, a patient develops obstruction early in the aftermath of an operation for adhesive SBO; this is a case par excellence for prolonged nonoperative management, with the patient maintained on total parenteral nutrition (TPN) until adhesions mature and the obstruction resolves. (See also Chap. 48.)

### *A Word About Patience*

You will understand by now that in some circumstances a laparotomy for SBO will be a long and difficult operation due to multiple adhesions or radiation enteritis, for example. If you begin an operation expecting a quick-and-easy procedure and are then confronted by a nightmare abdomen, the first thing you must do is reset your mental clock. Failure to do this may mean that you will attempt to rush the procedure, and this inevitably leads to disaster, with multiple inadvertent enterotomies, peritoneal contamination, and ultimately an even longer and more dangerous procedure. Upon entering such a disastrous abdomen unexpectedly, inform everybody immediately that the procedure is now going to take a few hours while you unravel all the loops necessary to get at the problem and fix it. And then, take your time and fix it carefully and slowly.

### *Gallstone Ileus*

Gallstone ileus develops typically in elderly patients with long-standing cholelithiasis. It is caused by a large gallstone eroding into an adjacent segment of bowel—usually the duodenum; then, the gallstone migrates distally until stranded at the narrow ileum. Presentation is usually vague as initially the stone may disimpact spontaneously, causing intermittent episodes of partial obstruction. You will never miss the diagnosis once you habitually and obsessively search for air in the bile ducts on any plain abdominal X-ray you order (Chap. 5). The air enters the bile duct via the enterocholecystic fistula created by the eroding gallstone. Treatment is operative and should be tailored to the condition of the patient. In frail and sick patients, deal only with the SBO: place an enterotomy *proximal* to the stone and remove it and search for additional stones in the bowel above; you do not want to have to re-operate. In patients who are younger and reasonably fit and well, you may want also to deal with the cause of the problem—the gallbladder. Perform a cholecystectomy and close the duodenal defect; place your suture line transversely to avoid narrowing of the duodenum. But again, *not* removing the gallbladder after dealing with the obstructing gallstone is a perfectly reasonable option.

### *Bezoars*

Bezoars are tightly packed collections, or "balls," of partially digested or undigested material forming in the stomach and then migrating distally, where they may obstruct the terminal ileum. You may encounter one of the following types of bezoars:

- **Phytobezoars:** partially digested agglomerations of vegetables or fruits that form in patients with altered gastric physiology (e.g., following gastric resection, vagotomy, or bariatric operation and even in patients with diabetic gastroparesis) or health food "crazies" and elderly forget-to-chewers. Many sorts of fruits and vegetables are implicated, particularly when consumed in large quantity (I once suffered partial SBO after consuming, within an hour, a whole bag of baby carrots), but consumption of *persimmons* is especially notorious in this regard, with patients developing multiple episodes of SBO.
- **Trichobezoars:** these most commonly occur in younger patients with psychiatric disturbances who chew and swallow their own hair. Trichobezoars form in the stomach and often reach a huge size; they break into smaller pieces and migrate into where they can obstruct at several points.
- **Parasitic bezoars:** consisting of conglomerates of parasites such as *Ascaris lumbricoides,* these may obstruct the distal ileum. Obviously, these are common in endemic areas.

Patients present usually with features of partial or smoldering SBO and a virgin abdomen. History is suggestive, and CT images—showing the actual intraluminal bezoars—are diagnostic. As mentioned, Gastrografin challenge can dislodge the obstructing parasites, or other types of bezoars, pushing them into the cecum. But when the obstruction is complete, you have to operate and deal with the obstructing bezoar like you did with the gallstone (see the section on gallstone ileus). It is crucial to palpate the entire small bowel, including the duodenum (and the stomach), for additional bezoars and remove all of them. Preoperative CT may be helpful in mapping such additional bezoars for you. You do not want the patient to develop early postoperative SBO caused by a missed bezoar—needing another laparotomy for removal—do you?

### *SBO After Gastrectomy*

With the disappearance of gastrectomies performed for benign disease and the declining rate of gastric cancer, there are not too many postgastrectomy patients to present with SBO, but some do. According to my friends Professor David Dent (Cape Town, South Africa) and Dr. Hernan Diaz

(Santiago, Chile)—both of them "old gastrectomists"—the reasons for SBO in these patients are:

- Simple adhesive obstruction—what is common is common
- Recurrent gastric carcinoma, with loops of bowel "frozen" by peritoneal carcinomatosis
- Bolus obstruction by bezoars
- Internal herniation of small bowel through defects of the mesocolon or behind the jejunal loop forming the Billroth II (or Roux-en-Y) gastroenterosotomy—be it antecolic or retrocolic
- Twisting or volvulus of redundant afferent or efferent jejunal loops

Obviously, the more complex the original postgastrectomy reconstruction, the more potential peritoneal defects created, and the "looser" the various intestinal loops, the higher the risk will be for bowel to kink, rotate, herniate, and obstruct. (Now you understand why we prefer Billroth I reconstruction after gastrectomy; Chap. 17.)

- Another specific type of obstruction is the jejunogastric intussusception. Both the afferent or efferent loops can invaginate into the gastric remnant, but the *retrograde* efferent loop intussusception is more common. This can occur from a few days up to many years after the gastrectomy. Sudden onset of epigastric pain, vomiting, and hematemesis and a palpable epigastric mass in a patient with previous gastric surgery are the classic triad.
- Obstruction of the afferent loop after Billroth II or Roux-en-Y reconstruction—by whichever of the mentioned mechanisms—produces a closed-loop obstruction (between the obstructing point and the duodenal stump). High intraluminal pressures are commonly associated with elevation of serum pancreatic enzymes (amylase) and, if the obstruction is not relieved, with necrosis of the involved loop and the attached duodenum. The clinical picture of epigastric pain, upper abdominal mass, and hyperamylasemia may confuse you to think that you are dealing with acute pancreatitis.

The proximal location of the obstruction is suggested by the frequent vomiting, lack of abdominal distention, and paucity of dilated small bowel on plain abdominal X-ray. CT with oral contrast is a superb diagnostic aid, showing the exact anatomy of obstruction and the ring sign of small bowel within the stomach in the case of jejunogastric intussusception. Occasionally, endoscopy is needed to clarify the picture. Do understand that acute afferent loop obstruction is a dire emergency; you have to operate before the closed-loop obstruction results in complete necrosis of the duodenum!

At operation, the anatomy has to be restored, and this entails resection of nonviable loops of bowel and reconstruction of the upper gastrointestinal tract, as you would do after partial or total gastrectomy.

### *Small Bowel Volvulus*

Small bowel volvulus is also called **midgut volvulus**, distinguishing it from *foregut volvulus* (Chap. 16) and *hindgut volvulus* (Chap. 25).

Volvulus, the "twisting strangulation" of an intestinal segment around an axis formed by a band or an adhesion, is a common occurrence in adhesive SBO. A narrow-based loop of small bowel suspended by a Meckel diverticulum can also undergo torsion. But, what about "spontaneous" volvulus, one that involves the entire, or almost entire, small intestine?

**Spontaneous volvulus of the small bowel**, while very rare in the "developed world," is not uncommon in rural areas of the Indian subcontinent, central Asia, and Africa. It seems more common in healthy farmers returning home for a large evening meal or, in Moslem countries, during the fast of Ramadan—when large meals are consumed at night after the day of fasting. The common pathway appears to be a huge load of high-fiber, indigestible food, arriving suddenly in an empty small bowel. The sudden distention creates rotational kinking forces. At operation, typically the twisted bowel is loaded with liters of claylike undigested food and is often suspended on an unusually long mesentery. Occasionally, small bowel volvulus occurs in combination with that of the sigmoid colon, forming the so-called **ileosigmoid knot**, in which the ileum and the sigmoid entangle each other to form a knot and become gangrenous. An arrangement of the small bowel and sigmoid colon on long, narrow mesenteries would appear to be a prerequisite.

Like in any other condition resulting in an acute vascular compromise of the bowel, patients present with severe central abdominal pain that is out of proportion to the abdominal findings; systemic signs of hypovolemia and toxemia are however dramatic and dominant. An urgent operation is indicated, during which the ischemic intestine is managed as discussed above and in Chap. 23.

### *Intestinal Malrotation*

Most cases of **midgut malrotation** present within the first weeks or months of life. The rest can present sporadically throughout childhood and even in adults. The anatomy of malrotation is depicted in Figure 21.2: note how close the D-J flexure (point X) is to the cecum (point Y) and how narrow the base of the mesentery is and thus prone to torsion. Strangulating midgut volvulus in these patients can present acutely, but more commonly, especially in older children and adults, volvulus is preceded by recurring attacks of upper and central abdominal colicky pain and intermittent vomiting of bile and is often relieved by diarrhea. Once again, patients presenting with acute midgut volvulus are in great pain and appear ill but have minimal abdominal findings on examination.

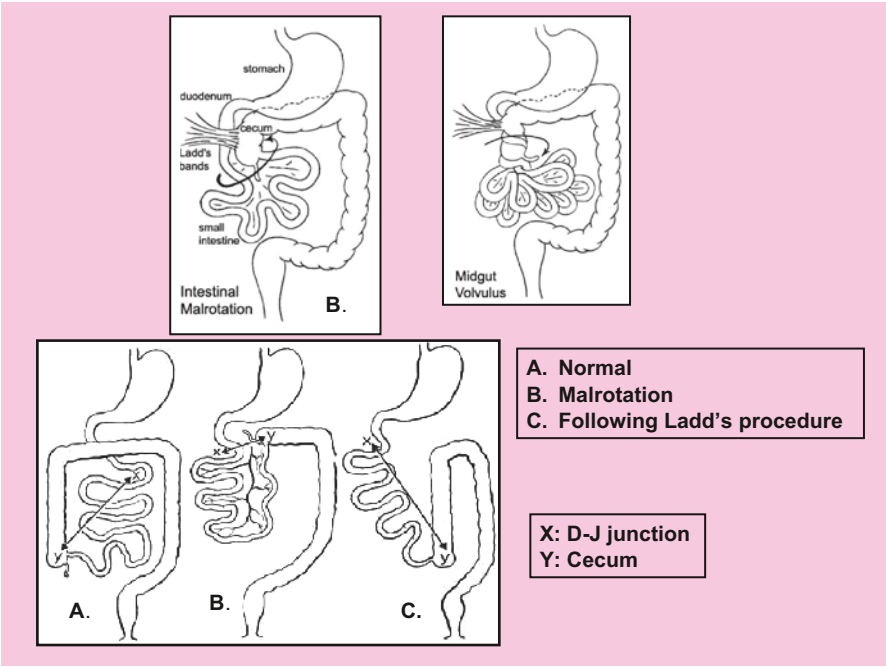


**Fig. 21.2.** Small bowel malrotation and volvulus. (Modified from George G. Youngson, Common Pediatric Disorders. http://www.rcsed.ac.uk/eselect/sig2.htm)

Classically, the diagnosis was achieved by contrast studies: upper gastrointestinal barium examination showing loss of the duodenal C (corkscrew duodenum) and the D-J flexure to the right of the midline. Barium enema would show the cecum riding high under the liver. CT, however, has become the optimal diagnostic modality, showing the small bowel located entirely within the right hemiabdomen and the colon situated on the left. Features of the twisted mesentery and intestinal wall ischemia are seen as well. Midgut volvulus can also be diagnosed on Doppler ultrasound by demonstrating the "whirlpool sign"—wrapping of the superior mesenteric vein and the mesentery around the superior mesenteric artery.

Emergency laparotomy is mandated. Remember that these patients are grossly hypovolemic and need aggressive fluid resuscitation. At operation, detort the twisted bowel, working in a *counterclockwise* rotation. Dead bowel needs resection, usually massive resection. Regarding whether to anastomose and whether second-look operation is necessary, see Chap. 23.

After resecting the dead bowel or convincing yourself that it is viable, you want to address the anatomical pathology of malrotation by doing what has been described by William E. Ladd (1880–1967):

1. Divide the peritoneal folds (Ladd bands) that cross from the cecum to the liver, compressing the duodenum.
2. Mobilize the right colon.
3. Mobilize the D-J flexure, freeing the ligament of Treitz—straightening the duodenal loop.
4. Divide any thick peritoneal folds compressing the SMA.
5. Place the bowel in a new pattern as depicted in Fig. 21.2c; note that now point X is far from point Y.
6. Remove the appendix to prevent "atypically situated" appendicitis.

Obviously, after having to resect most of the small bowel you do not worry about recurrence of the volvulus, and there is no impetus to correct the anatomy, except points 1, 4, and 6.

### Prognosis

Overall, about half the patients presenting with an adhesive SBO can be managed without an operation. About a third of patients operated once for adhesive SBO will have recurrent problems within 30 years. For patients admitted several times for adhesive SBO, the relative risk of recurrence increases with increasing number of prior obstructive episodes; more than two-thirds of patients with four or more SBO admissions will re-obstruct. In addition, the risk of recurrence is a bit lower in patients in whom the last obstructive episode was treated *surgically,* but this does not mean that those patients who were treated conservatively will have an increased need for operation during their future admissions for SBO. The aim is therefore to operate only when necessary but not to delay a necessary operation.

> The only thing predictable about small bowel obstruction is its unpredictability.

# 22 Acute Abdominal Wall Hernias

Paul N. Rogers

*"You can judge the worth of a surgeon by the way he does a hernia."* (Thomas Fairbank, 1876–1961)

## Acute Groin Hernia

In all parts of the world, many more hernias are now repaired electively than was formerly the case. In spite of this, surgeons are frequently confronted by acute groin hernias, and it is important to know how to deal with them.

**A word about terminology:** groin hernias, inguinal or femoral, may be described as reducible, irreducible, incarcerated, strangulated, or obstructed. This terminology can be confusing, and the words, which have come to mean different things to different people, are much less important than the concepts that underlie the recognition and management of acute hernia problems. The important concept to be grasped is that any hernia that becomes painful, inflamed, or tender and is not readily reducible should be regarded as a surgical emergency.

### *Presentation*

Patients may present acutely in one of two ways:

- Symptoms and signs related directly to the hernia itself
- Abdominal symptoms and signs, which at first may not seem to be related to a hernia

The **first mode** of presentation usually means pain and tenderness in a tense, irreducible hernia. A previously reducible hernia may suddenly become irreducible. This problem is usually obvious (› Fig. 22.1).

The **second mode** of presentation will be much more insidious. Beware the vomiting old lady! Treated at home for several days by the primary care physician as a case of gastroenteritis, she eventually comes under the care of the surgeons due to intractable emesis. By this stage, she is dehydrated and in need of

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**Fig. 22.1.** "This must be strangulated, eh?"

much resuscitation. It is surprisingly easy in these circumstances to miss the small femoral hernia barely palpable in the groin, trapping just enough small bowel as is required to produce obstruction. No abdominal symptoms or signs are present, and the plain abdominal radiographs are non-diagnostic. None of these difficulties saves you from the embarrassment of the following morning's round when the hernia is discovered.

Hernias are still one of the most common causes of small bowel obstruction (Chap. 21). A careful search must be made for them in all cases of actual or suspected intestinal obstruction. This may mean meticulous, prolonged, and disagreeable palpation of groins that have not seen the light of day, let alone soap and water, for a long time. In most cases, however, the diagnosis is obvious, with a classical bowel obstruction and a hernia stuck in the scrotum.

Beware the **Richter's hernia**, typical of femoral hernias, in which only a portion of the circumference of the bowel is strangulated. Because the intestinal lumen is not completely blocked, bowel obstruction may not occur, and presentation is consequently delayed and non-specific.

### *Preparation*

Surgery for acute groin hernia problems should be carried out without undue delay, but these patients must not be rushed to surgery without careful assessment and preparation (Chap. 6). As we suggested, some patients may be in need of quite a bit of resuscitation on admission to hospital.

Analgesia is an important part of the management of these patients. Opiate analgesia and bed rest with the foot of the bed slightly elevated may successfully manage a painful obstructed hernia of short duration. Gentle attempts at reduction of such a hernia are justified once the analgesics have taken effect. A successful reduction of the hernia means that emergency surgery at unsociable hours may be traded for a semielective procedure on the next available routine list, a benefit for both patient and surgeon. Note that manual reduction of the incarcerated hernia should be attempted only in the absence of signs of intestinal strangulation; it should be gently performed to avoid "*reduction en masse*"—when the herniated bowel with the constricting ring are reduced together, providing a false sense of achievement and a delay of necessary surgery.

## The Operation

### *Inguinal Hernia*

An inguinal incision is a satisfactory approach. Even if a bowel resection is required, it is possible to deliver sufficient length of intestine through the inguinal canal to carry this out.

The main difference in dissection in an emergency hernia operation compared to an elective procedure is the moment at which the hernial sac is opened. In the emergency situation, the hernia will often reduce spontaneously as soon as the constricting ring is divided. The site of constriction may be the superficial inguinal ring, in which case the hernia reduces when external oblique is opened. It is recommended, therefore, that the sac be opened and the contents grasped for later inspection *before* the constricting tissues are released. If the hernia reduces before the sac contents are inspected, it is important that they are subsequently identified and retrieved so that a loop of non-viable gut is not inadvertently left in the abdomen. Retrieval of reduced sac contents can be an awkward business via the internal ring, and occasionally a formal laparotomy may be required to inspect matters properly. It is for these reasons that great care should be taken to secure the sac contents for inspection as soon as possible during the procedure.

If the hernial sac contains omentum only, then any tissue that is necrotic or of doubtful viability should be excised, ensuring meticulous hemostasis in the process. If, on the other hand, bowel is involved, then any areas of questionable viability should be wrapped in a warm, moist gauze pack and left for a few minutes to recover. Irretrievably ischemic gut should be resected. If there is a small patch of necrosis that does not involve the whole circumference of the bowel, then this can sometimes be dealt with by invagination rather than by resorting to resection. In this situation, the injured bowel wall is invaginated by a seromuscular suture, taking bites on the viable bowel on either side of the defective area of gut.

Occasionally, particularly if a bowel resection has been necessary, oedema of the herniated gut makes its replacement in the abdomen difficult. Manoeuvers such as putting the patient into a marked Trendelenburg position and gently compressing the eviscerated gut, covered by a large, moist gauze swab, will almost invariably allow the bowel to be replaced in the abdomen. It is possible to minimize the chances of this difficulty arising if care is taken during any bowel resection not to have any more gut outside the abdomen than is absolutely necessary. Very rarely the herniated viscera will not return to the abdomen without pulling on it from **within**; in such instances, **La Rocque's manoeuver** may be useful: extend the skin incision up and laterally, then extend the split of the external oblique aponeurosis and follow this with a muscle-splitting incision of internal oblique and transverse muscles above the internal ring. Through this incision you enter the peritoneal cavity and reduce the herniated viscera from within.

The question of the type of hernia repair to be employed is a matter for the individual surgeon, with one proviso. In these days of tension-free hernia repair, it seems imprudent to place large amounts of mesh in the groin if necrotic gut has had to be resected. In this situation some other type of repair seems advisable to obviate the prolonged misery of infected mesh.

### *Femoral Hernia*

You can approach the acute femoral hernia from below the inguinal canal, from above, or through it.

- With the **low approach**, you place the incision below the inguinal ligament, directly over the bulge. You find the hernial sac and open it, making sure to grasp its contents for proper inspection. Strangulated omentum may be excised, and viable bowel is reduced back into the peritoneal cavity through the femoral ring. When the ring is tight, and usually it is, you can stretch it with your small finger, inserted medially to the femoral vein; occasionally, you will have to cut the lower fibres of the overlying inguinal ligament to let your finger enter the femoral canal. You can resect non-viable small bowel through this approach and even anastomose its ends, but pushing the sutured or stapled anastomosis back into the abdomen is like trying to squeeze a tomato into a cocktail glass. Therefore, when bowel has to be resected, it is advisable to do it through a small right (or left) lower quadrant muscle-splitting laparotomy (as for appendectomy).
- Some authorities favour an approach **via the inguinal canal** but we see little merit in this approach, which must disrupt the anatomy of the canal and presumably risk a subsequent inguinal hernia.
- Yet another approach is **McEvedy's**. This involves an approach to the extraperitoneal space along the lateral border of the lower part of rectus abdominis. The skin incision may be vertical, in line with the border of rectus,

or oblique/horizontal. A vertical skin incision has the merit of allowing extension to a point below the inguinal ligament and this may be helpful in reducing stubborn hernias, allowing traction from above and compression from below. Once the space behind the rectus muscle has been accessed the hernia can usually be freed from behind the inguinal ligament. The peritoneum can be opened as widely as necessary to permit inspection of the contents of the hernia sac and to carry out intestinal resection if necessary.

All these approaches are reasonable provided the contents of the hernial sac are examined and dealt with appropriately. As with inguinal hernias the implantation of large amounts of mesh should be avoided in patients who have contamination of the operative field with intestinal contents. With this caveat the choice of repair is not different from what you would do in the elective situation. **Our choice:** in the absence of gross contamination the femoral canal is obliterated with a mesh plug. When gross contamination is present we would "close" the femoral canal by suturing the inguinal ligament, above, to the pectineal fascia, below.

### *Incisional Hernias*

Incisional hernias are common but most are asymptomatic except for the unsightly bulge and discomfort they sometimes produce. It is the small incisional hernias with the tight neck that become acutely symptomatic—incarcerating omentum or intestine.

The presentation is well known to you: an old "silent" hernia or abdominal scar, which has now become painful. When bowel has been incarcerated there may be associated symptoms of small bowel obstruction (Chap. 21). The hernia itself is tense, tender and non-reducible.

It is important to distinguish between intestinal obstruction caused by the incisional hernia or simply associated with it. The latter situation, which is not uncommon, implies that the patient suffers small bowel obstruction due to adhesions, for example, and the obstructed and distended loops of bowel invade the long-standing incisional hernia. On examination, the bowel-filled tender hernia may mimic incarceration. It is for this reason that the contents of any hernia associated with obstruction must be examined carefully at operation to ensure that the hernia truly is the cause of the obstruction. (This applies to all kinds of hernias. We recall a case of obstruction that was addressed by reducing and repairing a tense femoral hernia, only for the obturator hernia, which was the true cause of the obstruction, to be discovered at laparotomy many days later when the patient failed to recover from the first operation.)

Any "acute" incisional hernia is a surgical emergency. This is also true with other types of abdominal wall hernias, such as paraumbilical or epigastric ones.

It should be noted, however, that epigastric hernias rarely, if ever, cause trouble. They contain only extraperitoneal fat from the falciform ligament and for this reason need not be repaired routinely in the absence of symptoms. Also the acutely incarcerated *umbilical hernia* is extremely unlikely to involve intestine. At operation the hernial sac has to be entered to evaluate the incarcerated contents, which are to be reduced or resected depending on the findings. And **the surgical findings should explain the clinical presentation**. For example, if you do not find strangulated omentum or bowel in the sac, you have to retrieve the whole length of the intestine in search for distal small bowel obstruction. If you find pus within the sac you have to look for the source. We have seen patients operated on for a "strangulated incisional hernia" when the underlying diagnosis was perforated appendicitis. We have operated for "strangulated femoral hernia" to find the hernia sac full of pus originating from a tuboovarian abscess.

After the contents of the hernia have been dealt with, identify the fascial margins of the defect. Use your conventional "best" repair but do not forget that placing a mesh in a contaminated field is potentially problematic. Not everybody agrees with such dogma and there are those who do report "reasonable" results with implantation of non-absorbable mesh in acute situations and even in grossly contaminated fields—after resection of bowel. A few **caveats** follow should you plan using synthetic mesh:

- In contaminated fields use polypropylene (e.g., Marlex or one of the new, lighter types of mesh) which is relatively resistant to infection rather then PTFE (polytetrafluoroethylene) (Gortex), which is not. Infected Marlex grafts are often salvageable while infected Gortex patches always have to be removed.
- Bear in mind also that leaving non-absorbable mesh in contact with the gut leads to difficulties and disasters later. Mesh repair of an incisional hernia should always aim to place the prosthetic material outside the peritoneum or, ideally, in the pre-peritoneal-retromuscular position. At the very least omentum should be placed between any unavoidable intraperitoneal mesh and the viscera. Experience with subsequent laparotomies in patients with intraperitoneal mesh shows that adhesions are much more dense than with extraperitoneal mesh and as a result small bowel resection is often required simply to access the abdominal cavity. And although uncommon we have all seen spontaneous intestinal fistulas developing at the contact point with the mesh. The manufacturers of the "dual"-type mesh (smooth on the inside, porous on the outside) claim that their products are safe for intraperitoneal use; however, injury to bowel has been observed also with such types of mesh.
- A relatively newly available product to repair abdominal wall hernias in contaminated fields is one of the biomaterials that, although resistant to infection, is prone to late formation of "weaknesses" and "bulges" of the abdominal wall.

In a critically ill patient, when the repair is deemed complex or is judged to increase the intra-abdominal pressure significantly, we would simply close the

skin, leaving the patient with a large incisional hernia. Remember: patients do not die from the hernia but from its intestinal complications or a closure that is too tight (› Chaps. 40 and 43).

"Always explore in cases of persistent vomiting if a lump, however small, is found occupying one of the abdominal rings and its nature is uncertain." (Augustus Charles Bernays, 1854–1907)

# 23 Acute Mesenteric Ischemia

Moshe Schein · Paul N. Rogers

*"Vascular surgery is peculiar because, above all, it is mainly surgery of ruins."* (Cid dos Santos)

*"Occlusion of the mesenteric vessels is regarded as one of those conditions of which the diagnosis is impossible, the prognosis hopeless, and the treatment almost useless."* (A. Cokkins, 1921)

Which of you has not been called by the internists or emergency room (ER) docs to the ER or medical floor or the intensive care unit to "rule out mesenteric ischemia" in some elderly patient? As a rule, on such occasions you will find a groaning patient with nonspecific abdominal complaints and a hospital chart that outweighs you. "Rule out mesenteric ischemia"—easier said than done!

Acute mesenteric ischemia usually involves the region supplied by the superior mesenteric artery (SMA). Thus, the small intestine is predominantly affected, but the right colon, which is also supplied by the SMA, can be involved as well. Isolated ischemia of the colon, which is much less common, is discussed separately under the heading of *ischemic colitis* in Chap. 24.

## The Problem

The problem is a sudden reduction in arterial perfusion of the small bowel, which quickly leads to central abdominal pain. If left untreated, the process progressively involves the muscular layer of the intestines, and it is only after some hours, when the serosa is affected, that peritoneal signs appear. In an attempt to simplify matters, let us divide acute arterial mesenteric ischemia (AMI) into three types:

- **Thrombotic**: due to an acute arterial thrombosis, which usually occludes the orifice of the SMA, resulting in massive ischemia of the entire small bowel plus the right colon—the area supplied by the SMA.
- **Embolic**: due to a shower of embolic material originating proximally from the heart (atrial fibrillation, post-myocardial infarction, diseased valve) or an aneurysmal or atherosclerotic aorta. Emboli usually lodge in the proximal SMA, but beyond the exit of the middle colic artery; therefore, as a rule,

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the most proximal segment of proximal small bowel is spared along with the transverse and (probably) the right colon. Emboli tend to fragment and re-embolize distally, producing a patchy type of small bowel ischemia.

- **Nonocclusive:** due to a "low-flow state," in the absence of documented arterial thrombosis or embolus. Note, however, that underlying mesenteric atherosclerosis may be a precipitating or contributory factor. The low-flow state is a product of low cardiac output (e.g., cardiogenic shock), reduced mesenteric flow (e.g., intra-abdominal hypertension), or mesenteric vasoconstriction (e.g., administration of vasopressors); usually, however, it is due to a combination of these factors, developing in the setting of a pre-existing critical illness.

Mesenteric venous thrombosis can also produce small bowel ischemia. The features and management of this entity differ drastically from the other three. It is discussed separately in this chapter.

The problem is that in clinical practice, outside the textbook, mesenteric ischemia is usually recognized when it has already led to intestinal gangrene. At that stage, the Pandora's box of SIRS (systemic inflammatory response syndrome) has been opened, and even removal of the entire gangrenous intestine will not always stop the progression to organ failure and death. Even if such physiologic consequences can be overcome, the patient commonly becomes an "intestinal cripple," suffering from the short-bowel syndrome.

Have we depressed you enough yet?

## Assessing the Problem

Typically, the early clinical picture is **nonspecific: the patient complains of severe abdominal pain—if able to complain at all—and the doctor finds little on physical examination.**

There may have been preceding symptoms of a similar sort of pain developing with meals and accompanied by weight loss, suggesting pre-existing *mesenteric angina*. History or evidence of systemic atherosclerotic vascular disease is almost the rule in patients with mesenteric thrombosis, while a source for emboli, such as atrial fibrillation, is usually present in patients with mesenteric embolism. Patients with low-flow state are commonly moribund due to underlying critical disease.

Nausea, vomiting, diarrhea, and hematochezia come late, if ever. You must resist the natural temptation to ascribe patients' nonspecific symptoms to some other benign condition, such as gastroenteritis, unless the associated history and symptoms for the alternate explanation are fully present. And by the way—in the

elderly the diagnosis of "acute gastroenteritis" is rarely the final diagnosis unless it was the wrong diagnosis.

**Physical examination** in the early stages of the process is treacherously benign; peritoneal irritation appears too late, when the bowel is already dead.

**Plain abdominal X-rays** early in the course of the illness are normal. Later, there may be a pattern of adynamic ileus, with visible loops of small bowel and fluid levels but with gas and feces seen within the normal colon and rectum. Likewise, **laboratory studies** usually are normal until the intestine loses viability; only then do leukocytosis, hyperamylasemia, and lactic acidosis develop.

The bottom line is that initially in acute mesenteric ischemia the physical examination and all commonly available X-rays and blood tests may be normal. At this stage, entertaining the diagnosis of mesenteric ischemia, you have two options: the first is to enter in the chart "abdominal examination normal; mesenteric ischemia cannot be ruled out; will reassess later." The second option is to order a computed tomographic (CT) scan, which has replaced mesenteric angiography as the initial, screening imaging modality in AMI. Although angiography is more specific and accurate, surgeons have been reluctant to offer such an invasive procedure in patients with a nonspecific clinical picture. Unfortunately, the first option is still common in the community—leading to procrastination, late diagnosis and treatment, and a very high mortality rate.

### *Computed Tomography*

To be diagnostic, the examination should include oral contrast and intravenous contrast ("CT angio"), with the focus on two areas: the bowel wall and mesenteric vessels. The most common finding is bowel wall thickening, which is nonspecific. The bowel wall may appear low in attenuation due to edema, or when submucosal hemorrhage is present, it may appear of high attenuation due to the blood products. Visualization of the dynamic enhancement pattern of the affected bowel loops may improve diagnosis. Affected bowel loops may demonstrate absence of enhancement, delay in enhancement, or persistent enhancement when compared to unaffected loops. *Pneumatosis* and *portal vein gas* are uncommon but specific, albeit late, signs—due to intraluminal gas dissecting into the friable bowel wall and then into the portal venous tributaries. CT angio can also visualize emboli within the SMA or thrombosis at its origin. From this description, one can easily appreciate that even the CT findings in this condition are subtle and easy to miss.

Magnetic resonance angiography (MRA) could be superb in imaging mesenteric vessels (with reduced risk of contrast nephrotoxicity) but, like CT angio, is far inferior to conventional angiography in visualizing distal branches. In addition, in how many hospitals would MRA be available in the middle of the night?

### *Mesenteric Angiography*

To be beneficial, the angiogram should be performed before the bowel has become gangrenous. The clock is ticking; every passing minute reduces the chances of the bowel and the patient surviving. Note that an acute abdomen with peritoneal signs is a contraindication to angiography. The radiologist should start with biplanar angiography (i.e., including a lateral view to show the origins of the SMA and the celiac axis). An *occluded ostium of the SMA denotes thrombosis* and calls for an immediate operation, unless there is evidence of a good collateral inflow, with the angiography providing the road map for vascular reconstruction. When the ostium is patent, the radiologist advances the catheter into the SMA. *Emboli* lodge distal to the takeoff of the middle colic artery, produce a smooth filling defect on the background of a normal SMA, and can be multiple.

## Nonoperative Treatment

In the absence of peritoneal signs, attempts at nonoperative treatment are justified, tailored to the clinical, CT, and angiographic findings. Selective diagnostic angiography can now become therapeutic, infusing a thrombolytic agent to lyse the thrombus or embolus with or without adding *papaverine*[1] to relieve the associated mesenteric vasospasm. Cessation of abdominal symptoms together with angiographic resolution means that the emergency is over, and any pre-existing mesenteric artery stenoses can be addressed electively, if indicated.

In the event of nonocclusive mesenteric ischemia, the approach involves attempts at restoring compromised hemodynamics. To relieve associated arteriospasm, a selective intra-arterial infusion of a vasodilator, such as papaverine, has been advocated. The few champions of this method have reported "favorable responses." When emboli are the cause, after successful transcatheter therapy, long-term anticoagulation is indicated. As a final point—while rushing to the arteriography suite remember to ensure adequate hydration of your patient to oppose the nephrotoxic effect of the contrast media.

## Operative Treatment

As we have told you, peritoneal signs, considered together with a suggestive clinical (and CT) picture, are an indication *not* to do arteriography but to operate; the same applies to failure of the nonoperative regimen. Through a midline

[1] That intra-arterial *papaverine* infusion is beneficial is a myth, originating in a retrospective study from one New York hospital more than 20 years ago and since perpetuated in reviews and texts, but it has never been further corroborated by a meaningful clinical experience.

incision, assess the viability of the intestine. In general, there are two main possible scenarios: one is that the bowel is *frankly gangrenous* (dead); the second is when the bowel *appears ischemic* (dusky) and of questionable viability.

- **Frank gangrene of the entire small bowel** is usually combined with the same problem in the right colon and signifies *SMA thrombosis*. Theoretically, a sporadic patient could survive resection of the entire small bowel and right colon. The patient may even tolerate a duodenocolic anastomosis while being nutritionally supported at home with total parenteral nutrition (TPN). But, the eventual mortality of such an exercise in the average elderly vasculopath approaches 100%, and the cost is immense. Our recommendation to you when involved in a similar situation is to walk out to talk to the family, explain that anything done will only increase the suffering of their beloved, return to the operating room, and close the abdomen over the dead bowel. Provide a lot of morphine and comfort. As with everything in life, there are exceptions: in a relatively young and active patient and when local circumstances are favorable (we doubt that there are facilities for home TPN in rural Afghanistan), you and the family may want to strive for long-term survival.
- **Frank gangrene of a shorter segment**, or multiple segments *of small bowel,* usually denotes *embolism*. After excising all dead segments, carefully examine the remaining bowel. Measure it: how long is it? Only about half of patients left with less than 1 m (3 ft) of small bowel will live without TPN (saving the ileocecal valve improves the prognosis). Now, observe the remaining bowel. Is it truly noncompromised? Are the mesenteric arcades pulsating well? Feel the SMA at its root; is it vigorously pulsating?
- **Dusky bowel.** When you are not happy with the remaining bowel or when the bowel is not dead but appears ischemic and of questionable viability from the start, proceed as follows. Wrap the bowel in warm, saline-moistened sponges and wait 15 min. Unscrub and have a coffee; surgeons cannot stare at an inactive field for that long without starting to fiddle. Failure of the bowel to pink up mandates its resection. When the length of remaining normal-looking bowel reduces toward 1.5 m (5 ft), it may be advisable to leave the doubtful bowel in situ, to be re-examined during a relook operation (see the second-look operations section). Salvaging even a short segment of small bowel may improve the chances of preserving a life worth living. Some authors recommend the use of handheld Doppler to examine the perfusion of the antimesenteric side of the bowel; others use intraoperative fluorescein angiography. You may choose to use such modalities if available to you, but your clinical judgment should be just as good as any gimmick (› Fig. 23.1).

### *Adjunctive Vascular Procedures*

The ideal setting to surgically improve the perfusion of ischemic small bowel is when the operation follows emergency arteriography (plus failed angiographic therapy) and the bowel is viable or doubtful. Obviously, when the bowel is dead, it


**Fig. 23.1.** "How much should I resect?"

cannot be revived. Arteriography serves as a road map; when the SMA is thrombosed at its origin, a vein or graft bypass, antegrade or retrograde, is indicated to reperfuse the SMA. Such a scenario is, however, rare; more commonly, you will encounter a picture of SMA embolism. Palpate for the SMA just at the base of the mesocolon; if nonpulsatile, you will find it, after incising the peritoneum, to the right of the large, blue superior mesenteric vein. After obtaining control, open the artery transversely and pass up and down a small Fogarty embolectomy balloon catheter. You may conclude the procedure with a shot of urokinase injected distally to lyse the clots in the distal branches, which are inaccessible to your embolectomy balloon catheter.

### *To Anastomose or Not?*

You should be **selective** in attempting an anastomosis following any resection of devitalized intestine. The patient has to be hemodynamically stable and have at least fair nutritional status. To be hooked up, the remaining bowel has to be unquestionably viable and the peritoneal cavity free of established infection. Most crucially, the cause of ischemia has to be solved. Another factor strongly bearing on your decision is the length of the remaining bowel and its predicted postoperative function. When more than half of the small bowel is resected, the resection is considered "massive." Restoring intestinal continuity in such cases would lead to poorly tolerated and *intractable diarrhea*. And finally, the chief

reason not to anastomose the bowel is the possibility that further ischemia may develop. In addition, a stoma provides an external window that allows you to assess viability of the remaining bowel.

We recommend, therefore, that whenever the mentioned favorable factors are absent or when resection is "massive," the two ends of the resected bowel should be exteriorized as an end enterostomy and mucus fistula, if possible via one abdominal wall site as a "double-barrel stoma" (this will allow a subsequent elective reanastomosis—after the patient has reached optimal nutritional status and the bowel remnant its maximal adaptation—without a major laparotomy). The postoperative appearance of the stomas will accurately reflect the status of the remaining bowel.

#### *Second-Look Operations?*

A routine planned second-look reoperation allows direct reassessment of intestinal viability at the earliest possible stage, before additional mediators of SIRS have been released and in a way that aims to preserve the greatest possible length of viable intestine. This concept, which in theory at least is attractive, motivates many surgeons to re-explore their patients routinely after 24–48 hrs. This is an ideal situation for an abbreviated laparotomy. The sections of bowel that are definitely dead are excised after stapling and dividing the bowel. The stapled ends are simply dropped back into the peritoneal cavity. A 24 hrs interval allows the patient's deranged physiology to recover before a second look. The finding of completely normal bowel at re-operation is of course reassuring and allows an anastomosis to be fashioned with confidence then, but the anastomosis may still leak 5 days later. If you plan a second-look operation, there is no need to close the abdomen at the end of the first procedure; instead, treat the abdomen as a laparostomy (Chaps. 43 and 52.2) until re-exploration, relieving any intra-abdominal hypertension to further improve mesenteric blood flow.

An alternative option is to close the abdomen, leaving a few laparoscopic ports adjacent to the bowel, through which a laparoscope may subsequently be inserted to assess the status of the bowel. Although this has been described, we do not have any experience with such an approach.

To sum, it appears, but has not ever been scientifically proven, that in most patients who at the end of the operation do not have stomas, a second-look procedure is indicated. Those with viable stomas who are otherwise well can be observed.

### Mesenteric Venous Thrombosis

In the rare condition of mesenteric venous thrombosis, the venous outflow of the bowel is occluded. The clinical presentation is nonspecific. Abdominal pain and varying gastrointestinal symptoms may last a few days until eventually the intestines are compromised, and peritoneal signs develop. Mesenteric venous

thrombosis maybe idiopathic (i.e., the doctor is an idiot, ignorant of the underlying reason), but commonly an underlying hypercoagulable state (such as polycythemia rubra vera) or sluggish portal flow due to hepatic cirrhosis is present. It has also been described in the postoperative phase after upper abdominal procedures such as splenectomy.

Typically, many of these patients are admitted to the medical floor with a surgeon consulted much later—to operate for nonviable bowel. However, an early trip to a contrast-enhanced CT scan may achieve an earlier diagnosis, helping to avoid an operation altogether and improving survival.

Characteristic findings on CT consist of a **triad**:

- A hypodensity in the trunk of the superior mesenteric vein
- Associated intraperitoneal fluid
- Thickened segment of small bowel

With these findings and in the absence of peritoneal signs, full systemic anticoagulation with heparin may result in a spontaneous resolution of the process. The role of systemic or selective-angiographic thrombolysis is not clear. Failure to improve or the development of peritoneal signs mandates an operation.

At surgery, you will find some free serosanguinous peritoneal fluid; the small bowel will be thick, edematous, and dark blue but not frankly dead, with the involved intestinal segment poorly demarcated. **Arterial pulsations will be present and thrombosed veins seen**. You will need to resect the affected bowel. Regarding whether to anastomose and considerations about the need for a second look, apply the same judgment as discussed for arterial ischemia. Postoperative anticoagulation is mandatory to prevent progression of the thrombotic process. Adding a venous thrombectomy is advocated by some, as is intraoperative thrombolysis; the real benefits of these controversial approaches are unknown.

### Conclusion

In most places, the mortality rate of acute mesenteric ischemia is still prohibitive. Why? Because surgeons **fail** to do the following:

- Suspect ischemia before intestinal gangrene develops
- Proceed with diagnostic/therapeutic angiography
- Improve intestinal perfusion during laparotomy
- Exteriorize the bowel or execute a second-look operation

So, here is the "catch 22": if you wish to see survivors of this horrendous condition, you will have to be aggressive.

On the other hand, the presentation of these patients is so nonspecific and, frequently, the CT findings so subtle that if an aggressive approach is taken, many patients with self-limiting minor abdominal complaints will have unnecessary investigations and operations, and yet cases will still be missed. Furthermore, these patients rarely have simple pathology. They commonly suffer from multi-system disease, and even in receipt of optimal care they will have a high mortality. Regrettably, in the majority of patients this condition seems likely to remain an agonal complaint. As our mentors taught us: "You can't save 'em all!"

"The man is as old as his arteries." (Thomas Sydenham, 1662–1689)

It is almost impossible to increase the current M & M associated with acute mesenteric ischemia.

# 24

# Inflammatory Bowel Disease and Other Types of Colitis[1]

Per-Olof Nyström

*When an internist wants you to operate urgently on his IBD patient, assume that the operation was indicated at least a week ago…*

**Ulcerative colitis** (UC) is a disease of the colonic mucosa only. **Crohn's disease** (CD) involves all layers of the bowel and can appear anywhere along the intestinal tract. Because of this difference, UC is curable with proctocolectomy, whereas CD is not amenable to surgical cure. For CD, surgical excision of the affected bowel segment serves only to reduce symptoms as nearly all patients will suffer recurrence. The need for emergency surgery in inflammatory bowel disease (IBD) patients has drastically diminished in recent years because patients are diagnosed earlier and are better controlled by gastroenterologists. In places where specialized care of IBD is lagging, emergency surgery is more common.

About a third of UC patients will eventually require an operation, while nearly all with CD will have one or more operations during their lifetime. Most general surgeons will not attend more than a few cases per year, and patients may be referred too late—unless gastroenterologists and surgeons co-operate and share a common philosophy of what medical and surgical treatments can and should provide. Gastroenterologists should know and appreciate that skilled surgery has a high rate of success when medical treatment fails. But, surgeons must appreciate that an operation may cripple the patient and turns some into intestinal invalids.

## Acute Attack of UC

There was a time when mortality was high for acute attacks of UC—with both medical and surgical treatment. It was British gastroenterologists and surgeons who led the way in the 1960s and 1970s in almost abolishing mortality by

[1]A comment by the editors on neutropenic and ischemic colitis is found at the end of the chapter.

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establishing criteria to measure the severity of the attack and the timing of operation. **The simple wisdom is that failure of medical treatment should be recognized early—being an indication for surgical treatment.** These developments have almost abolished emergency colectomy for UC as we are now able to schedule colectomy semielectively for nearly every patient. The skilled gastroenterologist is able to decide early when medical treatment is failing, and colectomy can then be discussed with the patient without haste. The surgeon should aim for this standard of care. Thus, the need for an emergency colectomy for UC in your practice implies a failure on the part of the treating team.

In the last few years, acute attacks of UC have been treated with "biologicals," which at this time means the anti-TNF (tumor necrosis factor) monoclonal antibody *infliximab*. It has been shown that the acute attack can be reversed in a large proportion of patients who would otherwise be candidates for surgery. Moreover, it seems that the need for surgery is reversed for at least a couple of years. Not all patients respond to this treatment, so you are likely to be called on to operate on a patient who has been given infliximab a couple of days ago but failed to improve. Ideally, you were informed about the decision to try infliximab, so you are aware that should this treatment fail you are likely to encounter a patient who is on a downhill course and thus more prone to complications.

### *Assessment of the Acute UC Patient*

When asked to assess a case of acute UC for colectomy, you should consider the following:

- **How extensive is the colitis, and how badly is the mucosa affected?** The acute attack has usually been progressing for several weeks. The patient has been given oral steroids, then admitted to the hospital and treated with parenteral steroids because of deterioration. Some gastroenterologists are unwilling to do a full colonoscopy for an acute attack, fearing the risk of perforation. However, a sigmoidoscopy suffices to demonstrate ulceration. From plain abdominal films, it is often possible to tell how extensive the colitis is by demonstrating no bowel contents in the affected colon. A little air injected through a rectal catheter will function as a contrast medium, giving a good demonstration of the extent of the colitis and often disclosing the presence of ulceration (› Fig. 24.1). The so-called **toxic megacolon**, an extreme dilatation and indication of impending perforation of the colon with systemic toxicity, is a problem of the past. It should never be allowed to happen in a patient under proper care, for whom an operation will have been scheduled long before such destruction of the colon has happened.
- **How has the colonic pathology affected the patient's physiology?** Colitis restricted to the left colon usually produces minor signs of systemic inflammation and wasting. Most such patients are not candidates for surgery unless it is obvious that the colitis cannot be controlled after extensive medical treatment has failed. But, we have seen acute attacks limited to the left colon causing perforation of the


**Fig. 24.1.** "Pancolitis, eh? Shouldn't we increase the steroids and add Imuran?"

sigmoid colon. In general, the extent and severity of colitis correlate with the physiological derangement of the patient. There will be fever, leukocytosis, and increased concentrations of C-reactive protein. The hemoglobin and albumin may drop significantly, often over just a few days. The patient has deteriorated while on a high parenteral dose of steroids, and now the patient's physiology is breaking down. It is time to decide on the operation. Table 24.1 will allow you to distinguish better between mild or moderate versus severe colitis that should be taken seriously. The APACHE II (Acute Physiological and Chronic Health Evaluation II) score is also useful in estimating the severity of illness in this situation (Chap. 6).

**Are there complications of colitis?** We pay little attention to the number of bowel movements because the actual counts are so dependent on tenesmus and urgency. There are patients who have 20 or more bowel movements per day because of the urgency, but the more common figure is around 10. Blood in the stools is

**Table 24.1.** Grading of ulcerative colitis

| | Mild/moderate colitis | Severe colitis |
|---|---|---|
| Temperature (°C) | <38 | >38 |
| Pulse (per min) | <90 | >90 |
| Diarrhea | Five per day or less | Six per day or more |
| Blood in stool | None or little | Large amounts |
| Anemia | None or mild | Severe (75% or less) |
| Albumin (g/L) | >3 | <3 |
| Abdominal pain | None or some | Severe |

common but try to get some objective information about how often and how much and compare with the hemoglobin concentration. Is the patient able to compensate for the blood loss? If not, it strengthens the indication to operate. Bleeding that requires several blood transfusions is an indication for urgent colectomy; fortunately, this has become very rare today. Considering that there may be extensive ulceration of the mucosa, it is remarkable that systemic sepsis with positive blood cultures is relatively rare. Associated pneumonia is occasionally present. With secondary infections, there is no haste as it is better to treat the infection with antibiotics and do the colectomy a few days later. We have seen several cases manifesting venous thromboembolic phenomena. One should probably view such complications as indications that the host defenses and homeostasis are breaking down, and that colectomy is necessary. Thrombosis, especially thromboembolism, is a troublesome complication as its treatment with heparin may increase any bleeding from the bowel, and the colectomy in itself is a distinct risk factor for further thromboembolism.

- **What is the general status of the patient?** One must evaluate how the colitis and its treatment have affected the patient over an extended period of time. It should be unusual to find obvious stigmata of steroid treatment apart from some edema and acne. If there is a moon face, muscle atrophy, hip adiposity, and cutaneous striae, the patient either has been treated too long or is too sensitive to cortisone. Any such patient, in our minds, should have a colectomy to get the patient off steroids. How alert is the patient? Is the patient out of bed, reading, or watching TV? At the first consultation, the patient may refuse surgery as an alternative, but as soon as the malaise associated with the disease activity appears, the patient is usually happy to consent to the operation. Both the short-term and the long-term consequences of the colitis should be considered: **the worse the previous course has been, the stronger the indication for a colectomy during the current attack.**
- **What is the nutritional status of the patient?** Withholding food and drink does not improve the acute attack, but eating increases the diarrhea, and most patients are unable to eat properly in the later stages of an acute attack. In general, in IBD patients, enteral nutrition is preferred over the parenteral route, but total parenteral nutrition may be indicated in the setting of a severe attack prior to the operation.

## The Operation for Acute Colitis

Schedule the operation for acute colitis for the next day if the patient is in reasonably good condition but do not delay it further. No preoperative bowel preparation is necessary. Antithrombotic prophylaxis with low molecular weight heparin should be given as for elective operations. Single-dose antibiotic prophylaxis is adequate. Do not forget to "cover" the perioperative phase with hydrocortisone.

The operation for acute colitis is **total abdominal colectomy**: in younger or leaner patients, the colectomy is easy and should take about 2 hrs; in a middle-aged male, it can be substantially more difficult. There are often only minor signs of

inflammation on the exterior of the colon; there may be some thickening of the wall and tortuous inflammatory capillaries on its surface. The segmental blood vessels may be enlarged due to the rich blood flow. You can begin the dissection on the right or left side as is convenient. Incise the peritoneal reflections laterally and identify the plane between the mesocolon and the retroperitoneal fascia. Divide the gastrocolic ligament so the omentum is removed with the colon but the gastroepiploic artery is preserved for the stomach. Once the colon has been freed laterally, it is time to divide the segmental arteries. Divide the ileum about 5 cm from the ileocolic junction and the rectosigmoid junction—just above the promontory—with the linear stapler. It is unnecessary to oversew the staple line. It is not necessary to spare the ileocolic artery and its ileum branches. The terminal ileum will have sufficient blood supply from the main branches of the superior mesenteric artery. However, on the left side it is clever to spare the inferior mesenteric artery and the superior rectal artery because this will prevent the rectum from sinking deep into the pelvis. Dividing the rectum with a linear stapler 5 cm above the promontory and then dividing the sigmoid mesentery in front of the superior rectal artery, preferably with a linear stapler with vascular cartridges, allows the rectal closure to remain just in front of the promontory. There is no role for drains. The closed terminal ileum is brought out through the rectus abdominis muscle on the right side. The stoma site should have been marked before the operation. Avoid suturing the ostomy or the ileal mesentery to the abdominal wall, which only creates more adhesions. Close the abdomen and then fashion the stoma. Cut the bowel 5 cm above the skin, evert, and suture to the skin, which results in a 2.5-cm long ileostomy.

A **proctocolectomy** for an acute attack of UC belongs to history as does the proctostomy (mucus fistula) with a long rectal remnant brought out through the wound or a separate incision. The short inflamed rectum will not keep the patient sick. After the operation, the diverted rectum becomes silent, but it is a good idea to finish the operation by turning the patient to the lateral position and evacuating the rectum transanally because retained blood may suppurate and cause problems in the postoperative course.

Your patient may be young and relatively well, and the operation may appear a "piece of cake" to you. But, resist the temptation to do anything more than a total abdominal colectomy by adding an ileorectal anastomosis or, God forbid, a restorative pouch ileoproctostomy. Those patients are catabolic and on steroids—the punishment for anastomotic complications is severe!

Colectomy for acute colitis is a delicate operation in a sick patient who, because of the systemic inflammatory response syndrome, is more likely to sustain postoperative complications. Watch over these patients carefully. The colectomy will be followed by a second **restorative procedure** within the next few months. The surgeon who does the colectomy can greatly facilitate the subsequent procedure. Most important, avoid creating adhesions, which can be extensive when all four quadrants of the abdomen have been touched. Precise surgery

in embryonic planes with minimal blood loss is important. Avoid suturing of peritoneum or stoma. A sheet of Seprafilm in the pelvis covering the closed rectal stump may do wonders to minimize adhesions in the pelvis, particularly important in females who plan to have children.

## Emergency Surgery for Crohn's Disease

Emergency operation in CD should be rare indeed. There are a few patients with acute colitis, which is clinically indistinguishable from acute UC and thus handled along the same lines, unless the **colitis is segmental**. Most of the time, however, the course and anatomical appearance of the colitis suggest that it is CD rather than UC. When small bowel is involved, a diagnosis of CD is obvious. Surgery for CD demands a lot more consideration because the patient will not be cured, and choosing the operation and its timing makes a difference to the future course. There is a growing understanding that repeated surgery contributes to and perhaps is the major factor behind the phenomenon of "Crohn's cripples" and even the premature death of these patients. It seems, however, that patients with recurrent or chronic symptomatic CD, like patients with chronic arthritis, slowly waste over the years, a problem to which steroids and repeated amputations of bowel contribute. For those of us who believe that any operation marks the patient permanently, both biologically and socially, it is a cause of concern that some patients with CD will have many operations during their life. It must be emphasized, however, that for the vast majority of the cohort, timely surgery is part of the optimal treatment. There are a few other instances, excluding acute colitis, when emergency surgery is considered in CD patients: suspected appendicitis, small bowel obstruction (SBO), and intra-abdominal abscess.

### *Acute Appendicitis*

If you operate for suspected acute appendicitis (Chap. 28) and encounter changes that are compatible with CD of the terminal ileum and cecum (e.g., serosal inflammation, thickened mesentery), what then? If the cecum is involved but the appendix appears normal, the best option is probably to leave it alone as appendectomy may result in an enterocutaneous fistula. The patient is then treated with steroids. An ileocecal resection in that situation may provide you with the histological diagnosis but is unnecessary or could at least have been postponed for several years. Almost every patient with an ileocolic resection will develop recurrent Crohn's inflammation of the anastomosis, usually within a year, yet another reason not to be blasé about the resection. But let us not forget that CD patients may develop acute appendicitis, which is treated with an appendectomy. In any

case, it is extremely important that the patient be made aware whether the appendix has been removed or still lurks in his or her belly.

### *Small Bowel Obstruction*

Small bowel obstruction is common in patients with CD. Usually, it is due to a narrow segment of diseased terminal ileum, but it may be caused by a more proximal stricture of a skip lesion. When the diagnosis of CD is known, you should treat the obstructive episode conservatively—SBO in CD is usually "simple obturation" of the narrow segment and resolves spontaneously, at least until the next exacerbation. In the absence of a previous diagnosis of CD, a careful history may reveal the typical previous abdominal symptoms, including episodes of transient obstipation, and systemic signs of inflammation that are compatible with a diagnosis of CD. A computed tomograph (CT) showing typical segmental bowel wall and mesenteric thickening, rather than a small bowel follow-through, can provide the diagnosis. Conservative management of SBO is discussed in Chap. 21. Steroids will be required.

**If you operate for SBO and find an inflamed and thickened terminal ileum compatible with CD, what then?** It is much better and simpler to operate on CD in the elective situation, when the bowel is empty and its inside can be inspected for strictures with intraoperative endoscopy through the wound. But now, the bowel is obstructed and distended. "Run" the bowel to identify any skip lesion that is more proximal and make sure there is a passage through it, that is, it is nonobstructing. Record any proximal skip lesion in your notes but leave the lesion untouched. Your task is to deal with the acute SBO. Obstruction in CD is very rarely complete or strangulating (Chap. 21); therefore, your best option is to close the abdomen and start the patient on steroids, thus sparing the patient's bowel.

Rarely, you will be called to operate on an acutely obstructed patient who has failed conservative treatment. Here, the operative options are resection of the ileocecal region, stricturoplasty, or a temporary proximal loop ileostomy. When the last option is adopted, the inflammation is medically treated until the acute phase resolves and an elective operation can deal permanently with the affected bowel.

### *Segmental Colonic CD*

Colitis in CD is often segmental and should not be treated by total colectomy. Contrary to what many surgeons think, the colon is an important organ with distinct functions of each part. Whenever possible, the resection should be limited, as it is for CD of the small bowel. Total colectomy in CD limits the options for later reconstructive surgery as the ileoanal pouch is a suboptimal option because of worse function and higher risk of complications than in UC.

### *Intra-abdominal Abscess and Fistula*

Intra-abdominal abscess and fistula represent more serious pathology. There is rarely a need for emergency surgery, and it is better to convert the acute situation to a semielective one. Most abdominal abscesses in CD patients can be drained percutaneously (Chap. 49). The patient is then treated with antibiotics, steroids, and nutritional support to allow resolution of the acute phase before undergoing elective resection of the involved bowel—the source of the infection. Complex abscesses which fail percutaneous drainage should be operated on. The involved segment of bowel has to be resected. Whether to restore bowel continuity with an anastomosis or exteriorize the bowel ends as a double-barrel stoma depends on the condition of the patient, the patient's abdomen, and the bowel (Chap. 13).

The risk of anastomotic failure is clearly higher in CD than in ordinary colorectal surgery. This is particularly true in CD patients who are operated on for intestinal fistula or abscess and those with several previous operations because of the inherent complexity of the anatomy. It is important to avoid any inadvertent bowel injury because it increases the risk of postoperative perforation and fistula formation. The best option is often to bring out the bowel ends in a combined ileo-ileal or ileo-colic stoma for later closure. Most anastomotic failures would necessitate a reoperation with resection of the failed anastomosis and stoma formation. Be mindful that a failed anastomosis in a CD patient will wipe out a year of that patient's social life and add two more operations in that period (see also Chap. 51).

## Perineal Crohn's Disease

About a fifth of the CD population will develop anal fistulae and abscesses at some time. About half of the abscesses and fistulae appear to be similar in character to those that affect the non-Crohn's population. In the typical "perineal Crohn's," the abscess is large with significant induration of the perianal area. The fistula is typically high, above the external sphincter, and has extensions. Not uncommonly, the external opening is far away from the anal orifice, not the usual 3–4 cm seen in cryptogenic fistula. There may be more than one external opening. There may be an undermining "sea of pus." There is often associated proctitis—a reason to do rectoscopy as part of the preoperative evaluation. Pain can be significant. The diagnosis of CD may have been established previously, and what you see around the anus is obviously a chronic process.

The anorectum in these patients is best evaluated and managed under general anesthesia. Do not attempt to do more than adequate drainage, which still may require quite extensive incision. Avoid incising close to the anus because the wounds may not heal; incise liberally over the peripheral abscess and external opening for adequate drainage. Place a loose **draining Seton** if the internal opening is identified and can be intubated. Your task is to provide source control of the abscess, which

can be difficult with inadequate incisions. Let the peripheral extension of the fistula tract guide you. Just avoid bringing the incision too near the anal orifice. And do not forget that oral Flagyl may prevent recurrences and offer non-surgical palliation.

### Clostridium Difficile Colitis

Although *Clostridium difficile* colitis (CDC) is not considered IBD, this is an acute colitis. With the prevalent overuse and misuse of antimicrobial agents by physicians and surgeons, CDC is a too common problem in hospitalized patients. CDC classically presents with diarrhea and abdominal pain following a history of antibiotic intake, with independent risk factors including age over 65, cephalosporin use, use of multiple antibiotics, prolonged hospital stay, and use of antibiotics for more than 7 days. The more antibiotics you give, the higher the chance that your patient will develop CDC, but it can occur even after a single dose. The tragedy is that patients may die from CDC after having received antibiotics for dubious indications.

The clinical spectrum of CDC is broad, ranging from mild diarrhea on the one hand to colonic perforation of the other. The gold standard for diagnosis is the stool cytotoxin assay for toxin B; however, the test results may take 1–3 days. Therefore, many institutions use the latex agglutination test, which has a faster return time but is less sensitive. **Or, even faster is sigmoidoscopy, demonstrating the typical ulceration and pseudomembranes for which the disease was named.** The preferred medical therapy for CDC includes oral metronidazole or oral vancomycin and, if the patient is unable to take oral medications, intravenous metronidazole. These therapies are highly effective in most patients, with only a minority eventually requiring surgical therapy. **However, systemic deterioration and peritonitis despite optimal medical therapy mandate urgent laparotomy.**

**A subgroup of CDC patients present from the beginning with an acute abdomen**, exposing them to a highly morbid and unnecessary exploratory laparotomy, which discloses viable and not perforated CDC. Therefore, **remember** that in any patient who presents with an acute abdomen with a history of recent or current antibiotic intake and without findings that mandate an immediate exploration (e.g., free air), CDC should be urgently excluded. Timely diagnosis of CDC through the use of sigmoidoscopy or CT scan—showing diffuse colonic wall thickening and colonic dilatation—will allow adequate medical treatment and could spare the critically ill patient an unnecessary and risky operation.

**At operation for fulminant CDC** that failed conservative treatment, the large bowel appears distended, inflamed, gray, and paper thin; sealed miniperforations may be present. There is no doubt that subtotal colectomy is the procedure of choice when the colon is nonviable or perforated. It is also a reasonable option,

albeit unproven, when operating on a patient with CDC who has failed to improve on medical treatment. But, whether a subtotal colectomy is advisable during an exploratory laparotomy in a critically ill patient for an acute abdomen, with a surprise operative finding of an undiagnosed CDC, is unknown. The construction of any bowel anastomosis is contraindicated when operating on CDC; the ileum is exteriorized as an ileostomy, and the rectum closed as a Hartmann's pouch. When the patient has recovered, an ileorectal anastomosis can be done. Consider, however, the risk of reactivation of the CDC at the time of stoma closure; remember that a single antibiotic dose is all it takes.

### *Summary*

**In acute UC:**

- Liaise closely with physician gastroenterologists
- Assess extent and severity of colitis and response to treatment
- Assess effects of colitis on the overall status of the patient
- Operate semielectively and do a total abdominal colectomy

**In CD:**

- Avoid surgery if at all possible
- Indications for emergency surgery include Crohn's colitis, suspected appendicitis, SBO, and abscess
- In surgery for suspected appendicitis avoid resection of CD
- In SBO operate only if the gut is truly completely obstructed
- Drain abscesses percutaneously and operate later in a planned, elective fashion

**In CDC:**

- Treat medically with metronidazole or vancomycin
- Operate if no response; resect but avoid anastomosis

## Editorial Comment

This is a good place to discuss **neutropenic enterocolitis** and **ischemic colitis.**

### *Neutropenic Enterocolitis*

Neutropenic enterocolitis is a transmural inflammation of the large bowel in myelosuppressed and immunosuppressed patients, usually suffering from myeloproliferative disorders, receiving chemotherapy or following solid organ or bone

marrow transplantation. Profound neutropenia appears to be the common denominator. The process involves mucosal damage and alteration in bacterial flora, which then invade the bowel wall. The cecum is primarily affected, but the process may extend to the ascending colon and even the ileum. The presentation may mimic acute appendicitis; watery or bloody diarrhea is present in only half of the patients. Right lower quadrant tenderness, a palpable cecum, peritoneal signs, and features of ileus may be present. Neutropenia is a pathognomonic laboratory finding. Plain abdominal X-rays are usually nonspecific, revealing an associated ileus, but may show *thumbprinting* of the right colon and intramural air (pneumatosis)—denoting severe involvement of the cecal wall. CT scan of the abdomen is the diagnostic procedure of choice, showing thickening of the cecum and free air if an underlying perforation exists.

Management should be initially supportive, including broad-spectrum antibiotics effective against colonic Gram-negative bacteria and anaerobes; granulocyte colony-stimulating factor may be considered. Clinical deterioration, evidence of free perforation, and, rarely, severe lower gastrointestinal hemorrhage may necessitate operation. At laparotomy, normal-looking serosal surfaces may hide mucosal breakdown and necrosis. Therefore, the whole involved segment of colon should be resected; anastomosis should be avoided in these debilitated patients. Mortality is obviously high. The key is to recognize the condition and avoid an operation in the majority of patients.

### *Ischemic Colitis*

Ischemic colitis is a poorly defined entity that encompasses a wide variety of conditions. Paradoxically, occlusion of the named arteries supplying the colon is not associated with ischemic colitis, but local vascular changes in the wall of the colon may play a role. Thus, a patient with sigmoid colon gangrene following repair of an abdominal aortic aneurysm and *ligation* of the inferior mesenteric artery has *colonic ischemia*—not *ischemic colitis*. But, a patient who undergoes treatment of a ruptured abdominal aortic aneurysm with preoperative hypotension who develops gangrene of the right colon postoperatively has ischemic colitis.

**Ischemic colitis develops in two different clinical settings:**

**Spontaneous:** in patients with underlying cardiac failure, chronic lung disease, renal failure, diabetes, and collagen disease—probably related to diseased intramural vessels

**Shock associated:** in patients who have experienced sustained shock regardless of etiology (e.g., ruptured aortic aneurysms)

Typically, the colonic process involves a varying depth of penetration. Transient mucosal involvement may or may not progress to partial thickness

necrosis, which may recover with or without a stricture or progress to full-thickness gangrene. Although most common in the "watershed" area of the splenic flexure and the left colon, the disease can involve any part of the colon and the rectum and rarely the entire colon; although usually focal, it may be patchy or diffuse.

Patients with spontaneous ischemic colitis present typically with nonspecific abdominal pain and lower gastrointestinal bleeding (hematochezia). Those with shock-associated ischemic colitis develop these features on top of their underlying critical disease.

As with mesenteric ischemia (› Chap. 23), the clinical picture, as well as laboratory findings, is entirely nonspecific, as is the commonly associated ileus. Abdominal X-rays may demonstrate an ileus and colonic dilatation proximal to the area of ischemia or a dilated ischemic colon. In the rare, advanced transmural cases, pneumatosis coli or free gas may be seen. Findings on **CT** include colonic wall thickening, free fluid, and pneumatosis coli. Lower gastrointestinal endoscopy (often bedside) is the best diagnostic test, visualizing a spectrum of hemorrhagic and ischemic changes that, although nonspecific and that may be confused with CD colitis (see discussion of CD colitis), are highly suggestive in the specific clinical setting.

#### Treatment

Clinical and radiographic evidence of colonic perforation or an endoscopic picture of dead bowel (black, paralyzed) necessitates a laparotomy and resection of the involved segment, but this is infrequently needed. Nontransmural ischemia is managed nonoperatively with supportive measures and wide-spectrum antibiotics as long as the patient is not deteriorating. Increasing or persisting abdominal pain, fever, ileus, leukocytosis, acidosis, and progressive changes on abdominal imaging may call for colonic resection.

Although most patients recover from the acute insult, some may progress to develop a chronic ischemic stricture—but this is beyond the scope of our story.

# 25 Colonic Obstruction

Per-Olof Nyström

*The only time human beings wish they could fart and defecate is when they are not able to do so.*

This chapter considers the most common cause of acute obstruction of the colon—**cancer**—but also mentions a much less common cause, which is **diverticulitis**. Also discussed is the condition that mimics obstruction: **pseudo-obstruction or Ogilvie's syndrome**. Finally, the chapter deals with **volvulus of the colon** affecting the sigmoid and cecum.

## Malignant and Diverticular Colonic Obstruction

The four "steps" you should consider in the approach to patients with mechanical colonic obstruction are:

- Establish the exact diagnosis

  Then, at operation:
- Decompress the colon
- Resect the obstructing lesion
- Decide whether there should be a primary anastomosis or a colostomy

### *Preoperative Diagnosis and Management*

The clinical hallmark of colonic obstruction is significant abdominal distention associated with recent onset of constipation and lack of flatus. The obstruction usually develops gradually over a few days, sometimes on a background of a change in bowel habit. The usual site of obstructing carcinoma is the sigmoid or left colon. The sigmoid is also the locus of any obstructing diverticular mass. Right colonic lesions become obstructing only at the ileocecal junction. Because of the wide caliber of the rectum, cancer here rarely presents with a complete obstruction.

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Most of these patients are elderly, and because the obstruction may have affected them for several days, they have not been eating and drinking properly, so they are dehydrated. Make a thorough examination of the abdomen. It is usually, but not invariably, grossly distended. Be especially observant of signs of peritonitis, which may indicate a manifest or pending perforation of the colon, usually proximal to the obstructing lesion. The site of perforation may be a preexisting sigmoid or left colonic diverticulum, but more commonly it is in the right colon. The right colon and cecum are the widest part of the bowel. They are also the most distended part with the highest tension of the bowel wall (Laplace's law), thus the most likely to perforate. When the *ileocecal valve is competent*, the small bowel will be only mildly distended, while massive distension and pressure affects the right colon. This pressure can tear the circular muscle layer or cause ischemic necrosis with subsequent perforation. Tenderness of the abdomen on the right side may be a sign of this development. If such tenderness is present and the abdominal X-ray shows a grossly distended right colon (in excess of 10 cm), then operation must not be delayed beyond the requirements of resuscitation.

Beware of obstructing cecal cancers. Because of the wide lumen of the cecum, they tend to present late—only after occluding the ileocecal valve—with features of distal small bowel obstruction (SBO), which commonly is incomplete and intermittent. Thus, whenever confronting distal SBO, especially in a "virgin" abdomen (Chap. 21), think about cecal cancer.

**Plain abdominal X-rays** (Chaps. 4 and 5) usually show a distended colon because the obstructing lesion is most often in the left colon. When the obstruction is in the right colon, at the cecal area, it can sometimes be difficult to differentiate between small bowel and large bowel obstruction. In long-standing left colonic obstruction, when the ileocecal valve is *incompetent*, the small bowel becomes dilated as well. Severely dilated loops of fluid-filled small bowel may then obscure the distended colon, a picture that may be misinterpreted as partial SBO. Regardless of the appearances on plain X-rays you must positively confirm the diagnosis by additional investigation and exclude pseudo-obstruction (discussed in a separate section of this chapter). What you have to do is document the site of the obstruction; this can be done either with **colonoscopy** or a **contrast enema**. For reasons explained in Chap. 4, our bias is against the use of barium in this situation and in favor of a water-soluble contrast such as Gastrografin. The site of the obstruction, but not the cause, will usually be evident. At this stage, "obstruction is obstruction"—the management is the same whether it is caused by a carcinoma or, less commonly, a diverticular mass. A preoperative **CT scan** is not mandatory but will usually give the diagnosis: a distended colon proximal to a cutoff point represented by a mass is an indication for operation without the need for colonoscopy or contrast enema. [Adding a Gastrografin enema to the CT would help in delineating the transition point! — The Editors] When clinical and laboratory features are suggestive of carcinomatosis or extensive hepatic

metastatic involvement, CT documentation of the advanced disease allows better planning of treatment together with the patient and family. You do not want to operate on a jaundiced patient whose liver is almost replaced with metastases for the patient surely will succumb to hepatic failure after the operation.

## Planning and Timing the Operation

In general, in the absence of signs of actual or impending compromise of the bowel wall, there is no reason for you to hurry with the operation. Daytime surgery, with all that it means in terms of the surgical team and supportive personnel, is the better option for the patient and yourself. There is plenty of time to prepare the patient for a definitive operation to relieve the obstruction. On the other hand, should the patient have peritonitis, systemic inflammatory response syndrome (SIRS), or free abdominal gas on abdominal imaging, an emergency operation is necessary. Antibiotic treatment should be started and the time of the operation decided according to the progress of the resuscitation-optimization (Chap. 6).

Obviously, in patients with colonic obstruction, bowel preparation is contraindicated. Any cleansing solutions administrated from above will accumulate proximal to the obstruction, further dilating the obstructed colon and making your life more miserable during the operation. Some surgeons like to administer enemas to clear the rectum and colon distal to the obstruction, but these sections of the bowel are usually empty. Again: do not forget to administer the usual dose of systemic antibiotic prophylaxis just before the operation (Chap. 7).

In general, the operation for acute colonic obstruction is a major procedure, often in a patient who is old and fragile. Consequently, the mortality and morbidity of these operations are significant (sorry, no percentages were allowed by the editors). To avoid complications and mortality, you have to exercise your best judgment along the lines presented here.

## The Operation

A long midline incision is nearly always preferable. The findings of ascites, peritoneal seedlings, "omental cake," and hepatic metastases will immediately tell you that the battle has been lost, and the operation is merely palliative. If the obstruction is in the right colon, there is usually not a lot of bowel distension. Then, the operation is a rather straightforward right hemicolectomy with primary anastomosis.

The **left colon or the sigmoid**, however, is the usual site of the obstruction. Here, the proximal colon is distended, making the operation more difficult. First, inspect the ascending colon to find out if there are tears or necrosis due to the distension.

If there are, they can be of any stage from minor to large with microperforation. The significance of the tears is that if they are extensive or necrotic it may suggest that a **subtotal colectomy** is indicated. Otherwise, proceed as follows:

- **Decompression**. Because of the distended bowel, it may be difficult to expose the lesion on the left side and to manipulate the bowel. Sometimes, it is better to make an enterotomy into the terminal ileum, or even through the appendix, and insert the suction device (a Poole sucker or a large sump drain) through the hole to decompress the small bowel and also pass the device through the ileocecal valve to decompress the right colon. Close the hole transversely with a suture. It should now be possible to expose the lesion that causes the obstruction. Often, in cases diagnosed and treated early, the colonic distention is caused by gas and not fecal matter; it can be relieved simply by inserting a large needle or angiocath connected to the suction tube and tunneled through the tenia coli.
- **Resection**. Whether it is cancer or diverticulitis-sigmoiditis (Chap. 26), the principles of treatment are the same. Mobilize the lesion the same way you would at an elective operation for cancer and resect it. Watch out for the ureter. If you are accustomed to linear cutting staplers (e.g., GIA), this is one of the best instances to use staplers. Transect the bowel on each side of the lesion and divide the mesentery and the segmental vessels with the linear stapler. You have resected the cause of the obstruction with complete control of the bowel ends and no leakage. Now is the time to decide whether the bowel ends should be joined or the proximal end should be brought out as a colostomy.

Do notice that it is considerably more difficult to operate on colonic obstruction than on a similar elective case. You will need the extra hands of an assistant to achieve exposure, and the decisions are much more complex during the operation. It is advisable to do the operation together with a colleague who can assist with the decisions [if you are a lonely country surgeon, you will have to be "assisted" by one of those fancy retractors; you can talk to the retractor as well—The Editors]. If it is a cancer operation, it should be the correct cancer resection, not just an operation that relieves the obstruction. A "simple" bowel resection is permissible only if the cancer is disseminated so that the type of resection has no influence on the prognosis of the cancer. In that situation, a colostomy is usually the better option because it is safer for the patient and has less risk of a new obstruction due to local recurrence of the tumor.

### *To Anastomose or Not?*

The judgment process regarding whether to anastomose is not much different from that considered after sigmoidectomy for acute diverticulitis (discussed in Chap. 26). What is different, however, is that here, usually, there is no associated

peritonitis and suppuration. In essence, after you have resected the lesion, you are left with a few options:

- End left colostomy—Hartmann's procedure
- Primary colocolic or colorectal anastomosis
- Subtotal colectomy with ileosigmoid anastomosis

If the cancer is situated in the *transverse* or *descending* colon, it is often better to do a *subtotal colectomy* and an ileosigmoid anastomosis. This usually means that empty or mildly distended and well-perfused small bowel is joined to normal colon below the obstruction. Most patients will manage an ileosigmoid anastomosis without incapacitating diarrhea and incontinence, while an ileorectal anastomosis requires that the patient had normal continence before the current illness. For cancers of the **sigmoid colon or rectosigmoid junction**, a **sigmoid colectomy** is adequate, and a subtotal colectomy should be considered only if the ascending colon is ischemic or perforated as mentioned.

### *Some Controversies*

The main dispute is the question of primary anastomosis and the means of obtaining that goal. It is only a problem for left-sided obstructions. **On-table bowel irrigation** has been proposed to facilitate primary anastomosis between clean proximal colon and the rectum. Its value is discussed in Chap. 26 on diverticulitis (in the section "Fecology"). The irrigation prolongs the operation substantially and therefore represents "negative damage control." **An alternative is the subtotal or total abdominal colectomy with anastomosis of the terminal ileum to the sigmoid colon or rectum.** This also is a bigger operation that takes longer. In a large Scottish randomized trial comparing the two means (subtotal vs. segmental resection) of obtaining a primary anastomosis, there was no difference in survival or anastomotic healing with either method (SCOTIA Study Group 1995). There are now several randomized trials of elective colonic resection with or without mechanical bowel preparation. Again, there was no difference in anastomotic healing. It may not be entirely valid to extrapolate the results with "residual feces" of the "elective" colon to the massive fecal load of the acute colon. It appears, however, that a primary anastomosis can be made safely on the obstructed colon after decompression and removal of feces with suction and milking the colonic end before joining it to the rectum. We, among others, make an anastomosis in an "unprepared bowel" in selective cases of obstruction.

**Why bother with a primary anastomosis at all when it increases the operation time and complexity of the operation?** A Hartmann resection and colostomy are quicker and simpler. It is not an all-or-nothing situation, but the

concerned surgeon will know that the Hartmann resection is often the better choice if the patient is in bad general condition or if the cancer cannot be radically removed. About half of the Hartman resections will never be reversed, often for very good reasons. For the less-experienced surgeon, we suggest that the Hartmann resection is always a valid option.

**Is there any role for a decompressive colostomy without resection of the obstructing lesion?** This staged management was commonly used only a few decades ago, usually consisting of a transverse colostomy as the first stage. Now, we would consider this option in three circumstances (but also see the discussion of stents in this chapter):

- The critically ill patient who will not tolerate a major procedure; for example, a patient developing an obstruction a week after a myocardial infarction. Here, a transverse colostomy or even cecostomy under local anesthesia will alleviate the obstruction.
- When there is preoperative evidence of widespread malignant disease, as discussed.

[We would also consider a diverting stoma when the obstruction is caused by a rectal tumor. This would allow for a proper staging of the tumor and subsequent elective resection, including, if indicated, adjuvant chemoradiotherapy.—The Editors]

### *The Colostomy (see also › Chap. 14)*

It should be understood that the creation of an emergency colostomy is potentially problematic. A common problem is *retraction* due to inadequate mobilization of the bowel. It frequently causes disruption of the mucocutaneous suture line in the early postoperative course, followed by retraction of the bowel end to a subcutaneous position and progressive stenosis of the skin orifice. Even retraction into the peritoneal cavity resulting in peritoneal soiling with feces occasionally occurs. To be safe, make sure that the left colon has been mobilized up to and sometimes including the splenic flexure. The closed proximal end should easily reach several centimeters beyond skin level and rest in that position without support. Do not settle for anything less or you may make the patient's remaining life an ordeal. The colostomy hole through the rectus abdominis muscle will have to be larger than normal because of the bowel distension. It is sometimes necessary to evacuate some of the gas and feces before the bowel can be brought out. A simple rule of thumb is that when the colostomy hole is kept open with retractors, the bowel end should pass "easily" between them, and it will not retract back if the retractors are removed. There is no need to close the lateral gutter or even to fix the bowel to the anterior abdominal wall if it has been sufficiently mobilized. Mucocutaneous suture of the colon to the skin with an absorbable suture is all that is needed.

You should choose either an anastomosis or a colostomy. The proximal "protective" ostomy for an anastomosis is a hybrid of disputable value. Should the anastomosis break, the protective colostomy is of little help because the colon was not clean and will leak all the residual feces distal to the protective stoma. Usually, a reoperation becomes necessary anyway. There is no study that proves that the proximal stoma prevents anastomotic failure.

### A Word About Stents

In recent years, endoscopically placed flexible, self-expanding stents have been developed and are increasingly applied in many hospitals as the preferred means of relieving the obstruction. There is a significant learning curve before the stent can be safely placed, so most surgeons will not have acquired the necessary skills. However, the technique may be available in your hospital, in which case it certainly should be considered in patients who do not require an urgent operation. The stents are used for two indications:

- As the definitive treatment in patients who have metastatic disease and therefore will not benefit from surgery or oncology treatment. This is for purely palliative treatment in patients with a short expected survival time.
- As a bridge to surgery in patients who have resectable cancer sometimes associated with resectable liver metastases.

Stenting requires that a CT scan is done to allow pretreatment staging of the cancer. Palliation can be excellent, avoiding an operation and its associated risks. The bridge-to-surgery option is less established, and there is yet much to learn about its advantage over operative resolution of the obstruction. However, if the obstruction can be safely relieved with the stent, it will allow an elective treatment pathway for the cancer. Stents are not an option for benign disease like diverticulitis.

### Our Preferences

We believe that resection of the obstructing lesion and a primary anastomosis can and should be achieved safely in most patients. For sigmoid lesions, we opt for a sigmoidectomy and colorectal anastomosis; if the proximal colon is excessively loaded or appears compromised, we proceed with a subtotal colectomy and an ileorectal anastomosis. The latter is also our preference for lesions in the proximal descending colon and the transverse colon. We reserve the Hartmann procedure for high-risk patients and those who appear poorly nourished.

## Acute Colonic Pseudo-obstruction (Ogilvie's Syndrome)

William Heneage Ogilvie (1887–1971) was not only a great British surgeon but also a keen surgical aphorist. For example, "Personal statistics are at the bottom of all unsound teaching; they are either too good to be true or too true to be good."

This is an important differential diagnosis of colonic obstruction. Pseudo-obstruction has the same symptoms, signs, and radiographic appearances of acute large bowel obstruction, but there is no mechanical blockage. The X-ray films are suggestive, but a contrast study or colonoscopy finds no obstruction. This pseudo-obstruction can be so intense that the right colon becomes ischemic and perforates due to the high intramural pressure. The distension may be extreme, resulting in acute abdominal compartment syndrome.

The mechanisms behind pseudo-obstruction are not known. It has been proposed that the condition may be due to sympathetic overactivity, parasympathetic suppression, or both. Most patients are already in the hospital for other reasons when the pseudo-obstruction develops. It is a rare but well-recognized sequel to giving birth, but more commonly is seen after major nonintestinal surgery or trauma or on the background of serious medical illnesses. Some patients may be sent directly from the nursing homes with this problem.

This entity is the reason why you should not operate on a suspected colonic obstruction without a preoperative colonoscopy, contrast enema, or CT. Taking an elderly patient with multiple premorbid conditions for a laparotomy to find "only" a distended colon, without an obstructing lesion, is a cardinal error. Avoid it. These patients should not have surgery but should be treated medically or decompressed with colonoscopy.

- For *medical treatment*, it is suggested that **neostigmine** (2 mg) intravenously will effectively induce bowel movements and colonic emptying within a few minutes. There are side effects to the neostigmine, including bradycardia, salivation, nausea, and abdominal cramps. The patient should therefore be under close surveillance during the treatment. We have tried this a few times; it does not always work, but when it does you look like a superstar.
- If medical treatment is ineffective, a careful *colonoscopy* may decompress the bowel. The target is decompression of the grossly distended cecum; occasionally, repeated colonoscopic decompressions may be needed. A large and long rectal tube can be left in situ after the colonoscopy for a few days. The diagnostic Gastrografin enema may occasionally also be therapeutic, with the hyperosmolar contrast medium promoting colonic peristalsis.

**Surgical treatment** is required if the cecum perforates or, very rarely, if medical treatment fails and the cecum reaches gigantic size, sometimes even producing an abdominal compartment syndrome! If the cecum becomes *necrotic* or perforates, a

right hemicolectomy is necessary. Because the functional obstruction must be in the left colon, a primary anastomosis is inappropriate. It is better to fashion an end ileostomy and bring out the distal end of the colon through the same colostomy hole, fashioning a "double-barrel" stoma. This arrangement makes it easy to restore bowel continuity later at the site of the colostomy without the need to reopen the abdomen.

When at laparotomy the cecum is distended but *viable*, most surgeons would opt for a **cecostomy.** Tube cecostomy is messy; it is associated with a high incidence of local complications, such as a fecal leak around it or even into the abdomen. To minimize these risks, use a soft, large-bore tube and surround its insertion site in the cecum with a double purse-string suture; the cecostomy site should then be carefully attached to the abdominal wall (as you do with a gastrostomy). Cecostomy tubes tend to obstruct with fecal matter and need regular flushing. A viable alternative to tube cecostomy is the formal—"matured"—cecostomy: simply exteriorize a portion of the cecum above the skin level and suture it to the surrounding skin. This, in medically ill patients with pseudoobstruction, can be performed under local anesthesia.

## Volvulus of the Colon

Though sometimes in a person who is fat
The diagnosis is not clear as that
'Tis then you get help from plain X-ray
Which gas within the gut should well display
So that the coil you see in the radiogram
Reaching from pelvis to the diaphragm.
(*The Acute Abdomen in Rhyme*. Zachary Cope, 1881–1974)

While volvulus accounts for only one-tenth of all instances of colonic obstruction, we tend to remember those patients. It is probably because of the spectacular appearance on abdominal X-rays and the equally spectacular way it is treated. Volvulus of the sigmoid colon is by far the most common, followed by that of the cecum. There is also volvulus of the transverse colon, but it is so rare that you will probably not see a case during your surgical life.

### *Sigmoid Volvulus*

In affected patients, the sigmoid is long, with a redundant mesentery that allows the sigmoid to rotate around its mesenteric axis, usually counterclockwise. It usually occurs after patients have reached seniority. It does happen at younger ages but then typically in an institutionalized patient. The rotation must be at least

180° to be symptomatic for obstruction, but if the rotation is 360° there is also a risk of strangulation. These circumstances account for two types of volvulus: a "slow" form in which obstruction develops gradually and a "rapid" form in which strangulation dominates. As the obstructing point is distally at the rectosigmoid junction, the propulsion of the proximal colon will blow up the obstructed sigmoid loop to impressive dimensions.

The typical patient presents with a history of recent onset of constipation and lack of flatus and a grossly distended belly. Because half of the patients have recurrent episodes of volvulus, the diagnosis may already be known. A **plain abdominal film** will suggest the diagnosis: a tremendously large loop of colon fills the abdomen from the pelvis to the upper abdomen. A contrast enema with Gastrografin will show the obstruction at the rectosigmoid junction. Typically, the contrast ends in a "beak-of-a-bird" sign that is very characteristic. It is the lower twist that causes this image. And of course, a CT would show the huge loop of sigmoid, including the "whirl sign"—typical for an intestinal volvulus—developing at the twisting point of the mesentery.

### *Treatment of Sigmoid Volvulus*

#### *Non-operative Approach*

Until around 1950, the treatment of sigmoid volvulus was surgical and associated with significant mortality. Then, it was demonstrated that the volvulus could be decompressed with much lower morbidity and mortality by passing a tube through the rectum. There are three ways of doing the procedure. If you are lucky to work in a hospital where the radiologist treats the patient, this is what they do. A large-bore, flexible but rather stiff tube, size 30–36 and 50 cm long, is passed through the anus and rectum to the site of obstruction. A bag of barium, or water-soluble contrast, is connected to the tube, and by letting in a little contrast, the hydrostatic pressure will open the twisted bowel sufficiently to pass the tube into the obstructed sigmoid. A flush of gas and feces signifies successful decompression. The whole procedure is done under X-ray imaging. Whether the tube should be left in place for a day or withdrawn immediately is a matter of debate (Fig. 25.1).

You might have to do the procedure yourself without the assistance of imaging. Then, use a rigid sigmoidoscope and pass it to the twist, which should be seen. The lubricated tube is introduced through the sigmoidoscope and carefully manipulated into the sigmoid. A third method is by means of a flexible colonoscopy and maneuvering the scope itself into the sigmoid. The eventual success of your manipulations is usually announced with a sudden rush of flatus and liquid feces at your face (watch out).

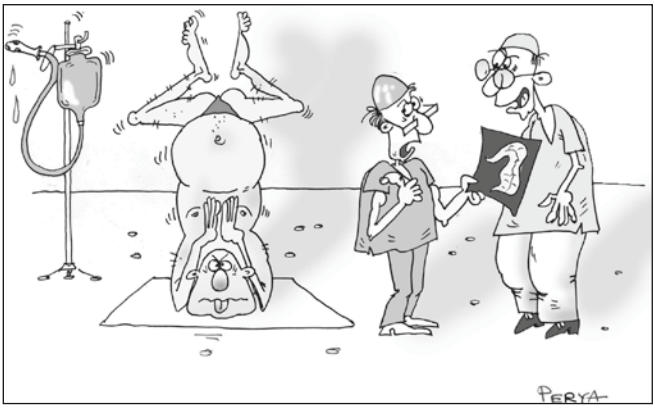

**Fig. 25.1.** Non-operative management of sigmoid volvulus

#### *Operative Treatment*

These non-operative methods are successful in the vast majority of cases because strangulation is uncommon. If strangulation and necrosis of the sigmoid are suspected on clinical grounds (evidence of peritonitis) or if attempts at nonoperative decompression fail, then an emergency laparotomy is required. At operation (modified lithotomy position), you will encounter a hugely distended sigmoid colon that has to be decompressed. This is best achieved by gently untwisting the sigmoid and advancing a prepositioned rectal tube into the dilated segment. Today, in most patients who undergo an emergency operation for sigmoid volvulus, the bowel will be nonviable or compromised. **Thus, the procedure of choice is sigmoid resection, with either a colorectal anastomosis or as a Hartmann's procedure.** The selection of what to do is essentially the same as discussed with regard to malignant colonic obstruction. Finally, we have to mention the option of **sigmoidopexy**, the fixation of the sigmoid to the lateral abdominal wall. This is a theoretical option when the sigmoid is viable and well decompressed, and you think that sigmoid resection with anastomosis is too much for the individual patient.

#### After Successful Non-operative Decompression

Elective sigmoidectomy to prevent recurrence, on the other hand, is very simple. It is done with a small transverse incision through which the hypertrophied mobile sigmoid loop is delivered and resected. There is no general agreement when patients should be offered a sigmoidectomy to prevent a recurrence. About half of the patients will have only one episode, but those with two episodes will frequently have a third. Most surgeons therefore offer resection after the second episode.

Anecdotally, a fragile lady in her mid-80s suffered one episode after another, but each time she was thought unfit for an elective operation on a benign condition. After her 12th volvulus, she had proved her case and was subjected to sigmoidectomy from which she recovered uneventfully and was discharged after 5 days.

### Volvulus of the Cecum

Volvulus of the cecum is much less common; you probably will not see more than a few cases during your career, but these will usually require an operation. The diagnosis is not as straightforward as that of the sigmoid volvulus. These patients have clinical and radiographic signs of SBO. In addition, typically the cecal "shadow" is absent from the right lower quadrant. Instead, the poorly attached and redundant cecum, which has flipped to the left and upward, is visualized in the epigastrium or the left hypochondrium, with its concavity pointing to the right lower quadrant. A single fluid level may be seen, representing the dislocated cecum and often confused with the gastric shadow. If in doubt, and in the absence of peritoneal signs, you may order a Gastrografin enema, which will demonstrate the characteristic "beak" in the right colon. Today, a CT would be the easiest and most accurate route to a diagnosis.

There are isolated reports of **colonoscopic decompression** of cecal volvulus, but the complexity of such a procedure and its doubtful results suggest that operation is the treatment of choice. What to do? There is an eternal controversy—probably never to be solved—between the proponents of cecal fixation-cecopexy and the advocates of mandatory resection. This is our selective approach: first, detort the cecum; the torsion is clockwise, so derotate the mobile cecum. If after detorsion the bowel appears gangrenous or of doubtful viability, then proceed with a **right hemicolectomy**. A primary anastomosis should usually be permissible, but occasionally circumstances suggest that a stoma is preferable. If so, bring out the small bowel as an end ileostomy and a corner of the closed colon end through the same hole. This combined double-barrel stoma allows simple closure and restoration of bowel continuity through the site of the stoma.

If the cecum is viable, we see no point in resecting it. Why remove a healthy organ that can be fixed? To prevent recurrence of the volvulus, fix the mobile cecum to the lateral abdominal wall (**cecopexy**). Start with decompression of the cecum by milking its contents toward a rectal tube for sutures hold poorly in a distended bowel wall. Cecopexy is accomplished by suturing the entire length of the cecum to the lateral abdominal wall. Use nonabsorbable material and take big seromuscular bites on the bowel and big deep bites on the abdominal side. Some surgeons elevate a flap of parietal peritoneum that is sutured to the anterior wall of the cecum.

Cecostomy, either a tube or matured to the skin, is an option that is mentioned in the literature as an alternative to cecopexy. However, we think that it is a bad idea: why convert a simple and clean procedure (i.e., cecopexy) to a contaminated and potentially complicated one (i.e., cecostomy)?

"Sometimes a bowel-coil gets out of place
By twisting round a narrow base
With gradual strangulating of the blood supply
And danger that th' affected coil will die.
This is a VOLVULUS which you should learn
Is from the Latin – volvere – to turn."
(*The Acute Abdomen in Rhyme*, Zachary Cope, 1881–1974)

## Reference

SCOTIA Study Group. (1995). Single-stage treatment for malignant left-sided colonic obstruction: a prospective randomized clinical trial comparing subtotal colectomy with segmental resection following intraoperative irrigation. Br J Surg 82:1622–1627.

# 26 Acute Diverticulitis[1]

Per-Olof Nyström

*Think about acute diverticulitis as a left-sided acute appendicitis which is, however, usually treated without an operation.*

Diverticula of the colon are not "true" diverticula but herniations of the mucosa through a weak spot of the muscular bowel wall. They can occur in all parts of the colon but are most abundant in the sigmoid colon. The mucosa bulges out through the points of entry for the blood vessels, which transgress the bowel wall on each side, where the mesentery joins the bowel. It is thought that the pressure inside the sigmoid colon, which can be very high, causes expulsion of the mucosa. The smooth muscle of the affected sigmoid colon, unlike that of the rest of the colon and rectum, is often hypertrophied. This thickening is always located at the summit of the sigmoid loop and rarely extends for more than 15 cm. The diverticula mainly appear within this thickened segment of the sigmoid but are not restricted to it. The thickening may reach the rectosigmoid junction but never extends into the rectum proper (15 cm from anal verge). However, it is common to find diverticula extending into the descending colon. Be aware that **diverticulosis**—the mere presence of sigmoid diverticula—is extremely prevalent in persons consuming a Western-type diet, while **acute diverticulitis**, inflammation of the diverticula-bearing segment of the colon, is relatively much rarer.

## Surgical Pathology

A wide spectrum of pathological conditions is covered by the term *acute diverticulitis*, each correlating with a specific clinical scenario, which in turn necessitates selective management.

[1]A comment by the editors is found at the end of the chapter.

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At operation for acute diverticulitis, the sigmoid usually feels like a thick fusiform tumor, with only a few diverticula. There are also cases of minor thickening with many diverticula, one of which has perforated and is the cause of the acute inflammation. Such observations make one think about the basic pathology of acute diverticulitis.

Basil Morson, the famous pathologist at St. Mark's, London, highlighted the hypertrophy of the bowel wall as the primary pathology, and we are inclined to accept this, with the addition that the mesenteric fat tissue also plays a role. It is this fat that creeps up the bowel wall, becomes inflamed, produces the phlegmon or abscess, and heals with fibrosis. In our experience, many cases of acute diverticulitis might better be termed *acute sigmoiditis*, recognizing that it is an acute inflammation of the thickened bowel wall and mesentery. When it is a diverticulum that has been eroded by a fecalith, one finds a localized inflammation, which identifies the site of the perforation. In cases of free fecal peritonitis, a perforated diverticulum is the cause, although more often it has been walled off by the mesentery or epiploic appendices to produce a pericolic abscess. Sometimes, the perforation occurs entirely within the mesentery, forming a mesenteric phlegmon or abscess. The latter may secondarily perforate into the free peritoneal cavity, but usually this variety only gives rise to minor abdominal and systemic signs but can occasionally produce septicemia in a patient who is unable to contain and isolate the perforation.

There is a strong tendency for diverticulitis and sigmoiditis to adhere locally and fistulate. The formation of fistulas has an obscure mechanism as most patients with such a fistula present as nonemergency cases and often do not even give a history of previous attacks of acute diverticulitis. Most often, the fistulas are into the bladder. The patient seeks attention for pneumaturia or persistent urinary tract infection. Fistulas can also communicate with the fallopian tubes, uterus, small bowel, or skin. It is usually thought that the fistula is the sequela of an abscess, but commonly there is no sign of an associated abscess; if there had been one, it must have been silent or drained spontaneously via the fistulous tract.

## Clinical Features, Diagnosis, and Approach

It is clinically pragmatic to think about acute diverticulitis or sigmoiditis as a "left-sided acute appendicitis." Unlike appendicitis, however, most episodes of acute diverticulitis are successfully managed without an operation. [As most episodes of acute appendicitis might be. See Chap. 28.—The Editors]

Practically, we find it convenient to think about the clinical scenarios of acute diverticulitis in order of increasing severity:

- Simple-Phlegmonous diverticulitis

and COMPLICATED FORMS:

- Pericolic abscess
- Free perforation with purulent peritonitis
- Free perforation with fecal peritonitis

### *Phlegmonous Diverticulitis*

Most patients admitted to the hospital with acute diverticulitis harbor a phlegmon; they are still capable of mounting an anti-inflammatory response that quenches the inflammation. Such patients are in good condition but suffer from acute pain and tenderness in the left lower quadrant and above the symphysis pubis. A mass may be felt on abdominal or rectal examination. There are signs of systemic inflammation with fever, increased CRP (C-reactive protein) and leukocytosis with left shift. For this stage, the diagnosis is clinical. The patient is treated conservatively and usually responds.

### *Conservative Treatment of Acute Diverticulitis*

Traditionally, patients with "mild" phlegmonous diverticulitis are admitted to the hospital; they are kept on nothing by mouth (nil per os, NPO) and on intravenous fluids. Wide-spectrum antibiotics are given and continued until local and systemic inflammatory manifestations subside. The colon, however, contains feces and will contain feces even after a few days of starvation. So, what is the rationale of the "traditional" regimen? We contend that in the absence of an associated intestinal ileus you may feed your patient or at least provide the patient with oral fluids instead of intravenous fluids. The same is also true concerning antibiotics: A perfectly adequate "coverage" of anaerobic and aerobic colonic bacteria can be achieved using oral agents such as metronidazole and ciprofloxacin. So, if intravenous therapy is not necessary, why admit the patient at all? And, in fact mild acute diverticulitis can be managed with oral antibiotics on an outpatient basis. Indeed, the necessity of any antibiotics for this condition is questioned. There are no randomized studies to demonstrate its advantage, but there are comparative studies that find no benefit with antibiotic treatment.

### *Complicated Diverticulitis*

In the minority of diverticulitis patients, local and systemic signs of inflammation will persist or increase over the next couple of days. This is when you should start considering the presence of complicated forms of diverticulitis. Now it is time to order abdominal computed tomography (CT; Chap. 5) to better define the pathological anatomy. Ambrosetti in Geneva has devised criteria to grade acute diverticulitis on CT in a clinically meaningful way (Ambrosetti et al. 1997):

- **Simple attack**: bowel wall thickness of more than 5 mm with signs of inflammation of the pericolic fat
- **Severe attack**: in addition, abscess, extraluminal gas, or leakage of contrast

About half of the patients found on CT to have a severe attack required an operation during the current admission or subsequent to it. Significantly, however, half of such patients did *not* require an operation, suggesting that CT findings are to be used **together** with the clinical picture in tailoring the proper management.

Should you order a routine CT of all patients suspected of suffering from acute diverticulitis? This is surely unnecessary overkill as most patients respond to conservative treatment. In addition, in some instances of clinically mild diverticulitis, the CT is negative.

#### Approach to Complicated Diverticulitis

A small number of patients present from the start with diffuse peritonitis, with free intraperitoneal gas on abdominal X-ray or CT (Chaps. 3–5). Here, of course, a CT scan is part of the modern standard of care. The CT scan will define the pathology so there is no question about the diverticulitis as the source of the peritonitis. The exception is the even smaller group with frank generalized peritonitis with increasing systemic inflammation accompanied by tachycardia, tachypnea, hypovolemia, oliguria, hypoxia, or acidosis for whom time is better spent in the intensive care unit for resuscitation and preoperative preparation (Chap. 6). The final diagnosis will then be established at the operation.

The CT manifestations of a **severe attack** (e.g., extraluminal gas, leakage of contrast, or abscess) in a patient who failed to resolve after a few days of antibiotics are not necessarily an immediate indication for an operation. Minor free intra-abdominal gas is also not an immediate indication for surgery if the patient is "stable." Instead, in the absence of spreading abdominal signs or systemic deterioration, even small (<5 cm) pericolic abscesses usually resolve without an operation (probably spontaneously draining back into the bowel). In such cases, we would therefore advise the continuation of conservative treatment under close clinical surveillance.

Larger pericolic **abscesses** (>5 cm) should be drained; this is best done percutaneously under CT guidance. After successful drainage, a semielective

resection of the sigmoid is recommended by some. We do not know, however, whether this is absolutely necessary since an unknown percentage of such patients would probably never develop another attack of acute diverticulitis.

## The Operation for Acute Diverticulitis

When you are forced to operate for acute diverticulitis, the procedure of choice is **sigmoidectomy.** It is usually best to open the abdomen with a lower midline incision, which should extend above the umbilicus to allow access to the descending colon and be extended further to reach the left flexure should it be necessary to mobilize it. The inflamed sigmoid has frequently folded itself into the pelvis, adherent to the left pelvic brim, and may rest against the bladder or uterus. At times, it will descend further into the pelvis between the rectum and bladder in the man and behind the uterus and upper vagina in the woman, depending on how deep the fossa is. The differential diagnosis of a perforated cancer easily comes to mind. A clue is to remember that the inflammation is always at the summit of the sigmoid loop. The rectum and the rectosigmoid junction anterior to the promontory are always unaffected. It is usually possible to reach the anterior rectum from the right side of the pelvis to identify the folding of the sigmoid. Try not to use sharp dissection in this inflammatory and adherent situation; using finger dissection is your best bet. Gentle finger pinching of the planes will separate the inflamed sigmoid from its attachments to the surrounding viscera.

This is not a cancer operation, and your aim is simply to remove the sigmoid colon, which is the source of the problem. Staying near the bowel wall helps you to stay out of danger, away from the left ureter and ovarian and spermatic vessels, which may be part of the inflammatory mass. It is best to start dividing the mesentery away from the inflammatory process below and above the sigmoid. After dividing and clamping (or using a linear stapler) the sigmoid at both ends, deal with the rest of the sigmoid mesentery. It is prudent to suture-ligate vessels within the thick, edematous mesentery rather then use simple ligatures that may slip. Using a vascular cartridge in a linear stapler to control the mesentery is another, albeit more expensive, alternative. Remove any residual blood, pus, or intestinal contents (Chap. 12) and consider the next step. (This contributor believes that the inflamed mesentery of the sigmoid should be removed as well.)

### *To Anastomose or Not?*

Should the two bowel ends be joined together, or is a Hartmann procedure with an end sigmoid colostomy to be preferred? An anastomosis is justified in the majority of patients, but there are a number of factors to consider. Localized peritonitis or an abscess is certainly not a contraindication to an anastomosis.

Generalized peritonitis is also not a contraindication in itself, but the surgeon needs to give it special consideration. Whether purulent or feculent, the generalized peritonitis signifies a greater insult to the patient, as reflected by the corresponding APACHE II (Acute Physiological and Chronic Health Evaluation II) score and the higher risk of dying (Chap. 6). Operative trauma adds to the postoperative SIRS (systemic inflammatory response syndrome) and MODS (multiorgan dysfunction syndrome) (Chap. 54). Most patients with generalized peritonitis due to perforated diverticulitis have an immunological defect that prevents localization of the process. Typically, they suffer from chronic obstructive lung disease or chronic arthritis with use of anti-inflammatory drugs or steroid dependence for years. Occasionally, they have received chemotherapy or are just recovering from major surgery, such as a coronary bypass. On the other hand, it seems that patients without such immunologic defects are capable of containing the inflammation and rarely have free peritonitis. Patients with free peritonitis will certainly not tolerate an anastomotic failure, and it is therefore all the better if there is no need to worry about the integrity of an anastomosis during the postoperative course. Therefore, in such patients we choose a **Hartmann's procedure:** sigmoidectomy, end colostomy, and closure of the rectal stump.

It is our impression that surgeons pay little attention to the consequences of the operative trauma added to the acute inflammation. We find surgeons blaming the unfavorable course of some of these patients on the diverticulitis and peritonitis, believing that residual infection is the problem. They should instead think about the operative trauma and postoperative SIRS. Consider this: if a sick patient is thrown out of the window (inadvertently, of course) and the surgeon then blames the subsequent course on the original illness, we would all say it is a misconception of the situation. The height the patient falls is the operative trauma. The longer the operation takes, the more dissection that is necessary, and the more bleeding it causes, the greater the operative trauma. This metaphor encapsulates the modern concept of damage control, and surgeons need to have a firm understanding of when enough is enough.

### *Fecology*

Reasonable amounts of feces in the colon are not a contraindication for an anastomosis. You can evacuate most of the fecal material from the left colon by milking it into a dish. Occasionally, however, the colon may contain large amounts of fecal material because the sigmoiditis has caused a relative obstruction in the days preceding the acute attack. Massive fecal loading is a factor against an anastomosis. To overcome this, it has been proposed that on-table antegrade bowel irrigation (through the cecum or appendiceal stump) be added to clean the colon before the anastomosis. Unless such irrigation is common practice in your hospital, with all the

equipment available, the irrigation will take at least half an hour and often much longer to accomplish. The subsequent anastomosis will add another 20–30 min to the operation. If this is the case, a stoma is quicker and gives better damage control.

In summary, consider an anastomosis in patients who are in reasonable health and without diffuse peritonitis. There should be no technical problems in making the anastomosis if the bowel ends are healthy and without tension. [For how to do it, see Chap. 13.]

## A Few Controversies

- Some surgeons believe that the inflamed mesentery should be anatomically resected together with the sigmoid, claiming that it usually provides for a better source control and anastomosis when there is no intervening mesentery left. Although the sigmoiditis affects the apex of the sigmoid colon, the mesentery is often shortened by the inflammation. The distal transection of the bowel should always be at the rectosigmoid junction because leaving a part of the distal sigmoid is the cause of recurrent diverticulitis. For these several reasons, some think that it is often better to resect the sigmoid in much the same way as one does a resection for cancer, and this is obviously the way to do it if one suspects the presence of cancer.
- Should the left flexure always be mobilized? No. This is indicated only in the minority of patients in whom the proximal colon fails to reach the rectum for a good anastomosis without tension or in patients in whom the blood flow in the marginal artery is uncertain. Diverticula of the descending colon are common, but we do not hesitate to anastomose diverticula-containing descending colon to the rectum. Recurrent diverticulitis proximal to the sigmoid is extremely rare.
- What should you do with phlegmonous diverticulitis accidentally discovered during operation with no frank perforation or suppuration present? Probably, you should do nothing at all, just close and treat with antibiotics. Most such patients will never return.
- Very rarely, the source of the perforation would be a *huge diverticular mass* densely adherent to, and obliterating, adjacent structures. (e.g., bladder, ureter, small bowel). A proximal diversion (with transverse colostomy or jejunostomy) and drainage of the perforation may be a safer management plan than resection, particularly when cancer is suspected or cannot be excluded. Definitive resection would become a much easier task later, when inflammation and infection have subsided.

## Newer Concepts

There were early reports of successful laparoscopic management with peritoneal lavage of perforated diverticulitis and generalized peritonitis—*without* the resection of the involved bowel. All patients recovered uneventfully and were well

during 12–24 months of follow-up. The concept that emerges is that the disease process can be reversed without a bowel resection, which can be postponed or not be performed at all. Larger experience has recently validated such an approach. This is so interesting that it calls for some details: in a prospective multicenter study (Myers et al. 2008), 100 patients with peritonitis from diverticulitis (the large majority had radiographic or CT evidence of free gas or fluid) underwent laparoscopy, during which they were found to have purulent peritonitis. Peritoneal lavage with 4 L of saline reversed the peritonitis without resection of bowel. Patients recovered and were discharged with only two instances of acute diverticulitis in the following 3 years.

With such results, we may be seeing the introduction of a new treatment principle with several advantages. It will also avoid the colostomy associated with the Hartmann procedure and the problems associated with its reversal. Only fecal peritonitis, which perhaps represents the true perforated diverticulitis, will require a Hartmann resection. All other forms, which respond well to resuscitation followed by laparoscopic lavage, seem to have a better prognosis than after a resection.

[**This nonresective approach seems so exciting that it calls for another opinion, which is offered by Dr. Jonathan Efron.—The Editors**]

Dr. Nyström raises the very exciting prospect of minimal intervention for perforated diverticulitis, which is indeed justified from recently published reports of great success with laparoscopic lavage for general peritonitis—without sigmoid resection. As greater than a third of stomas created emergently (when performing a Hartmann procedure) are not reversed, any procedure that reduces the morbidity of colostomy formation is to be applauded. I think, however, that several cautionary points need to be emphasized as our own very early experience with this technique has not been as successful as the reported data.

Laparoscopic lavage has not been performed in any of the published series for feculent peritonitis. Indeed, when identified, this was an immediate indication to proceed to sigmoid resection with Hartmann's pouch formation. Feculent peritonitis is commonly associated with an ongoing colonic perforation, with poor tissue quality making effective primary closure of the perforation difficult. With 30% mortality occurring in these patients, sigmoid resection with Hartmann's pouch formation is still the operation of choice. Similarly, patients with purulent peritonitis and significant signs of sepsis (hypotension, renal dysfunction, acute respiratory distress syndrome [ARDS], or pressor requirements) require removal of all potential sources of continued sepsis and therefore should undergo sigmoid resection. This same line of reasoning is applied to patients who have multiple comorbidities for whom persistent sepsis from ongoing diverticulitis may lead to further medical complications, and serious consideration should be given to sigmoid resection.

Stable patients who are suspected of having perforated diverticulitis are potential candidates for a laparoscopic exploration with lavage and drainage. On entering the abdomen laparoscopically, if the patient has minimal adhesions, exploration with four-quadrant irrigation should be performed. If there is a colonic defect or obvious perforation, an attempt should be made to laparoscopically close it; however, in most cases of purulent peritonitis no obvious "hole" in the colon is identified. Drains should be left in areas of an obvious abscess or near any suture repair; usually a 10F Jackson Pratt or Blake drain can be passed through one of the trocar sites.

Not all cases of diverticulitis are alike, and some simply do not respond to prolonged therapy with intravenous and oral antibiotics, and these cases will require surgical resection. This same rationale should be applied to patients for whom sepsis continues to progress after laparoscopic washout; in this case, sigmoid resection is required. Finally, thought should be given to resection with primary anastomosis and proximal diversion as opposed to Hartmann's pouch formation in many of these patients. As long as patients are stable at the time of surgical resection and the tissue quality of the proximal colon and rectum is sound, many feel it is safe to proceed with primary anastomosis and proximal diversion (or even without it). In the end, this may significantly decrease the number of patients who currently have to live with their "temporary stomas" forever.

[As a visible perforation is absent in most patients treated by laparoscopic lavage and postoperative fecal fistulas are not seen, it is clear that in most such patients control of the source of the purulent peritonitis has been achieved spontaneously—before the operation. We doubt, therefore, whether the lavage in these cases achieves more than what a course of antibiotics would achieve. We suspect that many of these patients who improve so rapidly with laparoscopic lavage and do not need any further surgery on follow-up did not need the laparoscopic lavage at all.—The Editors]

### After the Attack

Most patients with acute diverticulitis respond to conservative therapy; it is estimated that around one-fourth will experience a recurrence. Somewhat confusingly, this is variably interpreted as either confirming the need for elective surgery or indicating that the majority of patients do not require an operation. According to tradition, the second attack has been considered an indication for an elective sigmoidectomy, this being particularly true in the younger patient. Also, this approach has been recently questioned. Patients undergoing such elective sigmoid resections are not faring symptomatically better compared to patients who had no

surgery. In general, it is the initial attack that tends to complicate with "free" perforation, abscess formation, or fistula. Recurrent attacks tend to be relatively benign and to respond to medical treatment. The reasonable approach is to individualize the management. There will be a few in need of a sigmoid resection for persisting symptoms of fibrotic stenosis or complicating fistula, but the large majority would not benefit from elective surgery.

Looking at the "whole picture," it appears that we operate too early in acute diverticulitis, perform too many CTs, carry out too many percutaneous drainage procedures, remove too many colons, raise too many colostomies, reoperate electively on too many patients, treat too many with antibiotics, and perform too few randomized controlled trials in order to know what is right and what is wrong.

## Editorial Comment: Other Forms of Acute Diverticulitis

Although sigmoid diverticulitis is so common in our daily practice, other forms of diverticulitis should be kept in mind.

- With the horrendous amount of junk food consumed by "Western societies," we see a growing number of younger patients with colonic **pandiverticulosis** extending from the rectosigmoid junction to the ileocecal valve. Some of these present with acute diverticulitis in the right or transverse colon, which may mimic acute cholecystitis or acute appendicitis. The key to diagnosis here is an abdominal CT scan finding of a localized colonic phlegmon. This avoids unnecessary laparotomy and the temptation to proceed with colonic resection when the vast majority would respond to conservative treatment with antibiotics.
- "Solitary" cecal diverticulitis. This is a different entity: young, mostly male, patients present with one or two diverticula in the cecum—in the absence of diverticula distally. Once or twice a year, you will see a patient presenting with what you think to be "classical" acute appendicitis, but at operation you will find a cecal inflammatory mass or phlegmon of variable size. Free perforation and localized peritonitis are uncommon. On CT scan, a good radiologist should be able to distinguish cecal diverticulitis from acute appendicitis; if this is the case, you can treat conservatively as these patients would respond to antibiotics exactly like those with sigmoid diverticulitis. And, of course, recurrent cecal diverticulitis has been reported in conservatively treated patients. Most patients, however, come to operation either because CT is not done or its findings are mistaken for acute appendicitis. What to do at operation depends on the size of the process, ranging from diverticulectomy (place a liner stapler across the base of the diverticulum—including healthy cecal wall—and fire) to partial cecectomy (again, fire a stapler across and be careful not to narrow the ileocecal junction). Occasionally, when the diverticulum is situated just off the ileocecal valve, it is safer to excise and close the cecal hole by hand. Surgeons who are not aware of this condition or cannot recognize it are often carried

**Fig. 26.1.** "Which of these do we have to remove?"

away and perform right hemicolectomy. But, now you know that this is unnecessary. Surgeons who discover the process at laparoscopic appendectomy usually do not know what they see (one has to palpate it) and have to convert.

For the sake of completeness, let us mention here that acute diverticulitis *very rarely* affects patients with jejunal diverticulosis. These patients present with systemic signs of inflammation as well as with local peritoneal signs in the center of the abdomen. The key to diagnosis and subsequent nonoperative management and treatment with antibiotics (usually successful) is a CT scan, which shows an inflammatory mass affecting a segment of the jejunum and its mesentery. If forced to operate, all you have to do is a segmental small bowel resection and anastomosis.

Figure 26.1 will remind you that intestinal diverticula affect all of us; they may produce complications, but most can be treated without an operation. In the heart of Africa, you will rarely see a case of acute diverticulitis; people there do not yet eat the junk that we do.

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# 27 Massive Lower Gastrointestinal Bleeding

Per-Olof Nyström

*Whenever you encounter massive bleeding, the first thing to remember is that it is not* your *blood.*

*Massive bleeding* is defined as *exsanguinating* or hemodynamically significant bleeding that persists and requires at least four units of blood over a period of 24 hrs. Fortunately, truly massive bleeding from the colon and rectum is unusual. The vast majority of episodes of lower gastrointestinal bleeding (LGIB) are self-limiting and not hemodynamically significant. However, as with all types of gastrointestinal bleeding, never neglect it or think it is trivial until a period of vigilant observation tells you whether the bleeding is minor or major, whether it is likely to have ceased or is protracted.

## Sources of Bleeding

Most episodes of overt colonic bleeding never have the precise site and cause established. This is true even if colonoscopy is performed on the day of admission. Often, the bleeding is assumed to originate from an already-known pathology. Later, when the bleeding episode is over, a diagnostic workup may reveal previously unknown pathology as the cause or suggest, in retrospect, a lesion that may have been the source. Table 27.1 shows the most common causes.

A short comment about the causes mentioned in the table may help you to choose the most likely cause in your next patient with colonic bleeding. **Neoplasms**, whether cancer or benign polyp, rarely bleed massively but often have occult bleeding that can produce significant anemia. Rectal cancer commonly bleeds overtly and if associated with anemia can at first suggest a massive bleed until rectoscopy is performed. The patient with rectal cancer may give a history of tenesmus, and usually there will have been episodic minor bleeding with the stools for some time. Bleeding in **inflammatory bowel disease** (IBD) is

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**Table 27.1.** Causes of colorectal bleeding (not listed in order of frequency)

| |
|---|
| Neoplasm |
| Inflammatory bowel disease |
| Diverticulosis—diverticulitis |
| Ischemic colitis |
| Vascular malformation—angiodysplasia |
| Hemorrhoids |
| Postoperative—anastomotic |
| Meckel's diverticulum |
| Infectious |

almost never the first symptom of the disease and is rarely massive (› Chap. 24). The diagnosis will be known in most such patients, and the bleeding is associated with an exacerbation, for which diarrhea precedes the bleeding by several days. The exception is proctitis, which may present with bleeding, again easily identified at rectoscopy. The differential diagnosis of proctitis includes **infections** such as *Campylobacter* or amebiasis. The onset is then more sudden, with diarrhea and bleeding beginning together. **Radiation proctitis** may bleed significantly, but here the history is obvious.

**Diverticula** of the sigmoid colon are assumed to be the most common cause of acute major LGIB. Naturally, this occurs more often in elderly patients, particularly in those taking nonsteroidal anti-inflammatory drugs (NSAIDs) or anticoagulants. In middle-aged patients and also elderly patients with no other reason for hemorrhage, you must consider mucosal **angiodysplasia** as the possible explanation. The bleeding can be massive and recurrent. In elderly patients, **ischemic colitis** can rarely present with massive bleeding. **Postoperative** bleeding from colonic anastomosis, polypectomy, or after anal surgery should be easily identified. And finally, do not forget that internal hemorrhoids may bleed copiously; you do not want to diagnose an anal source only after laparotomy.

## Diagnosis

We find it very annoying to consult on bleeding patients for whom the referral note simply states: "Patient has melena." Anything can hide behind such a note. It tells us that not a lot of thought was invested in this request. There are two very

powerful tools to help you: the patient's history and the rectoscope. First, find out whether the blood is pink-fresh blood or maroon-almost-fresh blood. These two represent **hematochezia** (bloody stools) and signify a colonic (common) or small bowel (rare) source. We need not remind you that tarry black stools of **melena** signify an upper gastrointestinal (UGI) source above the ligament of Treitz (Chap. 17). Remember that with massive UGI hemorrhage and rapid intestinal transit, unaltered fresh blood may appear in the rectum. Insertion of a nasogastric tube followed by gastric irrigation may quickly direct you to a gastric bleed but always remember that bleeding duodenal ulcers may not show blood in the stomach (Chap. 17).

### *Rectoscopy*

For all cases of hematochezia, rectoscopy is the mandatory first step. It is amazing how often this step is omitted in "modern" practice—how often we see patients immediately referred instead for a "panendoscopy." Use a rigid rectoscope because the flexible instrument will be coated rapidly with blood, and you will see nothing. Have a good suction device available. It is not unusual to discover that there is simply too much blood to really see anything (Fig. 27.1). If blood can be aspirated and you do get to see the rectum, simple things like a rectal cancer or proctitis should be obvious. Do not decide on a diagnosis of proctitis too lightly because the mucosa may look all red from the fresh blood. The mucosa should be swollen, and there should be no visible mucosal blood vessels. The proctitis is often so distal that the margin between inflamed and


**Fig. 27.1.** "Hey, are you sure that all of this is coming from above?"

normal mucosa can be seen. The redder the blood is, the closer to the anus the source. Bleeding from the upper anal canal and lower rectum will reflux at least to the rectosigmoid junction, so do not be fooled by finding fresh blood at that level. If you have a good view, when there is not too much bleeding, fresh blood may be seen flowing on the wall or dripping from above, in which case bleeding from a more proximal source is likely. Quite frankly, in most patients with active bleeding you will not be able to see much at rectoscopy. But, at least you have the opportunity to exclude an anal source and to observe personally the character and magnitude of the bleeding.

Let us forget, at this stage, the majority of patients in whom the bleeding stops spontaneously. They will be further investigated with a colonoscopy performed in a well-prepared bowel. Let us concentrate instead on that problematic minority of patients—those bleeding massively or continuing to bleed. In such patients, more aggressive measures will be needed.

### *The "Sophisticated" Means of Diagnosis*

There are two "sophisticated" means of diagnosis in this situation: **technetium-labeled erythrocyte scan and mesenteric angiography.** Which of the two should be chosen roughly depends on the intensity of the bleeding and the availability of either test. The more profuse the bleeding, the better it is to start with angiography. Not only will it define the site of the bleeding, but also the bleeding vessel may be treated by embolization through the angiographic catheter. Both investigations require active bleeding at the time of the procedure; do not waste the radiologist's time with a non-bleeding patient. In angiography, the "window" for detecting active bleeding is just a few seconds; in scintigraphy, it is substantially longer. Many sources bleed intermittently, and the site may be "quiet" at the time of angiography. Remain vigilant after a negative study because it does not signify that the bleeding episode is over.

### *Emergency Colonoscopy*

To be of any diagnostic help, the colon must be cleaned as for an elective endoscopy. Oral cleansing solution in excess of 4 L is required and often mandates a nasogastric tube to make the patient ingest the volumes needed to produce a "clean" distal effluent. This investigation will identify the bleeding site with certainty in less than half the instances, but if seen the site can be treated with adrenalin injection or a Hemoclip. Endoscopy is probably more valuable in sigmoid diverticular bleeding than more proximal sites. However, few hospitals have an

endoscopy service experienced in dealing with massive colon bleeding out of hours. What about your hospital?

## The Operation

This is how to proceed if you elect to perform a laparotomy on a patient who fails to settle: make a quick examination of the colon to exclude obvious pathology. Then, inspect the small bowel, which may contain blood even if the bleeding comes from the right colon, although it would be unusual for the blood to regurgitate throughout the entire small bowel. If you find blood in the upper small bowel, direct your investigation to the UGI tract. Blood in the right colon, but not small bowel, does not definitely identify the bleeding as being in the right colon because blood will reflux long distances in the colon. Make your guess based on what you find because now comes the really difficult part. Are you going to take a chance on a right or left colectomy? Do you trust the preoperative localizing studies—if performed? Or, can you identify the bleeding spot with certainty? Not even if you open and clean the colon can you be sure to see the bleeding site. It is messy and takes time, which is a reason why traditional teaching proposed the "blind" right hemicolectomy (assuming angiodysplasia as the cause).

There are instances when the colon is so full of blood that a total or subtotal colectomy is the reasonable procedure. Temporary clamping of the three main vessels to the colon will reduce the bleeding while you mobilize the colon. What few statistics there are suggest that segmental resection is associated with higher rebleeding rate (no surprise), while subtotal colectomy has higher mortality. It is a delicate balance of judgment.

**A recent case of mine**

A healthy, 32-year-old woman presented repeatedly with significant maroon rectal bleeding; sometimes she had a hemoglobin around 6 g/dL. The bleeding episodes always stopped spontaneously. All attempts to visualize the bleeding site with certainty failed. The only positive finding was angiodysplasia of the right colon and terminal small bowel, so she was subjected to elective exploration with intraoperative endoscopy; right ileocolic resection was performed. A few months later, she was again admitted with a significant LGIB. This time, however, the bleeding did not stop completely, but it was observed that she passed normal-colored stools together with fresh blood, strongly suggesting a bleeding site at the anus, possibly hemorrhoids. Always think outside the box—everything is possible—and do not forget the humble hemorrhoid.

### A Pathway to Reason [The Editors]

The experience with, and perception of, LGIB differs slightly from one surgeon to another. This is understandable if one realizes that all published data on this topic are retrospective studies on poorly stratified patients. So, this is what we think:

- Let's face it—in nine-tenths of patients with LGIB, the bleeding stops spontaneously. Emergency localizing tests are unnecessary in this group; elective colonoscopy is indicated. Hysterical MDs tend, however, to overinvestigate this group—jumping on them with isotope scans and angiograms, all useless when the hemorrhage is not active.
- Each of us operates perhaps once or twice a year on "massive" lower LGIB (>4–6 units of blood over 24 hrs), which continues. Therefore, the collective experience of each hospital is small, not allowing any meaningful prospective studies. All that is published on this subject is therefore retrospective and biased by local dogma and facilities.
- Reports by radiologists boasting about high accuracy rates of isotope scans and angiography are often meaningless because such reports do not discuss the clinical benefit of such accuracy; that is, did it change the management and how?
- Most massive LGIB in elderly patients is either from colonic diverticula (in the left or, less commonly, the right colon) and angiodysplasia (usually of the right colon). True, angiodysplastic lesions are common, but we do not know how often they bleed. It is our impression that colonoscopists often overdiagnose these lesions as the source after the hemorrhage has ceased, whereas the true source of bleeding was elsewhere (e.g., diverticular).

**Based on these considerations, this is how we would approach a LGIB:**

- Start with supportive care. Exclude UGI bleeding. There is no need for a routine UGI endoscopy as fresh blood per rectum in a stable patient means that the source is not in the UGI tract. Do a rectoscopy to rule out an anorectal source.
- When the patient requires the second and third unit of blood, it is time to get a little excited. Angiography at this stage is indicated; if it localizes the source of bleeding in the left or right colon, so much the better. If it fails, it is not a big deal. An isotope scan requires time and anyway is clinically almost useless in actively bleeding patients. Blood migrates within the lumen of the colon and so does the extravasated isotope. We do not value this investigation. (Nuclear medicine = unclear medicine.)
- When the patient is on the fifth or sixth unit and blood is still dripping from the patient's rectum, it is time to take the patient to the operating room. If angiography has localized the source in either the left or right colon, we do a segmental colectomy—either right or left hemicolectomy. If angiography is not

available or is nonlocalizing, we do a subtotal colectomy with ileorectal anastomosis. "Blind" segmental colectomy may produce a rebleeding patient who will not tolerate a major reoperation.

- A few authors have described intraoperative colonoscopy after "on-table" colonic lavage. Theoretically, it appears attractive, but practically it is messy and time consuming. If the hemorrhage has stopped, it will not show much; try it and see what angiodysplasia is and what just some old clotted blood looks like.
- There is no doubt that in practice we are overinvestigating these patients and often waiting too long prior to operation. The bleeding either stops or continues; when it continues, you must operate—on a well-resuscitated patient who has not been allowed to deteriorate in a medical ward. A fast subtotal colectomy is a safe, definitive, and lifesaving procedure.

Whether we are right or wrong depends on which articles you read, on what you believe, your local facilities, and your own philosophy. We hope you will adopt ours.

Beware: in lower gastrointestinal bleeding, removing the wrong side of the colon is embarrassing. Removing any segment of the colon while the bleeding source is in the anorectum is shameful.

# 28 Acute Appendicitis

Moshe Schein · Ahmad Assalia

*Theoretically it would seem to be much better if we would cut down upon the appendix as soon as the diagnosis was tolerably certain, tie it above the seat of perforation, and remove from its neighborhood any concretion or decomposing material that might be the cause of irritation.* (Samuel Fenwick, 1821–1902)

*We all know: "Whatever the clinical presentation, whatever the abdominal findings, always keep acute appendicitis at the back of your mind."*

Acute appendicitis (AA) is discussed in any surgical text dating from the turn of the nineteenth century. Looking at the lengthy chapters devoted to this subject, we often wonder what there is to chat so much about. Knowing that you have been fed on AA *ad nauseum* since the early days of medical school, we do not intend to repeat here the whole "spiel" again. Instead, we promise to be brief and not to bore and perhaps teach a few things that have previously escaped you.

## Diagnosis

Acute appendicitis is an inflammation and subsequent infection of the appendix. This rudimentary structure varies in length and position, making matters complicated. Even a dentist (but not a gynecologist) can diagnose a case of *classical* AA (Fig. 28.1); the history of midabdominal visceral discomfort, shifting to the right lower quadrant (RLQ) and becoming a somatic, localized pain speaks for itself. Add to it the clinical and laboratory evidence of systemic inflammation/infection and, most important, the localized physical findings of peritoneal irritation. Unfortunately (or fortunately, otherwise dentists would be treating AA), for each classical case you will see two atypical cases. Sure, you know by now that the diagnosis of AA tends to be missed at the extremes of age, that in fertile females it is often confused with gynecological conditions (Chap. 33), that retrocecal and pelvic appendices tend to be more "silent," and that it should be "always on your

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mind"—at least second on your list of differential diagnosis. So, what can we add that you do not know? Perhaps nothing, but let us emphasize a few points:

- Never confirm or exclude the diagnosis of AA on the presence or absence of one or another symptom, sign, or finding "that must be there" because such an obligatory variable does not exist. Instead, suspect AA from a synthesis of the whole clinical picture and the various laboratory tests.
- New "diagnostic scores" for AA are popping up in the literature almost every year. But to us, what they describe is intuitive and thus clinically useless. We know that obvious RLQ peritonitis in a male teenager, accompanied by nausea and vomiting, fever, and elevated white cell count means AA. And of course, AA is much, much less likely in the fertile woman, presenting with minimal RLQ tenderness, preserved appetite, no fever, and normal white cell count—but can we absolutely rule out the possibility of AA in such a lady? No. So, those scoring systems are a good justification for writing papers—that is all.
- Every budding surgeon feels compelled to design a personal screening test for AA: the "cough test," the "jump sign," the "please bring your tummy to my finger test," and many others. They are all fun, but none approaches a sensitivity or specificity of 90% (oops, sorry, we promised not to use percentages). The truth is that it is impossible to be completely accurate in the clinical diagnosis of AA. Should your policy to operate be based only on clinical assessment and basic laboratory values, then one or two out of ten removed appendixes will be a normal, "white" appendix (in fertile females, this proportion will be much higher). More than that implies that you are a cowboy; fewer suggests that you are dangerously conservative.


**Fig. 28.1.** Even a dentist can diagnose classic appendicitis

So, you seriously suspect AA after having excluded, or at least you believe so, a gynecological complaint, urological pathology, gastroenteritis, the nebulous "mesenteric lymphadenitis," or the trash bin called "nonspecific abdominal pain." Should you now proceed directly to the operating theater or order fancy imaging?

> "Mesenteric adenitis" is code for "I thought it was appendicitis, but the appendix was normal." (David Dent)

## Caveat

The management of patients with suspected appendicitis has traditionally focused on the prevention of perforation by early operation at the expense of a high proportion of unnecessary operations. But, despite an increase in use of modern diagnostic modalities, the rate of perforation has not declined. Furthermore, population-based studies document that diagnostic accuracy decreases as the rate of appendectomy increases, *but the incidence of perforation does not change*. This teaches us that perforation is a different disease. Patients come to the hospital with perforation; they do not perforate while we investigate them or observe them. Sure, sometimes we miss a "masked" perforation, but that is another story.

> The proportion of perforated appendicitis is not a good measure of quality. The proportion of perforations may increase because you operate on fewer patients with nonperforated appendicitis. A high proportion of perforations may in fact be a good thing because it means you operate only on those patients who need surgical treatment. (Roland Andersson)

## Abdominal Imaging in Acute Appendicitis

While it is clear that we cannot modify the rate of perforated appendicitis (one in four will be perforated), we can decrease the number of unnecessary, negative appendectomies. It has been said that "a fool with a tool is still a fool." Indiscriminate and nonselective usage of modern diagnostic technology is not going to change this observation. What is needed is common sense and rational deployment of available investigations. Frankly, managing at least one case of AA per week, I do not recall when last I [MS] removed a normal appendix or missed an abnormal one. But then again, don't all my patients do well? ☺

**And, this is how I [MS] do it:**

1. Male patients with typical presentation: operate immediately or the next morning.
2. Male patients with atypical presentation: serial re-examinations; if not better or still atypical, order a computed tomographic (CT) scan (see Chap. 5).
3. Females in the reproductive age with typical presentation: start with transvaginal ultrasound (US), which frequently detects ovarian pathology (Chap. 33) and fluid in the pouch of Douglas to explain the clinical picture. If US is not helpful, they are sent for a CT.
4. Females with atypical presentation: see points 2 and 3.
5. As these approaches differentiate between those who need an operation and those who do not, I see no sense in using laparoscopy as a *purely* diagnostic tool. Diagnostic laparoscopy per se is a costly and invasive operation (some call it "controlled penetrating abdominal trauma") and despite assertions that normal appendices discovered during laparoscopy should be left alone, most surgeons still feel uncomfortable with this approach. Thus, commonly, "negative laparoscopy" means "negative appendectomy." And in fact, studies of laparoscopic appendectomy (LA) report a much higher rate of negative appendectomies.

In good hands, **US** has been reported to be accurate in the diagnosis of AA and is useful in excluding other diagnoses, which may require a different therapy (e.g., hydronephrosis), incision (e.g., acute cholecystitis), or indeed no therapy at all (e.g., ovarian cyst). Most of us do not work in an institution where we can be so confident of the radiologist's diagnosis of appendicitis on the basis of US. The value of **CT** in diagnosing conditions that mimic AA but may not need operative treatment (e.g., cecal diverticulitis; Chap. 26) is emphasized in Chap. 5.

### *Periodic Re-evaluation*

Many of you, however (in developing countries or in some "bush," for example), do not have a CT scanner readily available and thus cannot follow the advice given. This does not mean that you should have a high rate of negative appendectomies. *Periodic reevaluation* is a time-honored and proven diagnostic modality in the doubtful case. Unfortunately, the art of periodic re-examination and the virtue of patience are disappearing from the scene of modern practice, in which the emphasis is on obsessive activity and to prove oneself one has always to "do something." In the absence of clear peritonitis and toxicity, very rarely are attacks of AA a true emergency requiring an immediate operation. If undecided, admit the patient and periodically re-examine him or her over the day or night. In most instances, AA will declare itself, and if it is not AA, the "attack" will resolve. **Patients**

**do not perforate under surgical observation; but occasionally they lie with neglected perforations in the emergency room or pediatric wards.**

(Note: if you decide to observe the patient, do not administer antibiotics as they may mask the findings or "partially treat." However, they may cure the AA in most nonperforated cases, which in some specific circumstances could be a good idea.)

So, we order imaging selectively. Unfortunately, on our side of the Atlantic the diagnostic algorithm is increasingly driven by dogmatic emergency room personnel who perform CT scans in lieu of clinical evaluation. Such indiscriminate use of CT scanning leads to a new syndrome we call "CT appendicitis": you admit for observation a patient with RLQ pain and ambiguous clinical findings. Meanwhile, the emergency room doctor orders a CT, which is reported by the radiologist the following morning. At this stage, the patient feels much better, his or her abdomen is benign, and the patient wants to go home, but the radiologist claims that the appendix is inflamed ("cannot exclude …" or "suggestive of …"—these are the ambiguous terms they like to use to cover their ass). *But, should we treat the CT image or the patient?*

## Classification

Let us bring here a simple classification of AA to facilitate the discussion of management. In essence, AA is either **simple** or **complicated**. "Simple" AA implies inflammation of the appendix of any extent in **the absence of appendiceal gangrene, perforation, or peri-appendicular pus formation**. Define AA as "complicated" whenever any of these changes is present.

Another entity you should be familiar with is the **appendiceal mass**, developing late in the natural history of AA. The "mass" is an inflammatory phlegmon made of omentum or adjacent viscera, walling off a complicated appendicitis. A mass containing a variable amount of pus is an **appendiceal abscess**.

## Management

### *Antibiotics*

Judicious administration of antibiotics, to cover **Gram-negative and anaerobic** bacteria, will minimize the incidence of postoperative wound (common) and intra-abdominal (rare) infective complications. In simple AA, the antibiotics are considered **prophylactic**, while in complicated AA they are **therapeutic**. We encourage you to administer the first dose of antibiotics preoperatively just before you scrub. If at surgery the AA proves to be simple, no postoperative administration is necessary. Should you, on the other hand, discover complicated AA, additional postoperative doses are indicated. We suggest that you tailor the duration of

administration to the operative findings. Gangrenous AA, without any pus formation, represents a "resectable infection," which does not require more than 24 hrs of postoperative administration. Perforated AA with or without intraperitoneal pus should be treated longer, but **for no more than 5 days**—unless serious infectious complications develop. (Chaps. 7, 12, and 47).

### *Nonoperative Management of Acute Appendicitis*

Perhaps you are not aware that most attacks of simple AA respond to nonoperative management with antibiotics. Complicated AA may also respond to antibiotics or at least could mature into an abscess. So, why not treat most cases of AA initially conservatively, along the same lines as acute diverticulitis (Chap. 26) of the sigmoid colon? This is because the surgical management of AA is simpler and less morbid than that of diverticulitis and because we are dogmatic. However, there are several situations for which you should consider a nonoperative approach:

- In a patient who had just suffered a myocardial infarction
- In a morbidly obese patient
- During the first weeks of pregnancy
- On submarines
- On a trip to Mars

In 1961, Leonid Rogozov (1933–2000), a young Russian surgeon, on an Antarctic expedition, had to remove his own inflamed appendix—he used a mirror! However, in this day and age we would recommend antibiotics rather than autoappendectomy.

Also, the preferred management of an appendiceal mass (phlegmon) is conservative as discussed separately in this chapter.

## The Operation

"The appendix is generally attached to the cecum." (Mark M. Ravitch, 1910–1989)

"The point of greatest tenderness is, in the average adult, almost exactly 2 inches from the anterior iliac spine, on a line drawn from this process through the umbilicus." (Charles McBurney, 1845–1913)

### *When to Operate?*

You do not have to rush to the operating room as soon as possible with each patient diagnosed with AA. Obviously, if your patient is systemically sick and the abdominal findings are impressive (denoting a perforation), operate immediately.

Otherwise, a delay of a few hours while the patient receives antibiotics is acceptable. You do not rush to the operating room with acute diverticulitis (Chap. 26), so what is the difference?

### *Open Versus Laparoscopic Approach?*

As pointed out, liberal use of diagnostic laparoscopy for suspected AA leads to a high incidence of unnecessary removal of normal appendices, procedures that are not free of complications. But, what about LA if the diagnosis has been established? Evidence suggests that—and this is how one can summarize the voluminous literature steadily being published on this controversy—compared to the open procedure, LA is associated with some reduction in postoperative pain, marginally earlier discharge, and lower incidence of wound infection. However, LA is associated with a higher risk of intra-abdominal infective complications when performed for complicated AA. Concerning costs, the money saved by an earlier discharge after LA is spent on a more expensive and longer procedure. It appears thus that surgeons who prefer open appendectomy (OA) (MS among them) have the support of the literature, but it does not mean that they should avoid LA altogether; it surely has a place in very obese patients (avoiding a large incision) or in those with nonperforated appendicitis who specifically demand the laparoscopic approach.

### *Technical Points*

The open procedure is discussed here. Should you prefer to play with gas, sticks, and staplers, help yourself. (See the commentary in this chapter by coeditor AA.) We presume that you have done your share of appendectomies already as an intern. However, having seen many surgeons transform a customary appendectomy to an elaborate operation resembling a Whipple's procedure, we remind you of the KISS principle (keep it simple, stupid! ☺)

- **Incision**: you do not need the long, unsightly oblique incision except in the muscular young man whose possible retrocecal appendix may be unreachable through a transverse hole. Use the *transverse* one. A common error is to place it too medially over the rectus sheath; stay lateral to it. Incise the fascia, split the muscles, and open the peritoneum. Start with a mini-incision; it can always be extended by cutting the lateral edge of the rectus fascia or muscle.
- **Appendectomy**: you can remove the appendix in an antegrade or retrograde fashion, but there is no need to invert the stump unless you are hooked on useless rituals. Just ligate or suture-transfix the appendix at its base and chop the rest off. When the tissue is friable, overrun the divided mesoappendix with a running suture. The commonly performed fetishes of painting the stump with Betadine or burning it with diathermy are ridiculous. If the appendix has perforated just at its

base, to secure the stump safely you have to include in it some healthy cecal wall; just place a linear stapler across the cecum distal to the perforation and fire.

- **Peritoneal toilet**: just suck out the fluid with a Poole sucker and mop up whatever pus is present with a dry gauze stick (do not forget the pelvis). Peritoneal lavage through this keyhole incision is useless. But, if you are a dedicated irrigator, limit the lavage to the affected area (i.e., pelvis, peri-cecal). Why would you want to spread bacteria all over the abdomen?
- **Drains**: drains are never necessary. In theory, they may be indicated after the drainage of a large appendicular abscess, but in practice we never use them and have never regretted it.
- **Closure**: theoretically, closing the peritoneum is unnecessary because it adds no strength to the repair, and we know that the peritoneum repairs itself within 48 hrs; however, this step "covers" the bulging viscera, facilitating careful closure of the abdominal wall layers. Next, the muscles are approximated loosely with a few sutures of 2–0 vicryl to obliterate dead space, the fascia is closed with running 0 PDS, taking large bites at both edges. The subcutaneous layer, if thick, may be approximated with a few fine sutures of vicryl.
- **Instillation of an antibiotic** in the fat protects against wound infection (in addition to systemic administration). Our bias is for primary closure of the skin in all cases. A few will develop wound infection managed by removal of (a few) stitches. Isn't this better than delayed secondary closure, which condemns all patients to further manipulations and an ugly scar (› Chaps. 43 and 55)? We close the skin in continuous subcuticular fashion in cases of noncomplicated appendicitis; in complicated ones, we use interrupted nylon 4–0.

## The White Appendix

What should you do when the appendix proves to be pristine (white)? Well, you can rub it to allow the pathologist to diagnose mild acute inflammation (just kidding). The classical dictum is that whenever an abdominal appendectomy incision exists the appendix should be removed in order not to confuse matters in the future. What about a normal appendix visualized at laparoscopy? Should it also be removed? The emerging consensus is to leave it alone, informing the patient or the parents that the appendix has been left in situ. However, most laparoscopists do not feel comfortable with this recommendation, always worrying that what appears normal through the video camera may prove diseased at histology. Thus, for most surgeons, diagnostic laparoscopy for suspected appendicitis leads to appendectomy regardless of whether the appendix is normal or diseased.

Obviously, when the appendix appears normal you should search for alternative diagnoses such as Meckel's diverticulitis (› Chap. 35), adnexal pathology (› Chap. 33), perforated cecal diverticulitis (› Chap. 26), or mesenteric lymphadenitis (whatever that is). In most instances, however, you will find nothing. What

should you do if foul smelling, murky, or bile-stained peritoneal fluid is encountered, suggesting serious alternate pathology elsewhere? Bile should guide you into the upper abdomen. Close the incision and place a new one where the action is. Feces or its odor direct you toward the sigmoid; just extend the incision across the midline, and you are there. But of course, a preoperative CT (or laparoscopy) would have saved you all these old-fashioned headaches.

### The "Valentino" Appendix

Intraperitoneal inflammation from any cause can inflame-inject the appendix and the adjacent parietes from the outside, mimicking AA. This was the case with the famous movie actor and womanizer Rudolph Valentino, who underwent an appendectomy for suspected AA in New York (1926). He became gravely ill after the operation and died; autopsy revealed a perforated peptic ulcer. Old texts teach us that contaminants forming in the right upper abdomen tend to spread down along the right gutter into the lower abdomen, thus producing a misleading clinical picture. Therefore, the findings of peritoneal fluid and suppuration together with a mildly inflamed and nongangrenous and nonperforated appendix should raise your suspicions that the pathology is elsewhere. Look for it!

### The Post-appendectomy Appendiceal Stump Phlegmon

Your patient had an uneventful appendectomy for AA following which the patient happily went home. Seven days later, the patient re-presents with RLQ pain, a temperature, and high white cell count. The wound looks okay. This is a typical presentation of an appendix stump phlegmon. Now, the diagnosis is simple; a CT will demonstrate a phlegmon that involves the cecum, as opposed to a drainable abscess. A few days of antibiotic therapy will cure this relatively rare complication, which for some reason is not mentioned by standard texts.

**Stump appendicitis**: be aware that patients can develop classical AA at any time *after appendectomy*. Historically, this followed appendectomy for complicated appendicitis, often by a relatively inexperienced family doctor or surgeon. It is now becoming more common in the era of *LA*; during the procedure, surgeons may misidentify the cecal base of the appendix and consequently leave a longer than usual appendiceal stump, which is prone to stump appendicitis and requires a re-appendectomy.

#### *Fecoappendicopathy or the Painful Fecalith*

The patient presents with clinical features of early appendicitis. Abdominal X-ray may show a large fecalith. CT shows a fecalith or a grossly dilated appendix

with no surrounding inflammatory features. At operation, the enlarged appendix does not appear inflamed, but when you open it (after removal) you will find it stuffed with feces like a sausage or containing a large hard fecalith. So, do not feel bad when the pathology report mentions no "acute inflammation"—the distended appendix was responsible for the patient's symptoms, and the appendectomy was indicated.

## Appendiceal Mass (Phlegmon)

Typically, patients with an appendiceal mass present late in the course of the disease, with abdominal symptoms lasting a week or more. Occasionally, they report spontaneous improvement in their symptoms, reflecting localization of the inflammatory process. This is also more common in diabetic patients. On clinical examination, you will find a right iliac fossa mass. Overlying tenderness or obesity may obscure the presence of the mass. Therefore, suspect an appendiceal mass in the "late presenters" or those with an atypical smoldering picture. When palpation is not rewarding, obtain a CT scan, which is the best way to document an appendiceal mass. Another indication for CT is associated evidence of undrained pus, such a spiking fever and toxicity, signifying an **appendiceal abscess**.

Why should you distinguish between AA and appendiceal mass (or abscess) if the management of these conditions is the same (i.e., operation and antibiotics)? **This is because the appendiceal mass (and abscess) can (and should) be managed nonoperatively.** You could operate on both, as you operate on AA, but removal of the appendix involved in an inflammatory mass may be more hazardous than usual, occasionally necessitating a right hemicolectomy. On the other hand, conservative treatment with antibiotics leads to resolution of the mass in the vast majority of cases. Failure of the mass to respond to antibiotics signifies an abscess. CT or US-guided percutaneous drainage is the most rational approach (› Chap. 49). Failure to improve clinically within 48 hrs means that an operation is needed. At operation, drain the pus and remove the appendix if it is not too difficult.

### *Interval Appendectomy?*

As no more than one of ten patients treated conservatively for appendiceal mass will suffer a recurrence of AA (usually within 1 year and not a "complicated" attack), the dogma of routine interval appendectomy within 6 weeks has become obsolete. Interestingly, in many of these patients at interval appendectomy the appendix is found to be rudimentary and scarred. In patients over the age of 40 years, we suggest elective colonoscopy/colonic imaging after 3 months to exclude

the rare situation in which cecal carcinoma was the cause of the mass. Cecal cancer or inflammatory bowel disease will be detected in only 1 of 100 such patients.

With a high degree of suspicion, you can avoid an operation in the majority of patients with an appendiceal mass. *And remember: an appendiceal mass represents an unfavorable situation for your laparoscopic skills.*

## Appendicitis Epiploica (Appendagitis)

We mention the appendicitis epiploica condition here because of its name, because you probably have not heard much about it, and because it is not so rare and often imitates AA. Appendicitis epiploica (some call it "**appendagitis**") follows a spontaneous torsion of an appendix epiploica, the peritoneum-covered tabs of fat attached along the tenia coli. It is more common in obese individuals and in the cecum and sigmoid. Since the sigmoid colon often crosses the midline, the most common manifestation is localized tenderness and peritoneal signs in the right iliac fossa. Typically, patients do not feel or appear sick despite these findings. Thus, "AA on examination" in an afebrile and healthy-looking patient should raise your suspicions. The natural history is spontaneous remission as the appendix epiploica sloughs off, transforming into that loose calcified peritoneal body that you occasionally find during unrelated abdominal procedures. CT scan may identify the localized area of peri-colonic inflammation (Chap. 5). If you are misled into an operation, just remove the necrotic piece of fat.

## Laparoscopic Appendectomy

Ahmad Assalia

The open approach described is acceptable to most surgeons. But, I see things a bit differently and I would opt for LA in virtually every case of early AA. According to the current evidence and my personal experience, LA has immediate advantages as well as late ones. The immediate ones include less pain, speedier recovery (although not that dramatic compared to OA), and significantly decreased rates of wound infection. The long-term advantages include better cosmetic results (we cannot overlook this, especially in young women), lower rates of wound pain and hernia, and fewer adhesions, which is important for prevention of future small bowel obstruction and fertility problems in young women. The allegedly high rates of intra-abdominal infection in advanced cases of AA are no longer found in newer studies and in competent hands. On the top of this, let us not forget the preferences of patients in this modern era. As for the duration of operation, in experienced hands LA should not take longer to complete than OA.

There is no doubt that all of this is true provided you have solid laparoscopic skills. If you do not have them, play it safe and proceed with OA. This is good clinical practice, and no one will sue you for not doing LA, especially since there is still some controversy surrounding the issue.

I do agree with the notion that in the young thin male an OA with a keyhole incision is totally acceptable. My attitude dictates that if you are comfortable with LA, every case is suitable unless there is some problem with the laparoscopic approach, namely, previously operated lower abdomen, a patient on aspirin, advanced AA and generalized peritonitis.

Although in experienced hands virtually every case of AA is doable laparoscopically it should be emphasized that in the following conditions LA is preferable beyond any controversy (well … almost): young fertile women, obese patients, and cases with unclear diagnosis even after imaging (yes, there are such cases). The laparoscopic approach enables you easily to explore the whole abdomen, including the small bowel, the female genital organs, and every other possible pathology. Feel free to convert when you are not comfortable with the anatomy or have any possible conflict with the right ureter, uncontrollable bleeding, or an injury to the small bowel or cecum. This reflects a victory of your judgment over the inflated ego of most of us.

### *Technical Tips for Laparoscopic Appendectomy*

1. Make sure that the patient has voided shortly before the procedure and do not let your anesthetist overload the poor patient with fluids; a full bladder will interfere with your vision, and you may perforate the bladder while inserting your lower trocar.
2. The patient should lie supine with the left arm adducted. This will enable you and your assistant both to work on the patient's left side.
3. Secure the patient above the knees to the table. This will prevent the patient from sliding while rotating the table to the left and in steep reverse Trendelenburg position.
4. Use a good 5-mm scope. This will enable you to move the scope between the trocars for later retrieval of the appendix.
5. There are many ways to insert the trocars, depending on the habitus of the patient, but I find the following the most useful: place the first, 10-mm trocar, just under the umbilicus for the scope and later removal of the appendix; the second, a 5-mm working trocar is placed in the LLQ or suprapubic position; the third, a 5-mm working trocar, is at the upper midline, about 5–6 cm above the umbilicus. An acceptable alternative set-up of trocars would be: 5-mm at the umbilicus for the camera, 5-mm in the LLQ and a 10-mm suprapubically for retrieval.

6. The operating table should be tilted to the left with the head down, so that the entire small bowel moves to the left and cranially, thus exposing the cecum and the appendix.
7. Use an ultrasonic scalpel or 5-mm Ligasure for dissection and to take down the mesoappendix. Alternatively, you use bipolar diathermy or monopolar and clips. You do not need an endostapler. After dividing the mesoappendix and exposing the base arising from the cecum, ligate the appendix twice with an endoloop. If it is too wide or the base is involved in the inflammatory process, you may use an endostapler (35 mm), sometimes even including a normal-looking wall of the cecum in the bite. But, make sure you are not incorporating the ileocecal junction (valve).
8. If the appendix is in the retrocecal position, you may have to mobilize the cecum partially. Do not hesitate to do so. In the majority of cases, you will not have any conflicts with the right ureter.
9. Remove the appendix through the umbilical trocar. If it is a small one, there is no need for a bag. But, if it is bulky, you better use a retrieval bag. Do not hesitate to enlarge the umbilical opening for easy removal.
10. Aspirate (and irrigate if you wish) the gutter and the pelvis. For the pelvis, you will have to place the patient in a steep Trendelenburg position and retract the entire bowel out of the pelvis. Finally, before leaving the abdomen, do not forget to take a final look at the meso and the stump.

**Rebuttal:** Dr. Ahmad Assalia is trying to convince you (probably many of you have already been convinced) that the laparoscopic approach is almost always preferable. Perhaps in his own expert hands it is true, but in general, looking around us—beyond what is published in the literature—we see an epidemic of complications developing after LA that we have (almost) never seen before: intraperitoneal abscesses, intestinal obstruction, cecal fistula, recurrent appendicitis, bowel injury, bladder injury. AA mentioned decreased hernia formation with LA, but I have never seen a patient with post-OA hernia, and what about hernias developing in the trocar site? So, decide for yourself and play it safe.

The good thing about standards of care is that there are so many to choose from.

## Conclusions

Acute appendicitis, like any other surgical condition, has a *spectrum*. To reach the diagnosis, consider historical, physical, and laboratory findings together. No isolated variable can confirm or exclude AA; the more typical variables that are present, the higher the chance that you are dealing with AA. Whether you operate immediately or tomorrow, whether you observe or obtain additional tests is determined *selectively* based on your individual patient.

Never become blasé about AA; it can kill even today and may humble even the most experienced surgeon.

"The surgeon who can describe the extent of an appendiceal peritonitis has convicted himself of performing an improper operation."[1] (Mark M. Ravitch, 1910–1989)

There are two things in life that I will never understand: women and acute appendicitis.

[1]If you do not understand this aphorism feel free to e-mail us.

# 29 Anorectal Emergencies

Luis A. Carriquiry

*We suffer and die through the defects that arise in our sewerage and drainage systems.* (William A. Lane, 1856–1943)

Why have a chapter about anorectal emergencies in a book about emergency abdominal surgery? The easiest answer would be one based on anatomy: the rectum is an abdominal viscus, and the anus, although not strictly abdominal, belongs to the perineum, which *latu sensu* is the floor of the abdominal cavity. But, the main reason for inclusion is pragmatic: anorectal emergencies are managed by the general surgeon on duty, who has to provide optimal care to these frequent emergencies (› Fig. 29.1).

First, consider the three leading causes of acute anal pain:

- **Acute fissure**
- **Acute perianal hematoma**
- **Perianal abscess**

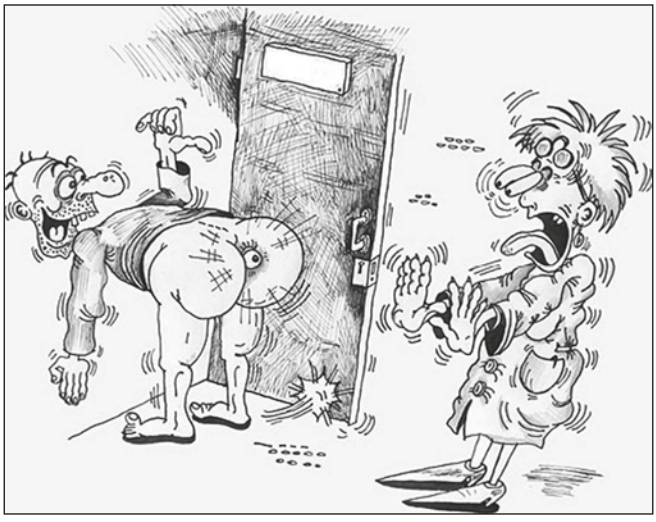

**Fig. 29.1.** "I know I'm a pain in the ass, but please help me!"

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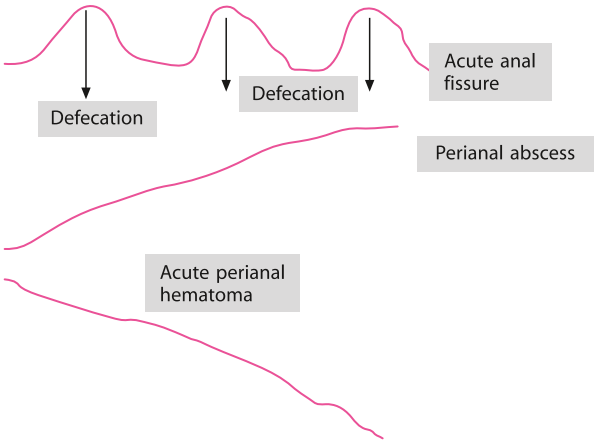


**Fig. 29.2.** Pattern of acute anal pain

Although none of these conditions is a life-threatening emergency, they should be treated without delay to relieve the pain and distress.

The differential diagnosis is easy and possible even before examining the patient. Figure 29.2 shows that each of the three conditions has a typical pattern of pain.

- In anal fissure, the pain is *sharp* and *intermittent*, aggravated by defecation, relieved thereafter, but sometimes lasting for 3 or 4 hrs as a dull ache. The patient begins to fear the act of defecation and frequently tries to postpone it, aggravating the symptoms.
- In perianal abscess, the pain is *constant*, *dull*, and *gradually increasing* until surgical or spontaneous drainage of the pus. Fever and chills may be accompanying symptoms but do not wait for them to make the diagnosis.
- In acute perianal hematoma, the pain is more often than not already *abating* when the patient presents to you, rarely lasting for more than 2–3 days.

Next, you want to examine the patient. This is done without resorting to a painful digital rectal examination. Place the patient in the lateral decubitus position or let the patient stand flexed forward with you sitting behind his or her buttocks. Gently spread the buttocks and look at the perianal region; now, you can easily visualize any perianal hematoma and often any fissure, sometimes even an abscess. If you see nothing, then assume that you are dealing with a perianal abscess and continue as discussed separately in this chapter.

## Acute Perianal Hematoma

You will recognize an acute perianal hematoma immediately after the buttocks are separated—a swelling the size and shape of a grape, bluish, tense, and situated at the anal verge. It is also known erroneously as a "thrombosed external hemorrhoid," although it is believed to represent a clotted perianal vein of indefinite etiology. If left untreated, the pain will subside gradually within a day or two, and the swelling will disappear within a week or so. From our own very personal experience, we know that stool softeners and local anesthetic cream alleviate symptoms rapidly. But, if the patient is hysterical, and you are one of those who always like to "do something," you may want to inject the lesion with lignocaine or numb it with ethyl chloride spray and evacuate the clot through a tiny radial incision through the overlying mucoderm. This relieves the symptoms, although you should be warned that we have seen patients return with an abscess or bleeding at the incision site. We therefore strongly favor nonoperative management of this condition.

## Acute Anal Fissure

An acute anal fissure is a linear superficial tear extending from the anoderm to the dentate line, most commonly at the 6 o'clock position, but in females an anterior midline location (12 o'clock) is not uncommon. The sentinel skin tag and hypertrophied papilla typical of *chronic* fissure will be absent. (Sometimes, the anal spasm and pain elicited by the simple separation of the buttocks make visual confirmation difficult.) Acute fissure almost never requires operative treatment. Your task is to interrupt the pain-spasm-pain cycle; the pain is caused by the fissure, which results in spasm of the internal sphincter, which in turn increases the pain. We would inject, using a fine needle, a few milliliters of local anesthetic solution (e.g., marcaine) just under the fissure. The pain will disappear quickly and with it the anal spasm. Now, the patient will allow you to insert a gloved finger into the anus. Gently introduce your finger coated with a generous dose of local anesthetic cream, dilating the anal canal gently. Do not try to make a further dilatation. Send the patient home and recommend stool softeners and the old hot sitz baths. Some would recommend local application of *glyceryl trinitrate* or *diltiazem cream* to relax the internal sphincter's spasm. If both creams are available, it seems the diltiazem should be preferred as it is equally effective and is less likely to provoke disturbing headaches. Management of recurrent, persisting, or chronic fissures, whether by prolonged applications of topical glyceryl trinitrate or diltiazem, by injection of botulinum toxin, or by a lateral internal sphincterotomy (which I prefer even now) is beyond the scope of emergency treatment.

## Acute Perianal Abscess

Sometimes, the acute perianal abscess is evident: a localized, very tender reddish swelling at the anal margin. At other times, you have to palpate the anal margin to elicit localized pain. Be careful to make this maneuver as brief and delicate as possible; repeating it many times or pressing your finger against the painful zone can be considered an act of torture. If you elicit localized tenderness, you do not need any other imaging technique to confirm the diagnosis, and you can treat it. In most cases, ordering a computed tomograph (CT) to diagnose a perianal abscess is a crime. However, in rare situations, when the abscess is situated *above the levator ani* or is *retrorectal*, with the patient presenting with dull perianal pain but no local findings on examination, then a CT of the pelvis may be diagnostic.

**The management is by incision and drainage**

*Where?* North American surgeons, for reasons of cost or ease of delegation, prefer to have the abscess drained through an incision under local anesthesia in the emergency department. Like many European surgeons, I prefer to complete my examination and perform the drainage under general or regional anesthesia in the operating theater. A proper exploration and drainage is too painful to be done in the awake patient, and local anesthesia does not work well in these circumstances. Most patients subjected to drainage in the emergency ward have bad memories of their ordeal. Perhaps, adopting a more eclectic posture, you can drain in the emergency room a small, well-defined, bulging perianal abscess, which is on the verge of spontaneous drainage, but in the case of bigger abscesses—especially those in the ischiorectal fossa—a trip to the operating room is mandatory.

*How*? I prefer to make a radial incision in the zone of the swelling. If a deeper abscess is not easily localized, tap it with a needle in search of pus. There is no need for the classical cruciate incisions or unroofing of skin. But, the incision must be wide enough to permit introduction of your finger to gently debride the cavity and look for unexpected extensions. Irrigation with normal saline is useful to remove residual pus or blood from the cavity. General or regional anesthesia also allows you to search for an associated *fistula in ano*—which should be present in more than half of the patients—and perform a primary fistulotomy or placement of a *seton*, depending on the type of fistula. The collected evidence shows that this line of management leads to fewer recurrences. This is what I do, but I think that it is unwise for the unskilled surgeon, in particular for the surgeon in training, to indulge in this practice, which may result in iatrogenic fistula tracts or damage to the sphincters, leading to incontinence.

There is no need to pack the cavity of the well-drained abscess or to leave a drainage tube, except in big cavities. The patient will experience almost immediate disappearance of pain and will be most thankful, although in subsequent

months—if you have omitted the search for it—approximately half will develop a fistula in ano, to be dealt with electively. And hey, please, these patients do not require any antibiotics!

**A caveat***:* the incidence of community-acquired perianal abscesses caused by methycilllin-resistant *Staphyloccocus aureus* (MRSA) is on the rise. You should suspect them when you find intense pain and extensive local inflammation but very little drainable pus. Take a swab for bacteriology study and start anti-MRSA antibiotics.

## Acute Strangulated Internal Hemorrhoids

As you may have noticed, until now we have not even mentioned hemorrhoids. Despite the commonly held opinion of family doctors, hemorrhoids do not usually cause acute anal pain. Acute perianal hematoma, which we mentioned, is not a complication of pre-exsisting hemorrhoids, although sometimes they coexist. But, there is an exception to this rule: **acute strangulated internal hemorrhoids**.

This is a relatively common occurrence in patients with grade III or IV hemorrhoids. The prolapsed hemorrhoids become irreducible because of swelling, and thrombosis frequently develops. The patient experiences intense pain and has serious difficulties sitting and walking. On examination you see the prolapsed *piles* (this is what the Brits call hemorrhoids)—blue with areas of mucosal necrosis.

Three options are available: nonoperative treatment, anal dilatation, and emergency hemorrhoidectomy. Most colorectal specialists prefer the last, which is the quickest solution to the problem, although they admit that the swelling may lead to an excessive excision of anal mucosa and to the subsequent development of anal stenosis. So, if you feel confident about your training in anal surgery, proceed to hemorrhoidectomy but always consider that a few residual skin tags resulting from insufficient removal of perianal and mucosal folds is a better result than stenosis as a consequence of an overenthusiastic excision. Some surgeons would remove the prolapsed piles with or without the addition of internal sphincterotomy to relieve the secondary anal spasm. A less-aggressive alternative to sphincterotomy would be the local application of glycerin trinitrate or diltiazem cream. If you are not too comfortable with emergency hemorrhoidectomy in this condition, you may safely resort to **anal dilatation** under general anesthesia; do it gently, especially in older people, and then reduce the prolapsed piles upward, where they belong. The third option—**nonoperative treatment**—is preferred by many surgeons and consists of bed rest (with the buttocks elevated) and analgesia until spontaneous resolution occurs. You can use sugar for accelerating this resolution [see Editorial Comment at the end of this chapter].

Before discussing the more serious conditions, a brief mention is made of a rare situation: **acute incarcerated full-thickness rectal prolapse**. This is an uncommon condition but most painful and distressing for the patient. It develops usually in individuals with weak sphincters. Examination makes the diagnosis quite obvious: you can see the bulge of the prolapse with the typical rectal mucosa and concentric folds, which must be differentiated from the above-mentioned acute prolapse of hemorrhoids (more irregular, with radial folds). Try to reduce the prolapse with local or general anesthesia. The use of sugar has been recommended also for this condition; it works by osmotically reducing the edema of the mucosa and thus allows easier reduction. When this fails or if there is extensive mucosal necrosis, I think that operative treatment is a better option; my choice is a perineal rectosigmoidectomy with a coloanal manual suture (Altmeier operation). This is obviously major, specialized surgery and therefore outside the scope of this book.

Now I discuss the really life-endangering anorectal emergencies: **trauma to the rectum and anus** and **necrotizing infections of the perineum.**

## Trauma to the Rectum and Anus

I have never seen any anal or rectal injury associated with blunt abdominal trauma. As a rule, any damage to the anorectum is as a consequence of penetrating trauma (almost exclusively from missile wounds), from perineal lacerations due to falls on irregular and pointed surfaces, or as a consequence of impalement or sexual abuse.

The exact assessment of damage following such injuries is best performed in the operating room, under general anesthesia with the patient in lithotomy position, using your fingers and proctosigmoidoscope. There is no need to remind you of the usual priorities of trauma care; oxygenation, hemostasis and vital organs come before the torn ass. Do not forget to "prep" the abdomen should laparotomy or colostomy prove to be necessary.

**Injuries to the intraperitoneal rectum** are usually caused by gunshot wounds (Chap. 38). They must be carefully looked for in the course of exploratory laparotomy, especially when the bullet trajectory is within the pelvis. Such injuries occur also after impalement with long poles, for which perforation of the high rectum or even the sigmoid is not exceptional, and other abdominal organs can be injured (I even know of a myocardial injury caused by impalement with a billiard cue). Intraperitoneal injuries can be treated almost always with simple suture, as with any colonic injury. Exceptionally, facing severe damage to the rectum that is not safely repairable, a proximal colostomy or a Hartmann operation may be necessary. Be that as it may, do not be afraid to suture the rectum with unprepared bowel; the rectum should be no more intimidating than, say, the cecum. An elegant way (suggested by Danny Rosin) to

close a low rectal perforation when access is limited due to obesity and narrow pelvis is to insert through the anus a circular, EEA stapler, connected to the anvil and in the *open* position; then, slowly *close* it to "side bite" the perforation, the edges of which are inverted into the open stapler. Two corner stitches help this manipulation. Finally, *close and fire* the stapler, thus excising the hole and stapling it closed.

- **Injuries to the extraperitoneal rectum** are more challenging. Any suspicion of extraperitoneal rectal injury suggested by the bullet trajectory must be confirmed or refuted by clinical examination. Discharge of blood and palpation of a hole in the rectal wall are confirmatory. Until recently, management was based on three basic principles developed for war injuries and demonstrated to be very effective in reducing mortality and morbidity: *diverting sigmoidostomy, presacral drainage*, and *rectal washout*. (Repair of the actual rectal wound was added when technically possible.) However, the routine use of these dogmas in civil injuries has been challenged in recent years. *Suture repair* of the rectum is a nice concept but has little to recommend it. Doing so through a transanal approach is not easy, and there is agreement that opening the pelvic peritoneum during abdominal exploration is indicated only to arrest hemorrhage from major vessels or for debridement in the face of extensive bony and soft-tissue damage. In most civilian rectal injuries, suture repair can be omitted without affecting morbidity and mortality (a similar situation exists in the case of full-thickness local excision of rectal tumors without suturing the rectal defect). *Rectal washout* has become the second victim of iconoclasts. Most recent series have omitted it with no change in results. The value of *presacral drainage* has also been questioned. Only proximal fecal diversion seems to remain a firm principle, but recent debates about its protective role in very low rectal anastomosis and the necessity of mechanical preparation in colon and rectal surgery are challenging even this status. I look at these developments with an open mind; probably, colostomy may be omitted in low-velocity missile wounds, but I am still inclined to use it in the management of most injuries. The colostomy should be created as distally as possible; a properly constructed loop sigmoid colostomy, with an adequate spur, has been demonstrated to be completely diverting, with no need for an end colostomy (see Chap. 14). The only recent development to be considered is the laparoscopic approach to look for associated intraperitoneal injuries and to exteriorize the sigmoid, without a formal laparotomy. Although not an unconditional fan of laparoscopic approaches, I think it may be a good idea and probably one of the better indications for laparoscopic colon surgery.
- **Injuries to the anal canal.** Hemostasis is achieved and lacerations are debrided while taking care to spare as much of the sphincter muscles as possible. The wounds are then left open. A sigmoid colostomy is recommended only for very extensive anal and perineal lacerations; in minor cases, it is not necessary. You can repair a partially torn sphincter when the injury is limited; however, I would not recommend attempts at sphincter reconstruction in grossly destructive injuries. Sutures do not hold well in the traumatized muscle, and nerves can be damaged during difficult dissection in a bloody field. All this can lead to failure, compromising the

success of further reconstruction. It is better to leave the job of anal canal reconstruction to the specialized surgeon, who can in due course perform a sphincteroplasty or even think about more complex techniques such as implantation of an artificial sphincter or creation of a stimulated gracilis neosphincter.

## Rectal Foreign Bodies

Rectal foreign bodies offer a particular kind of anal and rectal trauma. In the rarest case, they may result from accidental ingestion, with the foreign body making its way through the whole digestive tract and impacting on the rectal or anal walls (I have seen a toothpick transversely impacted in the anal canal, giving origin to bilateral anal abscesses). Most of them are inserted per anum and almost always by the patient attempting sexual gratification. By the way, do not assume this occurs only with flamboyantly gay people; in most cases, you will find middle-aged or even senior married men, who give the most incredible explanations for the unfortunate location of the foreign body. Self-inserted foreign bodies, whatever their shape and size, do not ordinarily cause rectal lesions that go deeper than the mucosa, but the same cannot be said when insertion is due to sexual assault, for which perforation at the level of peritoneal reflection or even at the rectosigmoid junction is not exceptional.

When the patient gives a history of impalement injury (fact or fiction), you must carry out a careful abdominal examination and consider abdominal imaging to confirm or rule out a visceral perforation, which may necessitate a laparotomy. In all other cases, an initial attempt to remove the foreign body through the anal canal is recommended under local, regional, or general anesthesia, which allows relaxation of the anal sphincters and prevents muscular disruptions due to forceful stretching. Many instruments and maneuvers for grasping the foreign body have been described, but if extraction is not easy, the risk of laceration of the rectal wall or the anal canal increases with time and effort, and laparotomy should be considered, always with the patient in the lithotomy position. In that case, you should try first to deliver the foreign body through the anus to the hands of the perineal operator by manipulating it through the rectal wall, but sometimes opening the rectum and removing the object from the top is, paradoxically, the least invasive way of solving the problem. A postextraction rectoscopy is mandatory to ensure the integrity of the rectal wall.

## Necrotizing Perineal Infections (Fournier's Gangrene)

Necrotizing perineal infections may be the consequence of neglected anorectal infections, but they also arise from trauma, skin infections and urethral instrumentation. A urethral source implies *Fournier's gangrene*—an eponym that has been incorrectly extended to the whole spectrum of this entity. But, more important than etiology is prompt diagnosis and treatment.

These patients are commonly diabetic, very obese, or inmunosuppressed. The synergistic action of gram-negative bacteria, anaerobes, and *Streptococcus* causes rapid dissemination of the infection along *superficial* fascial and subcutaneous planes, with secondary ischemic involvement of the skin. Pain may be the first symptom, but it may be vague. Swelling of the perineum, crepitus, local tenderness, and erythema of the skin—followed by its necrosis—are the typical elements found on examination.

There is no need for X-rays or CTs unless one suspects extension to fascial abdominal or retroperitoneal tissues. Only prompt treatment can prevent a fatal evolution; it should include supportive care, high-dose intravenous antibiotics to cover aerobic and anaerobic bacteria, and prompt surgical debridement, which is the mainstay of treatment. Necrotic skin must be resected, but as fascial and fat necrosis extend much further, extensive skin incisions are usually necessary to allow radical excision of fascia and fatty tissue until well-perfused and viable fat is found. If the infection extends to the perineal muscles, they must be sacrificed following the same criteria. Debride as much as necessary at your first operation but plan on taking the patient back to the operating room in the next days until you are satisfied that the infection is under control. Concerns about future reconstruction should be left to the plastic surgeon, but if it is necessary to excise scrotal skin, it is convenient to wrap the testicles, which are rarely compromised, in healthy tissues in the abdominal wall or the thigh.

Chop out everything that stinks or is dark, gray, or dead—irrespective of how large and horrendous the wound you create. And do it again and again, as many times as it is necessary. Eventually, it will all pink up, granulate, contract, and heal.

**Two controversial issues remain**: the necessity of a colostomy and the use of hyperbaric oxygen. Most authors think a diverting stoma is generally not necessary even in the case of a free-floating anus. Nevertheless, when ongoing fecal contamination is not easily manageable (e.g., incontinent patient, poor nursing facilities), I would consider proximal fecal diversion. The use of hyperbaric oxygen has been strongly recommended on the basis of the action of oxygen free radicals against anaerobic bacteria, but it remains controversial, cumbersome, and expensive and so cannot be considered a necessary component of the standard of treatment. Your knife should be the instrument to provide oxygen to the wound.

### Local Anesthesia to the Anus

I almost forgot. I have repeatedly mentioned "local anesthesia" but forgot to tell you how to anesthetize the anus. This is how I do it: with the patient in a comfortable prone position (my favorite position for anal procedures) and using a mixture

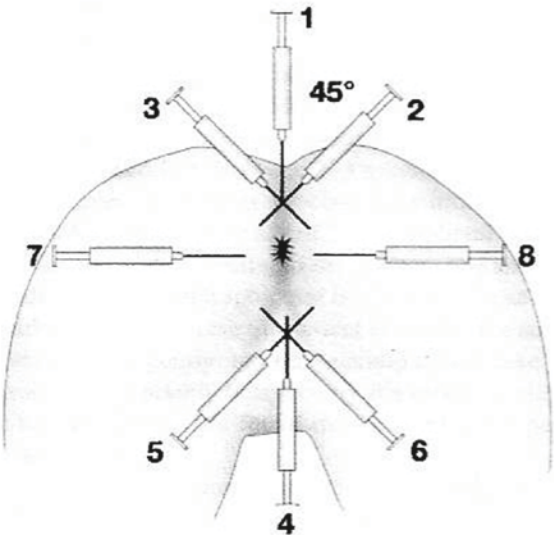


**Fig. 29.3.** Local anesthesia for anal procedures. See relevant text

of 20 ml of 0.5% bupivacaine, 10 ml of 1% lignocaine, 10 ml of normal saline, and a 6-cm needle, I inject 5 ml. of the local anesthetic mixture deeply behind the anus, peripheral to the external sphincter. Then, without totally removing the needle, I repeat the injection on both sides, with a 45° inclination (see Fig. 29.3, steps 1–3). A second injection is done anteriorly (steps 4–6), with the same fanning. Finally, two more injections are made following a similar pattern at 3 and 9 o'clock (steps 7, 8).

### "Tell Me About the Sugar"

Dr. Carriquiry recommends applying sugar to prolapsed strangulated hemorrhoids or prolapsed rectum. This is not a joke but an excellent idea. The hygroscopic sugar rapidly reduces the tissue edema, shrinking the prolapsed tissues and allowing manual reduction. Simply place the patient prone and pour a generous quantity of sugar on the strangulated parts until the tortured anus looks like a cake covered with icing sugar. Repeat as necessary following any sitz bath; you will not believe how fast the swelling will subside [The Editors].

> "An abscess near the anus should not be left to burst by itself, but … be boldly opened with a very sharp lancette, so that pus and the corrupt blood may go out. Or else … the gut which is called rectum … will burst … for then may it … be called fistula. And I have seen some who have seven or nine holes on one side of the buttocks … none of which except one pierce the rectum." (John of Arderne, 1306–1390)

# 30 Surgical Complications of Endoscopy

Ahmad Assalia · Anat Ilivitzki

*If you are too fond of new remedies, first you will not cure your patients; secondly, you will have no patients to cure.* (Astley Paston Cooper, 1768–1841)

Complications of endoscopy may be defined as *immediate*, occurring during the procedure or before the patient leaves the endoscopic suite, or *delayed*, occurring up to 30 days after the procedure.

## Some Basic Points

### *Complications*

- In the real world, complications are much more frequent than is suggested by the "beautiful" figures quoted in the books.
- Complication rates vary with expertise and case volume; expect more with less-experienced endoscopists.
- The risks associated with endoscopy are higher when the pathology is more complex and in *therapeutic* as opposed to *diagnostic* procedures.
- With complications of endoscopy, it is particularly important to know when not to operate rather than when to operate; many episodes of postendoscopy bleeding and perforation are best treated conservatively. It is unhelpful to carry out a laparotomy for postendoscopy complications and then be unable to identify the perforation or bleeding source.

**When called to see a "sick" patient after an endoscopic procedure**

- Suspect catastrophe! And, until proven otherwise, assume the patient has the most dreadful surgical complications.
- What is common is common! Adverse events following immediately after endoscopy are likely to be due to the procedure itself.

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- Always transfer these "sick" patients to the surgical service regardless of the immediate need for surgical intervention. In the interests of everyone, *especially the patient*, the best environment is the surgical floor where patients can be monitored and treated appropriately.
- Recognition and early management of complications is the key for a successful outcome. So, if you do not think about it, you will not diagnose it.
- Regardless of the etiology, always treat shock immediately and prepare the patient with obvious peritonitis for urgent laparotomy.
- Always **READ** carefully any admission and progress notes and the endoscopic report; **TALK** to patient, the patient's doctor, and *directly contact the gastrointestinal (GI) specialist who performed the "uneventful" procedure* (many clues for the nature of the complication are there); **VIEW**, personally, all images taken at the endoscopy and after it.

## Complications of Upper Gastrointestinal Endoscopy

Flexible esophagogastroduodenoscopy (EGD) is a relatively safe procedure with few complications. Almost half of the serious complications that occur are cardiopulmonary, related to aspiration, hypoxemia, vasovagal reflexes, and endocarditis. The surgical complications include:

- **Esophageal perforation.** The cervical esophagus is the area most at risk. Risk factors include anterior cervical osteophytes, Zenker's diverticulum, esophageal stricture or web, and a cervical rib. Most cervical esophageal perforations occur during rigid endoscopy or with blind passage of a flexible endoscope. Retching with an overinflated stomach and the endoscope occluding the gastroesophageal junction can result in Mallory-Weiss tears or transmural perforation. Cervical pain, crepitus, and cellulitis are all signs of high esophageal perforation. Halitosis develops rapidly due to overgrowth of anaerobic bacteria. Distal perforations cause chest pain. A cervical soft tissue X-ray and chest radiograph may be helpful in the initial stages for the detection of cervical air, pneumomediastinum, and pneumothorax or pleural effusion. The diagnosis is confirmed with water-soluble esophagography or computed tomographic (CT) scan. Do not waste time: obtain an urgent CT scan with oral contrast medium; it will detect minimal perforations and provide additional valuable information regarding the location and extent of the inflammatory process. The management of esophageal perforation is outlined in Chap. 15.
- **Post-EGD upper GI bleeding** (variceal and nonvariceal) is approached and treated according to the principles presented in Chap. 17.
- **Other complications.** Following sclerotherapy, and less frequently after band ligation for esophageal varices, up to half of the patients will experience one or more of the following: chest pain, pleural effusion, pulmonary infiltrates, and bacteremia

(without perforation). Bacteremia is especially common after esophageal dilatation, so antibiotic prophylaxis should be considered in an effort to prevent bacterial endocarditis in susceptible individuals. Esophageal placement of stents for malignant strictures may cause erosions, bleeding, migration, tumor ingrowth with recurrent obstruction, food impaction, or (if they are inserted across the gastroesophageal junction) reflux with aspiration. Remember—these patients have a short life expectancy; do no more than the minimum required for palliation. This may include repeated endoscopies for ablation of the tumor ingrowth or placement of a second stent.

## Complications of Percutaneous Endoscopic Gastrostomy

"DIB (death in bed)—a common early sequel of 'tracheostomy and gastrostomy.'"

Percutaneous endoscopic gastrostomy (PEG) tubes are commonly used as a feeding route in elderly and debilitated patients. In some cultures and places, it seems that patients are not allowed to die without having a PEG tube inserted. This is an invasive procedure, and complications after PEG insertion are not uncommon.

### *Leakage*

Leakage is by far the most important complication. It tends to present in the first days following the procedure. The clinical scenario ranges from asymptomatic leakage around the gastrostomy tube to overwhelming peritonitis and sepsis. The reason is inadequate fixation of the stomach against the inner abdominal wall or the separation of the two due to various factors, especially ischemia and necrosis of the gastric wall due to excessive tightness of the fixing device—whatever method is used.

Clinical features depend on whether the leaking gastric juice or feeding solution leaks only to the outside around the tube or whether the leak is into the peritoneal cavity. If the latter is the case, the clinical picture may range from mild pain and abdominal distention due to ileus to full-blown peritonitis and "sepsis."

**Diagnosis** ▶ The finding of free intraperitoneal air is not diagnostic because pneumoperitoneum may be present for weeks after uncomplicated PEG insertion. Intraperitoneal leak should be excluded by a contrast study, with contrast instilled through the PEG tube.

**Management** ▶ If a contrast study excludes intraperitoneal leakage, then the PEG tube has to be "rested," allowing the tissue around it to seal. Attach the PEG tube to gravity drainage, administer intravenous fluids and antibiotics, and follow the patient closely. Wait a week and then repeat the contrast study before attempting PEG feeding. This, with the addition of a nasogastric tube, should also be the approach when the PEG tube is pulled out inadvertently less than 2 weeks

after insertion, and there are no signs of peritonitis or sepsis and no evidence of intraperitoneal leak on contrast study. In cases with obvious leakage into the peritoneal cavity, your management should be guided by the clinical scenario. While minor and asymptomatic leaks can be treated conservatively, operative treatment is mandatory with free intraperitoneal leaks and signs of infection.

**Operation** ▶ Early on, in the absence of significant tissue edema, place a purse-string suture around the PEG tube and (carefully and all around) refix the stomach to the abdominal wall. But, if the surrounding tissues and the hole in the stomach look "bad," then take out the tube and carefully suture or staple off the hole. Based on the condition of the patient and the degree of peritonitis, consider whether you wish to insert a gastrostomy (or jejunostomy) tube in another, healthier location. Needless to say, thorough "peritoneal toilet" is mandatory (Chap. 12). This procedure could be accomplished laparoscopically if you have enough skills or by a minilaparotomy in the upper midline.

**Late Leaks** ▶ Less frequently, leaks may occur long after PEG insertion, particularly in patients with poor healing capabilities and occasionally also after inadvertent or planned removal of the tube. Most often, such late leaks behave like a controlled gastric fistula and will eventually seal spontaneously with conservative measures. However, an uncontrolled leak into the peritoneal cavity may occur and should be managed according to the principles outlined.

#### *Perforation of a Viscus*

Rarely, the colon or even small bowel can be "impaled" by the PEG tube during its placement. This could present early with a free leak and peritonitis or later with an abscess or colonic fistula (external or communicating with the stomach). The management (conservative vs. operative) depends on the acuteness of presentation, the anatomy of the complication, and the patient's general condition. Free leaks must be controlled, abscesses have to be drained, while controlled fistulas are managed conservatively. Cologastric-PEG fistulas usually subside when the tube is pulled out.

### Complications of ERCP

The ERCP procedure carries a relatively high incidence of complications. Were we not constrained by the editors, who forbade mention of percentages in this book, we would have told you that in decreasing order of frequency, the complications include pancreatitis (2–5%), bleeding (2%), cholangitis (1–2%), and perforation (0.5–1.2%). The mortality rate of the last-mentioned complication may be as high as 15%. Therefore, ERCP—especially therapeutic ERCP—should be viewed as a potentially risky endoscopic procedure.

### *Pancreatitis*

While hyperamylasemia may be seen in up to two-thirds of patients, clinical pancreatitis occurs rarely. The incidence is the same for both diagnostic and therapeutic procedures. The severity in the majority of cases is usually mild to moderate and self-limiting. Unfortunately, however, severe post-ERCP pancreatitis and even fatalities can occur. Interestingly, pancreatitis is more common in younger patients and has its highest incidence in patients having ERCP for suspected "sphincter of Oddi dysfunction" [one of those mystifying diagnoses seen only by those who write the articles—The Editors].

**Diagnosis** ▶ Any significant upper abdominal pain coupled with hyperamylasemia after ERCP should raise the suspicion of pancreatitis. Sometimes, the diagnosis is difficult to make since perforation (discussed separately) may give a similar clinical presentation. If cannulation of the duct was easy and no "precut" or therapeutic interventions were attempted, the likelihood of duodenal perforation is low. Even so, whenever you suspect a perforation, order a Gastrografin upper GI study, or preferably a CT scan, to exclude the perforation and to confirm the pancreatitis.

**Management** ▶ For management, intravenous fluids and nothing by mouth (nil per os, NPO) until the symptoms abate are usually all that is required. In a minority of patients, a more severe and protracted course may follow. The management strategy in such cases is discussed in Chap. 19. Obviously, impacted common bile duct stones may precipitate pancreatitis and protract its course; if so, repeated ERCP or operative common bile duct exploration may be indicated (see Chap. 20.3).

### *Hemorrhage*

Clinically significant hemorrhage may occur after endoscopic sphincterotomy (ES).

**Diagnosis** ▶ Bleeding may present as upper GI bleeding or mimic lower GI bleeding; the patient may develop hemodynamic compromise before hematemesis or melena appears. Admit the patient to the intensive care unit (ICU) or the surgical floor for close monitoring and apply all the principles of management of GI bleeding (Chap. 17).

**Management** ▶ Repeat endoscopy is indicated for accurate diagnosis, to confirm if the bleeding is in the form of oozing or brisk arterial "pumping," and to

achieve hemostasis. If endoscopic hemostasis fails, the patient's condition is still stable, and an experienced interventional radiologist is available, then *celiac angiography* with selective embolization of the gastroduodenal artery bleeding branch may avoid operative intervention. However, if this in turn fails or is unavailable and the bleeding continues or the patient is unstable, then operative intervention must be undertaken. After full Kocherization of the duodenum, a longitudinal duodenotomy in the second part will allow access to the papilla of Vater. The bleeding is controlled by suture-ligatures, being careful not to stenose the opening of the papilla or the *sphincterotomy* site (it may be advisable to convert the *sphincterotomy* to *sphincteroplasty*). In a "stable" patient in whom ERCP and ES has failed, one can proceed with a definitive surgical correction of the problem for which the ES had been attempted. Otherwise, the minimum should be done that allows drainage of the obstructed biliary system (e.g., cholecystostomy or a T tube).

### *Perforation*

Perforation is by far the most serious complication of ERCP and endoscopy in general, with up to one-fifth of the patients dying. The vast majority of perforations are into the retroperitoneum in the periampullary area. They are caused by a "precut" or ES. Less frequently, guidewire perforations of the common bile duct and the pancreatic duct may occur. Only a tenth of perforations are intraperitoneal and are caused by the endoscope itself (usually in the anterior wall of the second part of the duodenum). Risk factors for this include limited experience of the endoscopist, too generous precut or ES, therapeutic procedure, intramural injection of contrast material, repeated ERCP, and patients with a Billroth II gastrectomy.

**Diagnosis** ▶ Diagnosis is often apparent during the procedure or at the conclusion of it when the endoscopist suspects that something went awry. Abdominal and back pain during or immediately after ERCP together with the presence of retroperitoneal air on plain X-ray of the abdomen will confirm the diagnosis. Alternatively, injection of contrast medium by the endoscopist with demonstration of a leak is possible. The best single modality for the diagnosis is an abdominal CT scan detecting retro- or intraperitoneal air and contrast leakage. This prevents a mistaken diagnosis of pancreatitis, which could delay the appropriate management.

**Management** ▶ In highly specialized centers, a repeat ERCP with insertion of a stent, to "seal" the perforation, may be attempted for management, but most endoscopists are reluctant to have another go at these patients after endoscopy has caused the problem in the first place. Although there is lack of consensus

regarding the best management strategy, it seems that if the following conditions are met, the majority of these patients can be successfully treated nonoperatively:

- **Absence of free leakage of contrast**
- **Absence of clinical peritonitis or systemic inflammation** (hemodynamic compromise, high fever, and leukocytosis)
- **Absence of large pneumoperitoneum**

All other patients with a free leak, intraperitoneal air (denoting intraperitoneal perforation), peritonitis, or sepsis should be treated surgically.

If conditions for nonoperative management are met, a nasogastric tube should be inserted and broad-spectrum antibiotics with adequate Gram-negative coverage administered. Patients should be followed closely, and improvement should be expected within 12–24 hrs. Normally, these patients recover within 7–10 days, and repeated procedures, if still indicated, can be done at that time. Lack of improvement with the appearance of peritoneal irritation or signs of ongoing sepsis mandate immediate operative intervention. After fully "Kocherizing" the duodenum, the site of perforation is usually revealed at its posterior aspect. Depending on the degree of induration and inflammation of the tissues, either primary closure or an omental patch repair (see Chap. 18) are performed and a drain left in situ. The next step depends on the patient's condition, underlying pathology, failure or success of the "index" ERCP, and the adequacy of the duodenal closure. The principles are as follows: if the patient's condition is stable and the repair looks adequate (this occurs with early perforations), there is no need for a pyloric exclusion procedure. An obstructed biliary system should be decompressed, preferably by a T tube (after cholecystectomy and common bile duct exploration and clearance). **If you are already there, please do not leave the patient at the mercy of the endoscopist again!** If you are worried about the duodenal repair, or its lumen, do add a pyloric exclusion procedure. This is accomplished by making a gastrotomy just proximal to the pylorus and closing the pylorus from the inside with an absorbable suture, then forming a gastrojejunostomy (see Chap. 39.2). Finally, feed a narrow-bore nasogastric tube deep into the efferent loop of the gastrojejunostomy to feed your patient distal to the stoma and the duodenal repair.

Severe complications and deaths after ERCP are heartbreaking. But, what is tragic is that in many such cases it is clear in retrospect that the original procedure was not really indicated. (For example, MRCP could have excluded the suspected choledocholithiasis.)

## Complications of Colonoscopy

Colonoscopy is a relatively safe procedure, with the main complications perforation and hemorrhage. The complication rate is very low for diagnostic procedures and rises when the procedure is therapeutic, especially after polypectomy.

### *Bleeding*

Bleeding might occur immediately after the procedure or may be "secondary" or delayed from an ulcer developing at the site of the polypectomy or biopsy. The risk is higher with resection of polyps larger than 15 mm, recurrent or difficult procedures, or a bleeding tendency. Rarely, bleeding may occur due to mucosal injury caused by traumatic insertion and manipulation of the scope. Very rarely, vigorous manipulations in the region of the splenic flexure of the colon result in a *splenic injury* and intra-abdominal hemorrhage.

**Management** ▶ Management includes resuscitation and correction of any coagulopathy followed by an endoscopic attempt to treat the bleeding. If, after replacing fluids and correcting coagulation deficits, the patient has clearly stopped bleeding, one may elect not to repeat the colonoscopy to minimize the risk of a perforation at the biopsy site. In selected stable patients whose pathology does not require resection, an angiographic selective distal embolization may be attempted provided a highly skilled interventional radiologist is available. Just remember the (low) possibility of bowel ischemia following such an intervention. Persistence of bleeding after unsuccessful colonoscopic or radiological management mandates an immediate abdominal exploration. **Always have the endoscopist ready in the operating room to perform an intraoperative colonoscopy** (or even better, master the technique yourself). **Remember** that finding the bleeding spot could be a difficult task: an intraoperative colonoscopy will minimize blood loss and prevent unnecessary bowel resections. In most instances, after localizing the bleeding source, all you have to do is to place a colotomy and achieve hemostasis by oversewing the site of bleeding; then, close the colotomy. If bleeding originates from a source that requires resection (e.g., a large polyp or carcinoma), then an appropriate colectomy should be performed.

### *Perforation*

*The mechanism of perforation determines the size of the hole, which occasionally can then be managed selectively by the smart surgeon, not the "blind" gastroenterologist.*

Difficult, traumatic, and therapeutic colonoscopies are associated with an increased risk of perforation of the colon. Barotrauma from excessive insufflation of air, excessive use of cautery, or overzealous dilatation of strictures are common causative factors. In addition, prior surgery, diverticulitis, or preexisting intra-abdominal adhesions and a poorly prepared bowel may increase the difficulty of the procedure and the possibility of perforation.

When a colonic perforation occurs, the spectrum of consequences is wide and unpredictable. The *mechanism* of perforation matters; perforations that follow therapeutic colonoscopy (at a biopsy or polypectomy site) are usually small and more amenable to nonoperative treatment. On the other hand, perforations following diagnostic colonoscopy often result in sizeable rents in the colonic wall and thus require prompt surgical treatment.

**Diagnosis** ▶ The key to diagnosis is to suspect it. Think about the possibility of perforation in any patient who develops abdominal discomfort or pain at any time after colonoscopy. Presentation is varied: abdominal complaints and signs may develop immediately after the colonoscopy when there is a large colonic tear. On the other hand, patients may present a few days later with gradually increasing local and systemic manifestations of infection. Such delayed presentation is typical of perforations that are initially contained within the retroperitoneum or the mesenteric leaves and gradually leak or rupture into the free peritoneal cavity. Polypectomy with cautery necrosis of the bowel wall may also result in delayed perforation.

The abdominal-peritoneal signs and systemic repercussions of colonic perforation are well known to you. But, remember that loops of bowel—pumped up with air during colonoscopy—may still be tender many hours after the procedure.

Start with a plain upright chest X-ray and left lateral decubitus films of the abdomen and look for free air. The findings of free intra-abdominal air together with a clinical picture of local or systemic peritonitis are diagnostic of perforation. Pneumoperitoneum may be seen after colonoscopy with minimal or no clinical evidence suggesting perforation ("benign" postcolonoscopic pneumoperitoneum). Conversely, free air may be missing when the perforation is initially contained or retroperitoneal. Basing decision making on the absence or presence of free air reflects naïveté common to nonsurgeons (e.g., gastroenterologists) attempting to treat abdominal surgical emergencies.

Obviously, clinical signs of perforation and free air on abdominal X-ray are diagnostic of perforation. In the absence of free air, insist on obtaining a CT scan (or a Gastrografin enema if CT is not available). Not only is CT able to show free air not visualized by plain X-rays, but also it may show other details suggestive of injury, such as colonic wall hematoma or air in the colonic wall, the mesentery, or the retroperitoneum. When combined with rectal contrast, CT usually

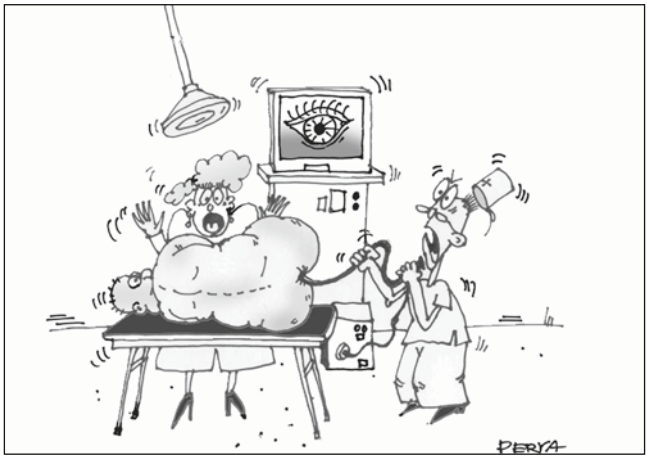


**Fig. 30.1.** "Nurse, is that the omentum?"

demonstrates the site and size of the leak and whether it is contained or not. Free fluid may reflect spillage of bowel contents or developing peritonitis.

**Remember:** the chief cause of death following colonoscopic perforation is delay in diagnosis and consequent delay in treatment. This holdup usually results from the failure of the responsible clinician (it is usually the colonoscopist to whom the patient presents with the complication) to consider such a diagnosis. Remember the "surgical ostrich" who cannot diagnose his or her own complications? Well, gastroenterologists are no different (› Fig. 30.1). We have to help them get their head out of the sand.

**Non-operative Management** ▶ Not all patients with colonoscopic bowel injury need a laparotomy. Patients who are minimally symptomatic, without fever or tachycardia, and in whom the abdominal exam is benign (i.e., no features of peritonitis) can be managed nonoperatively with nothing by mouth and broad-spectrum antibiotics (as you would manage acute diverticulitis; › Chap. 26). Patients who respond to conservative treatment typically have no or minimal pneumoperitoneum and no or minimal leak of contrast on CT.

As stated, perforation at the site of a polypectomy is more amenable to a trial of nonoperative management. Such an approach is often successful because these patients have had bowel preparation prior to colonoscopy; therefore, the potential for abdominal contamination is reduced. All such patients should be closely monitored for local and systemic progression of the process or failure to improve. Deterioration should prompt an urgent surgical intervention. If the perforation is at the site of pathology for which a colectomy will be recommended anyway, what is the point of sweating through conservative management? Go ahead and do the definitive surgery right away.

**Surgical Management** ▶ Patients who look sick, complain of localized or spreading pain, and have systemic sepsis and localized or generalized peritonitis should receive antibiotics and undergo an emergency laparotomy. In most patients undergoing early exploration, the findings are those of peritoneal *contamination* rather than established *infection*; all that is required is "peritoneal toilet" (Chap. 12) and primary suture of the perforation as you would do with any traumatic colonic injury (Chap. 39.2). The absence of feces in the colon helps to minimize the severity of contamination and infection. A diverting or exteriorizing colostomy may be indicated in selected patients, such as those with neglected established peritonitis or severe debilitating comorbidities such as malnutrition or steroid dependence. The role of laparoscopy in the diagnosis and treatment of colonic perforations is not defined yet. However, an experienced laparoscopist, with the help of intraoperative colonoscopy, may accurately diagnose and treat colonic perforations.

## Conclusions

The management of endoscopic injury to any hollow GI viscus, from the esophagus down to the rectum, can be summed up as follows:

- Always suspect disaster.
- Image for diagnosis.
- Those who are missed and neglected tend to die.
- Some can be managed conservatively.
- Some need an immediate operation.
- Some who are managed conservatively may eventually need an operation.
- **To achieve optimal results, be selective, alert, and always ready to change your mind. You are not a politician; you can be proud to be a flip-flopper!**

A fool with a tool is still a fool.

# 31 Complications of Bariatric Surgery

Ahmad Assalia

*Severe obesity restricts the movements and maneuvers of the body. It compresses blood vessels causing their narrowness. Breathing passages are obstructed and the flow of air is hindered leading to nasty temperament. … On the whole these people are at risk of sudden death. … They are vulnerable to stroke, hemiplegia, palpitation, diarrhea, fainting. … Any physical effort they make will weaken them.* (Avicenna, 980–1037)

Obesity is a modern epidemic. Bariatric surgery for **morbid obesity** is gaining acceptance and recognition. It seems absurd that in certain countries people now die of gluttony rather than hunger. Even more disturbing is that bariatric surgery is performed in countries where hunger and malnutrition are common. But, fortunately, philosophy is beyond our scope.

Luckily, most of the readers of this book do not engage in bariatric surgery. But, because it is performed all around the world, we all must be familiar with the diagnosis and treatment of the long list of complications that typically bedevil these procedures. Most, but not all, bariatric surgery is performed laparoscopically.

**Several basic points should be emphasized:**

- Obese patients have many related diseases, *but even in the absence of such diseases they are still considered to be sick!* They impose special anesthetic and medical challenges, and the surgical risk increases in tandem with their body mass index (BMI). There is an increased incidence of respiratory, cardiac, infectious, thromboembolic, and wound complications. In particular, some may have undiagnosed cardiac disease, which may lead to postoperative cardiac events and even death.
- Complications may be divided into those directly related to the surgical procedure (leaks, obstruction, etc.) and those related to comorbidity (deep vein thrombosis [DVT]), pulmonary embolism [PE], cardiac events, respiratory insufficiency, etc.). At times, diagnosis is difficult because both surgical and nonsurgical complications may produce similar clinical pictures (e.g., leak and PE). And, to compound matters PE and gastrointestinal (GI) leaks are the two leading causes of death after bariatric surgery. GI leaks in these patients may not present in the way with which you are familiar. This may be partly due to the thick abdominal wall. In practice, in many of these patients GI leaks do not produce either

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significant abdominal pain or abdominal findings. Instead, "soft" or nonspecific signs such as tachycardia, dyspnea, or fever are much more common. *Therefore, do not think and work "by the book"! Think beyond the accepted frames.*

- Even in developed countries, the majority of hospitals lack appropriate radiological facilities for "human hippopotami." Therefore, it may be necessary to rely on other modalities—your suspicion, clinical judgment, and laparoscopic re-exploration.

The focus of this chapter is directed to the common *acute* complications requiring urgent attention and not to *chronic* complication of bariatric surgery such as cholelithiasis, nutritional deficiencies, inadequate weight loss, esophageal dilatation, port problems after placement of gastric band, and others.

## Laparoscopic Roux-en-Y Gastric Bypass

Laparoscopic Roux-en-Y gastric bypass (RYGB) is the most commonly practiced bariatric procedure and the one considered to be the gold standard for weight loss surgery (› Fig. 31.1).

### *Early Postoperative Complications*

- **Intestinal (anastomotic) leak.** This is the most dreaded complication and a major cause of death in this population; leak rates as high as 7% have been reported. It can originate from either of the anastomoses (gastrojejunostomy or jejunojejunostomy), the staple lines of the gastric pouch or the bypassed stomach, or inadvertent intestinal perforation.

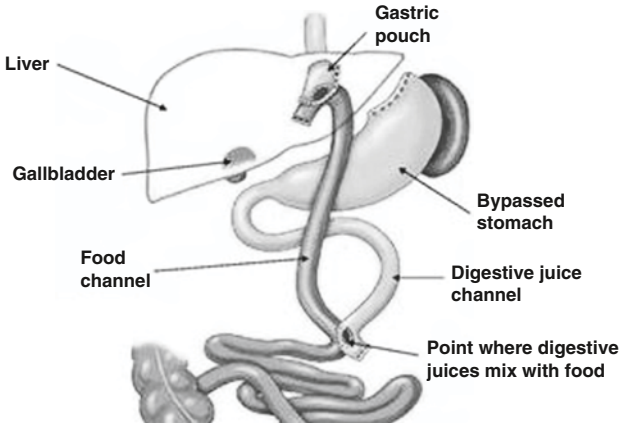


**Fig. 31.1.** Roux-en-Y gastric bypass (RYGB)

The clinical spectrum ranges from "minimal," with subtle presentation, to full-blown peritonitis and sepsis. **Again: you should suspect a leak whenever you are confronted with a patient with dyspnea, tachycardia or fever, abdominal tenderness, and signs of sepsis. Remember that abdominal pain and peritonitis are not reliable in this group of patients; most of the findings are nonspecific and can be confused with an acute cardiac event or PE.** Electrocardiography, blood tests for troponin levels, and even computed tomographic (CT) angiography of the chest may occasionally be necessary to rule out cardiac or pulmonary events. It is imperative that the appropriate tests be expeditiously performed to guide therapy. The diagnosis is made either clinically or by upper gastrointestinal (UGI) study. Be aware, however, that a high percentage of leaks could be missed by the UGI series. CT scan of the abdomen is more sensitive in this regard (if your scanner can accommodate the patient). **Notably, empiric abdominal exploration (usually through the laparoscope) may be the only appropriate diagnostic test. Failure to act quickly can result in the patient's demise.**

**Treatment** depends on the clinical situation. If the leak is contained and controlled and the patient is not septic, it can be treated conservatively with nothing by mouth (nil per os, NPO), broad-spectrum antibiotics, and intravenous nutrition. If the leak has resulted in a drainable abdominal collection, placement of a percutaneous drain is the desired treatment. **If the leak is not contained or the patient is septic, laparoscopic or open exploration is mandatory.** The exploration involves three basic principles: (1) adequate drainage, (2) repair of the leak if possible (of course, such repairs tend to break down, but what do you have to lose?), and (3) gastrostomy tube in the bypassed stomach. A jejunostomy tube (distal to the jejunojejunostomy) should be considered if the leak originates from the bypassed stomach or the jejunojejunal anastomosis (see also Chap. 50).

**Pulmonary embolus.** Obesity is a known risk factor for DVT and PE. Its incidence in RYGB is estimated around 1%. We do not need to repeat here our advice about DVT prophylaxis or discuss the investigation and management of pulmonary thrombo-embolism. Just remember that differentiation of PE from other bariatric surgery disasters can be difficult.

**Acute gastric dilatation.** Albeit rare, this complication can be dramatic. Dilatation of the *bypassed* stomach may occur spontaneously, as a result of obstruction at the "downstream" jejunojejunostomy or secondary to the interruption of the nerves of Latarjet during the creation of the gastric pouch. In the early post-op period, it is most commonly due to bleeding from the gastric staple line. You should suspect this in patients who develop hiccups and abdominal bloating accompanied by tachycardia; hemodynamic compromise may follow in extreme cases (remember: massive acute gastric dilatation produces abdominal compartment syndrome). A plain abdominal radiograph may reveal a large gastric bubble with an air-fluid level. If the stomach is filled with fluid, the radiograph may not be helpful; CT will be diagnostic.

Treatment is by urgent needle decompression of the excluded stomach (under fluoroscopy if time allows) or surgical gastrotomy for clotted blood. *If there is no obvious improvement or in cases of hemodynamic instability, suspect gastric blowout.* An urgent laparotomy is then mandatory. The patency of the jejunojejunostomy (an obstructed anastomosis could be the cause for the dilatation of the excluded stomach) should be verified in every case by a UGI study or during surgical exploration.

- **UGI hemorrhage.** Usually, the source is at the gastrojejunostomy. This should be treated by nonoperative methods and resolves in most cases. In rare instances when the bleeding continues or causes a hemodynamic compromise, open surgical re-exploration with direct control of the bleeding site is imperative. Access to the anastomosis may be gained either through an enterotomy in the jejunal Roux limb or by dismantling of the anastomosis. UGI endoscopy has limitations in this immediate postoperative period.

### *Late Postoperative Complications*

- **Intestinal obstruction**. This usually occurs after weight loss secondary to *internal herniation* through one of the mesenteric defects created by the surgery. With the antecolic technique, two potential defects are created: between the cut edge of the Roux limb mesentery and the mesocolon (Petersen's defect) and at the jejunojenostomy. With the retrocolic technique, there is a mesocolic opening through which the Roux limb is brought up to the gastric pouch. These defects (Fig. 31.2) should be closed during the primary procedure to prevent internal herniation. Following weight loss and disappearance of visceral fat, these defects become large enough to easily allow internal hernias. In addition, adhesions that could prevent these internal herniations are minimal after laparoscopic procedures.

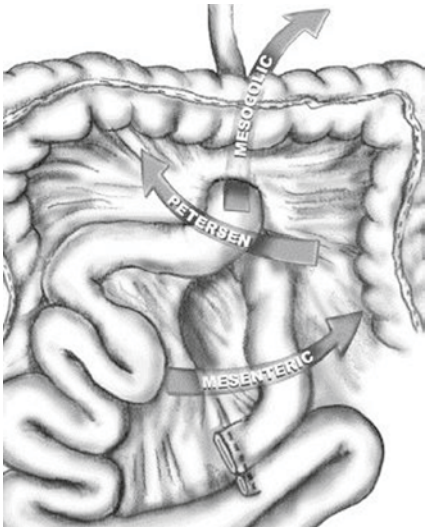


**Fig. 31.2.** Mesenteric defects after RYGB prone to internal herniation

- **The diagnosis of intestinal obstruction after RYGB can be frustrating.** The presentation is usually vague, with crampy midabdominal pain with or without abdominal distension. **Because of the configuration of the GI reconstruction, nausea and vomiting are usually absent.** If vomiting does occur, however, its quality gives some clue to the site of obstruction. Bilious vomiting implies holdup distal to the jejunojejunostomy anastomosis. Multiple episodes may occur with only transient symptoms (spontaneous "in-and-out" herniation). The diagnosis of internal hernia should therefore be suspected in every patient after RYGB presenting with unexplained abdominal pain.
- Some patients present with obvious intestinal obstruction with increasing abdominal pain, tenderness, and some degree of distention. Plain radiograph may be nondiagnostic, UGI series or CT scan are more helpful, but even these will likely be normal if symptoms subside spontaneously.

- The preferred treatment, even in equivocal cases, is laparoscopic exploration, reduction, and closure of the defects. Open exploration would be needed if the patient is hemodynamically unstable or there is necrotic bowel, confused anatomy, or poor visualization due to dilated small bowel loops.

- **Stomal stenosis.** Stricture of the gastrojejunostomy is not rare and presents with severe dysphagia, nausea, vomiting, and at times, odynophagia. Patients should undergo endoscopy for both diagnosis and treatment with balloon dilatation. Occasionally, there is an associated marginal ulcer that should be treated with acid suppression. Persistent ulceration, especially if combined with some weight gain, should raise the possibility of **gastrogastric fistula.** The diagnosis is achieved by UGI series, and it should be treated surgically.

## Laparoscopic Adjustable Gastric Banding (LAGB)

Once the most popular bariatric procedure in Europe and Australia, laparoscopic adjustable gastric banding (LAGB) has lost some appeal due to a high reoperation rate and disappointing long-term effectiveness.

### *Early Postoperative Complications*

- **Gastric perforation.** This has been described in less than 1% of patients undergoing LABG. It is caused by inadvertent gastric wall damage during dissection or the creation of the retrogastric tunnel (Fig. 31.3). The diagnosis and management should follow the general lines outlined for RYGB. Again, confirming the diagnosis could be very difficult even with UGI studies and CT scanning—so keep it in mind and suspect, suspect, and suspect. There should be a low threshold for laparoscopic re-exploration in any case with unexplained

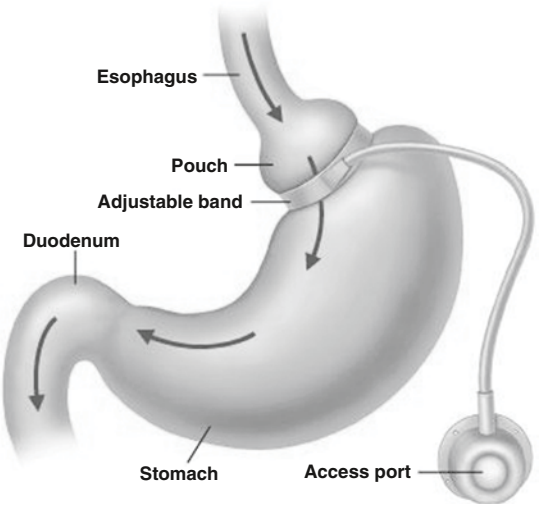


**Fig. 31.3.** Gastric banding

dyspnea, tachycardia, abdominal pain, or fever. **Treatment** includes removal of the band, repair of the perforation site, drainage, intravenous antibiotics, and nutrition, preferably through a jejunostomy feeding tube.

- **Gastric bleeding** is very rare. Unlike after RYGB, endoscopy may be utilized for the diagnosis and treatment.
- **Gastric pouch necrosis**. This rare complication is due to severe dilatation of the pouch proximal to a tight band and edema, possibly combined with operative damage to the gastric wall and its blood supply. The clinical presentation is vague, so you should keep this diagnosis in mind. UGI endoscopy may be helpful, and re-exploration is mandatory for the confirmation of diagnosis and treatment. Obviously, you will have to resect either the proximal stomach (proximal gastrectomy) or do a total gastrectomy.
- **Infection of the port site** usually presents as superficial cellulitis, which should respond to antibiotics. With deeper infections (manifesting with recurrence of the infection, persistent drainage, or periband fluid on imaging), removal of the port is necessary. In some cases, even removal of the entire LAGB system might be required to eradicate extensive infections. These principles apply also to late infections of the port site.

### *Late Postoperative Complications*

- **Band slippage**. This late complication has been associated with a particular technique of band insertion. There is prolapse of the anterior or posterior wall of the stomach. Anterior band slippage with consequent gastric herniation can occur if anterior fixation of the band was inadequate or as a result of early severe vomiting. The clinical presentation can be subacute or chronic, but occasionally

it is acute and dramatic. Symptoms and signs include epigastric pain, pouch dilatation, reflux, vomiting, dysphagia, or even acute obstruction. An upper GI study usually makes the diagnosis. Immediate band deflation followed by laparoscopic exploration should be performed with reduction of the herniated stomach, band repositioning, and anterior fixation of the band. If the slippage is posterior, the band should be removed and the *pars flaccida* technique used to reposition the band. This is obviously in the realm of the bariatric surgeon. Rarely, gastric wall necrosis may result from late diagnosis and treatment.

- **Band erosion**. In most cases, the presentation is subacute or chronic, but it may present as an acute infection of the port site or as intra-abdominal infection. Band erosion into the stomach has been reported in up to 7.5% of cases. Patients are usually asymptomatic, but they may rarely present with an acute abdomen. Diagnosis is made by UGI study or endoscopy. Late port site infection combined with weight gain (as a result of alleviation of the restriction of food after erosion) and occasional epigastric pain should raise suspicion of band erosion. As mentioned, this may cause an intra-abdominal abscess. Management of this complication is usually not an emergency and referral to a bariatric surgeon should be possible. The best treatment is not yet established, and details of this debate are outside the scope of this book, but essentially it entails removal of the entire band system and repair of the gastric wall. In case of an acute perforation with associated abscess, laparotomy and closure of the perforation and wide drainage should be performed. Note that band erosion can lead to port infection but not always. And, vice versa, port infection could occur primarily without band erosion.

## Laparoscopic Sleeve Gastrectomy

Laparoscopic sleeve gastrectomy (LSG) is a relatively novel, and increasingly popular, procedure that was introduced as a stand-alone procedure for morbid obesity. It involves vertical resection of around 80% of the gastric volume along the greater curvature (Fig. 31.4).

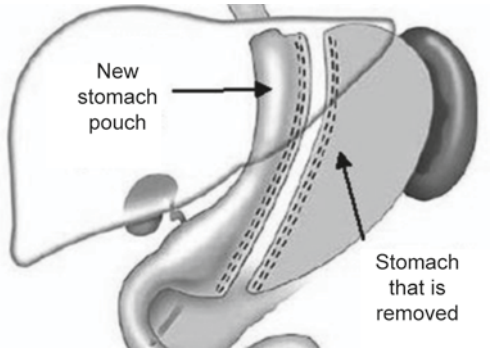


**Fig. 31.4.** Sleeve gastrectomy

### *Early Postoperative Complications*

- **Intra-abdominal bleeding.** This usually originates from the long gastric staple line. In most cases, it stops spontaneously and thus can be treated conservatively. It may be associated with UGI bleeding. Surgical re-exploration is rarely required and involves oversewing of the staple line. Occasionally, the hematoma could get infected and present as an intra-abdominal abscess that requires drainage.
- **Leakage**. This occurs more frequently at the upper part of the staple line, at or just below the gastroesophageal junction. The exact reason for this is not known. Diagnosis and management follow the same principles described for leakages after RYGB. Mild, contained or controlled leaks should be treated with percutaneous drainage, fasting, and intravenous nutrition. Uncontrolled leaks causing peritonitis and sepsis should be treated surgically by peritoneal toilet, attempt at reclosure of the leaking point (this will usually fail), and adequate drainage to achieve a controlled fistula (Chap. 50). In these cases, placement of a feeding jejunostomy is strongly suggested. Most resulting controlled gastric fistulas will close spontaneously.
- **Stenosis of the gastric tube (sleeve).** This is rare and may be seen either early or late. The early form is due to the creation of a too-tight gastric tube (when the staple line is being reinforced with sutures). Patients complain of severe dysphagia, odynophagia, and vomiting. UGI study confirms the diagnosis. Treatment is conservative, and this might require later endoscopic dilatations.
- **Necrosis of the gastric tube.** This is an extremely rare complication that occurs as a result of interruption of the blood supply to the lesser curvature. Early re-exploration with completion (total) gastrectomy is usually required.

### *Late Postoperative Complications*

Except for gastric tube stenosis with the "hourglass figure," all other complications do not require urgent attention. Late stenosis is the result of ischemia, and the diagnosis and management follow the same principles described. In the late form, patients may present with vomiting, regurgitation, and reflux symptoms.

## Biliopancreatic Diversion and Duodenal Switch

Biliopancreatic diversion and duodenal switch (BPD-DS) is a modification of the Scopinaro procedure. It entails the creation of sleeve gastrectomy, duodenal switch, and intestinal bypass (Fig. 31.5). In the acute setting, one can see the same complications described for other bariatric procedures. The same principles for the management apply here as well.

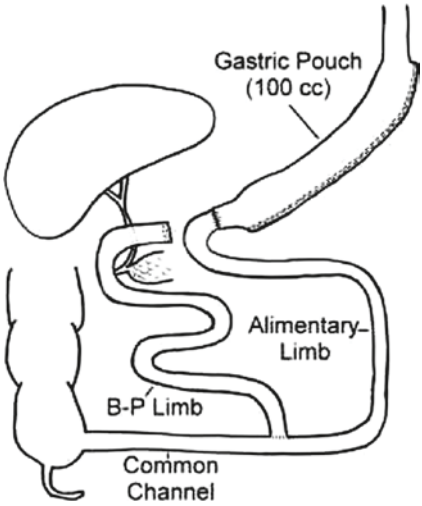


**Fig. 31.5.** Biliopancreatic diversion and duodenal switch (BPD-DS)

## Editorial Comment

While bariatric procedures performed in "good hands" should be safe, when complications develop the morbidity and mortality reach serious proportions. Surgical complications of bariatric procedures are far from being as funny as Fig. 31.6 would suggest.

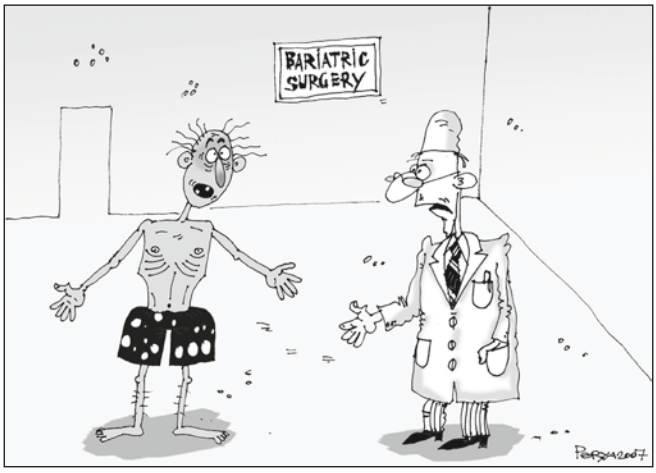


**Fig. 31.6.** "It seems that your band is a little too tight!"

Obviously bariatric surgeons are (it is hoped) those with the optimal expertise to treat all these complications. Thus, if your practice does not routinely include bariatric surgery, try to refer at least those patients who do not require an immediate lifesaving operation back to their original surgeons. Increasingly, however, patients shop online for these procedures and travel by air to boutique clinics elsewhere. After undergoing the operation, they fly back home and present with the complication on your doorstep; by that time, their original surgeon is scuba diving in the Bahamas or skiing in St. Moritz.

> "Bariatric surgeons should inhabit a special place in Hell, where they are condemned to deal with the complications they have created." (Angus Mciver)

# 32 Complications of Peritoneal Dialysis

Hany Bahouth

In some regions of the world, up to half of end-stage renal disease patients are maintained on peritoneal dialysis (PD). The curled and straight silicon *Tenckhoff* catheters still enjoy wide use and are considered to be the standard of care for PD use. But, as with any foreign body chronically implanted into the human body, these catheters tend to cause complications. And, it is you—the general surgeon who may or may not have inserted the catheter—who must now deal with these problems.

## Early Complications of PD Catheters

Early complications of PD catheters that occur within 30 days of insertion are mainly technical.

- **Wound infection/hematoma.** Treat as any other surgical wound infection with drainage and local care (but add antibiotics because of the proximity of the drain).
- **Exit site infection.** The main pathogens are ***Staphylococcus aureus*** and Gram-negative bacteria (*Pseudomonas*). If the presentation is in the form of erythema without purulent discharge, treat the exit site with your topical agent of choice. If it is accompanied by purulent discharge, then systemic antibiotics should be prescribed, guided by Gram stain and cultures. Withhold PD until recovery. In the absence of improvement after 2 weeks of treatment, the catheter should be removed.
- **Leakage.** Usually, this begins early after commencing PD. You may see fluid leaking at the exit site, subcutaneous swelling, or subcutaneous edema without signs of infection or any abdominal complaints. Ultrasound (US) can help in confirming the diagnosis by detecting fluid surrounding the tunnel and cuffs of the catheter. Computed tomographic (CT) scan with contrast medium injected

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with the dialysate can also make the diagnosis. This should be managed by holding PD for around 1–2 weeks. If leakage recurs after two or three trials, the catheter should be replaced by a new one, preferably through a new exit site.

- **Malposition.** You might be called for localized abdominal pain that starts shortly after the initiation of PD. If this is associated with a slow inflow of dialysate and with local swelling but without signs of local peritonitis or infection, then this may be a sign of malposition of the catheter (e.g., preperitoneal placement). This should be treated by removal of the catheter and reinsertion at another site.
- **Vascular injury.** Minor bleeding almost always settles spontaneously, but major hemorrhage signified by gross bloody effluent may need resuscitation and laparotomy to find and treat the source of bleeding. Whether to reinsert a new PD catheter depends on the general condition of the patient. Generally, our aim is to complete the job at the same session.
- **Bowel perforation.** Together with large vessel injury, this is the most feared complication. This is diagnosed by the appearance of gas and feces in the effluent or "osmotic" watery diarrhea if the dyalisate enters the bowel. There may be signs of systemic infection, various degrees of abdominal pain and tenderness, or generalized peritonitis. If the patient is septic or suffers diffuse peritonitis, an urgent laparotomy is mandatory for source control. Leave the catheter in place to guide you to the perforation site. Delay PD for 2–3 weeks after surgical repair (obviously, a new PD catheter will be needed). In the absence of diffuse peritonitis or sepsis and after ruling out an intra-abdominal abscess by CT scan or US, you may manage the situation nonoperatively. The rationale is that this is a limited injury to the bowel by the PD catheter and can heal spontaneously when managed like any "controlled" intestinal fistula. Keep the patient receiving nothing by mouth, start parenteral nutrition and broad-spectrum antibiotics, and stop PD. If fecal effluent continues for more than 2–3 weeks, this suggests that the catheter may be intraluminal. CT or injection of contrast under fluoroscopy will confirm this. In this case, gradually withdraw the catheter over a few days to allow for the creation of a controlled tract of the fistula until its complete removal. Then, treat this as any other fistula.
- **Urinary bladder injury.** The suspicion rises with the appearance of polyuria and glycosuria and perhaps slow inflow. This should be treated with early laparotomy for repair of the urinary bladder. Normally, you do not need more than one or two stitches to the wall of the bladder, and you do not have to call the urologist for that!

### Late Complications of PD Catheters

Complications developing more than a month after the insertion of the catheter are related to chronic catheter use. The most common is **peritonitis**. The usual catheter-related peritonitis is considered as *primary* (see Chap. 12) but do not

forget that these patients can develop **secondary** peritonitis as well (e.g., appendicitis or perforated bowel).

The causative organisms of **primary peritonitis** are mainly Gram-positive (*Staphylococci*) but Gram-negative organisms and fungi are occasionally responsible. The most common symptom is abdominal pain, which is diffuse and ill defined. Fever and leukocytosis may be present as well. Cloudy peritoneal effluent is a common finding. Send the fluid for analysis; a white cell count of more than 100/cc$^3$ (with more than 50% neutrophils) and positive Gram stain for bacteria will confirm the diagnosis.

The management is nonoperative and consists of appropriate intravenous antibiotics (obviously, adjusted for renal function or dysfunction), cessation of PD through the catheter, and close observation.

***When to remove the catheter?***

- *Refractory peritonitis*. Defined as peritonitis treated with appropriate antibiotics for more than 5 days without resolution. In such cases, US or CT scan of the abdomen is indicated to rule out another source of intra-abdominal pathology (i.e., *secondary* peritonitis).
- *Relapsing peritonitis*. Peritonitis with the same organism within 4 weeks of stopping antimicrobial therapy. In cases of relapsing infection with *Pseudomonas*, removal of the catheter is highly recommended.
- *Peritonitis with catheter obstruction*.
- *Fungal peritonitis*. *Candida* is the most common species.
- *Secondary peritonitis* (discussed in the next paragraph).
- *Mycobacterial infection*.

We wish to stress again that you cannot ignore the possibility of **secondary peritonitis**. Careful history, abdominal examination, and a high index of awareness are needed to rule out secondary peritonitis. Analysis of the effluent fluid might disclose multiple enteric organisms. Judicious use of imaging modalities can help to proceed to the right diagnosis. When operating for source control, removal of the catheter is mandatory.

Other less-frequent late complications that may require urgent attention include pericatheter or pre-existent **abdominal wall hernias** that become symptomatic and gradually enlarge after the initiation of PD. These may produce painful bulges, scrotal edema, and abdominal pain during PD. Normally, you have to stop PD and evaluate for possible incarceration. If this is ruled out, then repair is scheduled on a semi-elective basis. In this case, you have to allow for adequate healing before gradually resuming PD (usually 2–3 weeks).

The main message of this chapter could be summarized in a few words: "Treat infection and remove the catheter" (Fig. 32.1). But of course, whenever you stop PD or remove the catheter the patient has to be placed on hemodialysis.—[The Editors]

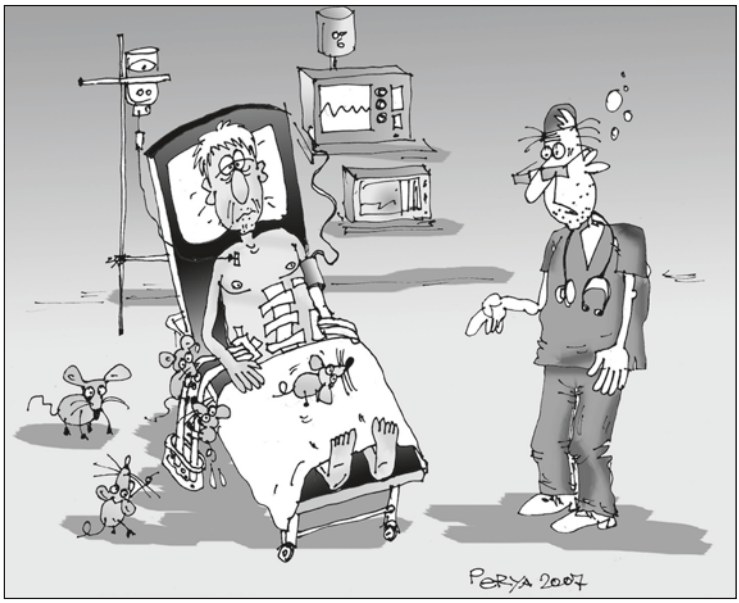


**Fig. 32.1.** "Gosh, they are all coming out of your tube; we'll have to remove it."

# 33 Gynecological Emergencies

Bernard Cristalli · Moshe Schein

*Have you ever seen a gynecologist who is convinced that the "acute abdomen" is gynecological in origin, and not due to acute appendicitis?* (MS)

*Have you ever seen a surgeon who is convinced that the "pelvic acute abdomen" in a woman is surgical and not due to a gynecological affliction?* (B. Cristalli)

The famous English writer and physician Somerset Maugham (1874–1965) wrote: "**Woman is an animal that micturates once a day, defecates once a week, menstruates once a month, parturates once a year and copulates whenever she has the opportunity.**" One could not pen such a politically incorrect statement today, but if allowed, we might have added to it some comment about "lower abdominal pain"…

In most locales, general surgeons are not expected to deliver babies, but you are likely to face a gynecological emergency that you should know how to handle. Acute abdominal pain is very common in women during their reproductive years. Such pain is as likely to be gynecological as it is to be "surgical." Your gynecological colleagues (excluding of course Dr. Cristalli) are generally good folk but typically possess a vision limited by the boundaries of the bony pelvis (› Fig. 33.1). Consequently, they are often reluctant to diagnose any acute condition as "gynecological" unless you have ruled out acute appendicitis. Occasionally, you operate for what you think is acute appendicitis, and the findings are gynecological. You should know how to deal with this. Another situation that provides you with the pleasure of interacting with gynecologists-obstetricians is dealing with the pregnant patient. As you know, pregnancy itself may be the cause of abdominal pain; at the same time, it may modify the presentation of common surgical disorders, making diagnosis difficult. It may also pose considerable challenges in the injured patient. For this edition, we have collaborated with a gynecologist and obstetrician—Dr. Berni Cristalli of Paris–and are happy to present a revised and much expanded version of this chapter.—[The Editors]

Acute pelvic emergencies are extremely common, and both surgeon and gynecologist must be able to understand what is going on and determine whether it is the former or the latter who will be in charge. If you happen to be on call and

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**Fig. 33.1.** "Call the general surgeon!"

see women, whether referred directly or via your friendly gynecologist, you will encounter mainly two kinds of syndrome: **pain** and **bleeding**. These two conditions may present alone or be associated with other symptoms, such as fever, vaginal discharge, and others. We are not going to address painless bleeding, which is the bread and butter of the gynecological practice.

The age of the woman is an important consideration as the gynecological pathology you are likely to encounter differs markedly among the following groups: premenstrual, menstrual-fertile, pregnant, menopausal—each group with its typical disease profile and consequently different clinical approach.

## Acute Abdominal Pain in the Fertile Woman

### *Assessment*

We do not have to remind you to take a history concerning *menstruation*, *sexual activity*, and *contraception*. Pregnancy, whether uterine or *ectopic*, should always be ruled out; this is done in most hospitals with a rapid pregnancy test. Any history of pain that occurs during the first days of the menstrual period hints at underlying *endometriosis* or *endometrioma* ("chocolate cyst"). Acute pain developing midcycle (*mittelschmerz*) may be due to rupture of the Graafian follicle at ovulation. Pain referred to the shoulder raises the possibility of *free intraperitoneal blood* irritating the diaphragm, with a likely source of bleeding being *a ruptured ovarian cyst* or an *ectopic pregnancy*.

We do not need to talk to you about physical examination. You surely know that the conditions to be discussed can produce signs of peritoneal irritation, often indistinguishable from those of acute appendicitis. However, the *site of pain and local findings on examination* are helpful in narrowing the differential diagnosis. When bilateral, consider pelvic inflammatory disease (*PID*); when on the right, think about acute appendicitis; when on the left, in an older lady, consider acute diverticulitis (Chap. 3). Bimanual vaginal examination performed by your gynecological friend (or by you) is an essential part of the assessment of these patients. You are palpating for masses or fullness in the cul-de-sac (pouch of Douglas) and looking for *excitation tenderness*—when moving the cervix produces a lot of pain (PID, ectopic pregnancy).

Ultrasound (it is hoped your gynecologist friend is armed with a transvaginal US) is the key investigation, allowing visualization of any free fluid, the uterus, and adnexae. When fluid is present in the cul-de-sac, it can be aspirated with a needle through the vagina (*culdocentesis*). When pus is present, think about PID or perforated appendicitis, while blood hints at a ruptured cyst or ectopic pregnancy.

Generally, most acutely *painful* gynecological conditions are treated nonoperatively. With all the information just given at hand, your job, together with the gynecologist, is to classify the patient into one of the following groups:

- "Benign" abdominal examination—most probably a gynecological condition. Treat conservatively.
- "Impressive" abdominal examination with no apparent gynecological pathology. This is perhaps the best indication for diagnostic/therapeutic laparoscopy.
- "Not sure." Admit and observe with or without a computed tomographic (CT) scan (Chaps. 3 and 28).

## Ectopic Pregnancy

The great French surgeon Henri Mondor (1885–1962) said:

> "When faced with an acute abdomen, consider ectopic pregnancy, think always about it always. Just thinking about it again is not enough, keep thinking about it."

*Ectopic* means that the fertilized ovum has implanted somewhere outside the usual location (i.e., the body of the uterus). The most common site for an ectopic is the tubes, but implantation may occur in the ovary, cervix, and abdominal

cavity. Heterotopic pregnancy (intrauterine and ectopic pregnancy at the same time) is so rare that if a normal pregnancy is seen, an ectopic can be ruled out. Abdominal pregnancy is a late ectopic pregnancy with development of a fetus.

Although the presentation of these patients varies tremendously, typically they have abdominal pain and vaginal bleeding. Many women do not even know about the pregnancy, ignoring associated symptoms of pregnancy such as a missed menstrual period. Some elements of history may be considered risk factors: previous history of ectopic pregnancy, PID, tubal surgery (including tubal ligation!), endometriosis. Contraception with an intrauterine device (IUD) is not a risk factor in itself, but an early pregnancy with an IUD in situ has to be considered ectopic until proven otherwise. An IUD prevents intrauterine pregnancies but not ectopics!

The diagnosis rests on a tripod of pregnancy, pain, and bleeding. Typically, the patient arrives with a sharp and sudden unilateral pelvic pain, mild brownish bleeding, and pregnancy (positive pregnancy test) with an empty uterus at US. The diagnosis comes easily when the woman knows she is pregnant and has vaginal bleeding. It can be a lot more difficult when pain is the only sign, and the pregnancy is yet to be discovered. Cataclysmic hemorrhage is very rare now, but any internal hemorrhage syndrome in a woman is a ruptured ectopic pregnancy until proven otherwise.

**Physical findings:** signs of hypovolemic shock and peritoneal irritation are proportional to the amount of blood loss. On pelvic examination, you may find a parauterine painful mass or at least a "little something" next to the uterus. The pouch of Douglas is tender and may contain a boggy mass (hematocele).

**Ultrasound is the imaging of choice** to show the ectopic gestational sac and free intraperitoneal bleeding.

**Management**: although some ectopic pregnancies may resolve and absorb spontaneously over time, the standard of care is an operative approach in all cases. As a general surgeon, you are more likely to be involved with the more dramatic scenario of a ruptured tubal ectopic (usually affecting the distal segment of the tube), which may occur as early as the fourth week of gestation. The sudden development of acute peritonitis and **hypovolemic shock** will force you to rush to the operating room without the gynecologist and perform a laparotomy. Whether to enter the abdomen through a midline incision or a Pfannenstiel incision depends on the urgency of the situation and the build of the patient. **Evacuate** the gestational sac, control the bleeding sites with suture-ligatures, and preserve the ovary. Less-dramatic presentations are usually managed by or in partnership with the gynecologist, preferably through the laparoscope. In early cases, the uterus is normal or mildly enlarged, and the ectopic pregnancy can be seen as a tube swollen by a blue "tumor"; there is a small-to-moderate amount of black blood in the pouch of Douglas. Note that in most ectopics at operation the bleeding has already stopped; when it is active, it may necessitate a simple salpingectomy. When the ovaries are left intact, the patient can still undergo in vitro fertilization even after bilateral salpingectomies.

## Ovarian Cysts

Ovarian cysts are common in young women; they are usually "functional" *cysts* (follicular or corpus luteum) and mostly asymptomatic. However, when cysts develop in postmenopausal women, ovarian cancer has to be suspected and excluded. Only complicated ovarian cysts, regardless of etiology, present as surgical emergencies.

*Acute pain develops when a cyst bleeds or undergoes torsion.* The intensity of pain and abdominal signs of peritoneal irritation are proportional to the amount of bleeding. Pain is severe in the case of torsion. In women of childbearing age, complications of ovarian cyst may mimic acute appendicitis. To prevent unnecessary operation, you have to image the abdomen (Chap. 28).

**Imaging:** typically, functional cysts are solitary, simple, and small (<8 cm). Free fluid in the pouch of Douglas suggests rupture and bleeding. Larger and more complex cysts suggest pathology, such as dermoid cyst. Absence of blood flow on US strongly indicates torsion. Commonly today, such patients initially undergo a CT examination "to exclude acute appendicitis," which in addition to showing a normal appendix may document the free pelvic fluid and the ovarian pathology. If this is the case, we would follow up with a transvaginal US, which is more accurate in delineating the pelvic pathology.

**Management:** small (<8 cm) simple ruptured cysts with minimal local and systemic findings should be treated conservatively. If, however, the rupture results in significant intraperitoneal hemorrhage and when another pathology cannot be ruled out (e.g., larger or complex cysts), surgical intervention is indicated. Laparoscopy is preferable for smaller cysts and when malignancy is not suspected, but for very large cysts (>10 cm) laparotomy allows removal of the intact ovarian mass without disrupting it. Whether you can do it through a Pfannenstiel incision depends on the build of the patient. *Torsion* is usually associated with more severe and persistent pain and more dramatic abdominal findings together with systemic manifestations; it is an indication for operation. At operation, if there is active bleeding from the cyst, obtain local hemostasis by whichever means. There is no need to aspirate or resect the cyst and, please, do not even think of removing the ovary. If viable, the tube and ovary can be detorted and conserved; only if clearly nonviable is the ovary resected. Dermoid cysts are resected. Discussion of ovarian malignancies is beyond the scope of this book.

## Pelvic Inflammatory Disease

Pelvic inflammatory disease is seldom a surgical emergency now, but it remains a frequent reason to visit the emergency room. Its is an infective syndrome that involves, to a greater or lesser extent, the endometrium, tubes, and

ovaries. The patient is commonly young and sexually active. The clinical spectrum of infection is wide, ranging from minimal pain, dyspareunia, fever, and vaginal discharge, associated with mild endometritis/salpingitis, to severe peritonitis and septic shock due to ruptured tubo-ovarian abscess. Likewise, physical findings depend on the disease process and vary from localized abdominal tenderness to generalized tenderness and rebound. Note that the pain and tenderness are commonly *bilateral*. Pelvic examination reveals purulent discharge with cervical motion tenderness. Ovarian or pelvic abscesses may be palpated or seen on US or CT.

**Treatment:** without treatment, the infection may develop into a tubal abscess and then spread intrapelvically and result in a true peritonitis. The late risk is tubal obstruction and pelvic adhesions, leading to infertility and chronic pelvic pain. The majority of mild cases should be treated with antibiotics. Outpatient treatment is appropriate for patients who can tolerate oral diet. Patients with severe abdominal and systemic manifestation should be admitted for intravenous antibiotic therapy. Antibiotic treatment is empiric, targeting the common causative organisms, which are, in isolation or combination, *Chlamydia trachomatis, Neisseria gonorrhea, Escherichia coli*, and *Haemophilus influenzae*. Many oral and intravenous agents are available.

Patients who do not respond to this regimen or in whom the diagnosis is uncertain are subjected to laparoscopy. This should be left to the gynecologist. The typical case you will be involved with is the ruptured tubo-ovarian abscess, causing severe pelvic or diffuse peritonitis. During laparotomy or laparoscopy, you will find pus; you read how to deal with peritonitis in Chap. 12. The abscess should be drained; whether to remove the uterus and ovaries depends on the age of the patient, the operative findings, and the patient's gynecologist. When talking about PID, "formal" textbooks usually mention the *Curtis-Fitz-Hugh syndrome* or "perihepatitis" as a late sequela—ascending from the pelvis. Although originally associated with *gonococcal* infection, nearly all present-day cases are associated with *C. trachomatis* infection. It may produce nonspecific abdominal complaints and has been reported to mimic acute cholecystitis, but in our experience it has never represented a specific entity warranting operative measures. We have seen it, however, as an incidental finding of perihepatic "piano-string" adhesions at laparoscopy or laparotomy for other conditions.

## Vaginal Tears

Vaginal tears are rare but may cause severe hemorrhage representing a true gynecological emergency. A vaginal tear can occur in young females at their first intercourse—the "bloody defloration" (what a way to spend her wedding night!). It can affect women of any age who experience violent or peculiar sexual relations

(instruments, bottles, and so on) alone or with a partner. Always suspect that rape may have been a causative factor. Clinically, the bleeding is obvious. Diagnosis is by speculum examination: there is a lateral laceration, beginning at the hymen and extending upward, and the edges are rather neat. In some cases, the tear is transmural and involves the cul-de-sac. Treatment consists of hemorrhage control and repair of the laceration with an absorbable continuous stitch in the lithotomy position; whether to do it under local or general anesthesia depends on the extent of the laceration and the individual patient.

## Acute Abdominal Pain in the Pregnant Woman

> "In men nine out of ten abdominal tumors are malignant; in women nine out of ten abdominal swellings are the pregnant uterus." (Rutherford Morrison, 1853–1939)

### *General Considerations*

A consultation about abdominal pain in a pregnant or immediately postpartum woman is frequently an anxiety-provoking experience for the general surgeon. We think that the following few paragraphs will help you to approach these difficult problems with a new understanding and confidence based on some simple concepts.

Abdominal emergencies in pregnant women pose a great challenge for the following reasons:

- The ascending uterus gradually distorts the normal abdominal anatomy, displacing organs and thus changing the typical clinical scenario.
- Physiologically, the pregnant woman is different; nausea and vomiting are not uncommon during the first trimester, thereafter, tachycardia, mild elevation of temperature, and leukocytosis are considered "normal."
- To a certain degree, abdominal "aches and pains" are common during pregnancy.
- When dealing with a sick pregnant woman, you automatically have two patients; the life and well-being of the fetus have to be considered also.

When it comes to treatment, there may be a conflict of interest between the mother and fetus. Early in pregnancy, the risk is of miscarriage, while at the end it is premature labor; in both cases, it is hard to determine which is more risky—surgery or nonoperative management.

Generally, acute abdominal conditions during pregnancy are either "because" or "in spite" of pregnancy.

**Abdominal emergencies specific to pregnancy are either:**

- **Obstetric**—Such as ectopic pregnancy (discussed separately in a previous section), abortion, and septic abortion (a septic uterus may present with an impressive acute abdomen); "red degeneration" of a fibroid; abruptio placenta; rupture of uterus; and pre-eclampsia. These conditions are not further discussed. Hey, we did not promise you a manual of obstetrics.
- **General**—Such as acute pyelonephritis, which is more common in pregnant women, or rupture of visceral aneurysm (e.g., splenic artery), which is rare but "typically" occurs during pregnancy. Another condition, which may be associated with pregnancy, is *spontaneous hematoma of the rectus abdominis muscle*. (This condition may also develop in nonpregnant men and women, particularly in anticoagulated patients.) The hematoma originates from a ruptured branch of the inferior epigastric artery and develops deep to the muscle. On examination, a tender abdominal wall mass is often felt; it will not disappear when the patient tenses his or her abdominal wall (Fothergill's sign). US or a CT can confirm the diagnosis. Treatment is conservative.

### *Abdominal Emergencies Randomly Developing During Pregnancy*

Any abdominal emergency may occur during pregnancy. Here are a few basic considerations:

- **"Think in trimesters".** During the *first trimester* the fetus is most susceptible to the potential damaging effects of drugs and X-rays. Abdominal operations at this stage may precipitate an abortion. Operations during the *third trimester* are more likely to induce premature labor, posing additional risk to the mother and fetus. Thus, surgery is best tolerated during the second trimester—if you have the luxury of choice.
- **The well-being of the mother overrides that of the fetus**. If maternal and fetal distress are present simultaneously on presentation, all therapeutic efforts should be for the benefit of the mother. A Caesarean section is considered only if the fetus is more than 24 weeks old and in persistent distress in spite of maximal therapy to the mother.
- **Pregnant women suffer from a chronic abdominal compartment syndrome** (Chap. 40). The abdominal emergency (e.g., perforated appendicitis or intestinal obstruction) will further increase the intra-abdominal pressure, reducing venous return and cardiac output. Place such patients in *a left lateral decubitus position* to shift the gravid uterus away from the compressed inferior vena cava.

**You should be aware of:**

- **Acute appendicitis.** You are commonly called to "exclude acute appendicitis" in a pregnant woman. Address the problem as discussed in Chap. 28; although the cecum is usually fixed in place, it may be displaced by the gravid uterus, and the omentum is "lifted" away and thus may not provide "walling-

off" protection to the perforated appendix, making free perforation more likely. US may help in excluding acute cholecystitis and ovarian or uterine causes of pain and may document an enlarged appendix. CT is not advisable because of the risks of irradiation of the fetus. Diagnostic laparoscopy or laparoscopic appendectomy during pregnancy have been reported safe to both mother and fetus but still remain somewhat controversial, particularly in late pregnancy. If you choose to operate, tilt the table to the left and place a muscle-splitting incision *directly over the point of maximal tenderness*, wherever it is (it may be higher than usual). And, remember that the "best" treatment of acute appendicitis occurring early in pregnancy may be nonoperative—with antibiotics (see Chap. 28).

- **Acute cholecystitis.** It is easily recognized clinically and ultrasonographically (Chap. 20.1) during pregnancy. During the first trimester, try conservative management, delaying the operation to the second trimester. If it occurs during the third trimester, try to postpone the operation, if possible, until after delivery. Laparoscopic cholecystectomy appears safe during pregnancy. Inflate the abdomen with the lowest pressure possible and rotate the table well to the left to decrease compression of the inferior vena cava by the uterus. When cholecystectomy is required late in pregnancy (when the uterus fills the entire abdominal cavity), we prefer an open approach through a small subcostal incision. This is perhaps the place to mention the *HELLP Syndrome* (hemolysis, elevated liver enzymes, and low platelet count). It is a relatively rare syndrome that may develop in a pre-eclamptic, preterm, patient and be confused with acute biliary disease (even "mild" HELLP may stretch the liver capsule, producing severe right upper quadrant pain). Liver hemorrhage and hematoma and even liver rupture are serious complications of the HELLP syndrome and represent surgical emergencies; the child should be promptly delivered and the liver managed based on trauma principles. In the unstable, coagulopathic patient, the liver should be packed (Chap. 39). Think about HELLP: a misguided cholecystectomy may kill the mother and baby.
- **Intestinal obstruction***: sigmoid* or *cecal volvulus* is more common during late pregnancy. The displacement of abdominal structures during pregnancy may also shift long-standing adhesions, producing small bowel obstruction or volvulus. Pregnancy tends to cloud presenting features and impedes early diagnosis. Notice that a few plain abdominal X-rays, with or without Gastrografin (Chaps. 4 and 21), are entirely safe even in early pregnancy. So, if you suspect a large or small bowel obstruction, do not hesitate. Remember that intestinal strangulation threatens the life of the mother and her child. This is no time for timidity.

## Trauma in Pregnancy

The management of abdominal trauma in pregnancy is identical to the management in the nonpregnant woman (Chaps. 38 and 39), except that in pregnancy there is concern for two patients—the mother and the fetus. Remember that

the pregnant woman has a marked increase in blood volume, a fact that tends to mask or delay clinical features of hypovolemic shock. Assessment of the fetal status either by Doppler or by continuous cardiotocodynamometry is mandatory when the clinical circumstances permit. The major clinical concerns in the injured pregnant female are uterine rupture and abruptio placentae. The former condition is suggested by abdominal tenderness and signs of peritoneal irritation, sometimes in conjunction with palpable fetal parts or inability to palpate the fundus. The latter is suggested by vaginal bleeding and uterine contractions. When the fetus is in jeopardy, a rapid Caesarean section is usually in the best interests of both the mother and fetus.

## The "Postpartum" Period

Abdominal emergencies are notoriously difficult to diagnose during the early postpartum or post-Caesarean section period. Abdominal pain and gastrointestinal symptoms are commonly attributed to "afterpain" and fever or systemic malaise to "residual endometritis." In addition, at this stage the abdominal wall is maximally stretched and redundant, such that guarding and other peritoneal signs may be missing. "Things move around" the abdomen during delivery, and a loop of bowel may be twisted or caught. We have treated perforated acute appendicitis, perforated peptic ulcer, and acute cholecystitis during the early postpartum days. Diagnosis is usually delayed and so is the treatment. Be aware.

Six men give a doctor less to do than one woman (a Spanish proverb).

# 34 Urological Emergencies

Jack Baniel

Urology is not associated with many emergencies. Actually, one of the main advantages enjoyed by the senior urologist is that most of our surgery is elective, and unlike his or her fellow general surgeon, nights may be spent out of the operating room (OR) and in the warm bed. Most acute urological problems are managed in the emergency room (ER) with the help of other disciplines. I discuss here those common scenarios that a general surgeon may encounter, and solve, while the urologist slumbers.

## Acute Renal Colic

Renal colic is quite easy to diagnose and usually involves intrinsic obstruction of the renal pelvis or ureter by a stone. The classic complaint is of acute flank pain in a restless patient. The pain radiates from the back forward, is spasmodic, and is recurrent. It is often associated with nausea and, less frequently, vomiting. The pain is caused by the dilatation of the urinary tract proximal to the stone. As the stone travels down the ureter by force of the forward pressure caused by the urine flow, the location of the pain changes, radiating toward the lower abdomen, inguinal area, and then the genitalia. As the stone reaches the lower ureter, the patient will complain of frequency and urgency, and then all abates as the stone is expelled into the bladder. Thus, one may trace the advancement of the stone by the patient's complaints. Stones need to pass three narrowings in the collecting system on their way to the bladder: the uretropelvic junction, the iliac vessels, and the vesicoureteral junction. These points are where calculi usually get stuck.

The most important factors to assess in this situation are the stone's width and its location within the urinary system. Most stones less than 5 mm in size and those in the lower ureter (beyond the iliac vessels) should be expelled spontaneously (80–90%) and thus are managed expectantly. Larger stones and those

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higher in the ureter need to be manipulated out. Most stones that pass spontaneously do so within 3–4 weeks.

### *Diagnosis*

Most stones are radiopaque and thus a regular plain abdominal X-ray is the initial diagnostic step. Looking at the X-ray, we use the rule of 4 *S*'s: when searching for stones check that the side corresponds to the pain, that the skeleton does not hold surprises (*metastasis*), and that there are no suspicious silhouettes (tumor).

Noncontrast computed tomography (NCCT) is the gold standard in the diagnosis of stones in the ER setting. NCCT may diagnose all stones regardless of their composition (uric acid, etc.). Ultrasound is helpful in the assessment of hydronephrosis and obstruction: urine flow into the bladder is visualized by urine jets in the bladder; its absence is a surrogate marker of obstruction.

### *Management*

The pain of ureteric colic is mediated by prostaglandins and therefore, intravenous nonsteroidal anti-inflammatory drugs (NSAIDs) are the drug of choice in the management of pain. Fluids are given to increase diuresis and force the stone down the ureter, and smooth muscle relaxants (e.g., intravenous papaverine) also have merit in alleviating acute pain. Steroids and calcium channel blockers have been tried in the past with minimal value. Lately, tamsulosin, an alpha-adrenergic blocker used for prostatism, has been found to facilitate stone expulsion.

**Remember:** on initial assessment of the patient, look for signs of **infection** or **renal dysfunction**. These along with **intractable pain** are indications for hospitalization.

Laboratory tests should include a complete blood count and those for creatinine and electrolytes. Some patients presenting with renal colic will be septic or in severe renal failure (beware patients with a single kidney). These patients must be admitted to the hospital and have emergency decompression of the collecting system as the penalty for delay in treatment may be death from sepsis. Decompression may be done by insertion of a self-retaining stent (JJ) (now, you will have to call your urologist) or by percutaneous nephrostomy by the radiologist. The available options to get rid of the obstructing ureteral stone disease are (usually) to insert a stent and fragment the stone later (shock wave lithotripsy) or to perform immediate ureteroscopy and stone fragmentation with laser. Ureteroscopy is the definitive solution for most lower ureteral stones. Stones in the upper ureter or in the renal pelvis are usually fragmented by ESWL (external shock wave lithotripsy).

## Torsion of Testis (see also Chap. 35)

As a general surgeon, you will see most "acute scrotal conditions" well before the urologist; some may present as depicted in Fig. 34.1. Torsion of the spermatic cord is the most dramatic "acute scrotum"; it requires emergency management, and if missed the testis will be lost. It commonly occurs in young boys but may appear at all ages, even in the neonate.

As the testis descends through the inguinal canal, it pushes ahead of it a sliver of peritoneum. As the testis reaches the scrotum, the peritoneum is sealed off, and only the part attached to the lower pole of the testis is left; this actually fixes the lower testicular pole to the scrotal wall. But, the peritoneum may adhere higher around the spermatic cord and wrap the entire testis within an isolated peritoneal sac. In this situation, the testis may rotate, twist itself around its vessels within the *tunica vaginalis* (the retained part of the peritoneum), and cause acute ischemia. This anomaly occurs equally on both sides of the scrotum. Medical literature from the 1960s reporting on this phenomenon observed a high frequency of delay in diagnosis and a very high orchiectomy rate. With more attention paid to the clinical symptoms and the adoption of an aggressive operative approach, most torted testes can be saved.

The classic symptoms are acute unilateral scrotal pain, swelling, nausea, and vomiting, without fever or urinary symptoms. Usually, there are difficulties in gait as the patient wishes to keep his legs apart to avoid pressure on the scrotum. Often, the presentation is not so clear, and pain and swelling are the only signs. The most common differential diagnoses are inflammatory intrascrotal conditions (e.g., epididymitis, orchitis), but in the young torsion is more frequent

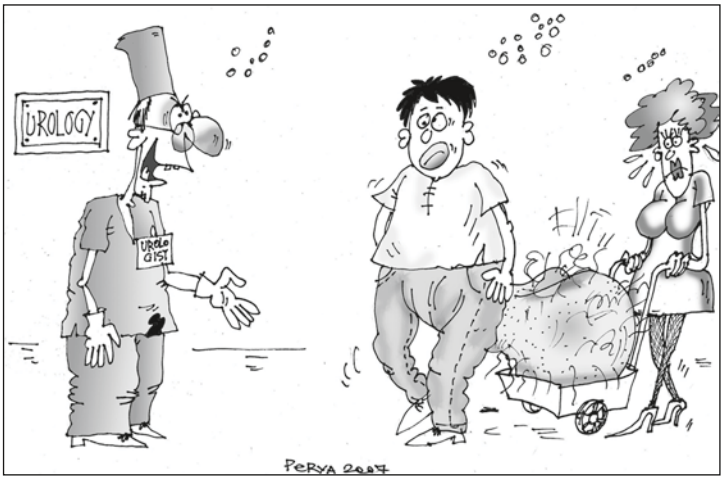


**Fig. 34.1.** "What's that? A watermelon?"

than inflammation. Torsion of a **testicular appendage** may also occur and confuse the examining physician. The testis has two appendages, emanating from the testis itself at the lower pole and from the epididymis. If they twist around their origin, a large scrotal swelling occurs that is very painful. In this case, the testis itself is normal. On examination, one may see a local enlargement called a "blue dot."

Signs of spermatic cord torsion on examination include a high-riding testis, transverse lie, a negative *dartos sign* (normally, stroking the thigh elevates the testis), and local pain and sensitivity.

The diagnosis may be assisted by a Doppler ultrasound, which may show reduced testicular perfusion—an indication for exploration. Equivocal ultrasound results along with indicative signs and symptoms warrant surgical exploration. The testis may withstand 4–6 hrs of ischemia, after which there are irreversible changes that result in atrophy. Practically, accurate assessment of time of onset of the torsion is usually difficult; thus, the recommendation is to explore the affected testis whenever signs are significant.

Exploration is performed trans-scrotally; the exposed testis is "untorted" and wrapped in warm pads. If blood flow returns (as seen by a pinkish color), the testis is fixed with nonabsorbable sutures to the scrotal wall at least at three places. If blood flow does not return, the testis must be removed. It is accepted that if left in situ the atrophic testis may produce autoantibodies, damaging the contralateral testis and causing infertility. At the same procedure, the contralateral testis must be explored as well and fixed as a prophylactic measure. Being "aggressive enough," one may expect a negative exploration in up to one-third of patients.

### Acute Retention of Urine

You will often have to deal with acute urinary retention in the ER or in your postoperative patients. Most patients with retention are men who suffer from benign prostatic hypertrophy (BPH) who give a history of lower urinary tract symptoms (LUTS), such as urgency, nocturia, double micturition, hesitancy, and the like. Other possible etiologies include urethral stricture and neurological disease (e.g., multiple sclerosis). Retention is manifested by severe lower abdominal pain, inability to void, and (not surprisingly) agitation. In some BPH patients, retention is precipitated by sympatomimetic drugs (ephedrine for flu) or anticholinergics (psychiatric drugs).

### *Management*

Relief of this situation is by simple insertion of a urethral (Foley) catheter. The rule in this case is to insert a catheter with a moderate caliber but not too large as it may have to stay in place for some time. A good choice is a 16F Foley catheter. Bladder neck stenosis, a large prostate, or urethral stricture may make it difficult to pass through the urethra. If insertion of a regular Foley fails, one may use a 14F Tieman Foley that has a special tip and an angle with a better chance of negotiating the bends and curves of the urethra. Third-line catheters are tougher and transparent, with a Tieman tip and no balloon and are of different calibers. Failure of all these measures would necessitate the insertion of a **cystostomy tube (suprapubic catheter)**. In most cases, we would first insert a large-bore needle in the midline above the pubic bone, and when urine pours out, we thread the cystostomy kit *du jour* into the bladder. Caution must be practiced if the patient has had prior surgery in the area, in which case cystostomy is best done under ultrasound guidance. Of course, renal function must be assessed as some patients may have chronic retention, causing renal failure.

It is very important to measure urine output for 2–3 hrs after insertion of a catheter. A common occurrence is **postobstruction diuresis** with output of very large amounts of urine. The pathophysiological basis for the polyuria is an acute washout of solutes that, due to retention, were not excreted properly, as in a hyperosmolar state. Other reasons are inability of the medulla to conserve water due to loss of urea and pseudo-diabetes insipidus, a temporary incapacity of antidiuretic hormone (ADH) receptors in the distal nephron. This situation is life threatening, especially in older patients, due to fluid and electrolyte imbalance. A patient with postobstruction diuresis (>200 ml/h) has to be hospitalized. Urine output is measured every hour and intravenous fluids (0.45% saline) are given. To avoid "chasing your tail," initially 80% of the voided volume is replaced; as urine output decreases, replacement is given at 50% of the voided volume. Usually, this is a self-limiting situation that should resolve within 24 hrs.

## Urological Trauma

### *Kidney*

Renal injuries may be blunt or penetrating, and they are commonly associated with motor vehicle accidents, falls from heights, and assaults. An important consideration is to check whether a deceleration injury is involved (fall from a height especially) as this may cause an arterial intimal tear leading to renal artery thrombosis, which is a real emergency. Otherwise, a tendency to conservative management has emerged over the years in all renal injuries.

The conservative approach to stab and low-velocity gunshot wounds emerged in South Africa. It was there that physicians managing masses of injured patients in cramped ERs first noticed that many of those with extensive renal injuries waiting for their turn for surgery survived without surgical exploration. The kidney has good recuperation potential, and most injuries heal with minor sequelae. Associated urine leaks are easily managed with drainage, which may be either internal via a stent diverting urine from the kidney to the bladder or by a percutaneous nephrostomy tube. Obviously, penetrating injuries are commonly associated with injuries to other nearby structures according to site of entry.

The hallmark of renal injury is hematuria. Microhematuria is defined as >5 RBCs/HPF (red blood cells/high power field). We do not need to tell you what gross, macroscopic hematuria is, right?

**Which patients need renal imaging?**

- Blunt trauma with gross hematuria
- Blunt trauma with microscopic hematuria and shock (blood pressure <90 mmHg measured at any time since the trauma occurred)
- Penetrating trauma: all patients with penetrating wounds in the anatomical vicinity of the kidneys
- Pediatric patients: use imaging more liberally because kids are more susceptible to significant renal trauma

**The preferred imaging study is contrast-enhanced CT.** Most spiral CTs are performed in a 2- to 3-min sequence and reveal an arterial and a venous phase. Urine excretion and possible injury to the collecting system may be seen only at 10 min; thus, a delayed image must be taken at 10 min as well.

Important findings are:

- Medial perinephric hematoma—suggesting vascular injury
- Medial extravasation of urine—uretropelvic junction avulsion
- Lack of contrast enhancement of the kidney—arterial injury

IVP (intravenous pyelogram) has been abandoned and is used for only one indication—a "single-shot" intraoperative IVP. If at laparotomy and without prior imaging a surgeon encounters an unexpected retroperitoneal, perinephric hematoma, the surgeon may order an IVP to assess the kidney. A single film is obtained 10 min after an intravenous push of 2 ml/kg contrast media. A kidney that looks normal may be left alone. Similarly, in an emergency nephrectomy it is always comforting to know that the contralateral kidney is intact. (Of course, in some developing regions IVP continues to play the role of the poor person's CT).

### *Grading of Injury*

As in most organs, there is a tendency to stage renal trauma and act according to the severity of injury. The staging system commonly used in urology is the American Association of Trauma organ injury severity scale for the kidney. Basically, grades I–III describe the magnitude of perirenal hematoma and laceration of the renal parenchyma. Stage IV entails either a laceration extending throughout the kidney from the cortex to collecting system or a vascular injury. Stage V includes a shattered kidney or avulsion of the renal hilum.

### *Managing the Renal Injury Patient*

- Well-staged injuries may be managed nonoperatively.
- Patients must be carefully followed in an intensive care unit (ICU) setting with frequent hemoglobin assessment.
- Grade IV and V injuries often require surgical exploration.
- If bleeding occurs on expectant management, angioembolization may be therapeutic.
- Renal artery occlusion by an intimal tear (deceleration injury) must be repaired within 6–8 hrs before the kidney dies.
- Experience shows that when in doubt it is better to explore and repair the injury rather than treat the complications.
- **Absolute indications** for operative management include persistent renal bleeding, expanding perirenal hematoma, and a pulsatile renal hematoma denoting arterial renal injury. **Relative indications** include incomplete staging, major urinary extravasation (with a medial urinoma; high probability of ureteropelvic junction tear that will not heal spontaneously), and >20% nonviable renal tissue.

### *The Operation for Renal Injury*

Exploration of an isolated renal injury is usually done for bleeding in an unstable patient or, rarely, for delayed complications. The appropriate incision is *midline* from the xyphoid to below the umbilicus. Although the classic access for elective nephrectomy is retroperitoneal, through the flank, in trauma one may need to approach the major vessels, and this is easier through a long midline incision.

The old dogma maintained that the renal vessels have to be controlled at their origin prior to exploring a perirenal hematoma, but this is easier said than done. Today, we know that there is no real advantage for early vascular control. In practice, the perirenal hematoma "dissects" all the planes around the kidney.

The surgeon opens the retroperitoneum lateral to or above the injured kidney and evacuates the clots. The immediate aims are to mobilize the kidney—lifting it forward and medially into the wound—and to identify the hilum to control the renal pedicle and assess the parenchymal damage.

Parenchymal tears are repaired, closing the collecting system with delicate absorbable sutures and then approximating the parenchyma over bolsters of *Surgicel* using blunt atraumatic liver needles (absorbable sutures again). Following repair of a major tear in the collecting system or a large partial or heminephrectomy, a double J stent may be best inserted retrogradely through the bladder into the collecting system to prevent urinary leak.

For a shattered kidney or when the major vessels are not amenable to repair, nephrectomy is the best option. Nephrectomy is also recommended if there is major trauma to adjacent organs such as the pancreas or bowel since urinary leak from an ill-performed partial repair may promote local "septic" complications. Studies in animals showed survival without dialysis on 33–50% of one kidney. Thus, a safe rule of thumb is that if one can save half or more of the kidney it is worthwhile.

### *Ureter*

Ureteric injuries are rare and frequently are recognized late when urine comes out of a drain after exploration for trauma. The ureter travels in the retroperitoneum covered on all sides by fat and is very evasive. Thus, it takes a lot of bad luck to have a ureter transected by a bullet or cut by a knife. Some of the injuries are partial tears due to a high-velocity missile traveling in the vicinity, injuring the ureter wall and causing a leak. The hallmark of ureteric injury is leakage of urine, and this is what appears on IVP or contrast CT. In the setting of penetrating trauma, suspect damage to the ureter if the injury is in the lateral retroperitoneal areas or in the pelvis. Search for traumatic ureteral injuries carefully; otherwise, they will be missed.

**Iatrogenic ureteric injuries** may occur during Caesarean sections and colorectal procedures. If diagnosed intraoperatively, they should be repaired immediately. Some accidental injuries occur during laparoscopy, and they are usually missed and diagnosed late. Sometimes, the ureter is obstructed by a stitch or a clip; the patient may complain of flank pain or develop asymptomatic hydronephrosis, detected on imaging.

#### Managing the Injured Ureter

The ureter must be carefully examined, and any necrotic segment should be debrided, although this may compromise its length. Always stent the anastomosis

to secure urine drainage and augment local tissue apposition. Urine always finds its way out through an imperfect anastomosis; this is why you see so many different types of stents and tubes in urology and why some urologists consider themselves plumbers.

Lower ureteral injuries (distal to the iliac vessels) are more common and are easier to fix. Usually, one sacrifices the distal part of the ureter and reimplants the ureter directly into the bladder. In this situation, if the ureter is too short, one may pull up the bladder by suturing it to the psoas (a psoas hitch) or use a flap fashioned from the bladder (the Boari flap).

Mid- and upper ureteral injuries less than 2 cm in length are fixed by end-to-end (spatulated) anastomosis with optimal apposition using fine absorbable sutures. Longer injuries may necessitate mobilization of the ipsilateral kidney and downward positioning, which may offer another couple of centimeters. If there is a large gap, several options exist; one is to connect one ureter to the other by tunneling it behind the peritoneum, a *transuereto-uretrostomy*. Another option, when a large gap is present, is to bridge it with small bowel, an *ileal ureter*. An extreme measure would be to autotransplant a kidney in the ipsilateral pelvis, thus bridging a large gap of missing ureter. In experienced hands, these measures are all done with a high rate of success.

***Two important notes:***

- If one encounters ureteral injury while dealing with an extensive and unstable trauma case, an easy solution is to clip the ureter above the injury. When the patient is stabilized and within 24 hrs, one may insert a nephrostomy tube and secure drainage of the kidney. Further repair is delayed to a more appropriate time.
- Another point that is often overlooked: if a ureteral injury is very extensive and necessitates a complex reconstruction, or diagnosed late, or when a complex urinary fistula already exists, then if the contralateral kidney has good function, nephrectomy may be the best option.

### *Bladder*

Bladder injuries are usually associated with pelvic trauma. Isolated bladder rupture occurs especially on holidays when a patient with a full bladder from overdrinking gets hit in the lower abdomen. Penetrating bladder injuries also are often associated with trauma to other organs. Iatrogenic injuries are common, and obstetricians and gynecologists are the main offenders.

Bladder rupture presents with suprapubic pain and tenderness with gross hematuria. A cystogram is diagnostic in nearly all cases. Care must be taken to fill the bladder appropriately. In an unconscious patient, a minimum of 300 ml of

contrast are instilled through a catheter. In a conscious patient, filling may be terminated when the patient complains of discomfort.

Bladder tears may be extraperitoneal (lateral flame shaped) or intraperitoneal (contrast outlines the small bowel contour). Extraperitoneal tears are managed by draining the bladder with a large-bore Foley catheter (20–22F) left in situ for 10–14 days until healing takes place. A cystogram should be performed prior to catheter extraction. All intraperitoneal injuries need to be explored and sutured primarily with absorbable sutures. Injury adjacent to the bladder neck needs careful assessment of the ureteral orifices.

### *Scrotum*

Blunt injury of any etiology may cause rupture of the tunica albuginea of the testis. Blunt injury usually involves a single testis, but penetrating trauma affects both sides of the scrotum in one-third of cases. Scrotal hematoma is a common clinical finding but may not correlate with the extent of damage to the testis itself as bleeding may originate from any of the other structures in the scrotum. Also, failure to feel the testis does not mean that it is damaged. Severe testicular pain radiating to the abdomen is suggestive that the testis has been injured. We have treated patients screaming with pain, and resistant to narcotics, who had almost no scrotal swelling or hematoma on examination; at operation, however, their tunica albuginea was found to be ruptured. Although ultrasound is the best imaging modality to assess the scrotum and testis, an unequivocal report does not rule out testicular trauma. When in doubt, it is best to explore the scrotum.

#### Management

Early exploration and repair of testis injury is the rule. Early repair is associated with increased testicular salvage, quicker convalescence, and preservation of testicular function. Explore the scrotum through a transverse incision; pass through the various layers (like cutting an onion) until reaching the tunica albuginea. When the tunica has been breached, you will see the *seminiferous tubules* flowing out like tiny spaghetti. Damaged tissues should be debrided and the tunica albuginea repaired. Even simple clot evacuation from a large hematocele will hasten recovery.

Urologists are just glorified plumbers.

# 35 Abdominal Emergencies in Infancy and Childhood

Wojciech J. Górecki

*Children are not small adults.*

The well-known phrase that children are not small adults is eminently applicable to pediatric abdominal emergencies, not only because of differences in physiology and metabolism, but also because of a different clinical spectrum of abdominal emergencies, their presentation, and management. This chapter focuses on abdominal surgical emergencies in infants and small children. Neonatal emergencies are omitted as you are unlikely to encounter them unless you are a specialist pediatric surgeon.

The first principle to remember is that you are less likely to commit an error if you consider an atypical presentation of a common condition than a typical presentation of a rare condition. In other words, a pediatric acute abdomen is intussusception in infancy or appendicitis in childhood—until proven otherwise. Another principle is that, much like with adults, watchful waiting is a prudent strategy in children.

## General Approach to Pediatric Abdominal Pain

The philosophy of classifying the multiple etiologies of the acute abdomen into several well-defined clinical patterns, presented in Chap. 3, works for children as well. The major pitfalls in assessing the pediatric acute abdomen are *timing*, *history*, and *abdominal palpation*.

*Children with abdominal pain* present to the emergency room at varying stages of disease because the timing of presentation depends on the parents. Some parents delay, while others rush their darlings to the emergency room (ER) at the slightest sign of trouble. As a general rule—as originally stated by Sir Zachary Cope—consider any abdominal pain lasting more than 6 hrs as a potential surgical problem.

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- *Younger children do not give you a history*, but listen to the parents because they know their kids so well. A classical example is intussusception, for which a description of the child's behavior and a glance at the stool can point you to the diagnosis even before the physical examination.
- *The importance of gentleness* during abdominal palpation cannot be overemphasized. The majority of children with a sore tummy object to abdominal palpation. Sometimes, a toy provides a temporary distraction that will allow you to examine the abdomen, but it is pointless to persist if the child is antagonized. Instead of the usual "head-to-toe" sequence of the physical exam in adults, take advantage of a spell of sleep or inattention to sneak a warm gentle hand underneath the blanket to palpate the abdomen.
- *An infant* who will not allow a gentle attempt even when held in his or her mother's lap should be sedated because sedation does not affect muscle guarding. Our preference is intranasal midazolam 0.1–0.2 mg/kg.
- *Examination of the scrotum* is essential for two reasons. First, an acute condition in the right testicle, such as torsion, can present with pain in the right groin and iliac fossa. Second, perforated appendicitis occasionally presents with a painful scrotal swelling because pus enters the *patent processus vaginalis*, causing acute *funiculitis*.
- *Rectal examination* is best left to the end of the physical examination, after looking at the throat and ears, and is not needed if there is a clear indication for laparotomy.

## Clinical Patterns of Acute Abdomen in Kids (see also › Chap. 3)

- The combination of **acute abdominal pain** and **shock** is rare in children and should make you think of occult abdominal trauma with rupture of an enlarged solid organ or a tumor (e.g., ruptured Wilms' tumor). Contrary to adults, urgent laparotomy is not always indicated.
- **Generalized peritonitis** in children is most commonly due to appendicitis. Do not try to elicit rebound tenderness as you will lose the confidence and cooperation of your patient. (This applies to adults, too!)
- **Localized peritonitis** in the left lower quadrant can be due to acute constipation, whereas right or left upper quadrant tenderness is commonly due to acute distension of the liver or spleen, respectively.
- **Intestinal obstruction** in a virgin abdomen is caused by intussusception or appendicitis. One of ten children with complicated rotational anomalies of the midgut presents after the neonatal period. The critical concern with malrotation is *midgut volvulus* with acute bowel ischemia. This life-threatening condition carries the risk of rapid transmural intestinal necrosis. Your surgical intervention should be prompt because simple counterclockwise detorsion of the bowel may save it. The two *major pitfalls* in pediatric small bowel obstruction are missing an incarcerated inguinal hernia and waiting too long with conservative management before surgery.

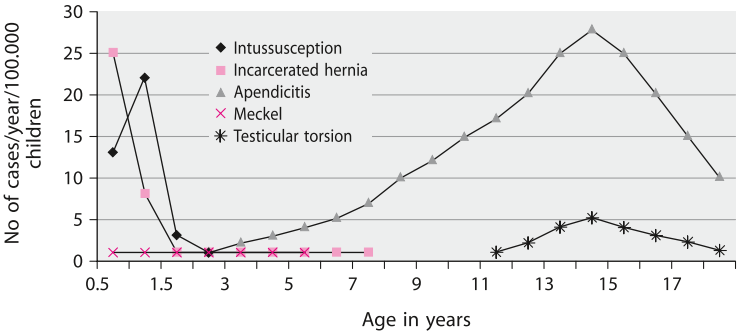


**Fig. 35.1.** Pediatric abdominal emergencies

A wide spectrum of **nonsurgical conditions** mimics abdominal emergencies. Particularly in infants, any acute systemic disease may present with apathy, vomiting, and stool abnormalities. Gastroenteritis is common in children and typically presents with acute abdominal complaints. The converse is also true. A child with an acute abdomen may present with a wide array of seemingly unrelated symptoms suggesting early meningitis, a neurological disorder or poisoning.

## Specific Pediatric Emergencies

The relative incidence of the conditions in the different age groups is depicted in Fig. 35.1.

### *Acute Appendicitis (see also Chap. 28)*

Acute appendicitis (AA) is rare during the first year of life and is uncommon during the second. Thereafter, the incidence rises and peaks between ages 12 and 20. AA in infancy typically presents as generalized peritonitis due to perforation. The infant looks unwell, with fever, tachycardia, and tachypnea. The abdomen is distended and generally tender with guarding. Diarrhea is more common than constipation. Pay attention to the useful "hunger sign"; it is rare to see a hungry child who turns out to have AA. Consider AA in the second place on your list of differential diagnoses for an infant with an acute abdomen and in the first three places in a child. The white cell count is normal in many cases of pediatric AA, but neutrophilia is more specific. Admitting children with equivocal signs for observation is a safe option as the chance of rupture under observation in a pediatric surgical ward is less than 1% (oops—the editors asked for no percentages).

*A limited helical computed tomograph (CT)* with rectal contrast has high accuracy in diagnosing AA in children, but clinical examination by an experienced pediatric surgeon is just as good. Even if the CT scan is "positive", appendectomy is not indicated if the child improves clinically.

*What is the role of laparoscopy* in the doubtful case? While it offers the advantage of a diagnostic modality that can be immediately followed by appendectomy, it will subject some children to an unnecessary operation. If you can get the child into a CT scanner without general anesthesia, this should be your preferred choice instead of diagnostic laparoscopy.

Although a valid alternative to the open technique, the value of *laparoscopic pediatric appendectomy* remains controversial because there are no good data to suggest that it confers an advantage in postoperative recovery. The short distances and thin abdominal wall of children allow a port-exteriorization appendectomy, performed via *two ports*, with the appendix exteriorized by pulling it out of the right iliac fossa port, and then the entire appendectomy is performed *outside* the abdomen. Or, the appendix can be pulled out of the umbilical port, and if you have a laparoscope with a working channel, you can perform a *single-port* appendectomy using the same technique. [This would be equivalent to a conventional "*no-port* appendectomy" through a 2-cm incision—The Editors].

There is no point in culturing the peritoneal fluid in case of obvious AA because the results are predictable, and antibiotics have usually been stopped by the time the culture results become available. Decide on the duration of postoperative antibiotics according to the degree of contamination or infection found in the peritoneal cavity (see Chaps. 12 and 47).

### *Intussusception*

Telescoping of one portion of the intestine into another (intussusception) can turn a healthy baby into a critically ill patient within a few hours. It typically occurs between the ages of 5 and 7 months, and the etiology is idiopathic. In children older than 2 years, look for an underlying pathology, the most common being a *Meckel's diverticulum*. Early intussusception is generally a benign condition, although it is a strangulating obstruction eventually leading to vascular compromise. Most cases start in the ileum as ileoileal intussusception and then progress through the ileocecal valve to become ileocolic intussusception.

The diagnosis is straightforward if the infant exhibits the classical clinical syndrome. A previously healthy infant suddenly starts to scream, pulls up his or her legs, and perhaps clutches the abdomen. The pain is then relieved, and the child may relax for a while only to have a similar bout 15–30 min later. This leaves the infant pale and ill. Vomiting and passing of "red currant jelly" stools is also characteristic, although salmonellosis may show a similar clinical picture. Atypical presentations are common and lead to diagnostic errors. The infant may be fretful and restless without either pain or vomiting. Pallor and peripheral coolness due to vasoconstriction, lethargy, and seizures may also confuse the picture. The crucial physical sign is palpation of an abdominal mass. The ultrasonographic findings of a "target sign" on cross section and "pseudo-kidney sign" in a longitudinal view are important adjuncts to the clinical diagnosis (Fig. 35.2).

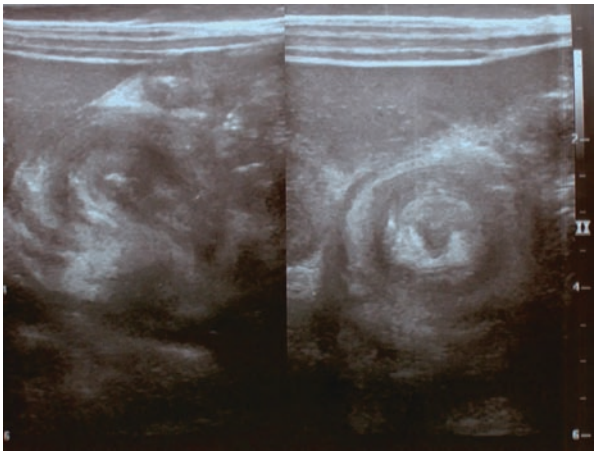

**Fig. 35.2.** Sonographic images of intussusception. On the *left*: longitudinal plane showing "pseudo-kidney sign." On the *right* a transverse plane showing the "target sign"

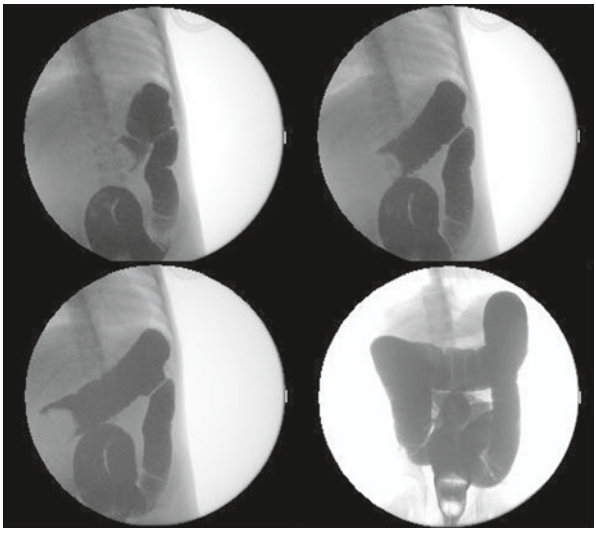

**Fig. 35.3.** Fluoroscopic monitoring of retrograde reduction of the intussusceptum by means of hydrostatic barium enema, with complete reduction (in the *lower* figure on the right, contrast is visible in the small bowel)

Children with diffuse peritonitis, perforation, progressive sepsis, and possible gangrenous bowel should undergo an urgent laparotomy. Early intussusception without peritonitis is reduced nonoperatively with pneumatic or hydrostatic pressure under radiographic or ultrasonic guidance. Water-soluble contrast is safer than barium in case of suspected perforation. Reduction is successful in the majority of cases but requires close collaboration between surgeon and radiologist (Fig. 35.3).

*Operative reduction of an early intussusception*: squeeze on the apex of the intussusception while the bowel is still within the abdomen so that the intussuscepted segment begins to slide out. When the reduction reaches the region of the hepatic flexure, it may become more difficult but after you eviscerate the proximal colon, the reduction can be completed under direct vision. After achieving complete reduction, remember to examine the entire bowel for a pathology serving as a lead point. If the intussusception is truly irreducible or if the bowel has suffered a serious vascular compromise—resect it.

### *Meckel's Diverticulum*

Two-thirds of Meckel's diverticula encountered by surgeons are incidental findings; the remaining one-third will present with a complication. Pediatric surgeons encounter different proportions as the incidence of these complications is maximal during the first 2 years of life and decreases thereafter, so that more than two-thirds of all complications occur in the pediatric population. These complications include bowel obstruction (adhesive obstruction, volvulus, or intussusception); complications with peptic ulceration in ectopic gastric mucosa (stricture, hemorrhage, or perforation); or acute inflammation ("second appendicitis"). There is also a distinct tendency for foreign bodies to penetrate and perforate a diverticulum. We have seen a 5-year-old girl with complete bowel obstruction by a Meckel's diverticulum filled with excessively ingested *Gummi Bears* candies. *Littre's* inguinal hernia contains a strangulated Meckel's diverticulum and, like *Richter's* hernia, may not produce signs of intestinal obstruction.

The treatment of a symptomatic diverticulum is resection. Diverticulectomy is possible if the base is wide and noninflamed but remember to check the base of the diverticulum and the adjacent ileum for ectopic mucosa because the bleeding source may lie within it. If in doubt, or if there is any technical difficulty, resect the involved segment of ileum.

What should you do with an *incidentally* found Meckel's diverticulum? Consider the degree of peritoneal infection (caused by the primary indication for laparotomy), the patient's age, and the shape of the diverticulum. On balance, the arguments *against* removing an asymptomatic Meckel's diverticulum are a little stronger than those in favor, and the strength of the argument increases with the age of the patient. Thin-walled, wide-mouthed, mobile (without a fibrous band to umbilicus or mesentery) diverticula should be left alone.

### *Irreducible Inguinal Hernia*

The emergency of irreducible inguinal hernia occurs primarily in boys during their first year of life. The fundamental difference between an irreducible inguinal

hernia in an infant and an adult is that the former presents a danger to the viability of the testis, whereas with the latter the major concern is the potential for bowel ischemia. Neonates with symptoms lasting for more than 24 hrs and with intestinal obstruction are at the greatest risk of testicular infarction. Necrosis of incarcerated bowel is extremely rare in pediatric hernias.

The diagnosis is straightforward because the baby cries and vomits, and the parents have usually noticed a tender lump in the groin. The major differential diagnosis is with torsion of a maldescended testicle, acute inguinal lymphadenitis, and a hydrocele of the cord. After making the diagnosis, sedate the infant and position the infant in a head-down position. In the majority of babies, this will result in spontaneous reduction within 1–2 hrs. Let the tissue swelling subside for a day or two and book the child for an elective herniotomy on the next available operative schedule.

*The operation for irreducible inguinal hernia* in an infant is fraught with danger and should be undertaken only by a surgeon with previous experience in pediatric surgery. The hernia sac is edematous and extremely fragile, and the ductus deferens is almost invisible. Simple herniotomy at the level of the neck of the sac is all that is required. Always make sure that the testicle is safely replaced into the lower part of the scrotum. In a *female infant*, a movable tender lump may be an irreducible ovary. The child may be almost asymptomatic yet require emergency herniotomy because of the risk of ovarian ischemia.

### *Testicular Torsion (see also ❯ Chap. 34)*

The key to successful treatment of testicular torsion is speedy detorsion, within less than 6 hrs of the onset of symptoms. The incidence of torsion rises sharply around age 12, with two of every three cases occurring between the ages of 12 and 18. Some boys with testicular torsion present with lower abdominal and inguinal pain, so you will miss the diagnosis if you fail to examine the scrotum. No clinical sign or test is foolproof, and because the price of delay is loss of the testis, the common wisdom is to have a low threshold for exploring an "acute scrotum."

If prompt surgery is not available, manual detorsion in a lateral direction under sedation or local anesthetic infiltration of the cord may restore testicular blood flow but is not a substitute for surgery. The operative procedure is bilateral orchidopexy to protect the ipsilateral testicle from recurrence and to secure the contralateral one as inadequate anatomic suspension is a bilateral phenomenon. After induction of anesthesia, first examine the scrotum to rule out incarcerated hernia or testicular tumor, both requiring an inguinal incision. Then, proceed with a scrotal exploration via a vertical incision in the median raphae of the scrotum or two transverse incisions to access both sides. Enter the serosal compartment of the scrotum to deliver and detort the testis. Place it in warm moist sponges while exploring

the opposite hemiscrotum. If the affected testis remains necrotic, remove it. Orchidopexy of the viable testis is performed by suturing the surface of the testis (tunica albuginea) at four points to the wall of the serosal compartment using nonabsorbable sutures. If you find torsion of the testicular appendage, simply excise it.

### *Ovarian Torsion*

Should you encounter pediatric ovarian torsion, be it deliberately or incidentally, the adolescent girl will be fortunate having you as the surgeon if you detort the "nonviable" adnexa and leave it alone. The macroscopic appearance of the ovary is not a reliable predictor of necrosis or the potential for gonad recovery. If an underlying lesion is found, then cystectomy, tumorectomy (even in cases with a very large teratomata, there is a rim of normal ovary at the hilum that can be preserved), or cyst aspiration with possible oophoropexy should be considered. If you are not comfortable with this situation, just detort the ovary and close the abdomen. Remember the key principle: you are more likely to preserve functional ovarian tissue than cause any morbidity by leaving in situ what appears to you a nonviable ovary in little girls.

### *Pediatric Abdominal Injuries*

Trauma is the major cause of death among children older than a year of age and is responsible for more deaths than all other causes combined. In one of seven injured children, the abdominal injury is paramount. The patterns of blunt abdominal trauma and the clinical pictures are similar to those in adults, with injuries to the kidneys, spleen, liver, and the intestines the most common (Chap. 39). Most cases can be treated conservatively, and laparotomy is required in only one child in four. The major deterrents for operative approach to abdominal trauma in children are the risks of nontherapeutic laparotomy and overwhelming postsplenectomy infection.

Even children with hemodynamic instability on admission often quickly improve with crystalloid administration and remain hemodynamically stable thereafter. If the situation stabilizes after three infusions of 20 ml/kg of fluid, then it is safe to observe the child in an intensive care unit. If the child continues to bleed and no other source of hemorrhage is apparent, a prompt laparotomy is indicated.

The Achilles' heel of this conservative approach is the possibility of missed injuries to hollow organs. Thus, if the child develops increasing abdominal tenderness or peritonitis, this also is an indication for laparotomy. A useful clinical marker of blunt bowel trauma is the triad of a fastened lap belt, a seatbelt sign on the abdominal wall, and fracture of a lumbar vertebra.


**Fig. 35.4.** "But … but I'm a pediatric surgeon…"

No discussion of pediatric trauma can be complete without emphasizing the need for always suspecting child abuse. While isolated abdominal trauma is a rare presentation of child abuse, unusually shaped or multiple bruises, associated long bone fractures, or inexplicable genital lesions should always raise the suspicion of this tragic and potentially life-threatening condition.

Children are not small adults but … see Fig. 35.4.

# 36

# The AIDS Patient[1]

Sai Sajja · Moshe Schein

*The AIDS patient can suffer from acute appendicitis.*

Human immunodeficiency virus (HIV) infection and its inevitable consequence of acquired immunodeficiency syndrome (AIDS) is a major public health problem worldwide that has affected the way surgery and medicine are practiced. With advances in medical treatment, people infected with HIV are living longer, so it is likely that most of you, wherever you practice, will encounter and treat patients with HIV/AIDS. While the general principles of emergency abdominal surgery described elsewhere in this book are relevant to the HIV patient, we highlight what is unique to this population.

## Natural History

This disease presents a spectrum ranging from asymptomatic HIV infection to advanced AIDS, including its associated opportunistic infections.

***Depending on the CD4⁺ count, HIV disease is categorized as follows:***

- Early stage (CD4⁺ count >500 cells/ μ l)
- Mid stage (CD4⁺ count 200–499 cells/ μ l)
- Advanced (CD4⁺ count 50–200 cells/ μ l)
- Terminal (CD4⁺ count <50 cells/ μ l)

A CD4⁺ count of <200 cells/μl now is defined as AIDS irrespective of the presence of symptoms or other illnesses. A long list of opportunistic infections and cancers, when present, place the HIV-infected patient in the category of AIDS.

[1]Why a separate chapter on HIV/AIDS? We're sure we are not alone in deploring the current trend in making some diseases (AIDS and breast cancer being the most notable) more "fashionable", and their sufferers more worthy of support and sympathy than regular patients. This chapter is emphatically not an addition to this regrettable development, but an acknowledgement that these patients and their illness may be different in a surgically relevant way. [The Editors]

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## Abdominal Pain

Abdominal pain and nonspecific gastrointestinal complaints are very common in patients with HIV/AIDS (Fig. 36.1). Clinical evaluation is difficult as many patients suffer from chronic abdominal symptoms, and for the physician encountering the patient for the first time, what may be the baseline status for the patient may appear very abnormal. Also, the list of differential diagnoses is much larger in this population. White blood cell count, which is very valuable in the normal population, is not reliable because of pre-exsisting leukopenia. Patients often have coexisting infections of the central nervous system, which makes evaluation of the abdominal pain difficult. Antiviral medications frequently cause chronic abdominal symptoms as well as acute pancreatitis. A thorough history, including the stage of the HIV disease, the presence of opportunistic infections and the antiretroviral therapy, and a careful physical examination along with an erect chest X-ray and abdominal X-rays and routine laboratory tests, including serum amylase and lipase, form the basis on which further management is planned.

When the initial examination is inconclusive, serial examinations often yield valuable information. In the absence of clinical peritonitis, free intraperitoneal air, and exsanguinating hemorrhage, computed tomographic (CT) scan of the abdomen and pelvis is indispensable for investigation in AIDS patients. It often identifies nonsurgical pathology and avoids a nontherapeutic laparotomy. Table 36.1 shows causes of abdominal pain in HIV/AIDS, and Fig. 36.2 shows the suggested clinical approach. (Note that the algorithm in that figure differs from the management of a patient not affected by HIV/AIDS really only in the early and uniform use of CT scanning in patients not scheduled for an emergency operation.)

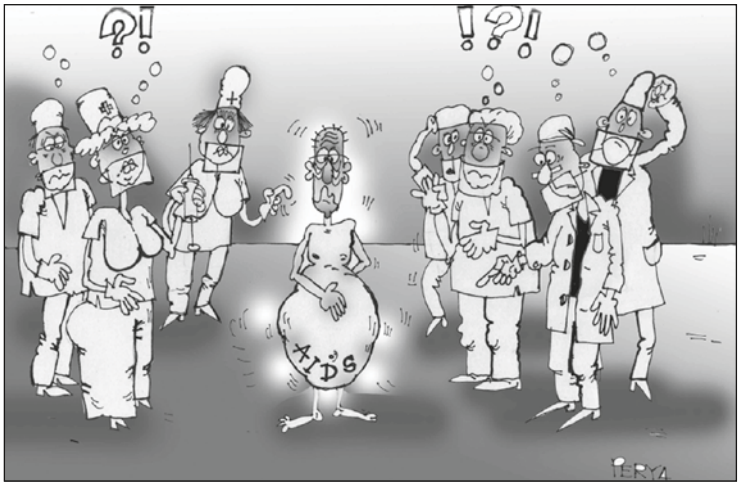


**Fig. 36.1.** "Is it appendicitis or CMV colitis again?"

**Table 36.1.** HIV-related and HIV-unrelated causes of abdominal pain according to the need for source control

| | HIV-related conditions | Non-HIV-related |
|---|---|---|
| Surgical procedure usually indicated | CMV bowel perforation | Appendicitis |
| | CMV-related toxic megacolon | Cholecystitis |
| | Acalculous cholecystitis | Secondary peritonitis |
| | Kaposi sarcoma | Intra-abdominal abscesses |
| | Lymphoma with bowel perforation | Intestinal ischemia |
| | Splenic abscess | Trauma |
| Usually conservative management | Uncomplicated CMV infection | Organomegaly |
| | *Mycobacterium avium* complex | Constipation |
| | *Mycobacterium tuberculosis* | Uncomplicated peptic ulcer disease |
| | Pancreatitis: infectious (CMV, MAC), drug induced (pentamidine, dideoxyinosine, trimethoprim-sulfamethaxazole) | Uncomplicated pelvic inflammatory disease |

*CMV* cytomegalovirus; *MAC Mycobacterium avium* complex

## Specific Conditions

**Acute appendicitis.** That a patient suffers from AIDS does not mean that he or she cannot develop acute appendicitis; in fact, the incidence of appendicitis in the HIV population appears to be higher than in the general population. While some patients present with typical symptoms and localizing signs in the right lower quadrant, often the presentation is atypical: diarrhea and vomiting are seen frequently, while fever and leukocytosis are not very reliable. CT scan is the diagnostic imaging study of choice when the presentation is atypical. Interestingly, CMV (cytomegalovirus) infection and Kaposi sarcoma of the base of the appendix have been reported to cause appendicitis. The operative and postoperative management is similar to that in the non-HIV-affected population (Chap. 28).

**Cytomegalovirus.** In the AIDS patient, CMV is found in every organ system in the body, is the most common opportunistic infection of the gastrointestinal tract, and often involves the colon—causing fever, diarrhea, and abdominal pain. CMV infects endothelial cells, leading to thrombosis of the submucosal blood vessels, which results in mucosal ischemia, ulceration, hemorrhage, perforation, and

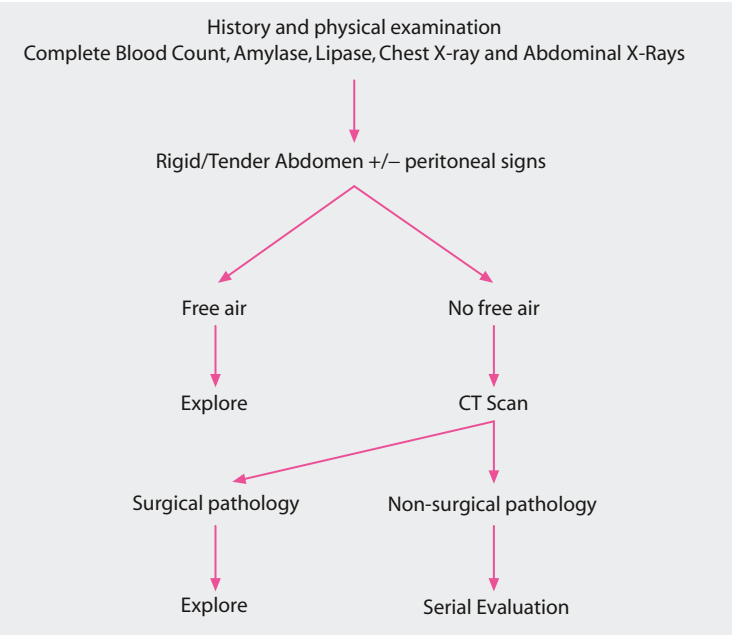


**Fig. 36.2.** An approach to abdominal pain in AIDS patients

toxic megacolon. Diagnosis is established by colonoscopy and biopsy, which shows characteristic intranuclear inclusion bodies. CT scan findings of thickening of bowel wall and mural ulceration are nonspecific. Once the diagnosis is established, treatment with ganciclovir or foscarnet is started. It is very important to keep these patients under close observation while they are on medical therapy to identify early the development of complications. Despite aggressive medical management, some patients develop perforation, toxic megacolon, and hemorrhage—complications that require urgent surgical intervention—following adequate fluid resuscitation and institution of broad-spectrum antibiotic therapy. The perforations related to CMV appear punctate when viewed from the serosal surface. Resection of the involved segment of bowel and formation of a colostomy or ileostomy—rather than primary anastomosis—is the treatment of choice. Toxic megacolon with impending perforation is best managed with a subtotal colectomy and ileostomy.

– **Acute cholecystitis.** Right upper quadrant abdominal pain associated with fever, nausea, and vomiting is a common complaint in patients with HIV/AIDS. While the cause of this pain may be due to hepatomegaly associated with granulomatous infiltration or colitis, the possibility of biliary pathology needs to be investigated. Although gallstones are present in many HIV/AIDS patients undergoing cholecystectomy, they are also believed to have a relatively high incidence of acalculous cholecystitis. CMV and *Cryptosporidium* are the most common opportunistic microorganisms isolated from the affected gallbladders; overwhelming growth of these pathogens seems to cause inflammation and

functional obstruction. This is different from the combination of hypotension, ischemia, and sepsis that is believed to be the cause of acalculous cholecystitis in the non-HIV-affected, critically ill patient. **Ultrasound** is the initial imaging study of choice; gallstones, size of the common bile duct, gallbladder wall thickness, pericholecystic fluid, and intramural air can be demonstrated. **CT scan and HIDA scan** (radioisotope hepatic iminodiacetic acid) are useful when the sonogram is inconclusive. Once the diagnosis is established, depending on the overall condition of the patient, surgical intervention is recommended. Laparoscopic cholecystectomy can be safely performed as experimental observations have not substantiated the concerns of aerosolization of HIV virus in the laparoscopy gas. To prevent blood spray during retrieval of the gallbladder, the pneumoperitoneum must be evacuated first. The routine use of specimen bags is recommended to prevent the accidental spillage of infected contents. The relatively high morbidity and mortality of cholecystectomy in these patients reflects the fact that acalculous cholecystitis occurs in the more advanced stages of AIDS.

- **Splenic abscess.** Splenic abscess is more common in patients with HIV/AIDS. Metastatic spread from other infections, secondary infection of a splenic infarct, and contiguous spread from an adjacent organ are the possible mechanisms of its development. CT scan or ultrasound establishes the diagnosis. In the absence of loculations, percutaneous CT-guided drainage of splenic abscess has a reasonable success rate. Splenectomy is the definitive treatment when radiological features do not favor percutaneous drainage or to salvage a failed radiological intervention.
- **Perianal sepsis.** Acute anorectal conditions are discussed in Chap. 29, but AIDS patients are different. Anorectal pathology is very prevalent in the HIV/AIDS population, especially in those who practice anal-receptive intercourse. While being susceptible to anorectal problems of the general population, HIV/AIDS patients are also prone to a variety of opportunistic infections like CMV, herpes, and benign and malignant neoplasms in the perianal area. Careful inspection of the perianal area, gentle digital rectal examination and a proctoscopic visualization will identify the perianal condition. **Examination under anesthesia** is an essential part of evaluation before definitive surgical therapy. As in the non-HIV-affected population, perianal sepsis in this population could result from cryptoglandular disease or, by contrast, be associated with HIV-related anorectal ulcers or result from secondary infection of anal proliferative lesions. The abscesses associated with HIV-related anorectal ulcers tend to be very deep—transgressing the sphincter planes—with variable destruction of the sphincter mechanism. Surgical intervention is usually necessary. Abscesses should be liberally drained, and specimens should be obtained for acid-fast staining and culture. Biopsy for histology is done if underlying malignancy is suspected. The principles of treatment are similar to the management of perianal sepsis in Crohn's disease—treatment has to be conservative. Damage to the sphincters is avoided, and noncutting setons and drains are used liberally. Delayed wound healing is a major concern with $CD4^+$ cell count of less than 50/μl a predictor of delayed wound healing.

**Remember**

- The general principles of surgical care described in this book are applicable to the HIV/AIDS patients; however, a thorough understanding of the natural history and the spectrum of HIV disease is essential. The pathology may or may not be related to the HIV status.
- Abdominal complaints are extremely common in the HIV population, and clinical evaluation is often difficult. Serial clinical examination and frequent use of CT scan are essential to prevent nontherapeutic interventions.
- Early diagnosis and prompt intervention are essential for non-HIV-related surgical pathology like acute appendicitis and cholecystitis. Surgical intervention is also essential for complications of opportunistic infections like CMV perforation. The morbidity and mortality for surgical procedures depends on the stage of the HIV disease and the nature of the pathology.
- Surgical intervention should not be denied to this population because of the risk of occupational transmission and the fear of high complication rates. Relief of symptoms and improvement in quality of life are the chief considerations.

# 37

# Abdominal Emergencies in the "Third World"

Robin Kaushik · Graeme Pitcher · Craig Joseph

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# Asia

Robin Kaushik

The spectrum of abdominal emergencies encountered in Asia is almost the same as that encountered elsewhere in the world but with a few regional differences in pattern and presentation. As the "conventional" causes of the acute abdomen are discussed in detail elsewhere in this book, this chapter focuses on what is specific to this vast continent—specific for example to India, as depicted in Fig. 37.1.1.

Duodenal ulcer perforation remains by far the most common cause of the acute abdomen. Although relationships with diet (predominantly rice based), seasonal variation, genetic, and environmental factors have all been proposed to

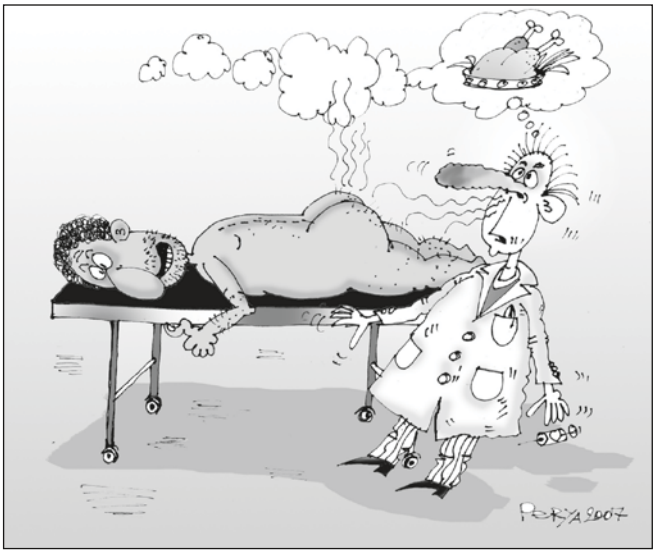

**Fig. 37.1.1.** "Doc, my anus is burning!" The surgeon taking a sniff: "Perhaps if your wife adds a little less curry powder..."

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explain the higher incidence of duodenal ulceration in this region, the exact cause remains unclear. Acute appendicitis, cholecystitis, and acute pancreatitis all occur in patients in this region. Other causes of the acute abdomen one may come across include colonic perforation, mesenteric vascular occlusion (and intestinal ischemia), and abdominal aortic aneurysms, but the incidence of these is much less than is seen in "developed" countries. Similarly, medical causes such as myocardial infarction and basal pneumonia may also occasionally be seen presenting as "acute abdomen."

Within this region, there exists a geographical variation in the disease-specific causes presenting as acute abdomen. Although duodenal ulcer perforation remains the most common cause of acute abdomen, the incidence of small bowel perforation can be very high, varying from nearly 40% in the Indian subcontinent to an almost negligible 6% in China and even less in Thailand. Enteric fever, tubercular, and nonspecific small bowel perforations are common in the Indian subcontinent, whereas Crohn's disease, Behcet's, radiation enteritis, adhesions, ischemic enteritis, and systemic lupus erythematosus (SLE) are the common causes of small bowel perforation in the "far eastern" countries like China and Japan.

I will dwell on the *acute presentations* of a few conditions seen commonly in Southeast Asia: abdominal tuberculosis, amebiasis, and parasitic infestations.

## Abdominal Tuberculosis

The abdomen is the most common site of extrapulmonary tuberculosis (TB). Abdominal TB can develop in the absence of pulmonary TB and does so in nearly two-thirds of cases. Although classified in many ways, intestinal problems due to TB are the most common presentation of abdominal TB. The disease can affect any part of the gastrointestinal (GI) tract from the esophagus to the rectum. The ileocecal region is most commonly affected (as a result of physiological stasis and the presence of more lymphoid tissue). Infected Peyer's patches ulcerate along the long axis of the terminal ileum, and *caseation* of the mesenteric lymph nodes may occur. Further disease progression usually depends on the host's immunological status.

Abdominal TB can present as an "acute abdomen" in two main ways: intestinal (small bowel) obstruction and peritonitis.

**Small bowel obstruction** is the most common complication of abdominal TB and is caused by stricture formation, adhesions, or external compression of the lumen of the bowel by caseating mesenteric lymph nodes. Classically, the patient gives a history of recurrent attacks of small bowel obstruction (the so-called subacute intestinal obstruction) prior to complete obstruction. Although some surgeons prescribe antitubercular drugs in this situation and recommend waiting for a month to see a response, a patient who does not respond to conservative measures needs to be operated on (Chap. 21). Not uncommonly, at operation

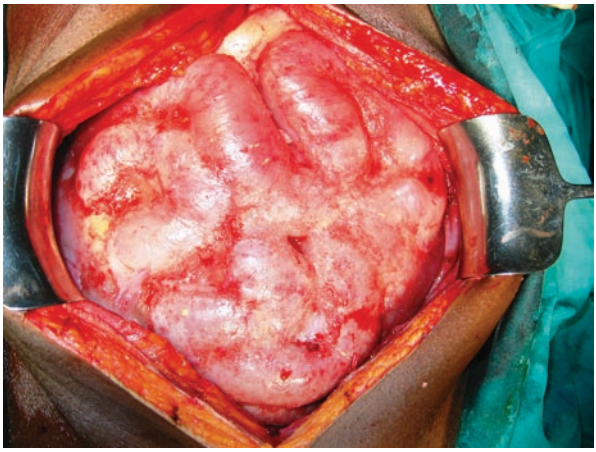

**Fig. 37.1.2.** Adhesions in abdominal TB

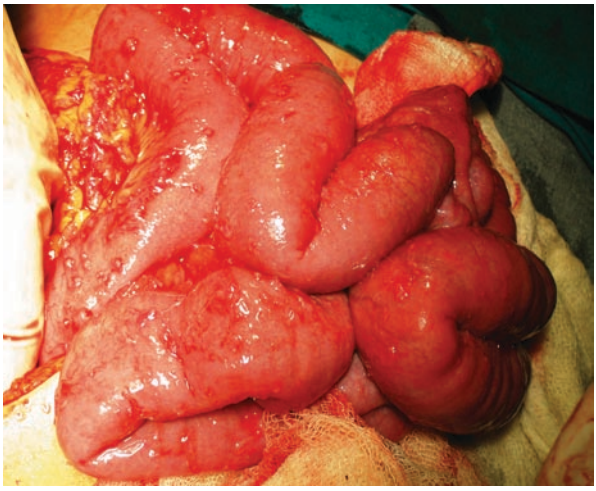

**Fig. 37.1.3.** Tubercles over the intestine and mesentery in abdominal TB

you may encounter dense adhesions (Fig. 37.1.2) that may be a challenge to even the most talented of surgeons. Patience, and calling for help when the case seems beyond your skills, is the key to successful management in this situation.

Caseating lymph nodes, tubercles on the bowel or mesentery (Fig. 37.1.3), and small bowel strictures (Fig. 37.1.4) should make you suspect abdominal TB in a patient undergoing laparotomy for intestinal obstruction. In such cases, the surgical strategy is to relieve the obstruction with resection and a primary anastomosis (ileoileal or ileocolic) whenever feasible. Stricturoplasty is another option.

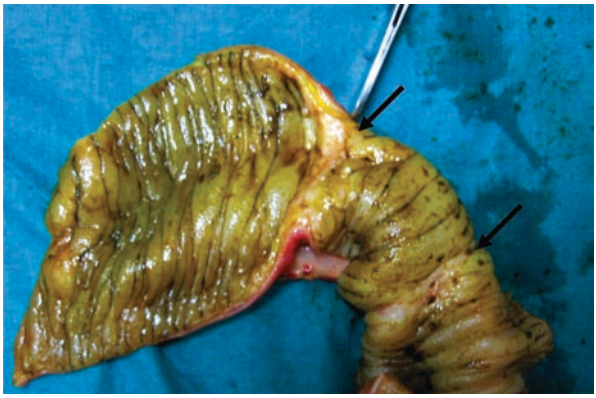

**Fig. 37.1.4.** Tubercular strictures of the ileum

*Bypass of the affected segment* is recommended only if resection is not possible due to dense adhesions or encasement of surrounding structures (such as the ureters, root of the mesentery) or, in a sick patient, if operating time can be saved by bypassing the diseased and densely adherent segment. Please remember to take some tissue for a biopsy during surgery; it not only will confirm your diagnosis, but also will rule out other diseases such as malignancy, which can mimic TB. Start antitubercular therapy for such patients in the postoperative period.

**Perforation of the intestine** is another way in which abdominal TB can present. The signs and symptoms are those of peritonitis, and it is rare to be able to make the diagnosis of a tubercular perforation pre- or even intraoperatively. These perforations usually occur in the distal small intestine, proximal to tubercular strictures. These are usually "blowouts" secondary to distension of the bowel. Occasionally, free perforations of tubercular ulcers can be encountered in the absence of strictures and distal obstruction; these carry a very high mortality. For such cases, resection and primary anastomosis whenever feasible are again the preferred surgical option, rather than simple closure (which is associated with a high incidence of leak and fistula formation). Exteriorization is an acceptable alternative in sick and debilitated patients.

## Amebiasis

The protozoan infection common in this region is amebiasis. This is caused by *Entamoeba histolytica* and spreads through the feco-oral route, usually leading to disease of the large intestine and liver. Although this remains primarily a medical disease, it can occasionally cause acute abdominal symptoms that necessitate surgical consultation and intervention.

As a surgeon, you may encounter acute abdominal conditions due to amebiasis in the following patterns:

- Amoebic liver abscess (possibly complicated)
- Peritonitis (secondary to colitis or rupture of a liver abscess)
- Intestinal obstruction or lower GI bleeding secondary to granuloma formation (rare)

A *liver abscess* is the most common complication of amebiasis and usually presents as an acute illness with right upper quadrant pain, moderate fever, and tender hepatomegaly. The abscess is usually solitary and confined to the right lobe (Fig. 37.1.5), but occasionally can be multiple and even in the left lobe (Fig. 37.1.6). Although the stools are often not positive for the amoeba, serology

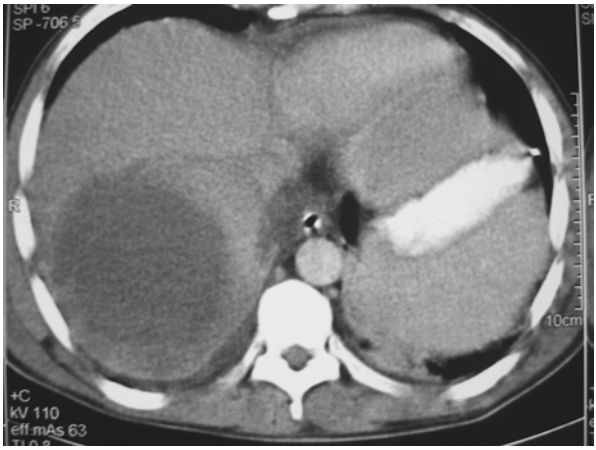


**Fig. 37.1.5.** Solitary amoebic liver abscess

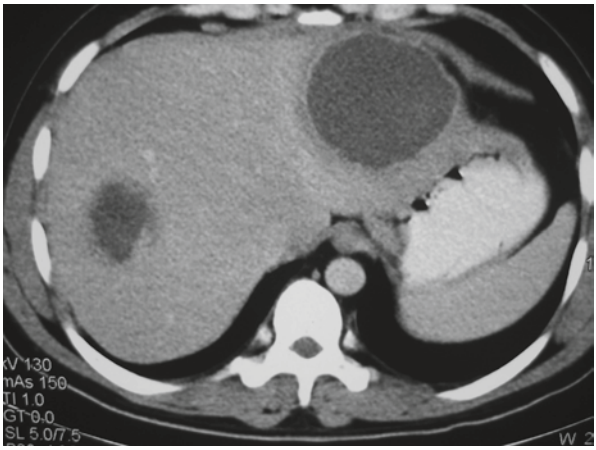


**Fig. 37.1.6.** Multiple amoebic liver abscesses

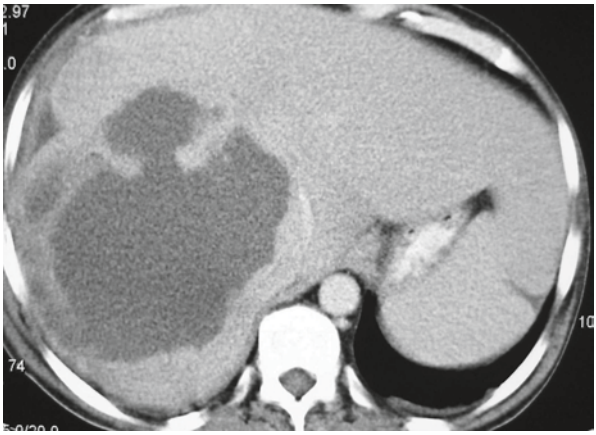

**Fig. 37.1.7.** Pyogenic liver abscess

is positive in the majority of cases, and negative serology safely excludes this diagnosis. On imaging (ultrasound [US] and computed tomographic [CT] scan), these abscesses appear as hypoechoic masses with smooth margins, in contrast to pyogenic abscesses (Fig. 37.1.7). Technetium-99m liver scanning can diagnose amoebic liver abscesses, which appear as cold lesions on the scan (i.e., they do not take up the radioisotope), but this investigation is rarely required.

The **management of uncomplicated amoebic liver abscess** is purely medical, with oral metronidazole 800 mg three times a day for at least 10 days the treatment of choice. The patient starts to respond within a couple of days, with clinical improvement occurring in the form of relief of fever, abdominal pain, and signs. If there is no improvement in the clinical condition or if the patient deteriorates, think of alternative diagnoses, such as secondary infection or pyogenic abscesses: aspirate the abscess under US or CT guidance and culture.

Routine percutaneous or surgical drainage of amoebic liver abscesses is rarely required, because medical therapy alone has been shown to be equally effective. However, aspiration may be necessary when there is a large sized abscess (>10 cm); when there is no improvement in the condition of the patient within 72 hrs; in left lobe or peripheral abscesses; or in cases of diagnostic uncertainty (e.g., when the abscess is associated with negative serology for amebiasis). The aspirate is the typical "anchovy sauce" pus, which is usually sterile, but may contain the characteristic trophozoites (scrapings of the wall of the abscess are a better source of trophozoites).

**Rupture of such a liver abscess** is a dangerous condition, with a high mortality. The rupture usually presents acutely with the classical features of peritonitis, and such a patient needs laparotomy (or laparoscopy) without undue delay to remove all the pus and muck from the peritoneal cavity. The abscess cavity in the liver is opened widely and irrigated completely to remove any residual pus.

A wide bore tube drain is then placed into this cavity, to be removed a few weeks later, once it stops draining and there is evidence of shrinkage in size on serial US. Occasionally, the abscess may rupture in a slow manner, leading to a slow leak and a localized collection that may be drained under radiological guidance, thereby avoiding a formal laparotomy.

**Peritonitis** in amebiasis can also occur from perforation of a colon that is usually severely affected. In fact, amebiasis is considered to be the most common cause of colonic perforation in this region and an important cause of fulminant colitis and toxic megacolon. These perforations are commonly multiple. The colon is dilated and friable and easily injured during surgery, compounding the surgeon's problems. After cleaning out the abdomen, deal with the perforation-bearing segment of the colon. This is usually achieved by limited resection of the affected segment of the colon (not necessarily a formal or classical anatomical resection) with exteriorization of the proximal and distal ends. Reconstruction is usually deferred in view of the poor condition of the patient and the risk of anastomotic breakdown, which could be fatal in such debilitated patients. A few authors have treated this situation with a diverting ileostomy without resection when there is limited contamination of the peritoneal cavity and self-sealing of the perforation by the omentum. They reported similar mortality to that following colonic resection; however, such "clean" cases are encountered very rarely.

## Parasitic Infestations of the Abdomen

A variety of parasites can cause chronic and acute abdominal symptoms. The important ones that can cause an acute abdomen are listed in Table 37.1.1.

### *Ascariasis*

By far the most common helminthic infestation is by *Ascaris* (ascariasis). This usually remains asymptomatic but may present with the passage of the worm through the anus or the mouth. As a surgeon, you may encounter *Ascaris* in the following situations:

- **Intestinal obstruction**. This is usually a partial small bowel obstruction but at times may become complete due to impaction of the worms (which may be further aggravated by spasm of the bowel) or by volvulus of the worm-containing obstructed segment of the bowel. The initial management is conservative, with nasogastric suction, intravenous fluids, antibiotics, and oral piperazine (dose of 75 mg/kg body weight). However, a few authors believe that antihelminthic therapy should be avoided initially as it could actually worsen the symptoms and convert a partial obstruction to a complete one. There are also reports of successful relief of partial worm obstruction by the instillation of Gastrografin through the nasogastric tube; because it is hyperosmolar, Gastrografin probably

**Table 37.1.1.** Parasites and the acute abdomen

| Name | Geographical distribution in Asia | Transmission | Acute conditions |
|---|---|---|---|
| *Ascaris lumbricoides* | Worldwide | Feco-oral | Intestinal obstruction<br>Bowel perforation<br>Cholangitis<br>Pancreatitis<br>Appendicitis |
| *Echinococcus granulosus* | Middle East, Indian subcontinent | Handling of infected dogs | Jaundice<br>Peritonitis<br>Infection<br>Anaphylaxis |
| *Anisakis simplex* | Japan | Ingestion of undercooked, raw infected fish | Intestinal obstruction<br>Anaphylaxis |
| *Clonorchis sinensis* | Orient | Ingestion of infected fish (undercooked, raw, frozen, dried, or pickled) | Cholangitis |

acts by dehydrating and separating the worm bolus (› Chap. 21). However, if the obstruction is complete or there is failure of conservative management, then these patients need to have an operation. The management at laparotomy should aim to milk the obstructing mass of worms into the large bowel, from where they will pass spontaneously through the anus. An impacted bolus that cannot be manipulated needs a longitudinal enterotomy to remove the worms; remember to close this transversely.

– Occasionally, **perforation of the intestine** may occur by the worm burrowing either through the normal wall or through areas of pre-existing lesions (ulcers, etc.). The management remains the same as for peritonitis from other causes.

– **Ascariasis of the biliary tract** is the most common extraintestinal site for the worm, and you may be surprised to hear that biliary ascariasis is the second most common cause of acute biliary symptoms (after calculi) in the world. The presentation can either be *uncomplicated* (clinical presentation similar to acalculous cholecystitis) or *complicated* (recurrent cholangitis, jaundice, or pancreatitis). The worm can usually be detected on US as a thin, echogenic tubular structure, which may even show movements, or as a worm bolus (the "spaghetti"

sign). The majority of patients with biliary ascariasis respond to conservative management (intravenous fluids, antibiotics, antispasmodics, and antihelminthics), and the worm spontaneously re-enters the bowel. Mebendazole (100 mg twice a day for 3 days) and albendazole (single dose of 400 mg) are considered the drugs of choice, but again, there are a few authors who believe that therapy against *Ascaris* should be deferred until it moves out of the biliary system and into the intestine (the dead parasite cannot migrate). Therapeutic ERCP may be considered in patients with complicated disease and in those who do not respond to conservative therapy. At ERCP, the worm is removed by the Dormia basket, taking care not to perform a sphincterotomy (as this may be associated with recurrent biliary ascariasis). If ERCP is unsuccessful in extracting the parasites, there has been fragmentation and partial extraction, or there are associated strictures, surgery is indicated. The aim of surgery is to achieve complete clearance of both the biliary tract (at common bile duct exploration) *and* the intestine of the worm, with adequate treatment of any associated conditions (such as hepaticojejunostomy when biliary stricture is present). If no further procedure other than ductal clearance is required, choledochotomy and closure over a T tube is recommended. Postoperative deworming is essential in all such cases.

### *Echinococcus*

*Echinococcus* is another important parasite that can cause acute abdominal symptoms. It gives rise to hydatid disease of the liver (Fig. 37.1.8), which is usually a chronic condition unless the hydatid ruptures freely into the peritoneal cavity (peritonitis) or into the bile ducts (jaundice, cholangitis). Asymptomatic, small (<4-cm), deep-seated, calcified, and uncomplicated hydatids of the liver can

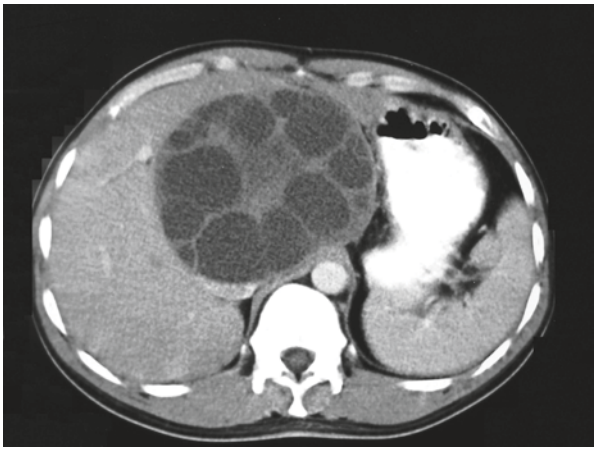

**Fig. 37.1.8.** Appearance of liver hydatid on CT scan

be managed adequately by nonsurgical means (chemotherapy, percutaneous aspiration, and injection), but complicated cysts usually need surgical intervention.

#### Free Ruptured Hydatid Cyst

The aims of surgery when dealing with secondary peritonitis from a ruptured hydatid cyst of the liver are:

- Elimination of the visible elements in the peritoneal cavity
- Sterilization of the peritoneal cavity using scolicidal agents such as 0.5% silver nitrate solution, hypertonic saline (20%), chlorhexidine solution, or cetrimide
- *Source control* to remove any residual parasitic element cysts from the liver, removing the germinal layer, suturing of any visible cyst-biliary communications, obliteration of the cavity (preferably by packing it with omentum), and drainage

#### Rupture of Hydatid into the Biliary Tree

Rupture of hydatid into the biliary tree usually produces obstructive jaundice and cholangitis and is often considered to be an absolute indication for surgery. However, ERCP may obviate the urgency of surgery by clearing the common duct of the parasitic cysts, providing free drainage of bile and lowering the intraductal pressures. Patients who fail ERCP or in whom there are recurrent symptoms can then be taken for surgery, where choledochotomy, clearance of the cyst remnants, and closure over a T tube are performed.

All emergency interventions must be followed up with albendazole (400 mg twice daily) for at least 3 weeks.

#### *Anisakis simplex*

Illness with the *Anisakis simplex* nematode can present with severe abdominal pain, nausea, and vomiting, typically within a few hours of ingesting raw or undercooked infected fish (sushi eaters be careful). The larva of *Anisakis* usually involves the stomach in humans but can also occasionally affect the small bowel. In the acute stage, the presentation has been confused with appendicitis, the patients submitted to surgery, and the true nature of the diagnosis revealed only on histopathological examination. The diagnosis is difficult to make with certainty in an emergency situation but can be suspected preoperatively if the patient spits out the worm or, in endemic regions, when a history of eating raw fish a few hours prior to the onset of symptoms can be elicited. In such cases, upper GI endoscopy

can visualize and remove the parasite, with relief of symptoms. As human infections are a dead end for the parasite, no further treatment beyond symptom relief is required if the diagnosis can be made. If undetected, the infection can present with small bowel obstruction after 2–3 weeks and invariably requires surgical intervention.

### *Liver Flukes*

*Liver flukes* (*Clonorchis sinensis*) are another significant cause of biliary symptoms, especially in the Far East. These live in the biliary tract for long periods before causing significant symptoms, classically known as oriental cholangiohepatitis or recurrent pyogenic cholangitis (pain, fever, jaundice). Chronic disease may give rise to secondary biliary cirrhosis and portal hypertension. An association of cholangiocarcinoma with this infestation has also been proposed. The treatment is medical (praziquantel), but clearance of the bile ducts (ERCP or surgical) may be required in refractory cases.

Although a wide variety of worm infestations can give rise to acute abdominal symptoms, it is important to realize that these diagnoses are often not made initially but after investigations or on histopathology, even in the endemic regions. For example, appendicitis can also arise as a result of luminal obstruction by worms (*Ascaris*, pinworms, or *Strongyloides*), but the diagnosis is rarely made pre- or intraoperatively. It is usually made retrospectively, on examination of the specimen by the pathologist. Therefore, it is important to realize that the symptoms of the patient can indicate the need for conservative or surgical treatment, and further therapy can be added once the specific diagnosis is made.

# 37.2 Africa

Graeme Pitcher · Craig Joseph

*Like the many paradoxes in Africa, emergency surgery illustrates the extremes: there may be helicopter transfer to twenty first century intensive care, or multiple cart and taxi transfers over several days to an under resourced mission hospital.* (David Dent)

The continent of Africa is as diverse as it is enormous. Its populace ranges from the some of the most rural, uneducated, and poverty stricken, with limited or no medical access, to wealthy first-world citizens who have access to modern, often private, health care. The full range of abdominal emergencies exists as in other countries, with trauma and obstetric emergencies masquerading as general surgical pathology. Though there are unique and interesting pathologies that are seen in Africa, the vast majority of patients with abdominal emergencies share their pathology with their Western counterparts, but because of poor access to health care, political strife, and civil wars they tend to present much later to the surgeon in the course of their disease, often with unique sets of surgical challenges. Delay in presentation is common to the point of being almost the norm. In this chapter, we discuss some of the unique conditions as well as share some tips to deal with the neglected case. Adequately treating these patients, often in suboptimal circumstances, remains the challenge of surgery in Africa.

## Intussusception: Lethal and Delayed

Intussusception remains the most common acute life-threatening abdominal condition of early life. The typical Western presentation depicted by Wojciech Górecki (Chap.35) is, in our experience, rarely seen in indigent African populations.

- Many patients present as having intestinal obstruction.
- Because of bowel loop distension, the typical abdominal mass may be difficult to palpate.
- Many patients are mistakenly treated for dysentery.
- Acute peritonitis from perforations, usually at the point of pressure of the lead point on the receiving bowel, is often present.

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- Colocolic intussusception is uniquely common in southern Africa, typically in older children.
- The intussusception may protrude from the anus and be mistaken for a rectal prolapse. Being able to pass one's finger up adjacent to the presenting bowel for a considerable distance on rectal examination should make this distinction (Fig. 37.2.1).

Intestinal obstruction in a child less than 4-years-old without evidence of an incarcerated hernia or previous surgery is intussusception unless proven otherwise.

Fewer patients with intussusception in our area can be treated by radiologically guided pressure reduction. In well-resuscitated patients in the age range 3 months to 2 years, with a nontender abdomen and no sign of established

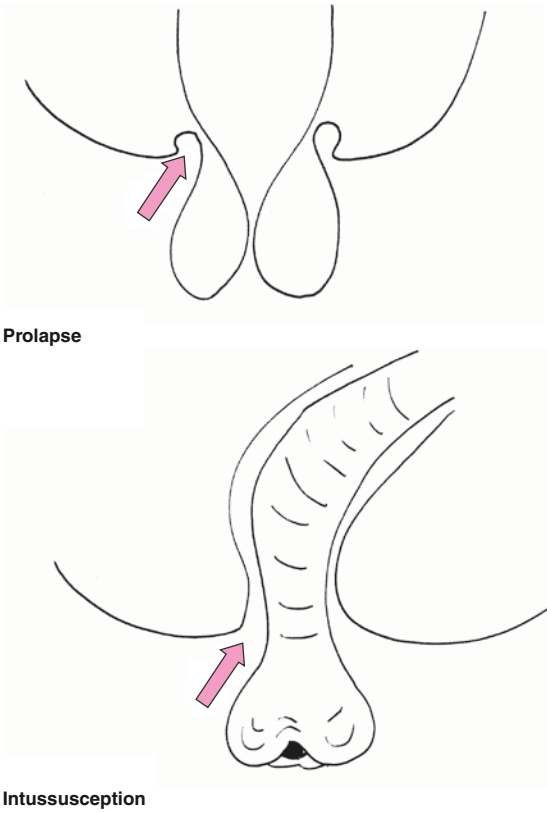


**Fig. 37.2.1.** Distinguishing between rectal prolapse and intussusception. In rectal prolapse, the examining finger cannot slide between the prolapsed bowel and the anal verge, whereas in intussusception the finger can slide in alongside the intussusception presenting at the anal verge

intestinal obstruction, reduction (preferably with air) can be attempted and is successful in about a third of patients. Duration of symptoms is not used as an absolute contraindication to air reduction because that would preclude almost all patients. Other patients are best treated by open surgical exploration as resection rates for bowel ischemia and perforation are high.

### Appendicitis: Neglected Perforation Still a Fatal Disease

The management of appendicitis in the developing world is no different from anywhere else. One possible exception is to recognize the patient with life-threatening long-standing perforation, often of up to 2–3 weeks duration. Such patients usually present with severe sepsis, dehydration, wasting, organ dysfunction, and a grossly distended abdomen with diffuse peritonitis and multiple loculated collections. They are best managed by very aggressive preoperative resuscitation, antibiotic treatment, and exploration by a midline laparotomy—not laparoscopically...

### *Ascaris* Infestations

The *Ascaris* parasite has certainly found a happy home in the intestinal tract of many Africans. The most common presentation to the surgeon is with intestinal obstruction. Most patients will report vomiting the worms or passage of worms rectally. Plain abdominal X-rays show a picture of intestinal obstruction with the typical curvilinear shadows of worms in the lumen of the bowel. Most patients can be treated conservatively with fluid resuscitation and nasogastric drainage. Vermicidal agents are traditionally not used in the acute stage of obstruction for fear of paralyzing the worms in the bolus and aggravating the situation (but this may be a myth!). Disobstruction is aided by the administration of Gastrografin (which is slippery and hypertonic), given usually from above via nasogastric tube (› Fig. 37.2.2) but also as an enema in cases of distal obstruction. Indications for surgical intervention include peritonitis, severe systemic toxicity, failure to respond in 24–36 hrs, and severe rectal bleeding.

### Leiomyopathy

Leiomyopathy is a common problem in African children. We have seen more than 50 cases. It is not well described in the textbooks or literature because it is virtually nonexistent in developed countries, and the cause is not known; we think that it is a toxic injury from enemas or other *muti* (see the section on enemas).

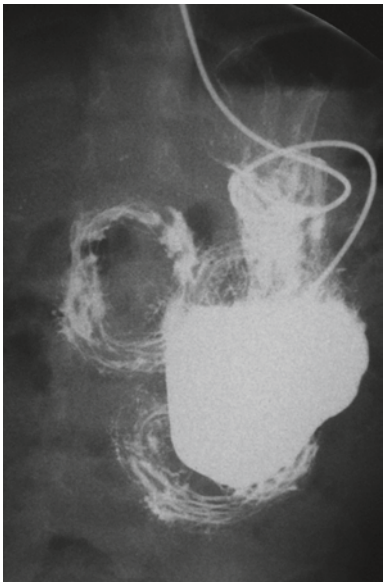

**Fig. 37.2.2.** Gastrografin administered via nasogastric tube in obstructed patient. Note *linear shadows* indicating worms in proximal bowel

This condition presents with an onset of (initially fairly benign and asymptomatic) abdominal distension at 3–7 years of age followed by progressive massive, predominantly gaseous distension with frank obstructive features and progression to death from intestinal failure, usually before the third decade of life. The cause is unknown, and pathology of affected bowel (usually starting distally and progressing more proximally) shows replacement of muscle in the muscularis propria with sheets of fibrous tissue. These patients are typically enormously distended clinically and radiologically but usually show no signs of intestinal obstruction and often arrive in surprisingly little distress, eating normally. The abdomen is soft and tympanitic to percussion.

The common pitfall with this condition is to look at the X-ray, assume a life-threatening obstruction, operate to find huge loops of colon, and then perform a stoma. These stomas are massive and bulky, prone to complications, and generally fail to adequately relieve distension. Instead, these patients are best served by rectal decompression by tube or endoscopy, a regular "bowel program" (mainly "from below"), and nutritional support. No form of surgery helps these patients significantly, although a small number can be palliated by total colectomy and ileorectal anastomosis for a few years.

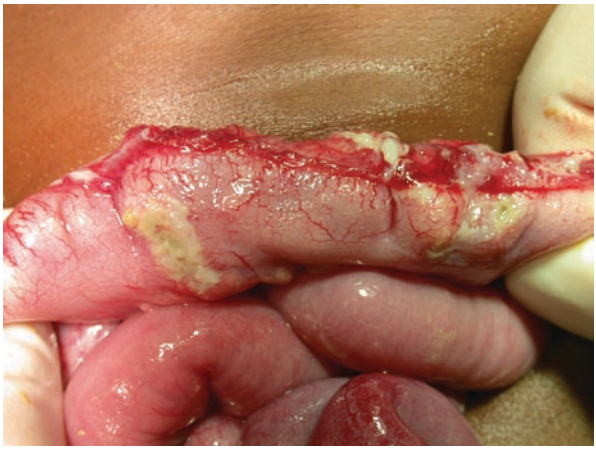

**Fig. 37.2.3.** Multiple colonic perforations in an HIV-positive infant with CMV colitis

### HIV-Related Abdominal Emergencies (see Chap. 36)

The high incidence of HIV/AIDS, and the associated opportunistic infections, in particular tuberculosis, greatly increases the possible spectrum of pathology. The most common abdominal complications of HIV/AIDS in our experience are cytomegalovirus (CMV) enteritis and perforation in the young infant (3–18 months of age) and abdominal tuberculosis in the older child.

Typically, CMV disease presents with acute abdominal distension, vomiting, and cardiovascular collapse in a severely immunocompromised and malnourished child. At laparotomy, multiple, sometimes hundreds, of small perforations (Fig. 37.2.3) are found along the entire length of bowel. Histology will sometimes reveal the typical intracytoplasmic viral inclusions. Even with aggressive treatment, including the use of intravenous ganciclovir, prognosis is poor.

### Traditional "Witch Doctor" Enemas

Although there is a paucity of literature on the topic, the use of traditional *muti* enemas is commonplace among many of the African tribes (see Fig. 37.2.4). The addition of toxic chemicals by the traditional healer or *Sangoma*, such as potassium dichromate (a potent caustic agent), can result in corrosive injuries causing tissue necrosis, perforations (Fig. 37.2.5), or late strictures. Mechanical instrumentation alone can result in direct rectal trauma, and any of these injuries may lead to peritonitis or retroperitoneal necrotizing fasciitis. The clinical picture may be further complicated by the addition of nephrotoxins, typically

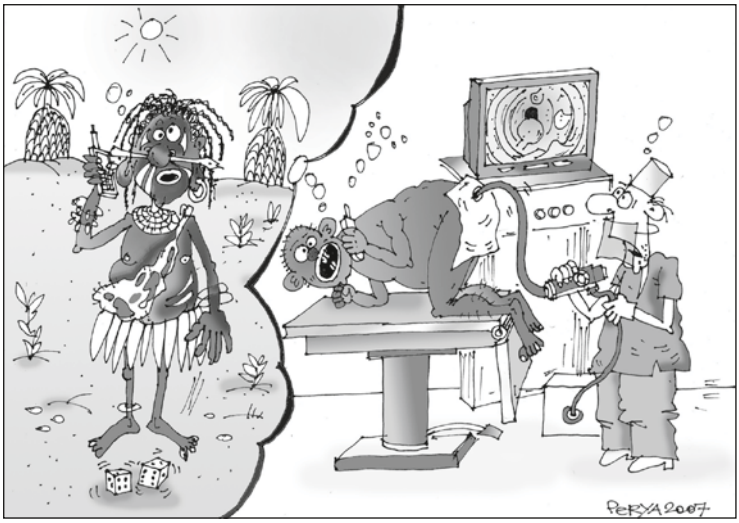

**Fig. 37.2.4.** "Hey Doc, let me just call for second opinion. Yes, I am calling my *Sangoma*"

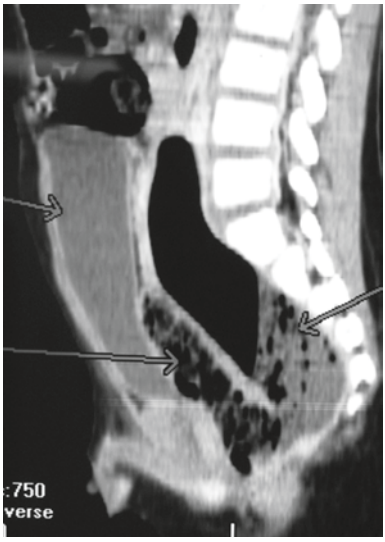


**Fig. 37.2.5.** A CT abdomen sagittal reconstruction of a 5-year-old boy treated erroneously for acute appendicitis found later to have a large posterior rectal perforation with a retrorectal collection—a complication of a "traditional enema." The *arrows* on *left* point out the bladder and rectum, whereas the large air-filled structure posterior to the rectum *arrowed* on *right* is the collection

Cantharidins (an extract that the Tswana tribe derive from grinding the bodies of dead blister beetles that is used to augment potency and as an abortifacient), resulting in acute renal failure. Patients are often not forthcoming with a history of having used traditional medicines, and a very high index of suspicion is required. Studies have shown that the majority of African infants and children admitted to hospital with gastroenteritis have already been administered traditional enemas. It is also vitally important to ascertain if the onset of illness pre-dated the administration of the muti or not. It is a common mistake to blame all the sequelae on the potion and therefore miss other underlying conditions.

### Sigmoid Volvulus and Ileosigmoid Knotting (see Chap. 25)

Unlike in the "developed," world where sigmoid volvulus typically affects elderly patients, in Africa it tends to occur in younger males. The African variety of sigmoid volvulus is associated with a degree of megacolon/rectum, and the bowel wall and mesosigmoid are thick and relatively resistant to ischemia. Bowel necrosis is therefore less common than in the thinner-walled colons of the older Western counterparts but still occurs when prolonged delay in presentation occurs. Patients presenting with bowel necrosis require prompt resuscitation and emergency resection. Primary anastomosis is considered safe if an experienced operator is present and the patient's condition is stable. Cases presenting without signs of bowel necrosis or perforation should undergo sigmoidoscopic decompression. This is best achieved via the use of a rigid sigmoidoscope inserted with the patient kneeling on all fours with the buttocks elevated above head and shoulders. Typically, a "flatus" (rectal) tube is inserted following successful decompression. Due to the high recurrence rate, patients should have a definitive procedure during the same hospital admission (Chap. 25).

**Ileosigmoid knotting** is a rare condition in which the ileum wraps itself around the sigmoid colon, causing a closed-loop obstruction with the potential for gangrene in both viscera. It occurs mainly in Africa, Asia, and the Middle East, typically in healthy individuals of middle age. It is thought to be caused by redundancy of the mesenteries of both the sigmoid and the ileum and possibly aggravated by a high-bulk diet. Principles of management include resection of bowel when necessary and applying judgment as discussed regarding the advisability of primary anastomosis.

### Typhoid Fever

Infection contracted by feco-oral transmission of the Gram-negative bacillus *Salmonella typhi* is still an important cause of small bowel perforation and peritonitis in poorer communities. Initially characterized by high fevers and a relative

leukopenia, a small percentage of patients will develop intestinal perforations, usually in the second to fourth weeks of illness. Following a perforation, mortality rises exponentially with delay to surgical intervention. Expeditious resuscitation and urgent surgical exploration are required. Perforations may be minimally debrided and simply closed. Multiple perforations may require segmental resection, typically of the terminal ileum. Primary anastomosis is deemed safe unless the patient is physiologically compromised, in which case the bowel is best exteriorized as a stoma. Appropriate antibiotic cover is vital to success. With increases in resistance to amoxicillin and chloramphenicol, a quinolone antibiotic is a safer first choice in very ill patients. The diagnosis may be confirmed by histology of the resected specimen, a positive blood culture, or positive serology in the form of a Widal test.

## Sickle Cell Disease

Sickle cell disease (SCD) is an autosomal recessive hemoglobinopathy that is relatively common in parts of Central Africa. Heterozygotes, who have only one abnormal gene, have sickle cell trait (SCT), a beneficial condition that confers a natural survival benefit against malaria. Unfortunately, individuals with a double dose of hemoglobin S (HbS; i.e., SCD) have no such luck. The abnormal hemoglobin becomes unstable under conditions of low oxygen tension and aggregates into large polymers. This results in distortion of the erythrocytes and a reduction in their deformability. The disease is characterized by a chronic hemolytic anemia and painful vaso-occlusive crises. Acute abdominal pain is a common mode of presentation and may be difficult to differentiate from other surgical or urological emergencies. Certain clues may point to a "sickle cell crisis":

- Pain occurs at multiple sites; typically the chest, back, and extremities.
- The pattern of pain is similar to prior episodes.
- Bowel sounds are preserved.
- Improvement occurs with general supportive measures such as hydration, oxygenation, and judicious use of analgesia.
- A precipitating factor (trigger) is present (e.g., a respiratory tract infection).

Most sickle cell crises will settle on supportive therapy, but very rarely ischemic bowel perforations may require surgery, and massive splenic or hepatic sequestration crises may prove fatal. The usual commonly encountered surgical pathologies also occur in this patient group, and abdominal computed tomographic (CT) scanning has proven to be a reliable modality that will identify most surgical conditions, but ultimately clinical observation of the patient's course will determine the appropriate management. Diagnosis of SCD in suspected cases is confirmed by observing sickled cells on a peripheral blood smear.

## Conclusion

Acute abdominal conditions challenge surgeons in Africa not so much because of their disease diversity but in the challenge of treating the often very severely ill, neglected patient in poorly staffed and equipped conditions. Sophisticated perioperative care, including intensive care, which has revolutionized the results of the treatment of these patients in the West, is simply not present in the vast majority of hospitals. Surgeons have to be innovative and in many instances must tailor the patient's treatment according to their facilities and circumstances. Patients' physiological reserves are frequently compromised by malnutrition and severe infectious diseases, particularly tuberculosis and HIV/AIDS, and the pressure is on the surgeon to "get it right the first time with the simplest and safest operation." There is no leeway for errors of operative strategy!

> "After climbing a great hill, one only finds that there are many more hills to climb." (Nelson Mandela)

# Penetrating Abdominal Trauma

38

Roger Saadia

*It is absolutely necessary for a surgeon to search the wounds himself, which are not drest by him at first, in order to discover their nature and know their extent.* (A. Belloste, 1701)

## General Principles

The crucial decision faced by the surgeon is whether an exploratory laparotomy is indicated. The decision to operate rests solely on the high likelihood that a significant injury is present; it does not require a precise inventory of all the possible intra-abdominal visceral injuries. In penetrating trauma, the role of clinical evaluation is primordial. Depending on the circumstances, it needs to be complemented sometimes by adjunctive diagnostic measures.

The surgeon's initial objective is to identify the patient requiring surgery while avoiding unnecessary laparotomies. These are termed *negative* when no injuries are present and *nontherapeutic* when the identified injuries would have healed spontaneously if left alone (for example, a minor hepatic laceration associated with a small hemoperitoneum but no active bleeding).

To fulfill this ideal requirement of timely necessary surgery with a zero rate of unnecessary laparotomies, numerous algorithms, some very complicated, have been devised incorporating various diagnostic tests. Not a single one is foolproof or has gained universal acceptance. In fact, the most experienced trauma surgeon does perform, from time to time, an unnecessary laparotomy; while such surgery is attended by some morbidity, this is a fair price to pay for not missing a significant intra-abdominal injury, provided that the frequency of such laparotomies is not unreasonably high.

In **civilian practice**, there are two main mechanisms of penetrating abdominal trauma: **stab wounds** and **gunshot injuries**. Owing to surgical tradition, these two categories have been treated differently, with mandatory surgery advocated

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for gunshot wounds. More recently, there has been a tendency to apply the same management principles irrespective of the injury mechanism.

After penetrating abdominal trauma, two possible clinical pictures can be found, in isolation or in combination: hypovolemic shock and peritonitis. The former is the result of bleeding from an injured solid organ (e.g., spleen, liver) or a sizable vessel. The latter is the consequence of soiling of the peritoneal cavity caused usually by an injured hollow viscus (gut, biliary system, urinary bladder).

## Abdominal Stab Wounds

> "Though shock may be temporarily alleviated by transfusion, it cannot be arrested or overcome; resuscitation divorced from surgery is folly." (William Heneage Ogilvie, 1887–1971)

The diagnosis of a stabbed abdomen is straightforward in the majority of cases: there is a visible wound on the abdominal wall, and the patient or witnesses usually confirm the circumstances of the assault. Do not be taken in by "fishermen's tales" about how long the steak knife was but remember instead the adage: "Treat the patient not the weapon."

It bears repeating that clinical evaluation of the patient (supplemented by an upright chest X-ray) is the most important step in the diagnostic workup. There are scenarios mandating an exploratory laparotomy without the need for additional confirmatory diagnostic procedures. The only required tests are those preparatory for a laparotomy (basic blood work, blood group and match, and when necessary electrocardiogram [EKG], βHCG, etc.).

***The following are indications for immediate surgery:***

- **Hemodynamic instability** in the absence of an associated extra-abdominal injury that could, by itself, account for shock. Aggressive fluid resuscitation must be started immediately. (Patients in extremis should be transferred expeditiously to the operating room since emergency room thoracotomy is not a useful maneuver in this context; as for emergency room laparotomy, it is extremely efficient in transferring the patient's total blood volume from the abdomen to the floor.)
- **Peritonitis** is frequent, and there is little diagnostic value in eliciting tenderness and even guarding on abdominal palpation in the immediate vicinity of the laceration. Signs of peritonitis need to be found at a distance from the wound to confidently establish the diagnosis. Always ensure that the bladder is empty before you prod for abdominal tenderness (these patients often arrive to the emergency room with a bladder ready to burst, courtesy of overenthusiastic paramedics).

- The demonstration of **free intraperitoneal air** on the upright chest X-ray. Abdominal X-rays are unnecessary in stabbed abdomens except for the lateral decubitus film in a patient who cannot sit up for a chest X-ray.
- **Omental or intestinal evisceration**. A laparotomy is advisable because of the high likelihood of visceral injury. Even if the laparotomy turns out to be negative, it would have served the double purpose of reducing safely the herniated viscera and allowing for a meticulous closure of the lacerated abdominal wall, preventing hernia formation.
- A **retained stabbing instrument**. This could be tamponading a sizable blood vessel and therefore should be removed in the operating room.

### *Abdominal Stab Wounds: When to Observe? How to Investigate?*

Reading the standard textbooks, one gets a little confused about how to manage the asymptomatic patient with, typically, an anterior abdominal wall laceration. In about one-third of patients, the wound does not extend into the peritoneal cavity, and in another third it does, but there are no significant visceral injuries. Exploring all these patients would not be a good idea.

Diagnostic procedures are sometimes advocated. **Diagnostic peritoneal lavage** is cumbersome and lacks accuracy; it is attended by a high rate of nontherapeutic laparotomies. **Exploration of the wound** under local anesthesia aims at identifying a breach of the parietal peritoneum. It is often difficult to determine with certainty the extension of the track: try it in an obese or combative patient in the rough-and-tumble atmosphere of a busy emergency department! **Laparoscopy** is a logistically demanding test since it requires general anesthesia. Here also, its main value is to ascertain intraperitoneal penetration. Do not be fooled by overconfident minimally invasive surgeons; a negative laparoscopic assessment of the peritoneal cavity cannot exclude a small intestinal laceration with minimal spillage or assess the retroperitoneum. In addition, postprocedure clinical or radiological abdominal assessments are made unreliable. About the selective role of laparoscopy for a suspected diaphragmatic injury, see the section on "difficult scenarios".

There remain two (we believe, complementary) approaches to the asymptomatic patient with an anterior abdominal stab wound: clinical reassessment and helical computed tomography (CT) scanning.

1. **Serial clinical reassessment of the patient**

This policy has been dubbed "selective conservatism" and has proved its worth in many centers. The patient is admitted, kept nil per os and given an intravenous infusion. The vital signs and urine output are closely monitored. The

abdomen is re-examined at frequent intervals, checking for the development of peritonitis; the initial area of tenderness around the wound can be circumscribed with a marker, and spreading tenderness is watched for over the observation period. Analgesia, antibiotics, or nasogastric decompression are not required. If, after an observation period of 18–24 hrs, no signs of hypovolemia or peritonitis are elicited, it is highly unlikely that a significant intra-abdominal injury is present. A very good indicator of this is a patient angrily demanding a meal tray. In applying this policy, keep an open mind at all times and do not persevere stubbornly with nonoperative management in the face of even subtle deterioration. Having to operate in a delayed fashion in a well-monitored patient is not a sign of personal failure but a tribute to your clinical acumen. An occasional unnecessary laparotomy will be performed; this is nothing to be ashamed of, and when in doubt it is safer to err on the side of surgical exploration.

2. **Abdominal CT scan**

In recent years, both the access to CT scanning and the quality of the images have improved dramatically. In many centers, the asymptomatic stabbed patient is often sent to the scanner by the emergency physician before the surgeon is even consulted. Whatever seasoned trauma surgeons may think of this practice, the train has long since left the station. Undoubtedly, this investigation is often valuable, even though its shortcomings in early intestinal perforation are well known. Some patients are shown to have only a superficial wound with greater ease and accuracy than by local tract exploration. They can be safely discharged from the emergency department. A small minority of asymptomatic patients are diagnosed with a significant visceral injury (which would have eventually declared itself under observation). Their trip to the operating room is thereby expedited. The remaining majority of the patients with a negative or equivocal CT scan need to be admitted and observed as described.

The primacy of clinical evaluation is unquestioned. However, as in the management of acute appendicitis (› Chap. 28), its interplay with the judicious use of the CT scanner can refine one's decision making a little further.

## Gunshot Abdominal Wounds: Dogma Versus Modern Imaging

> "It is highly desirable that anyone engaged in war surgery should keep his idea fluid and so be ready to abandon methods which prove unsatisfactory in favour of others which, at first, may appear revolutionary and even not free from inherent danger." (H.H. Sampson, 1940)

Traditional wisdom inherited from war experience has held that an exploratory laparotomy is always indicated in patients with abdominal gunshot wounds

irrespective of their clinical condition. This policy has been predicated on the higher likelihood of significant intra-abdominal injuries in gunshot than in stab wounds. This premise, if true, implies merely that shock and peritonitis are more frequently associated with the former than with the latter. Decision making is then easy. What about the benign-looking abdomen with a gunshot wound that is encountered now not that infrequently in many large urban trauma centers? There is accumulating evidence to suggest that initially asymptomatic gunshot victims can be managed safely along the same broad lines as stabbed patients. While the role of initial and serial clinical reassessments is here again very important, **we contend that an early CT scan of both the abdomen and chest is mandatory, not only in asymptomatic patients but in all gunshot victims who are stable enough to go to the scanner.** Bullets tend to travel longer distances than the length of a knife blade. Imaging of the whole torso is essential to document the trajectory of the bullet, which could extend beyond the confines of the abdominal cavity. A missing bullet should prompt the search for an extra-abdominal location or a hidden exit wound. Furthermore, a bullet entering the abdomen can significantly damage bony structures (thoracolumbar spine, pelvis, hip, etc.). The information gleaned from these images is often invaluable despite the occasional "scatter" caused by a retained metal fragment. Sometimes, it will be seen that **the missile's trajectory is tangential, missing the peritoneal cavity: a laparotomy can be avoided**, but semielective debridement of the abdominal wall may prove necessary in some of these cases.

### Difficult Scenarios: The CT Scan Reigns Supreme

Stab wounds to the lower chest, the flank, or the perineum pose the problem of possible but clinically occult injury to intra-abdominal viscera.

- **The diaphragm:** an isolated diaphragmatic laceration is often at first clinically silent but is sometimes complicated by a secondary diaphragmatic hernia. This complication is more likely to occur on the left than on the right side, which is relatively shielded by the bulk of the liver. Little is known about the natural history of diaphragmatic wounds, but very small ones are probably often missed with impunity. It is, however, standard of care to look for them whenever a stab wound is located in the lower chest or upper abdomen (especially on the left side). In this scenario, if there are no other clinical reasons to operate, then a thoracoscopy or a laparoscopy should be performed during the patient's hospital stay to check the integrity of the diaphragm; if a laceration is identified, it should be repaired at laparotomy. Reconstructed coronal CT scan views of the diaphragmatic domes can also be very helpful and may supplant laparoscopy in the future.
- **The flank:** a stab wound to the flank can involve the retroperitoneal portion of the duodenum or colon. Peritoneal signs are present only at a late stage (sometimes too late, associated with advanced retroperitoneal infection).

Therefore, a CT scan must always be obtained early (there is no more need for a combined contrast enema). An injury to the kidney is often benign and is usually associated with frank hematuria. The possibility of a ureteric injury is more serious and must be entertained in the presence of microscopic hematuria. The CT scan has now supplanted the intravenous pyelogram (IVP) as the screening investigation of choice in suspected injuries to the urinary tract.

- **The perineum:** abdominal penetration must always be suspected. A digital rectal exam looking for rectal bleeding is a mandatory component of the clinical examination. A CT scan is helpful and may need to be supplemented by a rectosigmoidoscopy.
- **Patients with multiple stab or gunshot wounds to both the chest and abdomen** may constitute a dilemma in the choice or sequencing of the operations if both the chest and abdomen are possible candidates for the source of severe hemorrhage; this is particularly the case if the patients are unstable and unfit for transfer to the CT scanner. Alternatively, one can come across a patient with a high epigastric stab wound and hypotension in whom the possibility of a cardiac tamponade should be entertained. In these cases, an ultrasound scan in the emergency room (FAST, focused abdominal sonography for trauma) may help formulate a logical management plan. FAST is used more frequently in blunt trauma and is discussed in Chap. 39.1.

## What to Do When CT Scanning Is Not Available?

Some of you readers from developing countries may not have unrestricted access to emergency CT scans. The great majority of penetrating trauma victims can be managed by the combination of three diagnostic modalities: clinical examination, upright chest X ray, and, yes, exploratory laparotomy, the last resorted to more liberally whenever in doubt. Keep the threshold for intervention low. The acceptable price to pay, in this context, is a higher rate of unnecessary laparotomies rather than missed injuries. In patients with a flank injury or hematuria, a one-shot IVP in the emergency room is easy to perform and very useful (especially in confirming the presence of a functioning kidney on the **uninjured** side).

## Conclusion

Clinical evaluation (including vital sign assessment and abdominal examination) retains to this day its primacy in the management of penetrating abdominal trauma. There are clear-cut clinical scenarios requiring immediate laparotomy. In other situations, clinical observation remains extremely valuable. In recent years, abdominal CT scanning has established itself as the best diagnostic adjunct. Know when to operate and when not to (Fig. 38.1).

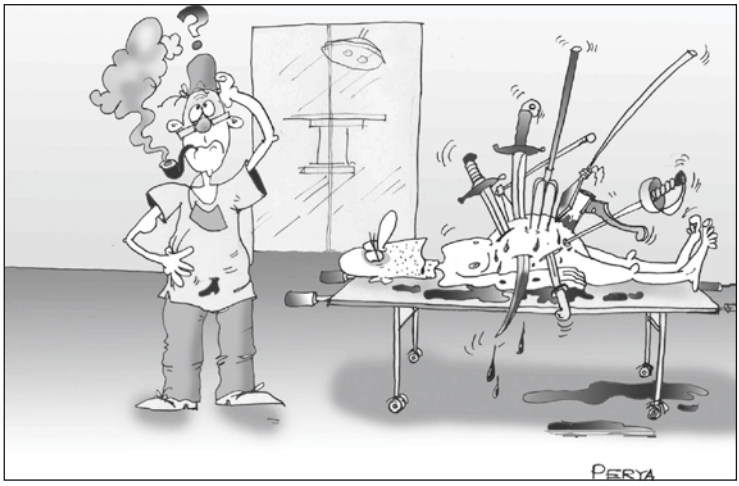

**Fig. 38.1.** "Let's be conservative!"

"Failure to promptly recognize and treat simple life-threatening injuries is the tragedy of trauma, not the inability to handle the catastrophic or complicated injury." (F. William Blaisdell)

# 39

# Blunt Trauma and Rx of Specific Injuries

Roger Saadia

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# 39.1 Blunt Abdominal Trauma

Roger Saadia

*Definition of a heavy trauma: somebody who arrives at the hospital in more than one ambulance.* (John Edwards)

*He should have a special love for the wounded persons as for his own body.* (Hans von Gersdorff, 1480–1540)

## Differences Between Blunt and Penetrating Trauma

There are several differences between blunt and penetrating trauma injuries:

- Penetrating abdominal trauma is made obvious by the presence of a wound. Blunt abdominal trauma is sometimes unequivocally identifiable by the presence of a visible contusion of the abdominal wall (e.g., seat belt sign), but more frequently, it is only suspected from the mechanism of injury.
- Penetrating abdominal trauma is usually confined to the abdomen. Common mechanisms of blunt trauma (vehicle accidents, falls, beatings, etc.) often result in polytrauma, the abdominal component being associated with other cavity or system injuries (head, chest, pelvis, vertebral column, long bones).
- The patterns of intra-abdominal visceral injuries are different. Hollow viscera injuries are common in penetrating trauma. They are very rare in blunt trauma, where solid organ injuries (to liver, spleen, pancreas) predominate.

## Clinical Evaluation Is Unreliable

Clinical evaluation is unreliable due to several factors:

- There is frequent presence of a head injury with decreased level of consciousness.
- The nature of multisystem trauma results in "distracting" injuries by which the pain experienced at other sites (chest, long bones, etc.) masks or distorts the patient's perception of abdominal pain and tenderness.
- While hypotension is frequently caused by an injured intra-abdominal solid organ, it is as frequently due to an associated long-bone fracture or a hemothorax. It may even not be the result of hypovolemic shock but represent a sign of cardiogenic (due to cardiac contusion, pericardial tamponade, tension pneumothorax) or spinal shock.

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**Fig. 39.1.1.** "So what's wrong inside your black box"?

- Tenderness on palpation may be the result of bruising of the abdominal wall rather than reflect a more severe intra-abdominal injury.

In blunt abdominal trauma, unlike in penetrating injuries, the reliance on clinical pictures of shock or peritonitis cannot constitute the sole justification for a laparotomy. The abdomen in blunt trauma has come to be seen as a "black box" (Fig. 39.1.1), in other words, an uncertain source for the patient's current instability or subsequent deterioration. It is therefore imperative to resort to additional diagnostic tests. The aims of these investigations are not only to confirm the presence of abdominal trauma, but also, whenever possible, to document as precisely as possible the nature of the visceral injuries since their treatment is not invariably surgical.

## Adjunctive Diagnostic Tests

There are three main diagnostic tests used in blunt trauma: diagnostic peritoneal lavage (DPL), ultrasound scan (referred to as FAST, focused abdominal sonography for trauma), and helical computed tomography (CT) scan. In modern, well-equipped centers, abdominal CT scan is the investigation of choice in the stable patient, while DPL and more frequently FAST are employed when the patient is hemodynamically unstable. The last two tests could also be used more liberally in facilities that cannot offer unrestricted access to CT scanning.

### *Diagnostic Peritoneal Lavage*

Both a nasogastric tube and a Foley catheter are first inserted in preparation for the DPL (it would be a pity to puncture the stomach or the bladder!). The technique entails the placement, under local anesthesia, of a catheter into the peritoneal

cavity. One liter of warmed saline is thereby infused, given a moment to mix with the intraperitoneal contents, and recovered by laying the bag on the floor.

**The DPL is deemed positive in cases of:**

- Aspiration of blood from the catheter on insertion ("grossly positive DPL").
- Presence of more than 100,000 red blood cells per cubic millimeter in the effluent ("microscopically positive DPL").
- Presence of bile, intestinal contents, or urine in the effluent.
- Presence of more than 10,000 white blood cells per cubic millimeter in the effluent (this is controversial).
- Flowing of the DPL fluid through the urinary catheter or the chest tube, indicating a bladder or diaphragmatic injury, respectively (these scenarios are rare).

Historically, DPL was the diagnostic gold standard for blunt abdominal trauma, but in recent times it has lost its shine for the following reasons:

- It is cumbersome and difficult to perform in a combative or obese patient.
- It has absolute or relative contraindications: previous surgery, pregnancy.
- It is invasive and attended by a small complication rate (bowel perforation).
- Most important, if laparotomies were to be performed for all instances of microscopically or even grossly positive DPL, the rate of non-therapeutic laparotomies would be unacceptably high because, in most cases, the source of bleeding could have been treated nonoperatively. Of course, an unnecessary laparotomy carries a significant morbidity in the context of multisystem trauma.

In modern centers, DPL is used only in the very unstable patient to confirm, preoperatively, the presence of a large hemoperitoneum. If your hospital lacks access to the more sophisticated investigations, do remember that a negative DPL is a crucial piece of information in ruling out the "black abdominal box" as a source for concern in a severe, multisystem trauma patient.

### *Focused Abdominal Sonography for Trauma*

The aim of FAST is to detect the presence of free fluid in the following areas:

- The pericardial sac
- Morrison's (hepatorenal) pouch in the right upper abdominal quadrant
- The splenorenal recess in the left upper abdominal quadrant
- The pelvis

FAST can assist in the diagnosis of pericardial tamponade (a rather rare finding in blunt trauma). In the evaluation of the abdomen, it duplicates somewhat the role of DPL with the advantages of being relatively cheap, totally noninvasive, and applicable at the patient's bedside. FAST is reliable only in the hands of personnel specially trained in the technique (surgeons, emergency room physicians, radiologists) and in centers with a high case volume. In modern centers, FAST plays an

important role in the assessment of the unstable trauma patient; a laparotomy is usually indicated in a hypotensive patient found to have a large amount of free intraperitoneal fluid. It is also commonly used in stable patients, but more as a practice exercise rather than a test allowing for definitive decision making. The use of FAST as a screening tool for abdominal CT scanning is more controversial.

### *Computed Tomography*

Computed tomographic scanning has become an essential part of the modern management of the stable blunt multitrauma patient. It is very common now to dispense with the cervical spine X-rays, the thoracolumbar spine X-rays, the pelvic X-ray, and even sometimes the chest X-ray; the patient is taken instead to a radiology suite adjacent to the resuscitation room, and a quadruple scan of the head, neck, chest, and abdomen (including the vertebral column and the pelvis) is obtained in a few minutes.

The abdominal component of this diagnostic workup is extremely valuable because:

- Both the peritoneal cavity and the retroperitoneum can be assessed.
- The integrity of bony structures (lumbar spine, pelvis) can be ascertained.
- A precise inventory of injuries to solid intraperitoneal (liver, spleen) and retroperitoneal (pancreas, kidneys) organs can be made; these injuries can be accurately graded.
- The new-generation scanners are able to detect intestinal injuries (suggested by mesenteric stranding, bowel thickening, or extraluminal air).
- Free fluid (with radiological blood density) in the absence of solid organ injury can be detected, suggesting the presence of a significant mesenteric injury.

In equivocal CT scan findings, clinical judgment is essential; a repeat CT scan 24 hrs later, clinical observation, or immediate laparotomy are the main options to be weighed.

The recourse to "total body scanning" has become so unregulated in some "high-tech" centers that a note of caution needs to be sounded:

- Cost aside, liberal "trauma scannograms" deliver a very high dose of radiation; this, combined with the recurrent need for CT scanning through an entire lifetime, carries a significant long-term cancer risk. Always ask yourself when sending a trauma patient to the scanner whether a quadruple test is essential in this particular patient. Could not, for example, the chest CT be replaced by a simple chest X-ray? An easy way to keep in mind this danger is to remember the acronym VOMIT (victims of modern imaging technology) coined by Hayward (BMJ, 2003).

- **Only stable or well-resuscitated patients can be put through the scanner. Borderline patients can decompensate catastrophically in the radiology suite.**

— CT images are as good as their interpreter. In the middle of the night, expert radiologists are rarely available. Always keep your clinical judgment on high alert, especially when there is discordance between clinical picture and CT images. Remember BARF (brainless application of radiological findings) and reach for an antiemetic.

## Nonoperative Management of Solid Organ Injuries in Blunt Trauma

The majority of patients with blunt splenic or hepatic injury (and almost all the patients with an isolated blunt renal injury) can be treated conservatively. Once such an injury has been identified on CT scan and provided there is no clinical or radiological evidence of an associated hollow viscus injury, nonoperative management can be attempted. The hemodynamic status rather than the radiological grade of the injury constitutes the basis for therapeutic decision making; the grade of injury has merely predictive value in the success of conservative management. The patient is admitted for the first 24 hrs to a high-care unit for close observation. Continuous vital signs and urine output monitoring, serial abdominal examinations, and repeated hemoglobin evaluation are conducted. Then, with every passing day on the ward with no sign of ongoing bleeding, the success of the conservative approach becomes more likely. Repeat CT scans are not required routinely on this admission but only if complications occur. On discharge, the patient is cautioned to avoid putting the injured organ at risk of a secondary rupture (e.g., contact sports, bar-room brawls) until a CT scan 8–12 weeks later documents complete healing. More subtle differences between splenic or hepatic injuries should now be pointed out.

### *Spleen*

Nonoperative management of a splenic injury should not be stubbornly continued in the face of an increased requirement for blood transfusion. When there are episodes of hypotension (unexplained by extra-abdominal injuries) or a sustained drop in hemoglobin (not accounted for by hemodilution), there should be a low threshold for splenectomy, especially in the adult. It is a real tragedy to lose a patient from splenic hemorrhage when definitive control of the bleeding can be achieved by a simple surgical procedure, namely, a splenectomy (acrobatic splenic salvage procedures belong to the past). The very small risk of postsplenectomy sepsis in the adult can be further minimized by patient education and vaccination (anti-*Pneumococcus*, anti-*Meningococcus*, and anti-*Hemophilus influenzae*). There is a range of opinions about the trigger for abandoning conservative management. Some believe that untreated hypotension alone (from a presumed splenic source) justifies intervention; others are prepared to transfuse up to a maximum of two units of blood before changing course. The message is clear: do not persevere with

multiple blood transfusions to treat ongoing splenic bleeding. The initial CT scan may reveal a contrast "blush" in the splenic parenchyma pointing to active bleeding; there is evidence to suggest that routine angioembolization of these bleeding vessels, in the stable patient, increases the success rate of nonoperative management.

#### *Liver*

The intraoperative control of hepatic bleeding is difficult. The loss of the tamponade effect at laparotomy followed by mobilization of the liver can result in renewed hemorrhage, sometimes torrential. In tackling a bleeding liver, there is no equivalent to a simple procedure like a splenectomy. Therefore, more diligence is called for in the pursuit of conservative management, as well as a greater reliance on aggressive transfusions of blood products and factors. There has been increasing recourse to angioembolization in attempts (often successful) to avoid surgery. With hepatic injuries treated nonoperatively, there is a higher complication rate than with splenic injuries. Increasing right upper quadrant pain, jaundice, melena, or sepsis should prompt specialized investigations (repeat CT scan, ERCP, angiography). Most of these complications can be treated by interventional radiology.

### When to Operate in Blunt Trauma?

The most common indications for surgery in blunt trauma are:

- The hemodynamically unstable patient with a significant hemoperitoneum preferably demonstrated by DPL or FAST. These investigations may be omitted when other extra-abdominal injuries are confidently ruled out in a hypotensive patient with a tense, distended abdomen.
- The patient with an acute posttraumatic diaphragmatic hernia demonstrated on chest X-ray or CT scan.
- The patient with or without peritoneal signs but with free intraperitoneal air demonstrated on an upright chest X-ray or abdominal CT scan.
- The patient with a hollow viscus injury (bowel, gallbladder, intraperitoneal urinary bladder) demonstrated clinically or on CT scan.
- The patient with CT evidence of a significant pancreatic injury.
- The patient with a significant hemoperitoneum in the absence, on CT scan, of solid organ injury; think of a severe mesenteric injury with a potential for bowel ischemia.
- The patient with signs of sepsis or a persistently tender abdomen in the presence of equivocal CT images.
- The patient in whom conservative management of a hepatic or splenic injury (identified initially by CT scan) has failed.

(For the operative management of these specific injuries, see ❯ Chap. 39.2.)

## Conclusion

Clinical evaluation is often unreliable in the management of blunt abdominal trauma. Great reliance is placed on the abdominal CT scan in stable patients and on DPL or FAST in hypotensive patients. The results of these investigations always need interpretation in the overall clinical context.

Things have changed since a century ago when it was stated:

> "Exploratory laparotomy offers, in our judgment, the quickest and the safest method of positive diagnosis. The emergency warrants a decisive step." (Albert Miles, 1893)

# Operative Management of Individual Organ Injuries

Roger Saadia

*We will always start with the most dangerously injured without regard to rank and distinction.* (Jean Larrey—Napoleon's surgeon; 1766–1842)

You have decided to perform a laparotomy. Currently, this is more likely for penetrating than for blunt trauma. Most solid visceral injuries in blunt trauma can be managed conservatively: often doing "less" is "better," with limited blood loss and avoidance of unnecessary tissue injury fueling the inflammatory response in a patient with frequently associated extra-abdominal injuries (Chap. 54). The incision and the assessment of the damage are described elsewhere (Chaps. 10 and 11, respectively).

## Diaphragm

A through-and-through diaphragmatic laceration requires suture repair with heavy, interrupted suture material. Lacerations with substantial tissue loss are rare and need repair with a synthetic mesh patch. A prosthesis may not be necessary when the tissue loss is at the periphery; instead, the diaphragm can be reimplanted to the ribs more cephalad. This is of particular benefit in the presence of extensive contamination. Remember that even in the absence of a preoperative pneumothorax, an ipsilateral chest tube must be inserted at some stage of the procedure. It is often said that minor diaphragmatic tears can be ignored on the right side because the bulk of the liver prevents future bowel herniation. However, large right-sided lacerations (seen usually in blunt trauma) must be repaired because the liver itself can, in time, be "sucked up" into the chest.

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## Liver and Biliary Tree

An irreverent classification of hepatic injuries follows:

- **Grade I:** Nothing should be done (treat conservatively)
- **Grade II:** Something should be done (local hemostasis)
- **Grade III:** Too much should not be done (packing only)
- **Grade IV:** Only God can do something (heroic measures)

The following are some practical management principles:

- Bleeding from small, superficial capsular tears can be controlled by cautery, individual vessel ligation, or clipping or by atraumatic suture repair of the fragile hepatic capsule.
- More severe bleeding from a deep or craggy hepatic laceration constitutes a surgical challenge requiring a stepwise approach. After a quick glance, bimanual compression of the hepatic parenchyma will control the bleeding temporarily, allowing the anesthesiologist to catch up with the blood loss. This must be followed by rapid mobilization of the liver by division of the falciform and left and right triangular ligaments—the liver can be literally dislocated into the abdominal incision. Additional exposure via a median sternotomy or right thoracotomy is rarely indicated. The *Pringle maneuver* (inflow occlusion of the undissected triad of portal vein, hepatic artery, and common bile duct) is sometimes useful and safe for up to 60 min. Deep parenchymal bleeding is controlled as well as possible by clipping visible bleeding vessels and by *conservative resectional debridement*. This rarely controls the hemorrhage completely—**supplementary packing is necessary.** Packs must be judiciously placed around (not into) the liver. The aim is to close the laceration by tight packing and thereby tamponade the bleeding. Excessive packing must be avoided because it can result in inferior vena cava compression or abdominal compartment syndrome with aggravation of the hypotension. A return to the operating room (OR) will be necessary in 36–72 hrs for pack removal. There is always a danger of losing sight of time and the amount of blood loss that is incurred while trying to achieve an elusive "perfect" result. More bleeding will require more transfusions and aggravate the coagulopathy in a well-known vicious cycle. **We strongly advise you to look at the clock before you tackle a nasty liver laceration: you should achieve both vessel control and packing ideally within 45 min.**
- Retrohepatic caval injuries are characterized by exsanguinating hemorrhage despite inflow occlusion. There are probably more techniques described for immediate hemostasis than there are survivors. It is perhaps best to resort to damage control with packing and come back to fight another day.
- Injuries to the porta hepatis require a wide Kocher maneuver for exposure. The injured portal vein should be repaired, or ligated as a last resort. Hepatic artery ligation is better tolerated than portal vein ligation. Suture repair or Roux-en-Y biliary enteric anastomoses are the treatment options for an injured common bile duct; the latter can

be performed either at the initial operation or at the reconstruction phase of a damage control strategy. Unilateral lobar bile duct injuries should be managed by ligation.

- An injured gallbladder should be resected.

### Spleen

The treatment at laparotomy of an actively bleeding spleen in the adult is splenectomy. Acrobatic surgical splenic conservation procedures belong to expensive surgical textbooks; they have no place in the OR. The risk of postsplenectomy sepsis is small and can be further minimized by vaccination, vigilance, and appropriate prophylaxis.

### Pancreas

The anterior aspect of the pancreas is exposed through the lesser sac by division of the gastrocolic omentum; the posterior aspect of the head is exposed by a Kocher maneuver, while the posterior aspect of the tail is achieved by splenic mobilization. The state of the pancreatic duct is a crucial determinant of the operative strategy in the injured pancreas. In some cases, the integrity of the duct may have been assessed preoperatively in the stable patient by endoscopic (ERCP) or magnetic resonance cholangiopancreatography (MRCP). If not, intraoperative pancreatography (through a duodenotomy and cannulation of the ampulla of Vater) is possible, **but in practice it is rarely performed**. In superficial pancreatic wounds, the main duct may be presumed to be intact, and drainage alone is sufficient. In deeper parenchymal wounds of the body or tail, ductal transection is likely, and a distal pancreatectomy (with splenectomy) is warranted. For deeper injuries of the head, wide drainage is indicated; the management of the inevitable pancreatic fistula in a stable patient is simpler than that of a leaking enteropancreatic fistula in the aftermath of a fancy, immediate, Roux-en-Y jejunopancreatic reconstruction. The Whipple procedure is reserved for massive injuries of the pancreatic head, with biliary ductal and duodenal disruption. This procedure is attended by a high mortality; it should be preferably "staged," with the definitive reconstruction performed only after the patient has been stabilized.

The following aphorism captures very graphically the management of this injury:

> "For pancreatic trauma, treat the pancreas like a crawfish: suck the head, eat the tail." (Timothy Fabian)

### Kidney, Ureter, and Bladder (for Much More, see › Chap. 34)

The intraoperative discovery of a perinephric hematoma is usually indicative of renal injury. A large proportion of these are self-limiting. Renal exploration is indicated in the presence of an expanding or pulsatile hematoma or when a hilar

injury is suspected. Moderate severity injuries can be controlled usually by cortical renorrhaphy and drainage; occasionally, a polar nephrectomy may be indicated. A shattered kidney or a vascular hilar injury is treated by nephrectomy; preliminary control of the renal artery and vein should not be attempted in the presence of hemodynamic instability. Attempts at saving a kidney, in these situations, are not warranted unless the patient has a single kidney.

Lacerations of the renal pelvis are repaired with fine absorbable sutures. An injured ureter should be carefully exposed, avoiding ischemic damage by overenthusiastic skeletonization. Primary repair with absorbable material over a stent is the rule. Very proximal or very distal ureteric injuries may require an expert urologic opinion.

An intraperitoneal bladder injury requires repair with absorbable sutures and catheter drainage. In an extraperitoneal rupture, catheter drainage alone is sufficient. A urethral Foley catheter is adequate in most cases. In severe, complex bladder injuries or significant bleeding, suprapubic drainage may be added to allow for efficient postoperative bladder irrigation.

### Stomach

Most gastric injuries are caused by penetrating trauma and are treated by simple, one-layer, suture repair. The posterior gastric wall should always be checked by opening the lesser sac. Blunt injuries are rare, and gastric resection is required only in exceptional cases.

### Duodenum

Intramural duodenal hematomas do not require evacuation; nasogastric decompression, fluid replacement, and adequate nutrition (usually parenteral) need to be instituted for up to 3–4 weeks.

Small, clean-cut lacerations can be safely repaired primarily. Extensive lacerations, the presence of significant tissue contusion (usually inflicted by blunt trauma), involvement of the common bile duct, or high-velocity gunshot injuries should be treated by duodenal repair and pyloric exclusion. This procedure consists of closure of the pylorus (by stapling or suture from inside the stomach) and re-establishment of gastrointestinal continuity by a gastrojejunostomy; the addition of a truncal vagotomy is not warranted. A feeding jejunostomy is a useful adjunct for the provision of enteral feeding. There is currently a feeling that this procedure is overused. In relatively extensive lacerations, we often supplement primary repair by tube duodenostomy inserted through the corner of the duodenal suture line. [Others insert it away from the suture line; some—us included—think that this is a gimmick—The Editors].

The Whipple operation is reserved for massive combined pancreatoduodenal disruptions. In an unstable patient, you should stage it: resect first and return another day for reconstruction.

### Small Bowel

Most lacerations can be treated with one-layer suture repair. Occasionally, a segmental resection may be required in injuries involving the mesenteric side of the intestine or for the treatment of multiple lacerations in close proximity. In the postresuscitation edematous intestine, hand-sewn anastomoses may be safer than stapled ones (Chap. 13). Neglected, long-standing lacerations (more than 24 hrs) with an established peritonitis may require the fashioning of a temporary stoma rather than primary repair. Rarely, an extensive mesenteric laceration may endanger a very large segment of bowel, which, if resected, would result in a short-gut syndrome; it is best then to decide on the extent of resection at a second-look operation in a well-resuscitated patient.

### Colon

Right- or left-sided simple colonic lacerations can be safely treated by suture repair in most cases. If the severity of the laceration warrants a resection, an ileocolic anastomosis (after a right hemicolectomy) is usually safe. A colocolic anastomosis (after a more distal resection) may not be as safe. In any case, a colostomy rather than repair is recommended in the presence of massive peritoneal contamination, severe associated injuries, or gross hemodynamic instability. In borderline cases, we advise you to err on the side of performing a colostomy; the stubborn resort to primary repair may turn out to be a costly act of surgical bravado: more trauma patients die from a leaking primary anastomosis than from a subsequent closure of a colostomy gone wrong. Extensive deserosalization (typical in seat belt injuries of the cecum or sigmoid colon) should be treated by serosal repair rather than resection.

### Rectum (see also Chap. 29)

In the absence of gross fecal contamination, minor lacerations can be treated by simple suture repair. In all other cases, a proximal diverting colostomy must be added; a loop sigmoid colostomy is usually adequate. Small lacerations of the intraperitoneal rectal segment do not require extensive mobilization of the rectum and repair; a diverting colostomy alone is sufficient. Washout of the distal rectal stump and presacral drainage are unnecessary except in very extensive injuries with wide dissection and soiling of the perirectal spaces.

## Intra-Abdominal Vascular Injuries

- **Aorta.** A most important step in the management of aortic injuries is exposure to achieve proximal and distal control. Depending on the level, this "medial visceral rotation" maneuver begins either lateral to the spleen or lower down, by incising the white line of Toldt lateral to the left colon. The viscera, including spleen, pancreatic tail, left colon, and if necessary left kidney are gradually mobilized medially. The suprarenal aorta can be approached through the gastrocolic omentum (via the lesser sac) with retraction of the stomach and esophagus to the left. For injuries of the supraceliac aorta, a left thoracotomy may be required. Aortic injuries are repaired with 3–0 or 4–0 sutures of polypropylene monofilament.
- **Infrahepatic vena cava.** The exposure is achieved by incision of the white line of Toldt lateral to the right colon with medial reflection of the right colon, duodenum, and if necessary right kidney. The bleeding site must be occluded by direct finger or sponge-stick pressure; vascular clamps may be used, but no attempt should be made to encircle the vessel. Venorrhaphy can be achieved with 4–0 or 5–0 monofilament vascular suture. Check for the presence of a posterior laceration: if present, it can be repaired by gentle rotation of the vena cava or from inside the lumen. In massive disruptions, a synthetic graft may be used, but more commonly the inferior vena cava is ligated. Ligation above the renal veins is not well tolerated.
- **Common or external iliac artery.** Suture repair or, if necessary, grafting is used. A synthetic graft may be used even in the presence of peritoneal soiling. In this case polytetrafluoroethylene (PTFE) is the preferred material. If gross contamination is present, consideration should be given to arterial ligation and restoration of the circulation by means of an extra-anatomical femorofemoral bypass. The internal iliac artery may be ligated with impunity.
- The exposure of the **iliac veins** is notoriously difficult and may require the division of the ipsilateral internal iliac artery or even a temporary transection of the common iliac artery. Iliac veins may be ligated with acceptable morbidity; compression stockings and limb elevation are indicated postoperatively.
- The **celiac artery**, the retropancreatic portion of the *superior mesenteric artery*, and the *inferior mesenteric artery* may be ligated. The infrapancreatic portion of the *superior mesenteric artery* should be repaired. The *superior mesenteric vein* should be repaired if possible since its ligation may cause bowel infarction, severe postoperative intestinal congestion, and intestinal varices. The *inferior mesenteric vein* may be ligated without risk.
- **Heroic attempts** at restoring flow by repairing a vessel in a patient *in extremis* are to be avoided. At times, ligation with later revascularization may be possible. A better approach is a temporary shunt across the injury with definitive grafting over the subsequent 24 hrs.

**Table 39.2.1.** Approach to traumatic retroperitoneal hematoma

| Type of hematoma | Penetrating injury | Blunt injury |
|---|---|---|
| Central (Zone I) | Explore | Explore |
| Lateral (Zone II) | Usually explore | Usually do not explore |
| Pelvic (Zone III) | Explore | Do not explore |

### Retroperitoneal Hematomas

The main issue is whether to explore a retroperitoneal hematoma discovered in the course of a trauma laparotomy.

As a general rule, all retroperitoneal hematomas in penetrating trauma should be explored, irrespective of size or location. In blunt trauma, a more selective policy can be applied, depending mainly on the location of the hematoma.

- A central abdominal location (Zone I), including the main abdominal vessels and the duodenopancreatic complex, always warrants exploration.
- Lateral hematomas (Zone II), including kidneys and retroperitoneal colonic wall, can be left alone unless they are very large, are pulsating, or are expanding.
- Blunt traumatic pelvic hematomas (Zone III) should not be explored. Breaching the intact retroperitoneum may result in the loss of the tamponade effect with catastrophic intraperitoneal hemorrhage (see Table 39.2.1).

### Management of Blunt Traumatic Pelvic Hematomas

With the exception of isolated fractures of the iliac crest, fractures involving the pelvic or obturator rings or sacrum have the potential for significant bleeding leading to shock and death. The pelvis is always imaged in severe blunt trauma, either by computed tomographic (CT) scanning (in stable patients) or by a simple anteroposterior radiograph (in unstable patients). Bleeding from a pelvic fracture arises from disrupted pelvic veins, from lacerated branches of the internal iliac arteries, and from cancellous bone, in various combinations.

In an unstable patient with a significant pelvic fracture who does not respond or responds partially to aggressive resuscitation, one must assume that the source of bleeding is pelvic in origin once an extra-abdominal source of hemorrhage has been ruled out. The first step is then to minimize the pelvic blood loss by increasing the tamponade effect of the pelvic retroperitoneum; this is best

achieved by the application of a specially designed pelvic sling (every emergency department should have one; otherwise, a sheet tightly wrapped around the iliac crests and tied might do).

This temporary stabilization of the pelvic bony fragments may result in hemodynamic improvement; if this succeeds, an abdominal CT scan may be obtained and will enable one to differentiate definitively between abdominal visceral bleeding and pelvic bleeding. The former warrants an emergency laparotomy. If the latter is present alone, a laparotomy should be avoided because it may increase the bleeding by loss of the tamponade effect. In that scenario, transfer of the patient to the angiography suite for attempts at angioembolization of pelvic arterial bleeding is the best strategy; throughout the procedure, maximal resuscitation must be pursued by the trauma team (the radiology staff, while excellent at what they do, have difficulty spelling the word "resuscitation"). If angiography facilities are not available, the application of an external pelvic fixator by the orthopedic team may be beneficial (it works best when the bleeding arises from a venous or bony source but may fail to make a difference in arterial bleeding).

A grossly unstable patient, unresponsive to resuscitation, is fit only for transfer to the OR. If pelvic bleeding alone is discovered at operation, the pelvis should be packed and the patient transferred as a last resort to the angiography room. The use of either supraumbilical diagnostic peritoneal lavage or abdominal ultrasound has been very disappointing in differentiating between intraperitoneal and pelvic bleeding in the particular setting of profound hemorrhagic shock. Mortality remains extremely high in that scenario.

## The Abbreviated Trauma Laparotomy (Damage Control)

When physiology is severely compromised, attempts at restoring anatomy are counterproductive.

In a small minority of patients, time-consuming organ repair cannot be undertaken safely when the physiological status is critically impaired. A bailout procedure consisting of essential temporary control of bleeding and contamination is the only viable option. These cases can be recognized either by a set of physiological criteria or by an anatomical pattern of injuries. In the former model, the presence of coagulopathy, hypothermia, and acidosis is and indication of impending physiological exhaustion. Each of these amplifies the other two in a vicious cycle that is aptly referred to as "the triad of death." In that scenario, a dogged determination to spend the time it takes to achieve definitive organ repair may result in the patient's demise. If the latter model is applied, the surgeon makes the decision for a bailout procedure by a flash assessment of the injury pattern. For example, an

injury to a major intra-abdominal vessel associated with a severe duodenopancreatic disruption is recognized immediately as a potential for massive blood loss should a prolonged, definitive, reconstructive procedure be undertaken. In these circumstances, there is only a place for a combination of packing, vessel shunting, tube draining, and the simplest means of preventing peritoneal contamination (by stapling or tying off with tapes the injured intestine). Abdominal closure consists of expeditious cutaneous approximation or is avoided altogether—preventing the commonly associated abdominal compartment syndrome (Chap. 40). The patient is then treated in the surgical intensive care unit, where secondary stabilization is conducted over the next 24–48 hrs. Delayed definitive organ repair (or resection) and abdominal closure are undertaken only in a patient who is hemodynamically more stable, is rewarmed, and has an improved clotting profile.

## Summary

Injured organs must be surgically repaired or resected as soon as possible. This being said, the surgeon should be able to recognize the potential for spontaneous healing of even severe visceral injuries (as in some cases of blunt trauma). Furthermore, the surgeon should know to temper enthusiasm for immediately restoring the anatomy in the face of severely impaired physiology.

# 40 The Abdominal Compartment Syndrome

Moshe Schein

*In surgery, physiology is the king, anatomy the queen; you can be the prince, but only provided you have the judgment.*

At Thanksgiving, a national holiday here in the United States, many millions of turkeys—also called "Thanksgiving birds"—are tightly stuffed with various sorts of ingredients (mine would include chickpeas, garlic, wine-soaked bread, and thyme) and served to the assembled members of American families. Granted, these large birds are stuffed postmortem, but what would happen if they were tightly stuffed alive? First, the bird would stop flying, and then gradually it would hypoventilate, collapse, and die. Of course, you could attribute the death of your stuffed avis to bad lungs, old heart, and toxins produced by the chickpeas and garlic; as a last resort, you could blame the anesthetist. The reality, though, proven by a large body of first-grade scientific evidence, is much more prosaic: **intra-abdominal hypertension (IAHT) secondary to increased intra-abdominal pressure (IAP) caused abdominal compartment syndrome (ACS).**

## Is ACS Real?

Much good evidence now supports the concept that elevated IAP or IAHT may impair physiology and organ function by producing the *ACS*. Complex, adverse physiological consequences of increased IAP develop as the pressure is transmitted to adjacent spaces and cavities, decreasing cardiac output, restricting pulmonary ventilation, diminishing renal function and visceral perfusion, and increasing cerebrospinal pressure (Table 40.1, Fig. 40.1). If you still doubt the existence of this condition, ask the anesthetist to monitor the airway pressure the next time you do a relaparotomy in a ventilated, critically ill patient with abdominal distension. You will note an immediate and dramatic fall as soon as the abdomen is reopened.

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**Table 40.1.** Physiological consequences of intra-abdominal hypertension

| | Increased | Decreased | No change |
|---|---|---|---|
| Mean blood pressure | – | – | × |
| Heart rate | × | – | – |
| Peak airway pressure | × | – | – |
| Thoracic/pleural pressure | × | – | – |
| Central venous pressure | × | – | – |
| Pulmonary capillary wedge pressure | × | – | – |
| Inferior vena cava pressure | × | – | – |
| Renal vein pressure | × | – | – |
| Systemic vascular resistance | × | – | – |
| Cardiac output | – | × | – |
| Venous return | – | × | – |
| Visceral blood flow | – | × | – |
| Gastric mucosal pH | | × | |
| Renal blood flow | – | × | – |
| Glomerular filtration rate | – | × | – |
| Cerebrospinal fluid pressure | × | | |
| Abdominal wall compliance | – | × | – |

## How Do You Measure IAP?

At the bedside, IAP is best measured through the urinary bladder catheter connected to a manometer or a pressure transducer. In fact, all you need to measure IAP is a Foley catheter: disconnect it from the urine bag; instill 100 ml saline into the bladder and elevate the disconnected catheter perpendicular to the supine patient and the patient's bed. The height of the water-urine column in the catheter is the IAP in centimeters of water (1 cm $H_2O$ = 0.735 mmHg). The level will fluctuate with the patient's respiratory cycle—up during inspiration, down during expiration—following the movements of the diaphragm. A neurogenic or small contracted bladder may render the measurements invalid. Errors can also occur if the catheter is blocked or if a pelvic hematoma selectively compresses the bladder.

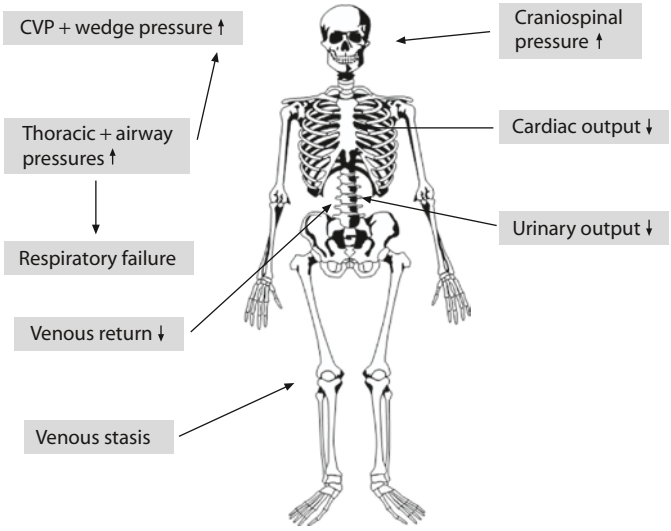


**Fig. 40.1.** The abdominal compartment syndrome

Because the Trendelenburg position (or its reverse) may affect intrabladder pressure, accurate measurements are best achieved in the supine position.

## Deleterious Consequences of Raised IAP Appear Gradually

At pressures less than 10 mmHg, cardiac output and blood pressure are normal, but hepatic arterial blood flow falls significantly; an IAP of 15 mmHg produces adverse, but spontaneously correctable, cardiovascular changes; an IAP of 20 mmHg may cause renal dysfunction and oliguria, and an increase to 40 mmHg induces anuria. In an individual patient, the effects of increased IAP are not isolated but usually superimposed on multiple underlying and coexistent factors, the most notable being hypovolemia, which aggravates the effects of increased IAP.

## Why Didn't We Notice IAHT and ACS Before?

Because you—or your mentors—did not know that this entity exists! (› Fig. 40.2). Any increase in the volume of any of the contents of the abdomen or the retroperitoneum elevates IAP. Clinically significant elevation of IAP has been observed in a variety of contexts (› Table 40.2), such as postoperative intra-abdominal hemorrhage; after complicated abdominal vascular procedures or major operations like hepatic transplantation; in association with severe abdominal

**Fig. 40.2.** "What? Abdominal compartment syndrome? Never heard of it!"

**Table 40.2.** Etiology of increased intra-abdominal pressure

| Condition | Etiology |
|---|---|
| ACUTE | |
| Spontaneous | Peritonitis, intra-abdominal abscess, ileus, intestinal obstruction, ruptured abdominal aortic aneurysm, tension pneumoperitoneum, acute pancreatitis, mesenteric venous thrombosis, fecal impaction |
| Postoperative | Postoperative peritonitis, paralytic ileus, acute gastric dilatation, intraperitoneal hemorrhage |
| Post-traumatic | Intra-/retroperitoneal bleeding, postresuscitation visceral edema |
| Iatrogenic | Laparoscopic procedures, pneumatic antishock garment, abdominal packing, reduction of a massive parietal or diaphragmatic hernia, abdominal closure under excessive tension |
| CHRONIC | Ascites, large abdominal tumor, chronic ambulatory peritoneal dialysis, pregnancy, morbid obesity |

The list cannot be considered "complete" as any increase, of any etiology, in the volume of the intra- or retroperitoneal space will increase intra-abdominal pressure

trauma accompanied by visceral swelling, hematoma, or the use of abdominal packs; severe peritonitis; necrotizing pancreatitis; the use of the pneumatic anti-shock garment; tense ascites in cirrhotic patients; or even extreme distension of

the colon (e.g., colonic pseudo-obstruction). Peritoneal insufflation during laparoscopic procedures is currently the most common (iatrogenic) cause of IAHT. Note that severe intestinal edema causing IAHT has been described following massive fluid resuscitation for **extra-abdominal** trauma. The combination of severe abdominal wall burns (producing a tight-constricting eschar) and fluid resuscitation causing visceral edema could lead to ACS in the burned patient.

Be aware that *morbid obesity* (Chap. 31) and *pregnancy* (Chap. 33) *are "chronic" forms of IAHT*; various manifestations associated with such conditions (e.g., hypertension, pre-eclampsia) are attributed to IAHT. Note that anything can cause IAHT and ACS—irrespective of the ingredients used in the "stuffing" or its flavor. The stuffing can even be composed of feces (definitely not recommended in turkeys).

An elderly lady presented with poor peripheral perfusion, blood pressure of 70/40, and respiratory rate of 36/min. Her abdomen was very distended and diffusely tender with guarding. Rectal examination revealed a large amount of soft impacted feces. Blood urea nitrogen (BUN) and creatinine levels were 30 mg% and 2 mg%, respectively. Arterial blood gases showed a metabolic acidosis with pH 7.1. Her IAP was 25 $cmH_2O$. Abdominal X-ray showed a hugely distended rectosigmoid. She survived following a decompressive laparotomy and resection of the partially ischemic and massive rectosigmoid (Fig. 40.3). So, you see: even s**t can cause ACS.

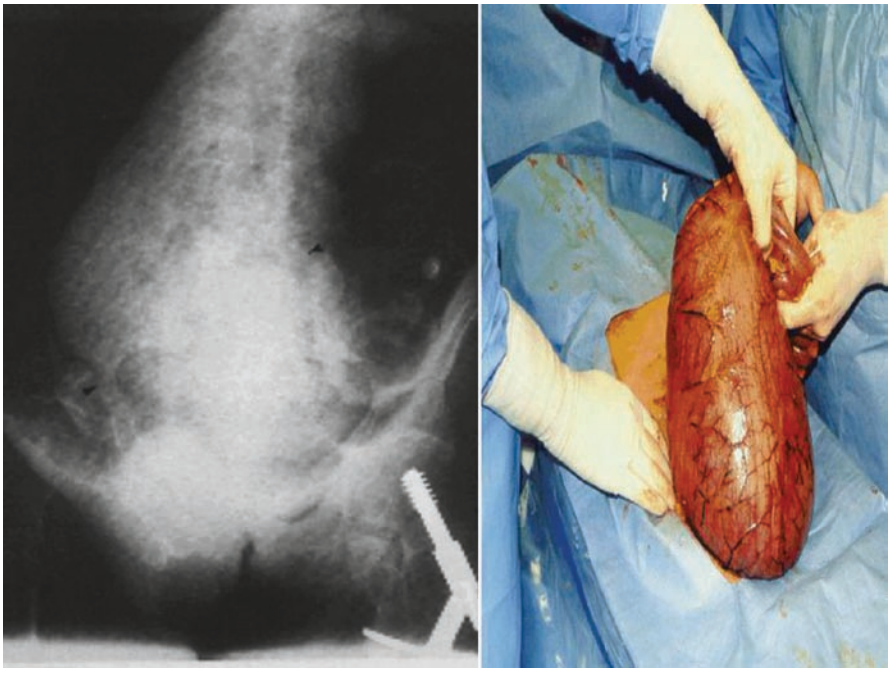

**Fig. 40.3.** Abdominal X-ray showing a massively dilated rectosigmoid and the corresponding findings at operation

Only a few years ago, we would have described this patient as suffering from "septic shock" due to "colonic ischemia." We would have attributed the cardiovascular collapse and acidosis to the consequences of endotoxemic sepsis. But today, it is clear to us that the mass effect created by the extreme dilatation of the rectum produced severe IAHT, causing cardiovascular and respiratory collapse and renal dysfunction—representing a typical ACS. This further decreased splanchnic perfusion, thus aggravating colorectal ischemia. Rectal disimpaction and abdominal decompression rapidly reversed the adverse physiological manifestations of the intra-abdominal hypertension. Being more aware that IAHT is a "real problem" and liberally measuring IAP, we are recognizing it with increasing frequency in our daily clinical practice.

### *The Mechanisms Culminating in ACS Are Usually Multiple*

The typical scenario of ACS occurs in a multiple-trauma or post-emergency laparotomy patient who receives a large volume of fluid for resuscitation, causing an increase in interstitial fluid volume. The ensuing visceral and retroperitoneal edema is aggravated by shock-induced visceral ischemia and reperfusion edema as well as by temporary mesenteric venous obstruction caused by surgical manipulation or the employment of hemostatic packs. The edematous abdominal wall is closed over the bulging abdominal contents under extreme tension.

## The Clinical Syndrome

The clinical syndrome of ACS consists of:

- Increased airway pressure
- Decreased cardiac output
- Decreased urinary output
- Abdominal distension

These abnormalities are often present despite apparently normal cardiac filling pressures because transmission of increased IAP to the thorax elevates central venous pressure (CVP), right atrial pressure, and pulmonary capillary wedge pressure. Cardiovascular, respiratory, and renal dysfunction become progressively difficult to manage unless IAP is reduced. Rarer consequences of ACS have been described, such as intestinal ischemia following laparoscopic cholecystectomy or spinal cord infarction in the setting of IAHT following perforation of a gastric ulcer.

## When Should You Consider Abdominal Decompression?

The decision to decompress the abdomen should not be taken based on isolated measurements of IAP without taking into account the whole clinical picture. Early or mild physiological abnormalities caused by IAHT can be managed by fluid administration or afterload reduction. (Note, however, that increasing cardiac filling offers only a temporary solution, and that fluid administration may in fact increase tissue edema and thus aggravate IAHT.) In patients receiving mechanical ventilation, *muscle paralysis* may decrease IAP by relaxing the abdominal wall.

Established ACS, however, mandates an emergency decompressive laparotomy, which, when performed in the well-resuscitated patient, should promptly restore normal physiology. To prevent hemodynamic decompensation during the laparotomy, intravascular volume should be restored, oxygen delivery maximized, and hypothermia and coagulation defects corrected. Following decompression, the abdominal skin and fascial edges are left open using one of the temporary abdominal closure devices (TACD) described in Chap. 52.2.

### *Prevention*

To avoid IAHT and ACS, forceful closure of the abdomen in patients having massive retroperitoneal hematoma, visceral edema, severe intra-abdominal infection, or a need for hemostatic packing should be avoided (Chap. 43). Leaving the fascia open, closing only the skin with sutures to protect the bulging viscera, is a good option! Occasionally, however, the skin closure alone may produce IAP of 50 mmHg or more. Certainly, leaving both fascia and skin unsutured offers maximal reduction in IAP but may result in fistula and evisceration. Bridging the fascial gap with a TACD circumvents most these problems (Chaps. 43, 52, and 53).

### *Would Decompression Benefit Patients with Only Moderate IAHT?*

That the "extreme" case of ACS as described necessitates an urgent abdominal decompression is obvious. But, what about a less-extreme case? Would decompression benefit a postoperative patient in whom the moderately increased IAP of 20 mmHg is compensated by appropriate fluid and ventilatory therapy? We believe that the available evidence suggests that the detrimental effects of IAHT take place long before the manifestations of ACS become clinically evident—just as nerve and muscle ischemia begins long before neuromuscular signs of the extremity compartment syndrome are evident. IAHT may cause gut mucosal acidosis at relatively low pressures long before the onset of clinical ACS. Uncorrected, it may lead to

splanchnic hypoperfusion, distant organ failure, and death. Prophylactic nonclosure of the abdomen may facilitate prevention of IAHT and reduce these complications. It seems sensible therefore that if postoperative IAHT seems likely, then delayed abdominal closure should be considered. It appears that "borderline" IAHT contributes to the overall morbidity, but in patients in whom the abdomen has already been closed, the risk-benefit ratio of abdominal decompression is not yet clear.

## Conclusion

Intra-abdominal hypertension is yet another factor to consider in the overall management of the patient needing emergency abdominal care. It may be obvious—"crying" for abdominal decompression. More commonly, however, it is relatively silent but contributing to your patient's SIRS (systemic inflammatory response syndrome), organ dysfunction, and death. So, now you know better; you know that your patient is not a "dead turkey to be stuffed." *Bon appetit!*

Be as aware of intra-abdominal hypertension as you are of arterial hypertension. It is much more common and clinically relevant than you have suspected.

[We asked Dr. Sugrue, who is a leading international authority on ACS, to comment.—The Editors]

## Invited Commentary

Michael Sugrue

The World Society of the Abdominal Compartment Syndrome (www.wsacs.org) defines ACS as sustained IAP >20 mmHg (with or without an abdominal perfusion pressure [APP] <60 mmHg) that is associated with new organ dysfunction or failure (Malbrain et al. 2006) and occurs in between 5% and 8% of intensive care patients. Not all hospitals have sophisticated transducers and monitoring equipment to measure IAP, so simple bedside monitoring can be undertaken; however, the more reliable gold standard is the modified Kron technique of instilling 25 ml into the urinary bladder, which is connected via a T piece and pressure transducer to the bedside monitor. Alternatively, commercial devices are available; simple ones are the Holtec or the Advisor. Alternatively, continuous IAP measurement can be undertaken using a three-way Foley catheter.

Recognition of ACS is increasing, although many units do not routinely measure IAP. This is now changing with the introduction of guidelines and

recommendations (Cheatham et al. 2007). The formation of the World Society of the Abdominal Compartment Syndrome and the success of the four initial world congresses will ensure greater worldwide awareness of ACS. The prevention of ACS is increasingly coming to the fore with greater emphasis on hemorrhage control rather than over zealous resuscitation—thus avoiding massive visceral and abdominal wall edema causing IAHT. Some units are reporting a reduction in secondary ACS due to fluid overload. And, prophylactic abdominal decompression remains a popular preventive option among trauma surgeons in particular (Ivatury et al. 1998).

The key to the management of both medical and surgical patients with impending ACS is treating the underlying cause, be it intra-abdominal hemorrhage or sepsis. Alternative techniques such as negative pressure and prone ventilation are unproven. In a significant number of patients, however, percutaneous drainage of intraperitoneal fluid has a role to play. Newer techniques (e.g., laparoscopic decompression of the fascia and linea alba [laparoscopic abdominal fasciotomy]) are being tried.

One of the greatest challenges, however, is managing the open abdomen. Early closure will reduce complications, particularly fistula. Currently, vacuum-assisted dressings offer the most manageable option for the open abdomen. There is increasing use of dynamic closure systems to apply "gradual" tension, which prevents further divarication of muscle mass (see Chap. 52.2).

In conclusion, the keys are prevention through timely hemorrhage control, excellence in elective and emergency abdominal surgery, and consideration for prophylactic decompression, particularly in trauma, aortic, and pancreatic patients. We must recognize the need for IAP monitoring as an adjunct to the diagnosis of ACS: no IAP, no ACS. The future will enlighten us further and provide greater understanding into the side effects of intra-abdominal hypertension since it was first described in 1865.[1]

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[1] For information about the history of abdominal compartment syndrome, access http://www.docschein.com/Compartment.html.

# 41 Abdominal Aortic Emergencies

Paul N. Rogers

*Abdominal/back pain and hypotension = a ruptured AAA unless proven otherwise. Urological and orthopedic wards are a cemetery for ruptured AAA cases.*

## Presentation

The diagnosis of a leaking abdominal aortic aneurysm (AAA) is usually not difficult to make. Typically, the patient presents with a sudden onset of acute lumbar backache, abdominal pain, and collapse associated with hypotension. On examination, the presence of a pulsatile abdominal mass confirms the diagnosis. In this situation, the patient proceeds directly to the operating room with a delay only to allow cross-matched blood to become available if the patient is stable.

### *Atypical Presentation*

Not infrequently, however, the diagnosis can be difficult to make. There may be no history of collapse, and the patient may be normotensive on admission. The only clue may be nonspecific back or abdominal pain. A pulsatile mass may not be palpable. Ruptured AAA patients are frequently obese; thinner patients tend to notice their AAA and present early for an elective repair. A leaking AAA may be mislabeled as "ureteric colic," but the absence of microscopic hematuria should alert one to the possibility that a leaking aneurysm is responsible for the symptoms. A high index of suspicion is important to prevent the diagnosis of a leaking AAA being overlooked. In appropriate individuals, particularly men in late-middle and old age, if significant and unexplained abdominal or back pain causes the patient to present acutely, abdominal aneurysms should be excluded by means of ultrasound or computed tomography (CT).

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## The Diagnostic Dilemma

A different diagnostic dilemma occurs in the patient who is known to have an aneurysm and who presents with abdominal or back pain, which may or may not be related to the aneurysm. The difficulty here is that a small, contained, "herald" leak from an aneurysm might produce pain without any hemodynamic instability. Examination in these patients may be unhelpful in that the aneurysm may not be tender. These patients are at high risk of a further bleed from the aneurysm, and this could be sudden and catastrophic. For this reason, it is important that they are identified appropriately and have an operation before a major, possibly fatal, hemorrhage occurs. The difficulty, of course, is that such a patient might easily have another cause for the symptoms, mechanical backache for example, that is unrelated to the aneurysm. Here, an operation is clearly not in the patient's best interests, particularly if his or her general health is poor. This dilemma, of operating without delay in patients who require it yet avoiding operation in those in whom it is not necessary, is a difficult one, sometimes even for experienced clinicians, to resolve. An emergency CT scan is indicated in this situation to delineate the AAA and presence of any associated leak, usually into the retroperitoneum. In general, however, in this situation it is safer to err on the side of operating on too many rather than too few patients.

## Who Should Have an Operation?

A useful rule of thumb regarding who should have an operation is that the chances of survival in a patient with a ruptured AAA are directly proportional to the blood pressure on admission. Profoundly shocked patients rarely survive; sure, they may survive the operation but usually do not leave hospital through the front door. Consequently, it has been proposed that operating on shocked ruptured AAA patients is futile and a waste of resources. Another view is that you should proceed with the operation unless the patient is clearly "agonal" or known to suffer from an incurable disease. You may be able to save the occasional patient and gain additional experience, which may help you to save the next rupture patient. These issues of philosophy of care are for the individual surgeon to resolve with his or her God, patients, and their families. A scoring system has been devised that aims to help with this decision making. The so-called Hardman criteria relate the presence of several easily determined variables to the likelihood of survival from surgery from a ruptured aneurysm.

***The Hardman Criteria (Hardman et al. 1996)***

- ✓ Age >76
- ✓ History of unconsciousness
- ✓ Hemoglobin <9.0 g/dl
- ✓ Creatinine >190 μmol/l
- ✓ Electrocardiographic evidence of ischemia

If three or more criteria are present, the mortality is 100%.
If two are present, mortality is 72%.
If one, mortality is 37%.

Perhaps not surprisingly, since these criteria were published other workers have demonstrated that it is possible to operate successfully on patients with three Hardman criteria (confirming the rule of "never say never"). Nevertheless, the criteria are a useful adjunct to the decision-making process in these patients.

## The Operation

Once the diagnosis of aortic rupture has been established or strongly suspected, the patient should be rushed to the operating theater without delay. Do not even bother with additional lines and intravenous fluids as what you pour in will pour out, and increasing the blood pressure will only increase the bleeding. Aim for stable hypotension in resuscitation.

**Preparation** ▶ "Prep and drape" (including the groins in case aorto-femoral bypass is necessary) for surgery while the anesthetic team establishes the appropriate monitoring lines. Do not allow them, however, to waste time by inserting unnecessary gimmicks such as the pulmonary arterial catheter. Anesthesia should not be induced until you are ready to make the skin incision; not infrequently, the administration of muscle relaxants at induction, and the subsequent relaxation of the abdominal wall, is sufficient to permit a further bleed from the aneurysm with an immediate hemodynamic collapse. Remember: your clamp on the aorta proximal to the aneurysm is more important than anything else.

**Incision** ▶ Open the abdomen through a long midline incision extending from the xiphisternum to a point midway between the umbilicus and the symphysis pubis. Occasionally, if the distal iliac arteries are to be approached, the

incision must be extended. In most cases, however, for the insertion of a simple aortic tube graft, an incision as described is adequate.

**Proximal control** ▶ Upon entering the peritoneal cavity, the diagnosis is immediately confirmed by the presence of a large retroperitoneal hematoma. The first priority is to obtain control of the aorta proximal to the aneurysm. In the majority of patients who are stable at this stage (with a contained retroperitoneal leak), there is time to approach the aorta above the aneurysm just below the level of the renal arteries. In patients who are unstable, rapid control of aortic bleeding may be obtained by approaching the aorta just under the diaphragm and temporarily applying a clamp there until the infrarenal aorta can be dissected.

**Subdiaphragmatic aortic control** ▶ Remember how you do truncal vagotomy? Of course, you do not! So, pay attention. Incise the phrenoesophageal ligament overlying the esophagus (feel the nasogastric tube underneath). With your index finger, bluntly mobilize the esophagus to the right; forget about hemostasis at this stage. Now, feel the aorta pulsating to the left of the esophagus, dissect with your index finger on both sides of the aorta until you feel the spine. Apply a straight aortic clamp, pushing it "onto" the spine. Leave a few packs to provide hemostasis and proceed as discussed next.

**Infrarenal aortic control** ▶ Returning to the matter of isolation of the aortic neck, note that the main principle to be observed is to avoid disturbing the retroperitoneal hematoma while gaining control of the proximal aorta. Once you enter the retroperitoneum at the neck's level, dissect bluntly using your finger or the tip of the suction apparatus to identify and isolate the neck of the aneurysm. Once the neck is identified, carry on down both sides of the aorta until the vertebral bodies are reached. Do not attempt to encircle the aorta with a tape. Apply a straight aortic clamp in an anteroposterior direction with the tips of the jaws of the clamp resting against the vertebral bodies. Placement of this clamp is facilitated by placing the index and middle fingers of your nondominant hand on either side of the aorta so that the vertebral bodies can be palpated. The jaws of the open clamp are then slid along the backs of the fingers until the clamp lies in the appropriate position. Now, you can remove the subdiaphragmatic clamp.

**Juxtarenal neck** ▶ Occasionally, the aneurysm extends close to the origin of the renal arteries. If this is the case, then the neck of the aneurysm will be obscured by the left renal vein, which may be stretched anteriorly. Care must be taken that the vein is not damaged. It may be divided to facilitate access to the aneurysm neck. This is done by very gently mobilizing the vein from the underlying aorta. It should be ligated securely as close to the vena cava as prudence

permits. If this is done, then the vein may be ligated with impunity, and the kidney will not be endangered because collateral venous drainage will take place via the adrenal and gonadal anastomoses. How do you know that effective proximal control has been achieved? Simple—the retroperitoneal hematoma stops pulsating. If it pulsates, your clamp is not properly placed. Reapply it!

**Distal control** ▶ The next part of the dissection to identify the common iliac arteries is often more difficult. Under normal circumstances, the pelvis is the site of accumulation of much of the retroperitoneal hematoma, and the iliac arteries are buried within this. The arteries are difficult to locate not only because they are buried in hematoma, but also because with the aorta clamped proximally there is no pulsation to guide the operator. In most patients, however, the presence of atheroma in the vessels makes palpation in the depths of the hematoma possible. Again, the use of the suction apparatus facilitates isolation of the iliac vessels. Otherwise, dig with your fingers within the hematoma and "fish" the iliacs out. As with the aorta, no attempt should be made to encircle the iliac vessels with tapes. This invariably produces damage to the iliac veins, which is a disaster. It is sufficient to clear the anterior and lateral aspects of the iliac vessels and apply clamps in an anteroposterior manner as before.

**An alternative—balloon control** ▶ After proximal control has been achieved and when the iliacs are immersed within a huge hematoma, you may also rapidly open the aneurysm sac and shove a Foley or large Fogarty catheter into each iliac artery, inflating the balloons to produce temporary distal control.

**Aortic replacement** ▶ Once the proximal and distal arterial tree is controlled, incise the aneurysm sac in a longitudinal fashion. Evacuate the clot and control back-bleeding from any patent lumbar arteries and the inferior mesenteric artery with sutures within the aneurysm sac. A small self-retaining retractor placed within the aneurysm sac to retract its cut edges facilitates this and the next few stages of the procedure. The proportion of patients in whom aortic replacement with a simple tube graft can be achieved varies widely from surgeon to surgeon and center to center. We believe that in the majority of patients insertion of a tube graft can be achieved quite satisfactorily. The advantages of this are that limitation of dissection in the pelvis minimizes the risk of damage to the iliac veins and damage to the autonomic nerves in the pelvis. Furthermore, there seems little point in extending the length of what is already a challenging operation by inserting a bifurcation graft unnecessarily. Obviously, there are circumstances when a tube graft is not acceptable, namely, when the patient has occlusive aortoiliac disease, when the iliac arteries are also significantly aneurysmal, or in some situations when the bifurcation is widely splayed so that the orifices of the common iliac arteries are far apart.

**Take care when fashioning the aorta to receive the graft**. The longitudinal incision in the aortic sac should be terminated at both ends by a transverse incision so that the incision becomes T shaped at each end. The limbs of the "T" at either end should not extend more than 50% of the circumference of the normal aorta.

**Suture the graft** in place using monofilament material so that a parachute technique can be used. This allows you to visualize clearly the placement of the individual posterior sutures. Large bites of the posterior aortic wall should be taken because the tissues in this situation are often very poor. Furthermore, leaks that occur after completion of the anastomosis are notoriously difficult to repair if they are situated at the back wall. Once the upper anastomosis has been completed, a clamp is applied to the graft just below the anastomosis, and the clamp on the aorta is then released. Assuming there are no significant leaks at the upper end, attention is turned to the distal anastomosis. This is completed in a similar fashion to the proximal anastomosis. Back-bleeding from the iliac vessels should be checked before the distal anastomosis is completed. Likewise, the graft should be flushed with saline and one or two "strokes" of the patient's own cardiac output to clear it of thrombotic junk. If there is no back-bleeding, it may be necessary to pass balloon embolectomy catheters into the iliac systems to check that there has been no intravascular thrombus formation. Once the distal anastomosis has been completed and found to be secure, the iliac clamps should be released individually, allowing time for any hypotension to recover before the second clamp is removed. The anesthesia team will appreciate a warning from you that the time is approaching for removal of the clamps, allowing them to be well ahead with fluid replacement. Inadequate fluid replacement at this stage will result in significant hypotension when the iliac clamps are released.

**A word about heparin** ▶ It is clearly not sensible to administer systemic heparin prior to cross-clamping in patients who are bleeding to death from an aortic rupture. In patients in whom surgery has been carried out for suspected rupture, however, and in whom no rupture is found at operation, systemic heparinization according to the surgeon's normal practice should be carried out. It is permissible, however, to heparinize locally the iliac vessels once the aneurysm sac has been opened and back-bleeding from the small vessels has been controlled. Heparinized saline may be flushed down each of the iliac vessels in turn before reapplying the iliac cross-clamps. No consensus on the need for this practice has been reached, and in the vast majority of patients it appears to be unnecessary.

**Abdominal closure** ▶ The large retroperitoneal hematoma and visceral swelling resulting from shock, resuscitation, reperfusion, and exposure commonly produce severe intra-abdominal hypertension, which becomes manifest after closure of the abdomen. Rather than closing under excessive tension, use temporary abdominal closure as discussed in Chaps. 40, 43, 52.2 and come

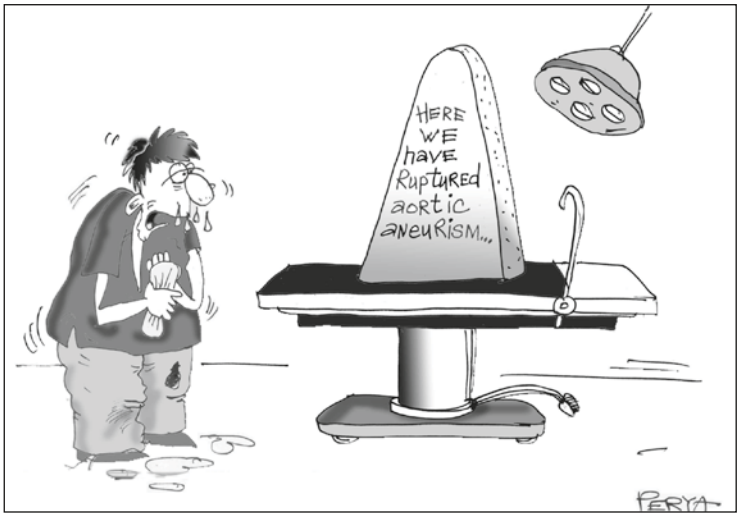


**Fig. 41.1.** AAA: common outcome

back to close the abdomen later. Avoidance of abdominal compartment syndrome is crucial in these physiologically compromised patients, in whom any further derangement may be the straw that breaks the camel's back.

**In emergency operations for AAA, simplicity of the operation is a key for survival: rapid and atraumatic control, avoidance of injury to large veins, tube graft, minimal blood loss, and rapid surgery.**

Many patients who reach the operating table will survive the operation only to die in its aftermath, usually from associated medical illnesses such as myocardial infarction. A successful outcome therefore requires excellent postoperative intensive care unit (ICU) care as well as competent surgery. The operation is only half the battle.

In ruptured AAA, the operation is commonly the beginning of the end—the end arriving postoperatively (Fig. 41.1).

## Endovascular Repair

As aortic stent grafting has become an established treatment for AAA in the elective patient, interest has developed in the use of the same techniques in patients with ruptured AAA in the hope of reducing the operative mortality from the current 40–50%. Emergency endovascular aneurysm repair (eEVAR) is now confined to a few major centers but may become more commonplace as familiarity with the necessary arrangements increases. The limitations of this treatment are the need for pre-op CT, an expensive stock of modular prostheses, and immediate

availability of appropriately skilled surgeons and radiologists. The patient needs to be stable enough to cope with the delay to obtain CT images that are required to obtain the measurements for the stent graft. This procedure is appropriate for only a minority of patients at present, but it is hoped that more will be suitable in the future as techniques improve.

## Free Intraperitoneal Hemorrhage (see › Table 11.1)

Most AAA patients with a free intraperitoneal rupture will not reach surgery. In the few who do, rapid proximal control is even more crucial. Other causes of nontraumatic intraperitoneal bleeding are rare and include **ruptured visceral artery aneurysms**. If this is encountered, then the commonsense principle of first stopping the bleeding by suture ligation or packing is followed by an assessment of the need for revascularization. Splenic artery aneurysms are the most common of these lesions; they occur most often in women, and rupture is a disaster particularly associated with pregnancy. When exposure and thus proximal and distal control are difficult, do not forget the option of **endoaneurysmorrhaphy:** open the sac of the aneurysm, control the bleeding with finger pressure or balloon catheters, and suture the proximal and distal openings from within. Currently, more and more of such aneurysms are diagnosed on CT and managed angiographically by the radiologist—in stable patients, of course.

### *Aortic Occlusion*

The emergency of aortic occlusion is characterized by acute ischemia of the legs with mottling of the skin of the lower trunk. It occurs for three reasons:

**Saddle embolus** ▶ A large clot originating from the heart occludes the aortic bifurcation. The patient most likely will have signs of atrial fibrillation or a recent history of acute myocardial infarction.

**Aortic thrombosis** ▶ The patient probably has a history of pre-existing arterial disease suggestive of aortoiliac involvement. Occasionally, this disaster will occur unannounced in a patient who is desperately ill for some other reason. Extreme dehydration, for example, may cause "sludging" of major vessels if there has been some preexisting atheroma. Malignancy may produce intra-arterial thrombosis.

**Aortic dissection** ▶ Suspect this if there is a history of interscapular pain associated with obvious hypertension. Look for evidence of other pulse deficits or signs of visceral ischemia suggesting involvement of other aortic branches.

### *Management*

Management depends on the etiology and the presence of any relevant underlying pathology. Embolism may often be dealt with easily by bilateral transfemoral embolectomy under local anesthetic. Thrombosis on pre-existing atheroma is a more difficult problem. Catheter thrombectomy is unlikely to be successful in either the short or the long term. If the patient is very fit (unlikely), aortofemoral bypass may be indicated. More likely, an extra-anatomic bypass (axillofemoral) may be feasible, always assuming that any underlying illness is not likely to cause the patient's demise in the immediate future. Often, these patients are not fit for any intervention, and the aortic thrombosis is an indication that the end is near.

Aortic dissection is a complex illness, and its management is variable. The mainstay is control of hypertension and relief of major vessel occlusion by endovascular "fenestration" of the dissection. The details of this therapy are beyond the scope of this book.

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# 42 Abdominal Drainage

Moshe Schein · Paul N. Rogers

*The more imperfect the technique of the surgeon the greater the necessity for drainage.* (William Stewart Halsted, 1852–1922)

The history of abdominal drainage is as old as the history of surgery. However, abdominal drainage was always a subject of controversy, practiced in confusion and subjected to local dogmas. Hence, 100 years ago there were ardent enthusiasts of drainage, like Robert Lawson Tait (1845–1899), who stated: "When in doubt drain!" There were the skeptics, like Yates (1905), who understood that "Drainage of the general peritoneal cavity is a physical and physiological impossibility." And, as always, there were the undecided, such as Joseph Price (1853–1911): "There are those who ardently advocate it, there are those who in great part reject it, there are those who are lukewarm concerning it, and finally, some who, without convictions, are either for or against it … as chance or whim, not logic may determine."

A century has passed, during which operative surgery and supporting care have progressed astonishingly, but what about drainage? Who should we drain after an emergency operation for abdominal contamination and infection tonight?

Percutaneous drainage of primary and postoperative abdominal abscesses and collections is discussed in Chap. 49.

## Classification of drainage

Surgeons may drain the abdomen for the following reasons:

**Therapeutic:**

- *To provide* egress for established intra-abdominal contamination or infection (e.g., periappendicular abscess, diffuse fecal peritonitis)
- *To control* a source of infection that cannot be controlled by other means by creating a "controlled" external fistula (e.g., for a leaking duodenal suture line)

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**Prophylactic:**

- *To prevent* recurrent infection (e.g., hoping that by evacuating residual serum and blood it will prevent abscess formation)
- *To control* "prospective" or "expected" leakage from a suture line (e.g., drainage of a colonic anastomosis, duodenal closure, or cystic duct closure)
- *To warn about* complications (believing that drains would sound the warning bell about postoperative bleeding or anastomotic leakage)

But, rather than dwell on the subject using rigid classifications, let us deal with it through the eyes of a general surgeon: **what is the current practice, and what should the current practice be concerning drainage after common abdominal procedures?**

## What Is the "Current Practice"?

The published literature is not much help when exploring the prevalence of abdominal drainage after emergency surgery. Therefore, we polled the opinions of general surgeons who are members of SURGINET (an international surgical discussion forum on the Internet) on their approach to abdominal drainage.

## Common Situations During Which Drains May Be Used

**Question:** *Should you place a drain after an appendectomy for gangrenous appendicitis?* This is not "simple" or "phlegmonous" appendicitis but gangrenous appendicitis: the appendix is black; there is some fluid around it or in the pelvis but no frank pus (Chap. 28).

**Answer:** Only 2% of responders would leave a drain in this situation.

**Question:** *Should you place a drain after an appendectomy for perforated appendicitis with local pus formation?* So, now the appendix is perforated, you remove it and suck out the pus floating around it. Occasionally, you break the adhesions formed by omentum or small bowel and expose a small abscess; when you insert the suction into the pelvis, you evacuate a few milliliters of pus. The procedure you did could have been open or laparoscopic (Chap. 28).

**Answer:** Only 20% of responders would consider drainage in this situation.

**Question:** *Would you place a drain after an appendectomy for perforated appendicitis with diffuse pus formation?* Here, we deal with one of those advanced, neglected cases, in which the perforated appendix is associated with pus "everywhere"—in the pelvis, right paracolic gutter, and even the upper abdomen (› Chap. 28).

**Answer:** Again, 80% of responders would not use a drain, but there was a geographical pattern: while almost none of the North American and Latin surgeons would drain, many of the surgeons in Asia would. This difference has to do with how surgeons view the value for drainage in diffuse peritonitis; see separate section for discussion.

## Drainage in Acute Appendicitis (› Chap. 28)

As elsewhere in this book, we are not going to burden you with a detailed review of the literature available regarding drainage in acute appendicitis short of mentioning one recent superb meta-analysis of such studies by Petrowsky et al. (2004), which concluded: "Drainage did not reduce postoperative complications and even appeared harmful in respect to the development of fecal fistula (the development of fecal fistula was observed only in drained patients) … drains should be avoided in any stage of appendicitis." We agree: drainage after appendectomy for phlegmonous or gangrenous appendicitis is unnecessary. It seems that most surgeons understand this. But, what about perforated appendicitis with *local pus* formation? Even though the literature cannot support—and even condemns—drainage in such situations, a fifth of our responders would leave a drain. "Formed" or "noncollapsible" abscesses are considered by many to be a good indication for drainage, and this is probably why some surgeons feel compelled to leave a drain in any collection of pus. But, the abscesses associated with perforated appendicitis are never noncollapsible; after you break down the walls and evacuate the pus, the potential space for the abscess is filled up by adjacent bowel, mesentery, and omentum. So, the source of infection has been removed, the peritoneum has been cleansed by "peritoneal toilet," now let the superb peritoneal defense mechanisms, supported by a short course of systemic antibiotics, complete the eradication of bacteria without being disturbed by a foreign body (i.e., drain).

Insecure closure of the appendix stump as a justification for drainage sounds anachronistic: "secure" closure is possible (even in the rare event when the appendix is perforated at its base) by including in the suture or stapler line a "disk" of adjacent cecal wall. Almost a quarter of our responders would use drains if the appendicitis is associated with *diffuse peritonitis*; but, as we discuss later in this chapter, those are the people who advocate drainage in generalized intra-abdominal infection, and drainage in this situation—*after the source control of infection has been achieved*—is an exercise in futility.

**Question:** *Would you place a drain following an open or laparoscopic cholecystectomy for severe acute cholecystitis?* Now, you are performing a "difficult" laparoscopic cholecystectomy on advanced acute cholecystitis. The dissection is not easy; it is time consuming and associated with irritating ooze from the liver. Or, perhaps you are forced to convert to an open procedure to complete the procedure. Would you leave a drain in the gallbladder bed or below the liver (› Chap. 20.1)?

**Answer:** A third of the responders would leave a drain.

## Drainage After Cholecystectomy for Acute Cholecystitis (› Chap. 20.1)

Based on a large body of data showing no advantage whatsoever for drainage, toward the end of the open cholecystectomy era routine drainage—once a holy cow of gallbladder surgery—was disappearing from many centers. But, if routine drainage is not beneficial in open cholecystectomy, why should it be in the laparoscopic one?

That most postcholecystectomy collections, whether composed of bile, serum, or blood, remain asymptomatic and are self-absorbed by the peritoneum was well known from ultrasonographic studies during the open cholecystectomy era. However, drains are much more effective in draining bile than evacuating feces or pus. Thus, it would be reasonable to leave a drain if the surgeon has a reason to worry about an unsolved or potential bile leak; for example, if the cystic duct opening cannot securely be controlled in subtotal cholecystectomy; bile staining in the lavage fluid or in the gallbladder bed (hinting at the possibility that a duct of Luschka has been missed), or what appears to be a nonperfect closure of the cystic duct for whatever reason. So, most patients do not need a drain, but if you are worried about the possibility of bile leak, leave a drain! Most drains produce almost nothing; only very rarely would the prophylactic drain become therapeutic by draining a large and persisting amount of bile. It is very important that drains with such hazy indications are removed as soon as possible. A dry drain after 24 hrs indicates that it has served its limited role. Lastly, Howard Kelly (1858–1943) said that, "Drainage is a confession of imperfect surgery." Do not confirm this statement in your practice; it may be better to convert to an open procedure and safely suture an ultrashort cystic duct than rely on faulty clip closure and a drain.

**Question:** *Would you place a drain following repair of a perforated peptic ulcer with an omental patch?* You have just repaired a perforated duodenal ulcer with a patch of omentum. Would you leave a drain (› Chap. 18)?

**Answer:** Eighty percent of the responders would not.

## Drainage After Omentopexy for Perforated Ulcer (Chap. 18)

The literature dealing specifically with drainage after omentopexy for perforated ulcer is scanty but does not support drainage. Omental patch repair, if correctly performed and tested (Chap. 18), should be leakproof. In addition, the presence of drains when a leak occurs is usually not a lifesaver.[1] Futile reliance on the drain when a leak develops simply postpones lifesaving reoperation and hastens death.

What about laparoscopic omental patch repair—an increasingly popular procedure: should it change the (non)indication for drainage? With leaks after omentopexy being so rare and large series comparing open to laparoscopic repair so scanty, it is difficult to appreciate whether leaks are more common after laparoscopic repairs. However, those of us used to open omentopexy should be alarmed to see the reported leakage following laparoscopic repairs. It may be that the "learning curves," the inability to *feel* the tension placed on the sutures to tie down the patch, or the reliance on suture closure rather than using the omentum make the laparoscopic approach more prone to leakage. But, would the drain help to avoid the ensuing disaster? We doubt it (Chap. 18). So, if you know how to do a proper and safe omental repair, draining it would be superfluous. If you are learning to do a laparoscopic repair (with the declining incidence of peptic ulceration, you may never reach the top of the learning curve), you may want to leave a drain. It will not avoid the need for reoperation should leakage develop, but it may warn you early that this is the case. On the other hand, a well-timed contrast study (with or without computed tomography [CT]) would provide you with more information than the often poorly placed and nonproductive drain.

**Question:** *Would you place a drain following a Hartmann procedure for perforated sigmoid diverticulitis or cancer?* Would you place a drain following a colectomy and primary anastomosis for perforated sigmoid diverticulitis or cancer (Chap. 26)?

**Answer:** These two questions, about drainage after emergency resection of perforated sigmoid colon without or with primary anastomosis can be discussed together. In both situations, *source control has been achieved by the colectomy*; thus, the rationale for drainage would be "therapeutic" (to help treat the associated intraperitoneal infection) or "prophylactic" (to prevent collections or to "control" potential leakage from a suture line, e.g., rectal stump closure). About two-thirds of responders to both questions would not drain routinely.

[1] A "side" leak from the duodenum is a very serious complication, almost impossible to control with simple drainage alone; instead, to improve chances of survival a reoperation is required to stop the leak (e.g., Billroth II gastrectomy) or at least convert the "side" duodenal fistula to the more manageable "end" duodenal fistula (e.g., gastrojejunostomy plus tube duodenostomy or "duodenal exclusion"—closure of the pylorus and gastrojejunostomy).

## Drainage After Emergency Left Colon Resection With or Without Anastomosis (› Chap. 26)

The topic of drainage after colonic resection has been subjected to intensive debate for the last 30 years; proponents claim that drains would avoid reoperation if anastomotic leaks develop, while critics contend that drains actually contribute to leaks. It would be difficult to improve on the review and meta-analysis by Petrowsky et al. (2004), which denied any benefits to drainage. Even the usually overly cautious Cochrane Review concluded that "there is insufficient evidence showing that routine drainage after colorectal anastomoses prevents anastomotic and other complications."

**The reasons given by those in favor of drains are varied:**

- The first is to help combat residual, or prevent recurrent, intra-abdominal infection by draining the pericolic abscess found and already drained during operation or by removing secretions. The futility of peritoneal drainage in achieving such goals has been discussed in this chapter (see acute appendicitis) and needs to be re-emphasized (see next question).
- The second is to drain the anastomosis should it leak. But, surely high-risk, leak-prone anastomoses should not be constructed in the emergency situation anyway; furthermore, as the literature points out, drains do not help much if leakage does develop—to say nothing about the false sense of security they tend to provide.
- The third reason given is to provide drainage to the rectal closure (Hartmann's pouch) should it leak. But, a solid stapler-or hand-closure of the healthy rectum away from the colonic inflammation should provide a leakproof closure. When, however, the closure is deemed "too difficult," then the rectal stump should be left partially open (around a tube) as advocated by the late John Goligher of Leeds. In any event, only a pathological optimist could hope that feces will climb up the drain and out of the pelvis, that is, if the drain is not already clogged by fibrin, clots, or feces. In conclusion: drains after emergency colonic resection are a waste of time!

**Question.** *Would you drain the peritoneum in generalized peritonitis (> Chap. 12)?*

**Answer:** Only about a third of responders would drain the peritoneal cavity in generalized peritonitis.

## Drainage in Generalized Peritonitis (› Chap. 12)

No comparative studies of drainage versus nondrainage in patients with diffuse peritonitis have ever been conducted because the futility of drainage in this situation was perceived long ago by experts in surgical infections. The modern view, endorsed by the Surgical Infection Society, maintains that:

"It is impossible to drain the peritoneal cavity in patients with diffuse peritonitis. Therefore, the use of drains in these patients is not indicated unless: (a) the drain is to be used for postoperative lavage; (b) the drain is placed into a well defined abscess cavity, (c) the drain is used to establish a controlled fistula."

We recall, when we were junior residents, postoperative patients with multiple rubber drains sticking out of each and every quadrant of their distended bellies. Those drains produced some old blood or perhaps a little pus or foul-smelling fluid. Then, the patient would die with the death blamed on "pneumonia." How stupid we were—believing that these drains were useful. We gradually understood how worthless they were: all intraperitoneal drains seal off by adjacent tissue within 24–48 hrs unless "perfused" by liquid effluent such as bile. So, in peritonitis, if you use a suction drain it drains almost nothing, and if you leave a wicking rubber drain (e.g., Penrose, "corrugated"), the drain simply drains the infected tract it has created.

The *only* indication to use a drain in general peritonitis is to control an uncontrollable source of infection such as a leaking duodenal suture line or a leaking gastroesophegal anastomosis. As pointed out, we are skeptical about the terms *well-defined* or *formed* abscess as an indication for peritoneal drainage. Such "abscesses" are pus collections that are part of the spectrum of peritonitis; after evacuation, they should be treated like the rest of the infected peritoneum. Let peritoneal defenses and antibiotics do the job. **In conclusion,** drains in diffuse peritonitis are senseless. Recurrent or persistent intra-abdominal infection, however, often develop and may need percutaneous drainage (Chap. 49), or a reoperation (Chap. 52). Drains will not change this.

**Question:** *In which situations would you always drain?*

**Answer:** Not many data are available to support any "scientific" opinion, but here are the situations considered by experienced surgeons as "obligatory for drainage":

- **High probability of leakage of bile or pancreatic juice.** This was the number one indication and rightly so. Bile and pancreatic juice are well collected and evacuated by drains. A drain placed for biliary or pancreatic leak may be lifesaving and curative.
- **Established pus-containing abscess.** This was the number two indication, showing that many surgeons believe that a well-formed collection of pus deserves a drain. Many responders emphasized the term *noncollapsible abscess* or *thick-walled abscess* as an indication for drains, but we wonder, does one really find such an animal within the abdomen?
- **Not satisfied with "source control."** This was the number three indication; it overlaps with other indications such as bile leak, urinary leak, or the impossibility of exteriorizing a leaking proximal jejunum or duodenum.

- **Difficult duodenal suture line.** The "difficult" or leak-prone duodenal stump after Billroth II gastrectomy is another reasonable indication for prophylactic drainage. The retroperitoneal duodenum is more susceptible to leakage; thus, draining it would make sense (e.g., after duodenotomy to control hemorrhage following endoscopic retrograde cholangiopancreatography [ERCP] and sphincterotomy. Chap. 30).
- **Other indications.** Prophylactic drainage when **leakage of urine** is likely is another good indication, as is drainage of **esophageal suture lines** (Chap. 15). About drainage for **expected bleeding**, it has been said: "If you have to use drains to take care of postoperative hemorrhage, then you did not finish the operation." In most cases for which you leave drains for bleeding or oozing, they are unnecessary and produce little; they also produce little when severe bleeding develops—showing only the tip of the iceberg.

**Question:** *Which type of drain do you use?*

**Answer:** The responders came up with a potpourri of drains. Sixty percent preferred "active" drains. While North American surgeons use predominantly "active" suction drainage (e.g., Jackson Pratt [JP] drain); many other prefer "passive" drains, whether round (hollow) or flat (e.g., Penrose or the corrugated rubber). But, which drains are best?

## The "Optimal" Drain

Preferably, all drains should be soft and malleable to minimize the *real* dangers of pressure necrosis and erosion of bowel and blood vessels. Passive drains work by capillary action, gravity, or overflow caused by slight pressure differences. *Active* drains are connected to a source of suction. *Passive* drains are considered to be an "open system," proven to be associated with contamination of the drain tract by retrograde spread of skin bacteria ("Drains drain both ways."). Theoretically, applying a sterile colostomy bag over a drain site should convert the open system to a closed one, but we doubt that this remains "closed" for more than a day. Whether, as some claim, passive drains are relatively inefficient in the upper abdomen because of the negative inward sucking pressures generated during respiration is controversial. Active drains tend to be clogged by tissue or clots, which are "sucked in"—the higher the sucking pressure, the more prone to blockage the drain is. "Sump" suction drains (double-lumen system) are more resistant to blockage but usually are of rigid construction and thus not considered safe for a prolonged stay in the peritoneal cavity. Evidently, the larger the drain, the wider the exit opening in the skin—the more effective is the drainage but also the more it is prone to complications.

**But, practically:**

- The flat and soft active JP is the only intraperitoneal drain that we use these days in "routine" practice, usually for the occasional case of difficult cholecystectomy.

**Table 42.1.** Complications of intraperitoneal drains

| Complication | Complication |
|---|---|
| Drain "fever" | Failure to retrieve (caught by fascial sutures, torn, or knotted) |
| Drain tract infection | "Lost" drain: migration into the abdomen or breakage |
| Drain tract hernia | Contamination of sterile tissues |
| Drain tract bleeding | Prevention of healing of fistulas |
| Intestinal obstruction | |
| Erosion of bowel | |
| Erosion of vessels | |

This is the drain we would use for indications such as a potential duodenal or pancreatic fistula.

- If you are one of those who drain peritonitis, remember that your suction drain will be plugged with fibrin and pus within a few hours, and your open passive drain would serve mostly as a one-way *autobahn* for skin bacteria.
- For those who place drains adjacent to *colonic anastomoses*, do you really believe that suction drains will evacuate feces? To form a channel capable of transferring fecal material to the outside, one has to use a large passive (e.g., corrugated) drain through a generous, two-finger, opening in the skin and abdominal wall. But by doing so, we would go back to the old days of drain site hernias, intestinal obstruction, bleeding, and drain site abscess formation.

For a list of complications of drains, look at Table 42.1. These complications are *real*; some are rare, but we have experienced each of them in the dark ages of excessive drainage. Such complications can be prevented by correct placement and management of drains (see Table 42.2) or, better, avoiding drains when not indicated.

## Regional Differences in Practice

From the international feedback we received, this trend is obvious: North American surgeons tend to be abandoning drainage for most indications, while surgeons in Asia and eastern Europe still seem to be enthusiastic about drainage. Such differences are particularly notable concerning drain placement in diffuse intra-abdominal infections and emergency colonic surgery. But, why do North American,

**Table 42.2.** The placement and management of drains

| |
|---|
| **Insertion** |
| Choose a suitable drain for the specific job but in general go with the softest and smallest |
| Place drain carefully in the desired region, trim it to remove excessive length but leave some "slack" |
| Place it away from bowel wall or vessels |
| Try to bring omentum between the drain and vital structures to prevent erosion |
| Bring drain out through the skin, away from the main wound, to prevent wound infection |
| Plan the shortest tract possible and, depending on the indication for drainage and type of drain, try to exit it in a dependent location |
| When closing the main wound, be careful not to catch the adjacent drain with your fascial sutures |
| Secure drain to skin with suture and tape |
| **Management** |
| Use a "closed" system whenever possible |
| Use a low suction to prevent sucking adjacent tissue into drain's holes |
| To keep small-caliber tube drains patent, they can be flushed twice daily with small amounts of saline under sterile conditions |
| When a fistula is established (e.g., biliary), suction can be disconnected and drain connected to a dependent bag, draining on gravity |
| Be careful that the drain's tip is not abutting the visceral defect it is draining—this would prevent the closure of the defect: check for drain position with a sinogram |
| **Removal** |
| Remove as soon as drain not productive or seems to have performed its prophylactic task |
| Long-term drains should be removed in stages to prevent abscess formation in the deep tract |
| Removal and shortening of drains could be guided (selectively) with sinograms or CTs |
| When shortening the drain, refix it to the skin to prevent proximal migration |

western European, and South American surgeons tend to rely less on drains? Such a shift in habits has surely occurred gradually and is due to multiple factors:

- With improved surgical techniques and antibiotic administration and better imaging, results of emergency abdominal procedures were improving. Thus, surgeons were noticing fewer complications that could have been allegedly prevented

by drains. This provided surgeons with a new sense of confidence: why should they leave drains if the drains seem mostly unnecessary?

- Readily available CT scanning added to the surgeons' confidence. Now, the mysterious postoperative abdominal cavity is no longer a black box. We do not need a drain to warn us that there is an abscess; we can see it on the CT.
- The immense success of image-guided percutaneous drainage of intra-abdominal collections and abscesses has obviously added to that confidence. And, it also taught us much about the methodology of drainage itself—that you do not need huge tubes, for many days, to get rid of an abscess. The elaborate rituals surrounding management of drains were evaporating as well.

So, modern surgeons found out that they do not need drains to "prevent or treat" persistent or recurrent infection after, say, perforated appendicitis. They learned that most patients would do well with source control (appendectomy) and antibiotics. And, if not, they would CT scan the patients and if necessary drain whatever was there under CT guidance.

## What Is Behind the Persisting Enthusiasm for Drains in Asia and Eastern Europe?

Is the persisting enthusiasm for drains in Asia and eastern Europe because the relative unavailability of postoperative CT in the "developing countries" makes the surgeons unable to gather the confidence to omit drains? Or, are the surgeons more forcefully subjected to local dogmas, entrenched by strict discipline? It seems so. It was during the mid-1980s that we abandoned routine drainage for the conditions discussed. At that time, we did not have CT and percutaneous drainage to bail us out, but we understood then what surgeons should understand today—that with CT or without CT, most drains are unnecessary and counterproductive.

Let us then repeat William Stewart Halsted's motto: "No drainage is better than the ignorant employment of it."

## Conclusions

The use of routine drainage in contaminated and infected abdominal surgery is declining but still practiced in some regions of the world. Drains should be used very selectively, when their placement is the only way to control the source of infection; to provide escape for highly predicted leaking fluids (bile, pancreatic juice, urine); to drain a noncollapsible abscess (a rare animal); or to drain, for

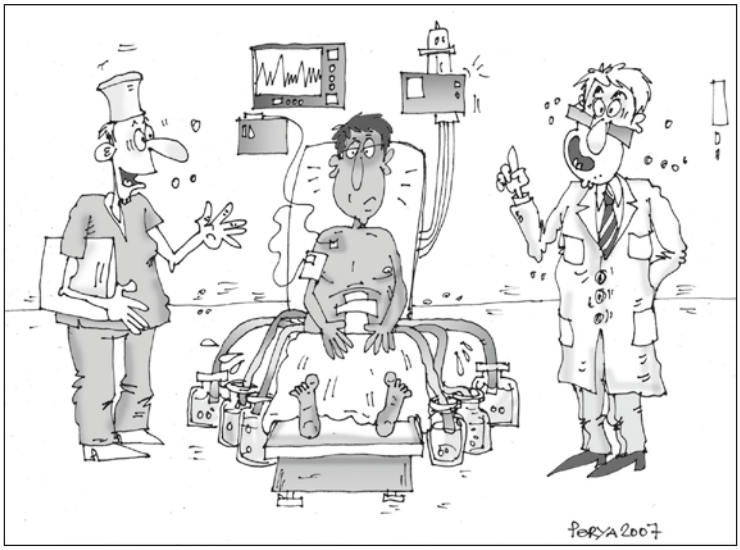


**Fig. 42.1.** Confused resident: "Boss, he's still sick." Old-fashioned surgeon: "Perhaps we should have placed more drains."

short duration, a very oozy surface (we are not sure about this last "indication"!). Prophylactic drainage of the general peritoneal cavity is senseless (Fig. 42.1), while drainage of an intestinal anastomosis may be dangerous.

> "Although more than five million surgical drains are used each year in the United States, their effectiveness, therapeutic indications, and efficiency remains an unsolved controversy." (J.P. Moss)

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# 43 Abdominal Closure

Moshe Schein

*Big bites, with a continuous monofilament suture and—above all—avoiding tension. This is how to avoid dehiscence and herniation.*

Finally, it is time to "get the hell out of here." You have been working all night, and it is tempting to finish hastily. Impatience, however, is inadvisable since correct abdominal closure protects the patient from abdominal wound dehiscence (and later on from the development of a hernia) and you from great humiliation ("everybody knows"). Yes, you are tired, but before closing, stop and think; ask your assistants: "Did we forget to do anything?" See the checklist in Chap. 44.

Generally, an abdominal closure fails because of poor quality of the tissues, increased intra-abdominal pressure, faulty technique, or a combination of all of these. Very rarely, a suture knot comes undone or a damaged suture breaks, but more typically, the fault lies with the tissue and not the suture. To achieve secure closure, keep in mind (and hands) the following discussion.

## Principles of Closure

### *Suture Material*

Use a nonabsorbable (e.g., nylon or Prolene) or "delayed" absorbable (e.g., PDS or Maxon) monofilament suture. Rapidly absorbed materials such as vicryl and Dexon are still widely used even though their use is illogical in view of wound repair kinetics. Those who fancy such suture material produce the hernias for the rest of us to repair. Nonabsorbed or slowly absorbable suture material, on the other hand, keeps the edges of wound together until its tensile strength takes over. Monofilament sutures are advantageous because they slide better, inflicting less "saw injury" to the tissues and, when used in the preferred continuous fashion, distribute the tension evenly along the length of the wound. The use of braided nonabsorbable material (e.g., silk) is associated with chronic infected sinus formation and belongs, we hope, to remote history. Monofilament material is not

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prone to breakage, but damage to the suture during insertion can make this more likely. It is therefore important to avoid grasping the suture material itself with forceps because this can damage its integrity and weaken it.

### *"Mass Closure"*

"Mass closure" is the preferred technique, as documented in numerous studies. It has been popularized for the closure of midline incisions but is as effective for the closure of transverse muscle-cutting incisions. For the latter, however, many surgeons still prefer layered (posterior fascia–anterior fascia) closure. We also do. For example, to close a subcostal incision, we would run a looped PDS 1 from the center laterally, taking the posterior sheath; at the lateral corner, we would lock the suture and run it back medially, taking the anterior sheath, and tying the knot at the medial corner of the incision.

Mass closure entails monolayered suturing of **all** structures of the abdominal wall in a continuous manner to provide "one strong scar." The secret here is to take large bites of tissue, at least 1 cm away from the wound's edges; the bites should be closely spaced so not to create gaps greater than 1 cm. Avoid the common mistake of carefully excluding muscle in your fascial bites; this may look cosmetically appealing as the muscle is hidden away under the fascia but does not produce the desired "mass scar." No less important is the issue of the correct tension to be set on the suture (› Fig. 43.1). If you pull the suture too tight, the tissue is strangulated and necrosed; if you keep the suture too loose, the wound edges gape. Bear in mind that the muscles are relaxed as you close


**Fig. 43.1.** "Jack, what are you doing?"… "The boss told me to close it tight"…

(or should be), and that postoperatively they will acquire their normal tone; the tissues in the wound will swell, and abdominal girth will increase. All of these changes make the wound closure tighter; if it is tight when you put the sutures in, then something must give way when these changes take place—the tissue tears. A ratio of suture length to wound length of at least 4:1 will ensure a moderate but secure tension of closure. The corners of the incision are the Achilles heels of closure, especially the corner that is closed last. Do not compromise complete closure of the corner because you are afraid of injuring the underlying bowel; there are good tricks to accomplish this endeavor—learn them from one of your mentors.

Do not harm the underlying bowel, which frequently bulges toward your large needle. At the end of the operation, the anesthetist always swears to God that the patient is "maximally relaxed"; the anesthetist lies. Make the anesthetist relax the patient again—do not compromise. Protect the bowel by whichever instrument is available; the best, in our experience, is the commercially available rubber "fish" retractor. The assistant's hand also may be useful for this purpose, but with all the hepatitis and HIV around, we do not find many volunteers willing to offer a retracting hand.

We recommend the use of a "looped" number 1 PDS suture. It is a slowly absorbable monofilament, usually long enough to provide a suture-to-wound ratio of 4:1. Threading the needle through the loop after the first bite replaces the need for the initial knot. The final knot in our hands would be the "Aberdeen" one. We cut it long—about 5 mm—and bury it in the subcutaneous space with any thin absorbable suture.

## The Subcutaneous Space

Now, when the fascia is closed, what do you do with the subcutis? Nothing. There is no evidence that the so-called dead space reduction using subcutaneous fat approximation reduces wound complications. On the contrary, subcutaneous sutures act like a foreign body and strangulate viable fat while not producing a more satisfactory wound. Subcutaneous drains increase the rate of infection and are almost never indicated. Plain *saline irrigation* has been shown to be useless but use of topical antibiotics (solution or powder) has been demonstrated to further decrease wound infection rate in contaminated wounds in patients who have already received systemic antibiotic prophylaxis.

## "Delayed Primary" or "Secondary Closure"

What about the well-entrenched ritual of "delayed primary" or "secondary closure" after contaminated or infected laparotomies?

We believe that these techniques are only rarely indicated. In spite of surgeons' obsession with tradition, lessons learned years ago under certain circumstances are not necessarily true today. Thus, 20 years ago when antibiotic prophylaxis was given incorrectly, heavy silk sutures were buried in the fat, and rubber drains were mushrooming through every wound, the infection rate in primarily closed wounds was intolerable. Today, on the other hand, with proper surgical technique and modern antibiotic prophylaxis, primary suture of the wound can be undertaken uneventfully in the majority of emergency laparotomy cases. When a wound infection develops, it usually responds to local measures. Thus, leaving all contaminated, potentially infected wounds gaping open—awaiting spontaneous or secondary closure—produces unnecessary physical and financial morbidity. On the rare occasion we decide to leave a wound open, usually in patients with gross established purulent or fecal peritonitis, in patients planned for further reoperations, or in the relaparotomized abdomen. In the vast majority of patients, we irrigate the subcutaneous tissues with antibiotics (after fascial closure) and close the skin with staples or interrupted sutures. Truly modern surgeons, however, are happy to close most wounds with a subcuticular stitch of absorbable material. This obviates the discomfort and expense of arranging staple or suture removal and gives a much neater scar. (This is the only part of your handiwork that the family and the patient see, and you would be surprised to discover how much this little thing matters to some patients.) An occasional wound infection is not a disaster and is simple to treat (Chap. 55).

## The High-Risk Abdominal Closure

Regarding high-risk abdominal closure, classically, in patients with systemic (e.g., cancer) or local (e.g., abdominal distension) factors predisposing to wound dehiscence (Chap. 53), "retention" sutures were and are still used by surgeons. Those heavy "through-and-through," interrupted sutures take bites of at least 2 cm through all abdominal wall layers—including the skin—preventing evisceration but not the occurrence of late hernia formation.

We do not find any use for the *classical retention* sutures, which cut through the skin and produce parietal damage and ugly skin wounds and scars. Instead, we suggest that in selected high-risk closures you place a few interrupted all-layers mass sutures (excluding the skin) to take the tension off the continuous mass closure. Should the latter fail at any point, the interrupted sutures would prevent separation of the fascial edges and evisceration.[1]

[1]Coeditor P.R. comments: there is no evidence supporting this. Moreover, if the mechanically sound pulley mechanism of the mass suture fails, then these itty-bitty interrupted sutures cannot survive.

The crucial consideration is, however, that the use of retention sutures together with abdominal distension results in intra-abdominal hypertension. Forceful closure under excessive tension may result in an abdominal compartment syndrome with its deleterious physiological consequences (Chap. 40). Thus, when the fascia is destroyed, as is often the case after multiple abdominal re-entries, or when closure may produce excessive intra-abdominal pressure, we suggest that you do not close the abdomen but cover it with a temporary abdominal closure device (TACD; "laparostomy") (Chaps. 40, 52.2, and 53).

### *Closing the Skin Only*

Occasionally, when we wish to avoid fascial closure but do not want to condemn the patient to the not insignificant morbidity associated with *laparostomy*, we leave the fascia unsutured but **close the skin**. Scenarios ideal for such an approach would be when you feel that no reoperation would be necessary but visceral "bulging" prevents fascial closure without excessive tension. Of course, all surviving patients will develop a large incisional hernia; the very old and infirm will live with the hernia for the rest of their lives. In others, an elective repair of the hernia is associated with lower morbidity than the staged management of laparostomy. Regarding how to do it: always spread the omentum, if available, over the viscera; the skin is closed with 2–0 nylon interrupted mattress sutures, taking bites at least 1 cm from the skin edge. Do not let anyone remove these sutures until you approve—usually not before 3 weeks. Remember: the patient's own normal skin is better than the VAC (Vacuum Assisted Closure) system or skin grafts.

In conclusion, remember: big continuous bites, with a monofilament, not too tight—this is how to avoid dehiscence and herniation.

> "Abdominal closure: if it looks all right, it's too tight—if it looks too loose, it's all right." (Matt Oliver)

# 44 Before Landing

Moshe Schein

*"Pilots may have more incentive than surgeons to be perfect, right or wrong. When they botch a landing, it's usually their last. Fortunately, modern day aircraft are a lot more predictable and reliable than any of our patients."* (Tim Eldridge (USAF ret)).

*Takeoffs are optional. Landings are mandatory.*

Everyone knows that a "good landing" is one from which you can walk away. But, very few know the definition of a "great landing." It is one after which you can use the airplane another time. Yes, we know that you are tired; you may have worked all night, and this may be the last of many long cases. But, any landing must be perfect, and even this last operation has to succeed.

Before closing the abdomen, you must be absolutely happy with what you did. You do not want to spend the next week in guilt and worry as your patient fails to recover promptly. Prevent "guilt-worry." Always ask yourself, "Am I totally satisfied with my procedure?" (› Fig. 44.1). Do not silence the little voice within you that informs you that the anastomosis is somewhat dusky, or it needs

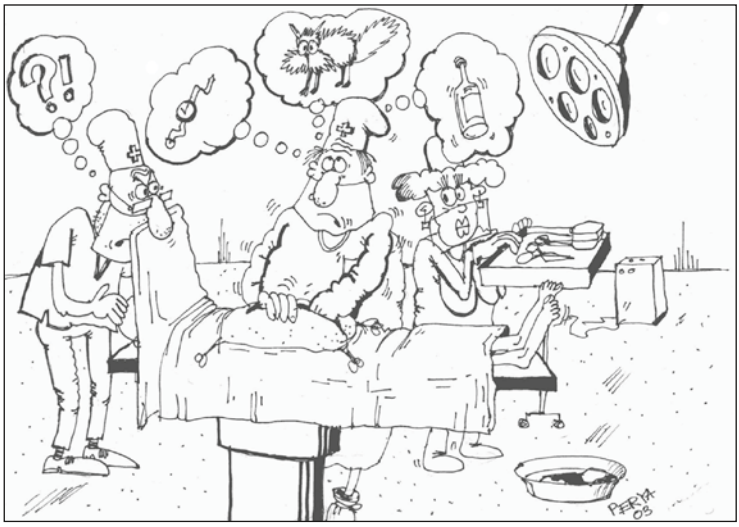

**Fig. 44.1.** "Am I satisfied?"

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another stitch. You must be absolutely convinced, at this stage, that you have done the best that your patient deserves. If not, swallow your pride, summon the last vestige of your patience, do it again or call for help. Hiding a potential problem will not solve it. And, you will go back to sleep so much better. However, bear in mind that—to paraphrase Voltaire—better is the enemy of good. You must be sure that any attempts to improve a less-than-perfect situation are justified. (Remember the philosophy of the abbreviated laparotomy.)

**You may want to go over a pre-closure checklist:**

**Hemostasis perfect?** This does not mean that you have to run after each red blood corpuscle.
**Source control** achieved?
**Peritoneal "toilet"** completed? All fluid sucked out?
**Anastomosis**: Viable? Not under tension, lying well?
Potential sites **for internal herniation** dealt with?
**Small bowel** comfortably arranged in place below the transverse colon?
**Omentum** placed between intestine and incision?
All additional **fascial defects** (e.g., trocar sites) closed?
**Nasogastric** tube in position (if needed)?
**Drains** (only if indicated) in place?
Need a **feeding jejunostomy?**
**Should I close** the abdomen at all? Or leave it open?

Do not compromise. Keep looking around; there is always something you have missed. Remember: **when the abdomen is open you control it; when closed, it controls you!**

There are old pilots, and there are bold pilots, but there are no old, bold pilots! There are, however, old bold surgeons—but their patients do not live long…

# After the Operation

C

# 45 Postoperative Care

Moshe Schein

*"When is a surgeon … nervous? Not during operations. But basically a surgeon's nervousness begins after the operations, when for some reason the patient's temperature refuses to drop or a stomach remains bloated and one has to open it not with a knife, but in one's mind, to see what had happened, to understand and put it right. When time is slipping away, you have to grab it by the tail."* (Alexander Solzhenitsyn)

*We repeat: "As long as the abdomen is open you control it. Once closed it controls you."*

The long operation is finished, leaving you to savor the sweet postoperative "high" and elation. But very soon, when your serum levels of endorphins decline, you start worrying about the outcome. And worry you must, for the cocksure, macho attitude is a recipe for disaster. We do not intend to bring here a detailed discussion of postoperative care or to write a new surgical intensive care manual. We only wish to share with you some basic precepts, which may be forgotten, drowned in a sea of fancy technology and gimmicks. The following are a few practical commandments for postoperative care.

## Know Your Patient

It is no joke! How often do we encounter a postoperative patient looked after by someone who has no clue about the patient's pre- and intraoperative details? Mistakes in management are more commonly made by those who "temporarily adopt" the case. Once you operate on a patient, he or she is yours. Shared responsibility means that no one is responsible!

## Touch Examine Your Patient

Touch examine your patient—do not examine only from the foot of the bed. Examining the chart or the intensive care unit (ICU) monitor is not enough. Look at the patient, smell and palpate the patient at least twice a day. Wouldn't it be embarrassing to load your patient with intravenous antibiotics or CT scan the

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patient's abdomen while an unsuspected abscess is cooking under the wound dressing, begging simply to be drained at the bedside?

Leo Tolstoy wrote in *War and Peace*: "When he came to himself the splintered portions of his thigh bone had been extracted, the torn flesh cut away and the wound bandaged. Water was being sprinkled on his face. As soon as Prince Andrei opened his eyes the doctor bent down, kissed him on the lips with not a word and hurried away." We do not ask you to kiss your patients—just touch them! And, you may hug their wives or husbands.

### Treat the Pain

You know the different drugs and their modes of administration. Sure, you always prescribe postoperative analgesia, but ordering is not nearly enough. Most randomly questioned postoperative patients complain that they are undertreated for pain. Nurses tend to be stingy with analgesia. You are the person on the spot; consider pain as the "fifth vital sign" and see that your patient does not suffer unnecessarily.

### Do Not "Crucify" Your Patient in the Horizontal Position

Typically, the "modern" patient is "crucified" horizontally, tethered by a **spaghetti** of monitoring cables, nasogastric (NG) tubes, venous lines, drains, leg pumps, and urinary catheters. Free the patient from these paraphernalia as soon as possible; the nurses will not do it without your order. The earlier your patient is out of bed, sitting or walking about, the faster the patient will be going home. Conversely, keeping the patient in the supine position increases the incidence of atelectasis/pneumonia, deep vein thrombosis (DVT), and decubitus ulcers and prolongs paralytic ileus, all adding fuel to the inflammatory fire of SIRS (systemic inflammatory response syndrome). Have your patient out of bed as soon as possible (ASAP), and this means commonly a few hours after the operation. If the nurses are reluctant or lazy, lift the patient out of bed by yourself—always provide the example.

### Decrease the Plastic and Rubber Load

Monitoring functions as an early warning system to detect physiological disturbances so that prompt corrective therapy can be instituted. The invasiveness of monitoring employed in the individual patient should be proportionate to the severity of disease*: The sicker the patient, the greater number of monitoring tubes used, the less likely is survival.*

Complete discussion of the continuously growing number of monitoring methods available today is beyond the scope of this chapter. However, please note:

- To be able to respond to monitoring-generated warning signs, you must fully understand the technology employed. You should be able to distinguish between real acute physiological changes and electrical or mechanical artifacts of observation.
- Understand that all methods of monitoring are liable to myriad potential errors specific to the technique or caused by patient-related variables. Alertness and sound clinical judgment are paramount.
- Because of improving technology, monitoring is becoming more and more sophisticated (and expensive). Furthermore, monitoring techniques are responsible for a significant number of iatrogenic complications in the surgical ICU. Use monitoring discriminatingly and do not succumb to the *Everest syndrome:* "I climb it because it is there." Before embarking on invasive monitoring ask yourself: "Does this patient really need it?" Remember there are safer and cheaper alternatives to invasive monitoring; for example, in a stable patient, remove the arterial line as the blood pressure can be measured with a conventional sphygmomanometer, $PO_2$ determined transcutaneously, and blood tests drawn by phlebotomy. Each time you see your patient, ask yourself which of the following can be removed: NG tube, Swan-Ganz catheter, central venous line, arterial line, peripheral venous line, Foley catheter.

**NG tubes** ▶ Prolonged postoperative NG decompression to combat gastric and intestinal ileus is a common ritual. The concept that the NG tube "protects" distally placed bowel anastomosis is ridiculous as liters of juices are secreted each day below the decompressed stomach. Nasogastric tubes are extremely irritating to the patient, interfere with breathing, cause esophageal erosions, and promote gastroesophageal reflux. Traditionally, surgeons keep the tube until the daily output drops below a certain volume (e.g., 400 ml); such a policy often results in unnecessary torture. It has been repeatedly demonstrated that most postlaparotomy patients do not need nasogastric decompression—not even following upper gastrointestinal procedures—or need it for a day or two at most. In fully conscious patients, who are able to protect their airway from aspiration, NG tubes can be safely omitted in most patients. Following an emergency abdominal operation, nasogastric decompression is compulsory in mechanically ventilated patients, in obtunded patients, and after operations for intestinal obstruction. In all other cases, consider removing the NG tube on the morning after surgery. If in doubt, you may want to cap or clamp the tube for 12 hrs before removing it and observe how this is tolerated by the patient. A small percentage of patients will need the tube to be reinserted because of early postoperative small bowel obstruction or persistent ileus (Chap. 48).

**Drains** ▶ Despite the widely publicized dictum that it is impossible to drain the free peritoneal cavity effectively, drains are still commonly used and misused

(Chaps. 12 and 42). In addition to the false sense of security and reassurance they provide, drains can erode into intestine or blood vessels and promote infective complications. We suggest that you limit the use of drains to the evacuation of an established abscess, to allow escape of potential visceral secretions (e.g., biliary, pancreatic), and to establish a controlled intestinal fistula when the bowel cannot be exteriorized. Passive, open-system drainage offers a bidirectional route for microorganisms and should be avoided. Use only active, closed-system drainage systems, placed away from the viscera. Leaving a drain close to an anastomosis in the belief that a possible leak will result in a fistula rather than in peritonitis is a long-enduring but unproven dogma; drains have been shown to contribute to the dehiscence of a suture line. A policy like "I always drain my colonic anastomoses for 7 days" belongs to the dark ages of surgical practice. Remove drains as soon as they have fulfilled their purpose.

### Obtain Postoperative Tests Selectively

*Unnecessary* diagnostic procedures or *interpretative* errors in *indicated* diagnostic procedures commonly result in *false-positive* findings, leading in turn to an increasingly invasive escalation of diagnostic or therapeutic measures. Added morbidity is the invariable price. If the results of a test are not going to affect your management, do not order the test.

### Realize That the Problem Usually Lies at the Operative Site

The cause of fever or "septic state" in the surgical patient is usually at the **primary site of operation unless proven otherwise**. Do not become a "surgical ostrich" by treating your patient for "pneumonia" while the patient is slowly sinking in multiple organ failure from an intra-abdominal abscess (Fig. 45.1).

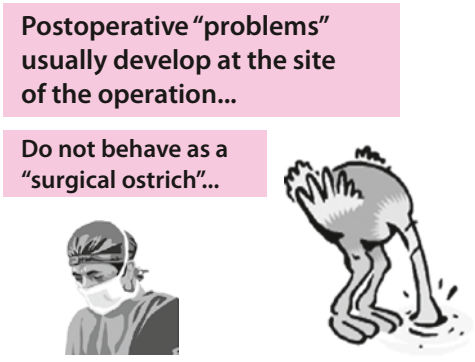


**Fig. 45.1.**

### Temperature Is Not a Disease; Do Not Treat It as Such

Postoperative fever represents the patient's inflammatory response (SIRS) to different insults, including infection as well as surgical trauma, atelectasis, transfusion, and others. SIRS does not always mean sepsis (Sepsis = SIRS + Infection). Therefore, fever should not be treated automatically with antibiotics. It also should not be stifled with antipyretics as the febrile response has been shown beneficial to the host's defenses. You will have to argue with your nurses about this. "The patient will be more comfortable," "he'll convulse," "we always give Tylenol," they will claim. The absolute level of temperature is of less importance than its trend, and it is difficult to assess this important sign when you are artificially suppressing it.

> "Fever is, in a measure, a beneficial process operating to protect the economy." (Augustus Charles Bernays, 1854–1907)

### Avoid Poisoning Your Patient With Antibiotics

Tailor antibiotic administration to the patient. Avoid the common practice of administering antibiotics for as long as the patient is in the hospital and beyond (Chap. 47).

### Be Frugal With Blood Product Transfusions

Generally, the amount of blood or derived products transfused inversely and independently correlates with the outcome of the acute surgical disease. Donated blood is immunosuppressive and is associated with an increased risk of infection, sepsis, and organ failure, not to mention the other well-known hazards. Cancer patients in particular fare worse in the long term if they receive a transfusion. Transfuse your patient only if absolutely necessary. A patient requiring only one unit of blood does not require any at all. For the vast majority of patients, a hematocrit of 30% is more than satisfactory. We would rarely transfuse a postoperative patient with a hemoglobin above 8 g% unless the patient is critically ill or suffers from an underlying cardiorespiratory disease.

### Do Not Drown Your Patient in Salty Water

The current, exaggerated "protocols" of postoperative fluid management provide too much water and salt, resulting in obligatory weight gain and swelling of tissues. And, edematous tissues do not function well and do not heal well—causing

a higher rate of medical and surgical complications (see Editorial Comment section in ◉ Chap. 6). All your patient needs is enough water to replace insensible losses (500–1,000 ml) and provide for urinary flow of 0.5 ml/kg/h. Additional losses (e.g., via NG tube) should be replaced selectively on an ad hoc basis but writing an order for 150 ml/h of saline and going to sleep will result in a swollen patient. You have to read the article by Brandstrup et al. (2003) to see how postoperative fluid restriction may help your patient. And, get rid of the intravenous line as soon as possible!

> Fluids given intravenously bypass all the defenses set up by the body to protect itself against excess of any constituent, against bacterial entry. … They give the patient what the surgeon thinks his tissues need and what they are damned well going to get. (William Heneage Ogilvie, 1887–1971)

### Do Not Starve or Overfeed Your Patient; Use the Enteral Route Whenever Possible (◉ Chap. 46)

Please do not torture your patient with the useless and baseless ritual of slowly increasing the permitted consumption of oral fluids from 30 ml hourly to 60 then 90 and so on over several days.

### Recognize and Treat Postoperative Intra-Abdominal Hypertension (◉ Chap. 40)

### Prevent Deep Vein Thrombosis and Pulmonary Embolism

It is easy to forget DVT prophylaxis in the preoperative chaos of emergency surgery. As a pilot goes over a checklist prior to any flight, you should be the one to inject the subcutaneous heparin and place the anti-DVT pneumatic device—**before** the operation. DVT prophylaxis should be continued **postoperatively** as long as the patient continues to be at high risk of thrombosis. Selected patients (e.g., after operations for cancer) may need to continue DVT prophylaxis at home.

### Be the Leader and Take Responsibility

Many people tend to dance around your postoperative patient, giving consults and advice. But remember, this is not their patient; he or she is yours. At the mortality and morbidity meeting (or in court), the others will say, "I just gave a consult" (◉ Chap. 59). The ultimate responsibility for all aspects of your patient's management falls squarely in your hands. Know when you need help and request


**Fig. 45.2.** "Who is missing, guys? Where is the podiatrist?"

it, preferably from one of your mentors. As Francis D. Moore said: **"Seek consultation even if it is not sure to help; never be a lone wolf."** But, solicit advice judiciously and apply it selectively. Relinquishing blindly the care of your postoperative patient to anesthesiologists, medical intensivists, or other modern "experts" may be a recipe for disaster. It is much better in this modern surgical age to form close working relationships with colleagues who share your philosophy of care and who have expertise in areas beyond your own. We all need help with patients suffering multisystem failure; while we can take care of the abdominal problem, we do need assistance and advice to manage cardiac, respiratory, and renal failure appropriately. As Mark Ravitch said: **"The problem with calling in a consultant is that you may feel obliged to take his advice" (Fig. 45.2).**

## Analyze Your Care

When all is said and done, step back and assess your management. Ask yourself, "What did I do well?" and "What could I do better the next time I'm confronted with a situation like this?" How else will you get any better?

> "The operation is over when the patient is eating a cheeseburger and can't remember your name." (Leo A. Gordon)

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# 46 Nutrition

James C. Rucinski

*"In every disease it is a good sign when the patient's intellect is sound, and he is disposed to take whatever food is offered to him; but the contrary is bad."*
(Hippocrates, circa 460–377 BC)

*God created man with a mouth, a stomach, and a gut—not a TPN line.*

The relatively brief interval available to you to prepare an emergency abdominal patient for an operation does not allow for nutritional considerations. This issue, therefore, is addressed only *during* and *after* the operation. Toward the end of the laparotomy, you should ponder whether there is a need to provide *enteral access* to facilitate postoperative feeding. After the operation, the issues to think about are how early, and by which route, the patient should be fed.

## Starvation

Starvation results in a state of adaptation. After hepatic glycogen stores are consumed in 24–48 hrs, the liver synthesizes glucose, using amino acids derived from protein breakdown. This "autocannibalization" of functional protein stores is ameliorated, to some degree, by conversion to ketone metabolism of the two major "obligate" glucose users, the central nervous system and the kidney. Fat stores help by providing ketones and, through glycerol metabolism, adding a small amount of glucose. Injury, illness, or operation, though, greatly increases the demand for glucose to answer the hypermetabolic demands made by SIRS (systemic inflammatory response syndrome) and to provide energy for wound repair and for the bone marrow and its offspring, the leukocytes. The end result, then, is the breakdown of protein leading to general debility, impaired reparative processes, attenuated immune function, and respiratory muscle weakness, which in turn may cause atelectasis, pneumonia, ventilator dependence, and death.

The need for nutritional support, then, is based on:

- Your physical and laboratory assessment of the patient's *nutritional reserves*
- An estimate of the *associated stress of the underlying illness*
- An estimate of the *time interval* that will pass before the patient can resume a normal diet

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## Assessment of Need for Nutritional Support

You must ask the patients how long they have felt sick and how much weight they have lost, if any, in the weeks prior to the operation. You must also ask when they last ate. By looking at the person, you can estimate what their ideal weight might be and make a "guesstimate" regarding the percentage that has been lost. (Your rule of thumb standard is the fabled "70-kg man.") *A loss of more than 10% is associated with a higher rate of complications and death after abdominal surgery*. This will give you the first two pieces of information necessary for decision making:

- Percentage weight loss and available reserves
- Time since normal feeding was stopped

*Serum albumin level* reflects the balance of synthesis and degradation of one of the products of hepatic metabolism. In the emergency setting, the albumin level and total lymphocyte count will be the only laboratory parameters available to you to estimate *available reserves.* Serum albumin level of <3 mg/dl and a total lymphocyte count <1,500 are associated with a higher rate of complications and death in abdominal surgery.

*The associated stress of illness* may be roughly estimated as minimal, moderate, or maximal. It is better, though, to characterize stress by the use of a physiologic scoring system that measures the severity of the acute illness—such as the APACHE II (Acute Physiological and Chronic Health Evaluation II) system (Chap. 6). An increased level of stress is associated with a higher rate of protein breakdown, as well as complications and death, in abdominal surgery.

The third piece of information necessary for decision making is *the time interval that will pass before the patient can resume a normal diet*. This estimate is based on the nature of the primary illness and the type of operation that is required or has been performed. For example, a person with "simple" acute appendicitis will experience cessation of normal feeding for a period of 24–72 hrs, whereas a person with perforated diverticulitis, with generalized peritonitis, may experience cessation of feeding for a period as long as 10–14 days.

With this information, then, you can decide which patients will be most likely to benefit from nutritional support.

- At one end of the spectrum, the patient with *normal reserves* by history and examination, with *minimal-to-moderate associated stress*, and with *less than 7–10 days estimated before resumption of a normal diet* is *unlikely* to benefit from parenteral nutritional support.
- At the other end of the spectrum, the patient with depleted available reserves, moderate-to-severe stress, and with more than 7–10 days estimated before resumption of a normal diet is likely to benefit from nutritional support.

## Enteral Versus Parenteral Nutrition

Nutritional support may be provided by *enteral* (through the alimentary tract) or *parenteral* (intravenous) routes. The advantage of enteral nutrition is that it is associated with reduced rates of infection, sepsis, length of hospital stay, and costs. Although the exact reasons for the effectiveness of enteral over parenteral nutrition are not clear, almost all outcome studies of acutely ill adults with functioning gastrointestinal tracts fail to document improved outcomes from parenteral nutrition. The advantage of parenteral nutrition is that it can be used when and if the gastrointestinal tract is not functional.

This is no longer controversial; when the gut functions, use it! Clearly, *enteral* feeding is safer, cheaper, and more physiologic than *parenteral* nutrition! Alexander Solzhenitsyn knew it 40 years ago, writing in *Cancer Ward*: "If I need grape sugar, give it to me through the mouth! Why this twentieth century gimmick? Why should every medicine be given by injection? You do not see anything similar in nature or among animals, do you? In a hundred years' time they'll laugh at us and call us savages."

## Enteral Nutrition

Tasty food given by mouth is the ideal. Oral feeding requires the co-operation of the patient, a normal swallowing mechanism, and normal gastric motility. Unconscious and intubated patients, however, cannot swallow, but the main problem is that following abdominal operations the stomach is lazier than the intestine. In other words, after laparotomy the small bowel recovers motility before the stomach. The gut is ready to absorb nutrients in the first postoperative day, whereas the stomach may have delayed emptying for a few days (Chap. 48). It is clear then, that when early postoperative feeding is deemed necessary, or when oral intake is inadequate, the food should be instilled distally—beyond the esophagus and the stomach.

### *Routes*

In general, when the mouth is not available, the following feeding routes are options:

- *Nasogastric and nasoenteric*. The former is of course not usable when the stomach is not functioning. The latter delivers the nutrients directly into the duodenum and jejunum. *Transnasal intubation* in conscious patients is only tolerated with narrow-bore and soft tubes. Rare complications are nasal trauma,

sinus infection, and even (very rarely) misplacement into the bronchial tree with inadvertent instillation of the feeding solution into the lungs.

- *Gastrostomy and transgastric jejunal tube*. The feeding tube is operatively placed directly into the stomach and then through the pylorus into the jejunum. This is a surgical procedure that violates the gastric wall. The chief complication is leakage at the insertion site: around the tube, which is not uncommon, or into the peritoneal cavity, which is rare but potentially fatal.
- *Jejunostomy tube*. The feeding tube (or a catheter) is inserted directly into the proximal jejunum as discussed next.

Feeding directly into the jejunum, as opposed to gastric feeding, is supported by randomized controlled trials and is intuitively associated with less risk of aspiration, better delivery of food, and fewer problems with gastric retention.

### *Should I Place a Jejunal Feeding Tube?*

This is the question you should ask yourself at the end of the emergency laparotomy. It is much more convenient to do it at this stage as opposed to doing it postoperatively. You should consider the three questions mentioned above: what is the likelihood that this patient will be eating in 7–10 days? Is the patient malnourished or not? What is the magnitude of this illness?

A malnourished alcoholic patient who requires a total gastrectomy with esophagojejunal anastomosis for massive upper gastrointestinal bleeding represents a classical indication for a jejunal (J) feeding tube. A case of multitrauma involving the thorax, pelvis, and long bones who undergoes a laparotomy for hepatic injury could also benefit from immediate J-tube feedings. After a partial gastrectomy in a previously well-nourished patient, J-tube placement is not indicated as the potential risks override the assumed benefits. You do not want to place a J tube in a patient who will not need it.

**There are three methods to place the J tube during the operation:**

- *Transnasally:* into the stomach, from which you can manipulate it by palpation into the proximal jejunum. The advantage is that it does not require a gastrotomy or enterotomy; disadvantages are its nasal presence and risk of accidental dislodgment.
- *Transgastric:* "combined" gastrostomy/jejunostomy tubes are available to allow gastric aspiration and jejunal feeding at the same time. Obviously, gastrostomy has its own complications, mainly leakage around the tube, leakage into the peritoneal cavity, and abdominal wall cellulitis. A meticulous fixation of the stomach onto the abdominal wall is mandatory.
- *Jejunostomy:* a 16F or larger tube may be placed through a purse-string controlled enterotomy and then suture-tunneled with serosa over the site of entry

extending 5–7 cm proximal (Witzel technique). Alternatively, a 12- or 14-gauge catheter may be "tunneled" into the jejunal lumen through a needle (needle catheter technique). Both techniques require suture fixation of the bowel to the site of catheter entry in the abdominal wall to prevent intra-abdominal leakage of small bowel contents or leakage of feedings if the tube is accidentally removed before an enterocutaneous tract is developed (in 7–10 days). An additional useful trick is to fix the efferent and afferent portions of the loop to the abdominal wall to prevent kinking and obstruction at the site of the jejunostomy. The needle and catheter should pierce the abdominal wall obliquely in line with the bowel wall tunnel; this will prevent kinking—followed by breaking—of the fine tube at the bowel-skin junction.

Continuous J-tube feeding may be instituted immediately following operation in most cases. Diarrhea is a common problem requiring adjustment of the volume and concentration of the specific solution you prefer to use. Be aware that nasojejunal tubes can be inserted across suture lines, and that feeding can be instilled proximal to suture lines.

> "There is no way a patient is going to eat a hole in the anastomosis." (P.O. Nystrom)

Note also that cases of *massive intestinal infarction were reported in critically ill patients* receiving early postoperative jejunal feeding, possibly due to increased metabolic demands on an already poorly perfused gut. Therefore, hold J-tube feedings in unstable patients and those on vasopressors. Small bowel ileus can prevent adequate J-tube feeding; always consider that behind the nonresolving or reappearing ileus there may be a treatable cause (› Chap. 48).

You may have been approached by the manufacturers of the new "immunoenhancing diets." These are tube feeding formulas that contain high concentrations of certain nutrients and are claimed to "increase immunity," thus reducing the postoperative infection rate. The value of such expensive diets is questionable, as is the value of enteral supplementation with the amino acid glutamine.

### *Postoperative Placement of Transnasal J Tube*

You can place a transnasal J tube also *after* the operation—if indicated. This however is not easy and requires prolonged manipulation under fluoroscopy. An alternative is to use a gastroscope, with a long tube (e.g., nasobiliary) placed into the distal duodenum through the biopsy channel of the scope and under vision. Clearly, intraoperative placement is much easier. Please do not forget this option before closing the abdomen.

## Parenteral Nutrition

Patients who cannot eat and will not tolerate enteral feeding may need parenteral nutritional support, and in that circumstance it may be lifesaving. Parenteral nutrition comes in three "flavors":

- *Protein-sparing hydration* takes advantage of the fact that 100 g of glucose a day suppresses hepatic gluconeogenesis by supplying much of the obligate daily glucose need. Two liters of 5% dextrose provide this amount of sugar. For the average "not-so-stressed" patient, this is more than enough for the first seven postoperative days.
- *Peripheral parenteral nutrition* (PPN) contains amino acids in addition to a low concentration of glucose and may provide an additional protein-sparing effect when "stress" is added to starvation. It is useful in maintenance nutrition for an intermediate period of postoperative starvation, 7–14 days, or as long as the patient's peripheral veins last. This is so because PPN is a "vein destroyer," which often requires frequent change of the venous access. (The editors asked me not to cite references, but I cannot resist and wish to recommend an excellent 2003 review on this subject by Anderson et al.)
- *Total parenteral nutrition* (TPN) contains amino acids and a concentrated dextrose solution, into which a lipid solution is usually added, which can provide for an indefinite duration the total amount of nutritional requirements even in the face of maximal stress. As usual, bypassing physiology has a price: TPN is associated with a long list of mechanical, catheter-related, infectious, and metabolic complications and is rather expensive.

Do not forget that replenishing electrolytes (e.g., magnesium, phosphorus), trace elements, and vitamins is crucial in patients in need of parenteral nutrition.

## Control of Hyperglycemia

Data derived in the last 5–10 years suggest that optimal control of blood glucose is far more important than the route of nutrition in critical illness. Maintenance of the blood glucose below 110 mg/dl has been shown to decrease morbidity (particularly the length of stay in the ICU and the need for ventilator support and renal dialysis) and to decrease the rate of mortality among critically ill patients. Tight maintenance requires the use of a constant intravenous insulin infusion and is easier to accomplish with enteral, rather than parenteral, nutritional support. [Please do not be carried away with "tight control of hyperglycemia"; remember that hypoglycemia is dangerous as well!—The Editors]

## Measurement of Effectiveness of Nutritional Support

Prolonged overfeeding and underfeeding must be avoided. In the long term, the optimal amount of nutrition can be calculated by observing the balance

of protein synthesis and degradation reflected in serum protein levels such as albumin (half-life 17 days), transferrin (half-life 8 days), or transthyretin (prealbumin, half-life 48 hrs). In the short term, particularly in the critically ill, nitrogen balance can be assessed by comparing the amount of nitrogen that is produced in the urine (24-hrs urine specimen analyzed in the laboratory) with the amount of nitrogen that is given by nutritional support (written on the package).

### So, What Should You Do?

- First, decide if nutritional support will be helpful by estimating *nutritional reserve, degree of stress, and time interval to normal diet*.
- Hold off starting nutritional supplements until perioperative intravenous fluid resuscitation has attenuated the effect of third-space fluid sequestration and the initial hypermetabolic, hyperglycemic physiologic picture has abated somewhat (usually within 24 hrs).
- Calculate the nutritional requirement by formula (there is no shame in looking this up) or indirect calorimetry.
- Institute nutritional support. Enteral nutritional support should be the first option. Parenteral nutritional support should be instituted if the nutritional goals cannot be achieved with enteral support within 7 days.
- Closely control the serum glucose with insulin and reassessment every 1–4 hrs.
- Measure the effectiveness of treatment by analysis of urinary nitrogen loss compared with the amount of nitrogen provided by the treatment.

### "Routine" Oral Feeding

Fortunately, most of your patients needing emergency abdominal care recover from the ileus, induced by the underlying disease and its surgical treatment, within a few days. Traditionally, resumption of oral intake was completed in stages. First, there was the nasogastric tube, which was kept in situ for variable periods (Chap. 45); then, the tube was removed (according to the rules established by the local dogma-guru). After the patient professed the blessed sounds of flatus, the patient was started on "sips," thereafter gradually advanced from "clear fluids" to "full fluids" to "soft diet," until the great day when "regular diet" was allowed, usually indicating that discharge home was imminent. Is such a ritual or its variant still practiced in your environment? If yes, you should know that its value is based on no evidence at all. In fact, there is scientific evidence to prove that starting the patient on solid feeds is as "safe" and tolerable as the staged method still practiced by many.

On the other side of the coin, there are surgeons who maintain that a patient who devours a beefsteak a day after a colectomy is a testimony to their superb surgical skills. This attitude is probably wrong as well: what is the point of

**Fig. 46.1.** Postoperative day 1: "Let him eat as much as he wants…"

force-feeding a patient who does not have an appetite? The physiological postoperative ileus is a response that must have some purpose; appetite and desire to eat return when intestinal motility recovers. Our approach is therefore to let the patient decide when to eat, what to eat, and how much; the patient will tell you when his or her stomach is ready for a steak or the cornmeal (Fig. 46.1). But of course this does not apply to the morbidly obese patient who asks for a giant pizza an hour after undergoing appendectomy for perforated appendicitis—not an uncommon scenario in the "fatlands" of northern America.

## Concluding Remarks

Before we finish, let us share a few truths with you:

- We know that *prolonged* starvation may be harmful, but there is no definite proof that early refeeding after surgery is beneficial.
- We know that when compared to postoperative TPN, enteral nutrition is associated with better results. However, in the absence of a nonfed control group in any of the studies, it is not clear whether enteral nutrition provides specific benefit or that TPN is associated with an increased rate of complications.
- There is some evidence that early postoperative enteral nutrition may adversely affect respiratory function.
- There is some evidence that tight control of hyperglycemia is associated with a better outcome in the critically ill surgical patient.

Abdominal catastrophes and their operative treatment are often complicated by compromised nutritional reserve, stress, and a long interval before a normal diet is resumed. The result of these factors is the production of immunoparesis by autocannibalization of functional protein with associated morbidity and mortality. Nutritional support in selected patients may help to attenuate these effects. Driven by manufacturers, nutrition hospital services, or "TPN teams," the current trend is toward unnecessary overfeeding of the surgical patient—provoking additional morbidity and costs. Artificial feeding is a double-edged sword. Thus, be selective and cautious.

"Some people never seem able to allow their patients to use the channels designed by nature to receive nourishment. … Food and fluids given by the alimentary canal allow the tissues to select and keep what they want, and to reject what is harmful or surplus to requirements." (William Heneage Ogilvie, 1887–1971)

"In most conditions, foods that agree with the patients may be eaten, those which do not, should not be eaten." (Mark M. Ravitch, 1910–1989)

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# 47 Postoperative Antibiotics

Moshe Schein

*No amount of postoperative antibiotics can compensate for intra-operative mishaps and faulty technique, and they cannot abort postoperative suppuration necessitating drainage.*

## The Issue

Perhaps an issue as apparently banal as postoperative antibiotics does not deserve a separate chapter. Already in Chap. 7 you read about preoperative antibiotics, and in Chap. 12 you were introduced to the concepts of contamination and infection and their therapeutic implications. Why not just administer postoperative antibiotics routinely for any emergency abdominal operation until the "patient is well"? In fact, this is a common practice in the surgical community in this country and around the world: patients receive postoperative antibiotics for many days, many of them are even discharged home on oral agents "just in case". What is wrong with this approach? One important problem with this approach is that thoughtless antibiotic administration has complications that include antibiotic-associated diarrhea, colitis, and the emergence of resistant strains (methicillin-resistant *Staphylococcus aureus* [MRSA] and *Clostridium difficile* colitis are major worldwide health problems). The other problem is cost—not only of the drugs themselves but also of the expense of administration and the treatment of complications. Our aim is to convince you that indiscriminate postoperative antimicrobial administration is **wrong** and to provide guidelines to approach this issue in a more rational way.

Only recently has the topic of duration of administration been addressed in the literature; for years, we endured the common laconic recommendation that antibiotics should be continued until all signs of infection, including fever, leukocytosis, and even ileus subside, and the patient is clinically well. No evidence existed, however, to prove that indeed the continuation of antibiotics along these lines could abort an infection-in-evolution or cure an existing one (Fig. 47.1).

During the last decade, we learned that fever and white cell response are part of the patient's inflammatory response to a variety of infective and noninfective causes. We realized that sterile inflammation is common after any operation, manifesting itself as a local inflammatory response syndrome (LIRS) or a

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**Fig. 47.1.** "This will cure your fever…"

systemic one (SIRS, systemic inflammatory response syndrome) (Chap. 54). Is there a need to administer antibiotics after the bacteria are already dead?

The evolving policy of **minimal antibiotic administration** (strongly supported by the Surgical Infection Society; see Mazuski et al. 2002) represents a trend away from the use of postoperative therapeutic courses of fixed and often long duration; rather, you should attempt to stratify the infective processes into grades of risks and to tailor the duration of administration to the severity of infection.

## Duration of Postoperative Administration

We recommend the policy summarized in Table 47.1. It is based on the following arguments:

- Conditions representing contamination do not require postoperative administration since the infectious source has been dealt with at operation; bacteria and adjuvants of infection are effectively removed by the host's defenses, supplemented by peritoneal toilet, and adequate tissue levels of pre and intraoperative prophylactic antibiotics. By definition, **prophylaxis** should not be continued beyond the immediate operative phase.
- In processes limited to an organ amenable to excision (**resectable infection**), the residual bacterial inoculum is small. A postoperative antimicrobial course of 24 hrs should suffice to sterilize the surrounding inflammatory reaction and deal with gut bacteria, which may have escaped across the necrotic bowel wall by translocation.

**Table 47.1.** Duration of postoperative antibiotic therapy

| |
|---|
| **Contamination: no postoperative antibiotics** (assuming appropriate preoperative prophylaxis administrated) |
| Gastroduodenal peptic perforations operated within 12 hrs |
| Traumatic enteric perforations operated with 12 hrs |
| Peritoneal contamination with bowel contents during elective or emergency procedures |
| Appendectomy for early or phlegmonous appendicitis |
| Cholecystectomy for early or phlegmonous cholecystitis |
| **Resectable infection: 24-hrs postoperative antibiotic course** |
| Appendectomy for gangrenous appendicitis |
| Cholecystectomy for gangrenous cholecystitis |
| Bowel resection for ischemic or strangulated necrotic bowel without frank perforation |
| **"Mild" infection: 48-hrs postoperative antibiotic course** |
| Intra-abdominal infection from diverse sources with localized pus formation |
| "Late" (more than 12 hrs) traumatic bowel lacerations and gastroduodenal perforation with no established intra-abdominal infection |
| **"Moderate" infection: up to 5 days of postoperative antibiotics** |
| Diffuse, established intra-abdominal infection from any source |
| **"Severe" infection: more than 5 days of postoperative antibiotics** |
| Severe intra-abdominal infection with a source not easily controllable (e.g., infected pancreatic necrosis) |
| Postoperative intra-abdominal infection |

– **Nonresectable infections** with a significant spread beyond the confines of the involved organ should be stratified according to their severity. A therapeutic postoperative course of more than 5 days is usually not necessary. However, certain complex situations may need **extended** courses of postoperative antibiotics. A typical example is infected pancreatic necrosis, for which the nidus of infection is not readily eradicated in a once-and-for-all surgical procedure. Similarly, patients with postoperative peritonitis, for which the control of the source of infection is questionable, should be considered for prolonged antibiotic therapy.

It should be quite clear that the commonplace blind, extended antibiotic administration for as long as fever or leukocytosis is present should be abandoned. Pyrexia and white cell response usually represent a sterile, peritoneal (LIRS) or systemic (SIRS), cytokine-mediated, inflammatory response; admittedly, they may on occasions indicate the presence of a focus of persistent or recurrent infection. The former situation is self-limiting and resolves without antibiotics. The latter usually represents suppurative infection, which should be treated by drainage of the intra-abdominal abscess (› Chap. 49) or the infected wound (› Chap. 55). Antibiotic treatment can neither prevent nor treat suppurative infection; it may only succeed in masking it.

By now, you should understand that the persistence of inflammation beyond the appropriate therapeutic course is not an indication to continue, restart, or change antibiotics. What should be avoided is complacent reliance on the advice of the average infectious disease (ID) specialist; this can only lead to an expensive and often unnecessary diagnostic workup and, even more alarmingly, to the prescribing of the latest antibiotic agent on the market (e.g., "dinnericillin," "lunchicillin"). What should instead be done first is to stop the antibiotics. The fever will subside spontaneously in most patients within a day or two with little more than chest physiotherapy. At the same time, a directed search is undertaken for a treatable source of intra or extraperitoneal infection. Surgeons are best placed to anticipate complications in their patients, and this is what is meant by a directed search: a search that is conducted with the full knowledge of the patient's initial disease process, the operative findings, and the natural history of the surgical disease—in brief, a corpus of information that usually eludes the ID specialist.

We have nothing personal against the so-called medical ID specialists, who, at least on this side of the Atlantic, are considered the gurus on antibiotic therapy. But, we have reasons to believe that many of them do not understand the concept of "surgical" infection and how it differs from "medical" infection (see › Table 47.2).

**Table 47.2.** Differences between medical and surgical infections

| Medical infection (e.g., pneumonia) | Surgical infection (e.g., appendicitis) |
|---|---|
| **Not amenable** to surgical source control | **Amenable** to surgical source control |
| Antibiotics mainstay of treatment | Antibiotics only an adjunct to source control |
| A host of potential causative organisms | Predictable causative organisms |
| Prolonged formal course of antibiotics | Antibiotics tailored to operative findings |

So, when was the last time the ID "expert" asked you about your operative findings? And by the way, in a questionnaire study we asked ID specialists whether they would recommend obtaining peritoneal cultures during operation for a "fresh" penetrating wound of the colon; 100% said yes, as if we do not already know the bacterial composition of s***! So, let them focus on HIV and TB ☺

We hope that you realize that unnecessary antibiotics are wrong because anything unnecessary in medicine is bad medicine. In addition, the price to be paid is high, not only financially. Antibiotics are associated with patient-specific adverse effects (the list is long, think of the gravity of *C. difficile* colitis) and ecological repercussions such as drug-resistant nosocomial infections in your hospital.

Are you convinced?

Start antibiotics prior to any emergency laparotomy; whether to continue administration after the operation depends on your findings. Know the target flora and use the cheapest and simplest regimen. The bacteria cannot be confused, and you should not be.

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# 48 Postoperative Ileus Versus Intestinal Obstruction

Moshe Schein · Sai Sajja

*The postoperative fart is the best music to the surgeon's ears.*

Five days ago, you removed this patient's perforated appendix (› Chap. 28); you gave him antibiotics for 2–3 days (› Chap. 47), and by today you expected him to eat (› Chap. 46) and go home. Instead, your patient lies in bed with a long face and a distended abdomen, vomiting bile from time to time. And, the family is asking you what you are asking yourself: what is the problem?

## Definitions and Mechanisms

The term **ileus** as used in this book, and in daily practice, signifies a "paralytic ileus"—the opposite of mechanical ileus, which is a synonym for intestinal obstruction. In essence, the latter consists of a mechanical stoppage to the normal transit along the intestine, whereas the former denotes hindered transit because the intestines are "lazy."

In previous chapters, you noted that ileus of the small bowel, colon, or both can be secondary to a variety of intra-abdominal (e.g., acute appendicitis), retroperitoneal (e.g., hematoma), or extra-abdominal (e.g., hypokalemia) causes that adversely affect normal intestinal motility. Following abdominal operations, however, ileus is a "normal-physiological" phenomenon—its magnitude directly proportional to the magnitude of the operation. In general, the more you do within the abdomen, the more you manipulate, the more prolonged will be the postoperative ileus.

## Ileus

Unlike mechanical intestinal obstruction, which involves a segment of the (small) bowel, postoperative ileus concerns the whole length of the gut, from the

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stomach to the rectum. As mentioned in Chap. 46, physiological postoperative ileus resolves gradually. The small bowel resumes activity almost immediately, followed, a day or so later, by the stomach; the colon, being the laziest, is the last to start moving.

The magnitude of the postoperative ileus correlates to some extent with that of the operation performed and the specific underlying condition. Major dissections, prolonged intestinal displacement and exposure, denuded and inflamed peritoneum, residual intra or retroperitoneal pus or clots are associated with a prolonged ileus. Thus, for example, after simple appendectomy for nonperforated appendicitis, ileus should be almost nonexistent, whereas after a laparotomy for a ruptured abdominal aortic aneurysm (Chap. 41) expect the ileus to be prolonged. Common postoperative factors that can aggravate ileus are the administration of opiates and electrolyte imbalance. **While the "physiological" postoperative ileus is diffuse, ileus due to complications may be local.** A classical example of a local ileus is a postoperative abscess (Chap. 49) that may "paralyze" an adjacent segment of bowel. In another example, a localized leak from an ileo-transverse anastomosis after right hemicolectomy may paralyze the adjacent duodenum, mimicking a picture of gastric outlet obstruction.

## Early Postoperative Mechanical Intestinal Obstruction

You became familiar with small bowel obstruction (SBO) in Chap. 21. Early postoperative SBO (EPSBO) is defined as one developing immediately after the operation or within 4 weeks. Two primary mechanisms are responsible: **adhesions** and **internal hernia.**

Early postlaparotomy **adhesions** are immature, inflammatory, poor in collagen (thus "soft"), and vascular. Such characteristics indicate that early adhesions may resolve spontaneously, and that surgical lysis may be difficult, traumatic to involved viscera, and bloody. Postoperative adhesions may be diffuse, involving the whole length of the small bowel in multiple sites, as is occasionally seen following extensive lysis of adhesions for SBO (Chap. 21). Localized obstructing adhesions may also develop at the operative site with the bowel adherent, for instance, to exposed Marlex mesh or raw peritoneal surface. The operation also may create new potential spaces into which the bowel can herniate to be obstructed, forming **internal hernias**. Typical examples are the partially closed pelvic peritoneum after abdominoperineal resection or the space behind an emerging colostomy. The narrower the opening into the space, the more likely the bowel is to be trapped.

## Diagnosis

**Failure of your patient to eat, fart, or evacuate his or her bowel within 5 days after a laparotomy signifies a persistent ileus.** The abdomen is usually distended

and silent to auscultation. Plain abdominal X-ray typically discloses significant gaseous distension of both the small bowel and the colon (Chaps. 4 and 5). The diagnosis of EPSBO in the recently operated abdomen is much subtler. Textbooks teach you that on abdominal auscultation "ileus is silent and SBO noisy"; this may be theoretically true but almost impossible to assess in the recently operated on belly. *If your patient has already passed flatus or defecated and then ceases to manifest these comforting features, SBO is the most likely diagnosis*. **The truth is that in most instances the patient will improve spontaneously without you ever knowing whether it was an EPSBO or "just" an ileus.**

The natural tendency of the operating surgeon is to attribute the "failure to progress" to an ileus rather than SBO and to procrastinate. Procrastination is not a good idea, however. A distended and noneating patient is prone to the iatrogenic hazards of nasogastric (NG) tubes, intravenous lines, parenteral nutrition, and bed rest (Chap. 45). Be active and proceed with diagnostic steps in parallel to therapy.

## Management

A management algorithm is presented in Fig. 48.1. Pass an NG tube—if not already in situ—to decompress the stomach, prevent aerophagia, relieve nausea and vomiting, and measure gastric residue. Carefully search for and correct, if present, potential causes of prolonged ileus:

- Opiates are the most common promoters of ileus; pain should be controlled but not excessively and for too long.
- Measure and correct electrolyte imbalances.
- Consider and exclude the possibility that an intra-abdominal complication is the cause of the ileus or EPSBO. A hematoma, an abscess, an anastomotic leak, postoperative pancreatitis, postoperative acalculous cholecystitis—all can produce ileus or mimic EPSBO.
- Significant *hypoalbuminemia* leads to generalized edema, also involving the bowel. Edematous and swollen bowel does not move well; this is called *hypoalbuminemic enteropathy* and should be considered.
- Some claim that manual abdominal massage, positional changes, or chewing gum hastens the resolution of ileus. We carry chewing gum in our pockets and distribute it generously to our postoperative patients. Even if it does not alleviate ileus, it will surely promote salivary flow and oral hygiene in the fasting patient and improve his or her mood. (A tiny sip of a single malt Scotch may help too…)

Practically speaking, if on the fifth postlaparotomy day your patient still has features of ileus or EPSBO, we recommend a plain abdominal X-ray to assess the gas pattern (Chaps. 4 and 5). If the X-ray suggests an ileus or EPSBO, a **Gastrografin challenge** as described in Chap. 21 may be useful in relieving both conditions.

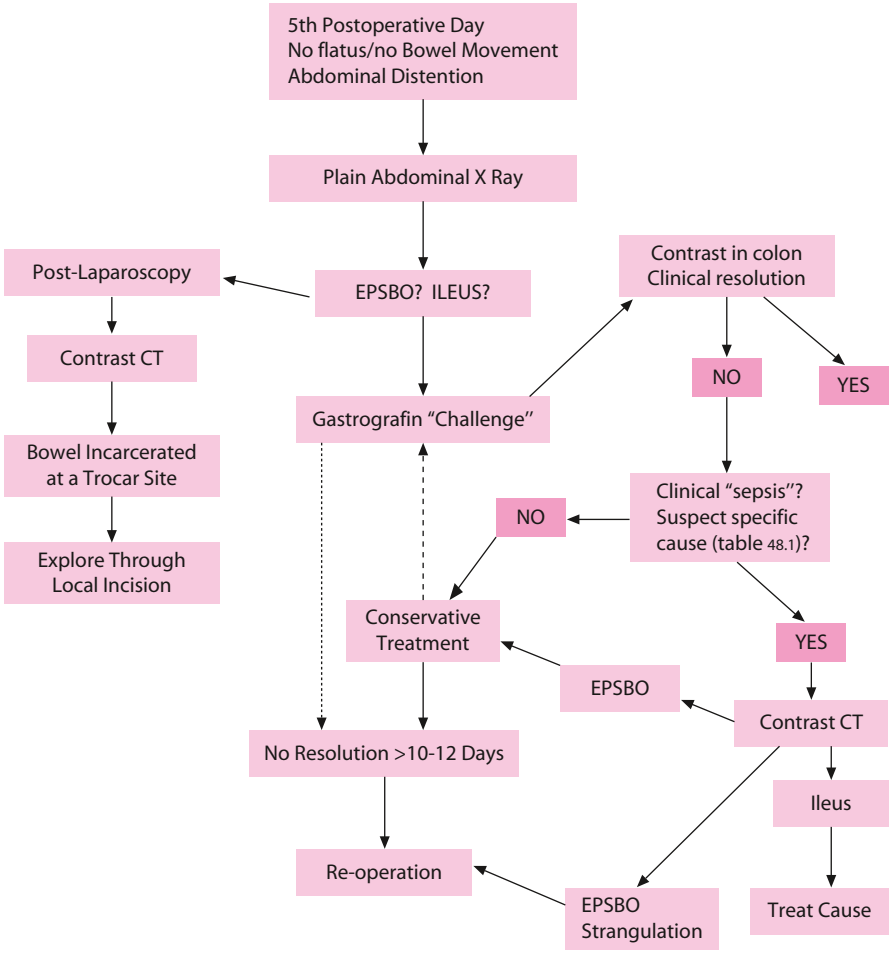


**Fig. 48.1.** Management algorithm

When the clinical picture suggests one of the mentioned intra-abdominal causes of persistent ileus, an **abdominal CT (with oral Gastrografin-no Barium!)** is indicated to pinpoint the problem and, at times, to guide treatment.

Failure of the Gastrografin to arrive at the colon denotes an EPSBO. In the early postoperative phase, this is not an indication for a laparotomy. **Intestinal strangulation almost never occurs in this situation, and spontaneous resolution is common. Resolution of SBO, however, rarely occurs beyond postoperative days 10–12.**

In the absence of intra- or extra-abdominal causes for ileus, and when the "ileus" does not respond to the Gastrografin challenge, the diagnosis is EPSBO. Do not rush to reoperate; treat conservatively while providing nutritional support (Chap. 46). **Lack of resolution beyond 10–14 days is an indication for**

**relaparotomy**, which in itself may be difficult and hazardous because of the typical early dense and vascular adhesions cementing the bowel at many points.

## Specific Considerations

The various primary operations may result in different and specific types of postoperative obstruction as summarized in Table 48.1.

**Table 48.1.** Early postoperative small bowel obstruction (EPSBO): special consideration

| Primary operation | Question | Consideration |
|---|---|---|
| Laparotomy for SBO | Did you deal with the obstructing point? | If not—consider an earlier reoperation |
| Abdominal-perineal resection | Is the small bowel prolapsing into a pelvic space (CT)? | If yes—consider an earlier reoperation |
| Colostomy, ileostomy | Is the small bowel caught behind the stoma (contrast/CT)? | If yes—consider an earlier operation |
| Appendectomy | Is there a pelvic abscess or stump phlegmon? | If yes—consider percutaneous drainage or antibiotics |
| Laparoscopy | Is the bowel caught in a trocar site (CT)? | If yes—operate immediately |
| Radiation enteritis | How severe and extensive was the process?<br>Is it resectable? | If no—consider prolonged nonoperative management |
| Carcinomatosis | How severe and extensive was the process?<br>Is it resectable? | If no—continue prolonged palliative/symptomatic approach |
| "Frozen" abdomen | Was the abdomen "frozen" during index operation? | If yes—consider prolonged nonoperative management |
| Intestinal anastomosis | Anastomotic obstruction: a bowel anastomosis at any level may cause early postoperative upper gastrointestinal, small bowel or colonic obstruction. A self-limiting "mini"-anastomotic leak, associated with local phlegmon, is often responsible but underdiagnosed. Diagnosis is reached with a contrast study or CT. Most such early postoperative anastomotic obstructions are "soft" and edematous—resolving spontaneously within a week or so. | |

### *EPSBO Following Laparoscopy*

Cholecystectomy, transperitoneal hernia repair, and appendectomy are the three most common procedures associated with postlaparoscopic EPSBO. Adhesions are the culprit in half of the patients and small bowel incarceration at the port site in the other half. All port site herniations involve the use of 10- or 12-mm trocars, and the umbilical port is the most common site. In the majority of port site herniations, adequate fascial closure was thought to be achieved at the initial operation. Adequate closure of the fascial defect does not preclude the possibility of trocar site incarceration of bowel: a strangulated Richter's hernia may develop, with the bowel caught in the preperitoneal space behind a well-repaired fascial defect. Another cause for EPSBO following laparoscopic surgery is spilled gallstones during cholecystectomy, which can lead to the development of an inflammatory mass to which the bowel adheres.

Therefore, remember that when EPSBO follows laparoscopy the first question on your mind should be whether the bowel is caught in one of the trocar sites. Because physical findings suggestive of this condition, such as a mass or exceptional tenderness at the trocar site, are rarely present, CT examination of the abdomen is recommended to provide an early diagnosis. CT detects the trocar site responsible for the EPSBO, allowing immediate operation to relieve the obstruction. Surgery can be carried out through the (extended) actual trocar site itself, obviating the need for a formal laparotomy. Unlike EPSBO following open procedures, postlaparoscopy obstruction usually will not resolve without a reoperation. You have to understand that postlaparoscopy EPSBO is a specific entity that calls for immediate action. See also Chaps. 31 and 58.

### *The "Hostile" Abdomen (see also Chap. 21)*

Any mixed series of patients with EPSBO includes a subgroup of patients in whom the index operation has disclosed a "hostile" peritoneal cavity suggesting that any further surgery to relieve the obstructive process would be hazardous and futile. To this group belong patients with **extensive radiation enteritis** in whom persisting obstruction can be defined as "intestinal failure" and who are best managed with long-term parenteral nutrition. Indiscriminate reoperation in such patients often leads to massive bowel resection, multiple fistulas, and death and should be avoided. Patients with evidence of **peritoneal carcinomatosis** at the index operation also belong to this group. In general, only one-third of patients with "malignant" bowel obstruction from peritoneal carcinomatosis will have prolonged postoperative palliation. Thus, EPSBO in such patients is an ominous sign; abdominal reoperation should be avoided and future palliative treatment planned based on the individual patient's functional status and the burden

of cancer. Finally, every surgeon has some personal experience with a little reported entity, the *frozen abdomen*, in which intractable SBO is caused by dense, vascular, and inseparable adhesions—fixing the bowel at many points. The astute surgeon knows when to abort early from a futile dissection before multiple enterotomies—necessitating massive bowel resection—are created. This surgeon also knows not to reoperate on such patients even if persisting EPSBO develops after what appeared to be a successful adhesiolysis. Prolonged parenteral nutrition over a period of months, with complete gastrointestinal rest, may allow the adhesions to mature—with resolution of the SBO or at least allowing a safer reoperation.

### *Anastomotic Obstruction*

A bowel anastomosis at any level may cause early postoperative upper gastrointestinal, small bowel, or colonic obstruction. Faulty technique (Chap. 13) is usually the cause. A self-limiting "mini"-anastomotic leak is often responsible but underdiagnosed (Chap. 50). Diagnosis is reached with a contrast study (water-soluble please) or CT. Most such early postoperative anastomotic obstructions are "soft" and edematous, resolving spontaneously within a week or two. Do not rush to reoperate; gentle passage of an endoscope—if practical—may confirm the diagnosis and "dilate" the lumen.

### *Delayed Gastric Emptying*

Often, the stomach fails to empty following a partial gastrectomy or a gastrojejunostomy performed for any indication. This is more common when a vagotomy has been added or when a Roux-en-Y loop has been constructed. A Gastrografin study will show that the contrast persistently sits in the stomach. The differential diagnosis is between a gastric ileus (gastroparesis) and mechanical obstruction at the gastrojejunostomy or *below* it (yes, do not miss the mechanical obstruction in the small bowel just "below" the stomach). A complete discussion of the various postgastrectomy syndromes is beyond the scope of this volume, but remember this fundamental principle: **postoperative gastric paresis is self-limiting**—it will always resolve spontaneously but may take as long as 6 weeks to do so. Exclude mechanical stomal obstruction with an endoscope and contrast study and then treat conservatively with NG suction and nutritional support. Try to pass a feeding tube distal to the stomach (Chap. 46). Parenteral *erythromycin* has been shown to enhance gastric motility and is always worth a trial in this situation. Resist the devil within you—tempting you to reoperate for gastric paresis for it will eventually resolve, while reoperation may only make things worse.

## Prevention

It is imperative to emphasize that you can, and ought to, prevent prolonged postoperative ileus or SBO by sound operative technique and attention to detail. Gentle dissection and handling of tissues, careful hemostasis to avoid hematoma formation, not using the cautery like a blowtorch, leaving as little foreign material as possible (e.g., large silk knots, spilled gallstones during laparoscopic cholecystectomy), not denuding the peritoneum unnecessarily, not creating orifices for internal hernias, carefully closing large port sites, and not catching loops of bowel during abdominal closure are self-explanatory essentials. We are not yet too impressed with the evidence supporting recently developed expensive commercial products that allegedly "prevent adhesions."

**Summary:** exclude and treat causes of persistent ileus, treat EPSBO conservatively as long as indicated, think about specific causes of SBO (e.g., herniation at a laparoscopic trocar site), and reoperate when necessary. In most instances, ileus or EPSBO will resolve spontaneously (› Fig. 48.2).

Better to leave a piece of peritoneum on the bowel than a piece of bowel on the peritoneum.

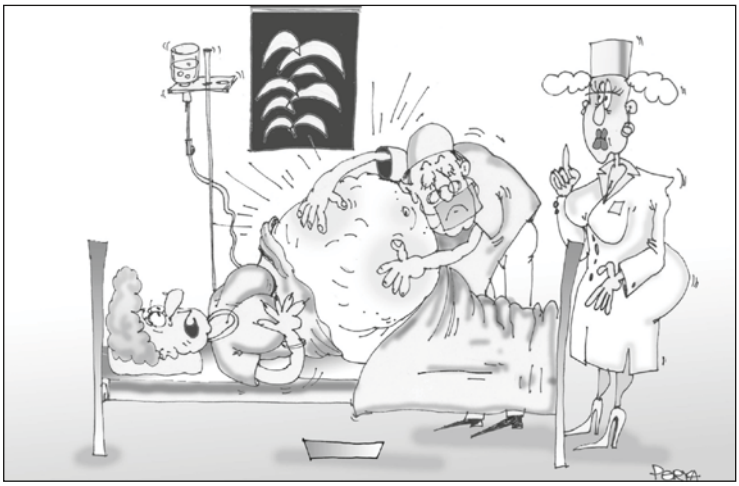

**Fig. 48.2.** "Doctor, is it mechanical obstruction or ileus?" … "Shh … let me hear."

# 49 Intra-Abdominal Abscesses

Moshe Schein

*"Signs of pus somewhere, signs of pus nowhere else, signs of pus there—under the diaphragm." This was 100% true when I was a student, 50% true when I was a resident. Today, it is irrelevant…*

The contents of this chapter could have been summarized in a sentence: an abscess is a pus-containing, confined structure that requires drainage by whichever means available. We believe, however, that you want us to elaborate.

Abscesses may develop anywhere within the abdomen, resulting from numerous conditions. Specific types such as diverticular or periappendicular abscesses (Chap. 26 and 28) are covered elsewhere in this book; this chapter introduces you to general concepts—with emphasis on what is probably the most common abscess in your practice: the **postoperative abscess**.

## Definition and Significance

Erroneously, the term *intra-abdominal abscess* has been and still is used as a synonym for secondary peritonitis (Chap. 12). This is not true as abscesses develop as a result of effective host defenses and represent a relatively successful outcome of peritonitis.

To be termed an abscess, the confined structure has to be walled off by an inflammatory wall and possess a viscous interior. In contrast, free-flowing, contaminated, or infected peritoneal fluid or loculated collections, which are deprived of a wall, represent a phase in the spectrum/continuum of peritoneal contamination/infection and not an abscess.

## Classification and Pathogenesis

The myriad forms of intra-abdominal abscesses make their classification complex (Table 49.1), but practically, abscesses are **visceral** (e.g., intrahepatic or splenic) or **nonvisceral** (e.g., subphrenic, pelvic), **intraperitoneal** or **extraperitoneal**.

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**Table 49.1.** Classification of abdominal abscesses

| Classification | Examples |
|---|---|
| Visceral vs. nonvisceral | Hepatic vs. subphrenic |
| Primary vs. secondary | Splenic vs. appendiceal |
| Spontaneous vs. postoperative | Diverticular vs. perianastomotic |
| Intraperitoneal vs. retroperitoneal | Tubo-ovarian vs. psoas |
| Simple vs. complex | Complex |
| | Multiple (liver) |
| | Multiloculated |
| | Communication with bowel (leaking anastomosis) |
| | Associated with necrotic tissue (pancreatic) |
| | Associated with cancer |
| Anatomical | Subphrenic, subhepatic, lesser sac, paracolic, pelvic, interloop, perinephric, psoas |

**Nonvisceral abscesses** arise following the resolution of diffuse peritonitis during which loculated areas of infection and suppuration are "walled off" and persist or arise after a perforation of a viscus, which is effectively localized by peritoneal defenses. **Visceral abscesses** are caused by hematogenous or lymphatic dissemination of bacteria to a solid organ. **Retroperitoneal** abscesses may result from perforation of a hollow viscus into the retroperitoneum as well as by hematogenous or lymphatic spread. Another distinction is between the **postoperative abscess**—for the development of which we surgeons feel responsible—and **spontaneous abscesses**, unassociated with a previous operation. A further clinically significant separation is between **simple abscesses** and **complex abscesses** (e.g., multiple; multiloculated ones; or associated with tissue necrosis, enteric communication or tumor), which require a more aggressive therapy and carry a poorer prognosis. The **anatomical classification**, based on the specific anatomical location of an abscess, which typically develops in one of the few constant potential spaces, has diminished in significance since the advent of readily available modern imaging and percutaneous (PC) drainage techniques.

Note that abscesses signify an **intermediate** natural outcome of contamination/infection. At one end of the spectrum infection persists, spreads, and kills; at the other, the process is entirely cleared by host defenses—assisted by your

therapy. Abscesses lie in no-man's-land, where the peritoneal defenses are only partially effective—being disturbed by an overwhelming number of bacteria, microenvironmental hypoxemia or acidosis, and adjuvants of infection such as necrotic debris, hemoglobin, fibrin, and barium sulfate. An untreated abdominal abscess will not kill your patient immediately, but if neglected and undrained, it will become gradually lethal unless spontaneous drainage occurs.

## Microbiology

**Generally, abdominal abscesses are polymicrobial.** Abscesses that develop in the aftermath of secondary peritonitis (e.g., appendiceal or diverticular abscess) possess the mixed aerobic-anaerobic flora of secondary peritonitis (Chap. 7 and Chap. 12). It appears that while endotoxin-generating facultative anaerobes, such as *Escherichia coli*, are responsible for the phase of acute peritonitis, the obligate anaerobes, such as *Bacteroides fragilis*, are responsible for late abscess formation. These bacteria act in **synergy**; both are necessary to produce an abscess, and the obligate anaerobe can increase the lethality of an otherwise nonlethal inoculum of the facultative microorganisms. The vast majority of visceral abscesses (e.g., hepatic and splenic) are polymicrobial—aerobic, anaerobic, Gram-negative, and Gram-positive. This is also true for retroperitoneal abscesses. Primary abscesses (e.g., psoas abscesses) often are monobacterial, with staphylococci predominating. Postoperative abscesses are often characterized by the flora typical of tertiary peritonitis, representing superinfection with yeasts and other opportunists (Chap. 54). The low virulence of these organisms, which probably represent a *marker* rather than a cause of tertiary peritonitis, reflects the global immunodepression of the affected patients.

## Clinical Features

The clinical presentation of abdominal abscesses is as heterogeneous and multifaceted as the abscesses themselves. The spectrum is vast; systemic repercussions of the infection vary from frank septic shock to nothing at all when suppressed by immunoparesis and antibiotics. Locally, the abscess may be felt through the abdominal wall, the rectum, or the vagina; in most instances, however, it remains physically occult. In our modern times, when any fever is an alleged indication for antibiotics, most abscesses are initially partially treated or masked, presenting as a systemic inflammatory response syndrome (SIRS) with or without multiorgan dysfunction (Chap. 54). Ileus is another not uncommon presentation of abdominal abscess; in the postoperative situation, it is an "ileus that fails to resolve" (Chap. 48).

## Diagnosis

Life has become simple. Modern abdominal imaging has revolutionized the diagnosis of abdominal abscesses. Yes, you still need to suspect the abscess and carefully examine your patient, but the definitive diagnosis (and usually the treatment) depends on imaging techniques. Computed tomography (CT), ultrasound (US), and various radioisotope-scanning techniques are available. Which is the best?

Radioisotope scanning, regardless of the isotope used, does not provide any anatomical data beyond vague localization of an inflammatory site; it is not accurate enough to permit PC drainage. The usefulness of these methods is limited, therefore, to the continuous survival of nuclear medicine units and an excuse to publish papers (Nuclear medicine = Unclear medicine). Practically, these tests have no role at all. Both US and CT provide good anatomical definition, including the site, size, and structure of the abscess; both can guide PC drainage. US is portable, cheaper, and more accurate at detecting abscesses in the right upper abdomen and pelvis. It is, however, extremely operator dependent. We surgeons are better trained to read CT scans rather than US; hence, we prefer CT, which allows us to visualize the entire abdomen, independently assess the anatomy of the abscess, and plan its optimal management. *CT, enhanced with intravenous and intraluminal contrast, is also helpful in classifying the abscess either as simple or complex* (› Table 49.1).

It appears that performing multiple tests—adding a CT to a US—is not productive. Do understand that CT or US scanning during the very early postoperative days is futile because neither technique can distinguish between a sterile fluid collection (e.g., residual lavage fluid) and an infected fluid collection before the development of a frank, mature abscess. The only way to document the infective nature of any visualized fluid is a **diagnostic aspiration**, subjecting the aspirate for a Gram stain and culture. CT features suggestive of a proper abscess are a contrast-enhancing, well-defined rim and the presence of gas bubbles. Please bear in mind that not all fluid collections that are detected in the postoperative abdomen require active management; be guided by the patient's clinical condition at all times. Be resistant to the offers of aggressive radiologists to drain all accessible collections, particularly in the early post-op period.

## Treatment

Abdominal abscesses should be drained; when an "active" source exists, deal with it. Antibiotic treatment is of marginal importance.

### *Antibiotics*

The truth is that no real evidence exists to prove that antimicrobial agents, which penetrate poorly into established abscesses anyway, are necessary as an adjunct to the complete evacuation of pus. Think about the good old days, not many years ago, when pelvic abscesses were observed until reaching "maturity" and then drained through the rectum or the vagina; no antibiotics were used, and the recovery was immediate and complete. The prevalent "standard of care," however, although lacking evidence, maintains that when an abscess is strongly suspected or diagnosed, then antibiotic therapy should be initiated. The last should initially be empirically targeted against the usual expected polymicrobial spectrum of bacteria; when the causative bacteria are identified, the coverage can be changed or reduced as indicated.

**How long to administer antibiotics?** Again, there are no scientific data to formulate logical guidelines. Common sense dictates that prolonged administration after effective drainage is unnecessary. Theoretically, antibiotics may combat bacteremia during drainage and eradicate locally spilled micro-organisms, but after the pus has been evacuated, leading to a clinical response, antibiotics should be discontinued. The presence of a drain is not an indication to continue with administration.

### *Conservative Treatment*

Traditionally, multiple hepatic abscesses, as a consequence of portal pyemia, which are not amenable to drainage, are treated with antibiotics, with a variable response rate. There are those who claim that nonoperative treatment, with prolonged administration of antibiotics, is also effective in children who develop abdominal abscesses following appendectomy for acute appendicitis. The problem with such "successes" is that the alleged "abscesses," which were imaged on US or CT, were never proven as such. Instead, they probably represented sterile collections—the majority requiring no therapy at all—or early, unwalled, infected fluid collections into which antibiotics do penetrate. In addition, small (<5 cm) pericolic "diverticular" abscesses can be resolved with antibiotics only—without the need for drainage (Chap. 26).

### *Drainage*

**Philosophy and timing.** Presently, the prevailing paradigm when an abscess is suspected on a CT or US is to hit the patient with antibiotics and rush to drainage. In this hysterical hurry to treat, clinical lessons learned over centuries are often

ignored. Only a generation ago, a patient who spiked a temperature after an appendectomy was patiently but carefully observed without antibiotics (which did not exist); usually, the temperature, signifying residual local inflammatory response syndrome (LIRS) (Chap. 54), subsided spontaneously. In a minority of patients, "septic" fever persisted, reflecting maturing local suppuration. The last was eventually drained through the rectum when assessed as mature. Today, on the other hand, antibiotics are immediately given to mask the clinical picture, and imaging techniques are instantly ordered to diagnose "red herrings," which in turn promote unnecessary invasive procedures. Remember, in a stable patient fever is a *symptom* of effective host defenses, not an indication to be aggressively invasive (Chap. 45).

## Practical Approach

When an abscess is suspected, a few dilemmas arise and should be dealt with stepwise:

- **Is it an abscess or a sterile collection?** The aforementioned CT features may be helpful, but the clinical scenario is as important, especially if postoperative abscesses are concerned. Abscesses are rarely ready for drainage in the first postoperative week, and 3 weeks after the operation the cause of sepsis is rarely within the abdomen. When in doubt, image-guided diagnostic aspiration is indicated.
- **PC versus open surgical drainage.** During the 1980s multiple retrospective series suggested that the results of PC drainage are at least as good as those achieved by an operation. It was also said by some that, paradoxically, despite the attractiveness of a PC technique for abscess drainage in the most ill patients, a better chance of survival is achieved with surgical treatment, and that surgical treatment should not be avoided because the patient is considered to be too ill. Be that as it may, there is no clear evidence to attribute lesser mortality or morbidity to PC drainage versus surgical drainage. The former, however, is a minimal access procedure that can spare the patient the unpleasantness and obvious risks of yet another open abdominal operation.
- **The concept of a complex abscess is clinically useful.** Abscesses that are multiple, multiloculated, and associated with tissue necrosis, enteric communication, or tumor are defined as **complex** and are less likely to respond to PC drainage, whereas most **simple** abscesses do. However, in gravely ill patients with **complex** abscesses, PC drainage may offer significant **temporizing** therapeutic benefits, allowing a definitive semi-elective laparotomy in better-stabilized patients.
- **It appears that PC drainage and surgical drainage techniques should not be considered competitive but rather complementary.** If an abscess is accessible by PC technique, it is reasonable to consider a nonoperative approach to the problem. You, the surgeon, should consider each abscess individually together with the radiologist, taking into the consideration the "pros and cons" presented in Table 49.2.

**Table 49.2.** Intra-abdominal abscesses: percutaneous (PC) vs. open surgical drainage. Considerations in selecting the approach

| | PC drainage | Open drainage |
|---|---|---|
| Surgical accessibility | Hostile abdomen | Accessible |
| PC accessibility | Yes | No |
| Source controlled | Yes | No |
| Location | Visceral | Interloop |
| Number | Single | Multiple |
| Loculation | No | Yes |
| Communication with bowel | No | Yes |
| Associated necrosis | No | Yes |
| Associated malignancy | No | Yes |
| Viscosity | Thin | Thick debris |
| Invasive radiologist | Available | Not available |
| Severity of illness | "Stable" | Critically ill |
| Failed PC drainage | No | Yes |

- **PC aspiration only versus catheter drainage?** A single PC needle aspiration may successfully eradicate an abscess, especially when it is small and contains low-viscosity fluid. There is good evidence, however, that PC catheter drainage is more effective.
- **Size of PC catheters or drains?** Some claim advantage for large-bore trocar catheters for PC drainage, but the evidence indicates that size 7F PC sump drains are as effective as size 14 F.
- **Management of PC drains.** There is not much science here; these are small tubes and should be regularly flushed with saline to remain patent. The drain site should be regularly cleaned and observed; there are single case reports of necrotizing fasciitis of the abdominal wall around a PC drain site. PC drains are removed when clinical SIRS has resolved and the daily output (minus the saline injected) is below 25 ml. On average, after PC drainage of a simple abdominal abscess, the drain is removed after 7 days.
- **Re-imaging.** Clinical improvement should be seen within 24–72 hrs following PC drainage. Persistent fever and leukocytosis on the fourth day after PC drainage correlates with management failure. Nonresponders should be re-imaged with CT

combined with water-soluble contrast injected through the drain. Depending on the findings, a decision should be made by you—the surgeon—in consultation with the radiologist regarding the next appropriate course of action: a re-PC drain or an operation. Persistence of high-output drainage in a patient who is clinically well can be better investigated with a tube sinogram to delineate the size of the residual abscess cavity. Abscess cavities that do not collapse commonly tend to recur.

#### *Failure of PC Drainage: When to "Switch Over" to Surgical Drainage?*

Patients who deteriorate after the initial attempts at PC drainage should be operated on promptly; further procrastination may be disastrous.

In stable nonresponders to the initial PC drainage, a second attempt may be appropriate according to the considerations mentioned in Table 49.2. Inability successfully to effect the second PC drainage, or its clinical failure, mandates an open procedure.

### Surgical Management of Intra-abdominal Abscesses

About a third of intra-abdominal abscesses are not suitable for PC drainage and require an open operation. A few practical dilemmas exist:

- **Exploratory laparotomy versus direct surgical approach**. A "blind" exploratory laparotomy to search for an abscess "somewhere," so common less than 20 years ago, is currently very rarely necessary. A direct approach is obviously more "benign," sparing the previously uninvolved peritoneal spaces and avoiding bowel injury and wound complications. It is almost always possible in spontaneous abscesses, which are so well defined on CT. But, those are also the kind of abscesses that usually respond to PC drainage. Now, although postoperative abscesses are anatomically well localized on CT, those that fail PC drainage are usually "complex" and therefore are often not amenable to a direct approach (e.g., interloop abscess) or require additional procedures to control the intestinal source. Criteria for choosing the correct approach are summarized in Table 49.3.
- **Direct approach: extraperitoneal versus transperitoneal?** There are no significant differences in overall mortality and morbidity between the two approaches; however, the transperitoneal route is associated with a higher incidence of injury to the bowel. It is logical to suggest that the extraperitoneal approach should be used whenever anatomically possible. Subphrenic and subhepatic abscesses can be approached extraperitoneally through a subcostal incision or—if posterior—through the bed of the 12th rib. Old-timers are still familiar with these techniques, which are currently rarely utilized, having been replaced by PC drainage. Pericolic,

**Table 49.3.** Exploratory laparotomy vs. "direct" open drainage of abdominal abscesses

| | Exploratory laparotomy | Direct open drainage |
|---|---|---|
| Abscess accurately localized on CT | – | √ |
| Early postoperative phase | √ | – |
| Late postoperative phase | – | √ |
| Single abscess | – | √ |
| Multiple abscesses | √ | – |
| Lesser sac abscess | √ | – |
| Interloop abscess | √ | – |
| Source of infection uncontrolled | √ | – |
| Subphrenic/subhepatic | – | √ |
| Gutter abscess | – | √ |
| Pelvic abscess | – | √ |

appendicular, and all sorts of retroperitoneal abscesses are best approached through a loin incision. Late-appearing pancreatic abscesses (› Chap. 19) also can be drained extraperitoneally—through the flank—but occasionally need a bilateral approach. Pelvic abscesses are best drained through the rectum or vagina.

**Drains?** Classically, at the end of the open procedure a drain has been placed within the abscess cavity, brought to the skin away from the main incision. The type, size, and number of drains used depended more on local traditions and preferences than on science. Similarly, the postoperative management of drains involved cumbersome rituals with the drains sequentially shortened, based on serial contrast sinograms, to ascertain the gradual collapse of the cavities and drain tracts. House surgeons and nurses forever changed dressings and irrigated the drains, again according to the locally prevailing ritual. Our experience is that this elaborate nonsense should belong to history. With adequate surgical drainage, when the source of infection has been controlled, when the abscess cavity is "filled" with omentum or adjacent structures, and prophylactic perioperative antibiotics are administered, no drains are necessary. Trust the peritoneal cavity to deal with the residual bacteria better in the absence of a foreign body—the drain. We do not recall the last time we had to "shorten" a drain or to obtain a drain sinogram. Oh,

the sweet memories of naïve youth. For a more detailed discussion of "surgical drainage," go to Chap. 42.

## Summary

Tailor your approach to the anatomy of the abscess, the physiology of the patient, and the local facilities available to you. Do not procrastinate, do not forget to deal with the source, do not over-rely on antibiotics, and get rid of the pus. Sepsis, the host-generated systemic inflammatory response to the abscess, may persist, and progress to organ failure, even after the abscess has been adequately managed (Chap. 54). Try not to be too late.

> "No drainage is better than the ignorant employment of it. … A drain invariably produces some necrosis of the tissue with which it comes in contact, and enfeebles the power of resistance of the tissues toward organisms." (William Stewart Halsted, 1852–1922)

# 50 Anastomotic Leaks and Fistulas

Moshe Schein

*If there is a possibility of several things going wrong, the one that will cause the most damage will be the one to go wrong.* (Murphy's Law, Arthur Bloch)

*Somebody's leak is a curiosity—one's own leak is a calamity.*

There are two chief clinical patterns of postoperative intestinal leak:

- The leak is obvious: you see intestinal contents draining from the operative wound or from the drain site (if a drain was used).
- You suspect a leak but do not see one.

## Scenario 1: The Obvious Leak

It is postoperative day 6 after a laparotomy for small bowel obstruction (Chap. 21). The procedure was uneventful except for two accidental enterotomies, which were closed with interrupted vicryl 3–0 in one layer. During morning rounds, the patient complains: "Look, doctor, my bed is full of this green stuff." You uncover the patient's abdomen to see bile-stained intestinal juice pouring through the incision. Now, you are very upset. True, the patient's recovery was not smooth; he was running a fever and a high white cell count—and now this terrible disaster. It is a disaster indeed, for even today up to one-third of patients with intestinal suture line breakdown die.

Your first reaction is: "Let's get him immediately to the operating room and fix this mess." Is this advisable?

### *The Controversy*

There is little controversy that established postoperative external enterocutaneous fistulas, which usually result from leaking anastomoses or incidental enterotomies, should initially be managed *conservatively*. As noted in previous chapters, there is also little controversy that acute gastrointestinal perforation, whether spontaneous or traumatic, is an indication for an emergency laparotomy to deal with the source of contamination/infection (Chap. 12).

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So, what about the "early postoperative small bowel leakage"? Is it like a "simple perforation" requiring an immediate operation, or like a "fistula" to be managed conservatively? We contend that this scenario represents both conditions and should therefore be managed selectively in the individual patient.

### *The Role of Non-operative Management*

With proper supportive management and in the absence of distal obstruction or loss of bowel continuity, about a third of postoperative **small bowel fistulas** will close spontaneously within 6 weeks. Those that fail to close by this time will require elective reoperation. This, when performed on an anabolic, non-SIRS (systemic inflammatory response syndrome) patient, in a less hostile peritoneal environment, will restore the integrity of the gastrointestinal tract with an acceptable risk of complications.

A crucial issue when deciding on a trial of conservative management is the presence or absence of peritonitis or sepsis; clinical peritonitis is an indication for an immediate operation. Even when clinical peritonitis is not present, any evidence of SIRS or sepsis should trigger an aggressive search for drainable intra-abdominal pus. This is best done with a computed tomographic (CT) scan; associated abscesses should be drained percutaneously (PC) or at laparotomy (Chap. 49).

**Remember: in unselected series of postoperative enterocutaneous fistulas, a third of patients die—the vast majority from neglected intra-abdominal infection.**

### *The Role of Early Operative Management*

As stated, peritonitis or a complex intra-abdominal abscess not suitable for, or not responding to, PC drainage are indications for laparotomy. But, why not operate on all such patients? Why not just surrender to the temptation buzzing in your brain: "I know where this leak is coming from; let me just return to that abdomen and fix this frustrating problem with a few more sutures"? Why won't resuturing the leak solve the problem?

**Because primary closure of a disrupted intestinal suture line is doomed to fail.**

We can all remember an isolated success in closing an intestinal leak (see specific indications to do so in this chapter), but the collective experience points to an overwhelmingly high rate of failure. Attempts to close an intestinal leak, after a few days, in an infected peritoneal cavity are doomed to fail. Redoing an intestinal anastomosis in the presence of postoperative peritonitis is an exercise in futility. Obviously, if successful the surgeon is a hero who either saves the patient's life or at least prevents prolonged hospitalization and morbidity. If, however, a leak redevelops, as it usually does, it produces a tremendous "second hit"—added to

the insult of the reoperation—which strikes an already primed, susceptible, and compromised host (› Chap. 54). Sepsis and death are then almost inevitable.

### *Suggested Approach to Early Leaks/Postoperative Intestinal Fistula*

**Trial of conservative management is warranted when:**

- There is no clinical peritonitis.
- There are no associated abscesses on CT, and you know the leak is "controlled."

**An immediate relaparotomy is warranted when:**

- There is evidence of clinical peritonitis.
- There is SIRS/sepsis with proven or suspected intraperitoneal abscesses that cannot be drained PC.
- Somebody you do not trust, or know, performed the primary, "index," operation, and you cannot obtain accurate information about what was done. Bitter experience has taught us that in such (transferred from elsewhere) patients "anything is possible," and it is better to reoperate; you never know what the findings will be.

### *What to Do During an Emergency Re-laparotomy?*

There are three things to consider during an emergency re-laparotomy: (1) the condition of the bowel, (2) the condition of the peritoneal cavity, and (3) the condition of the patient.

**Very rarely** in a stable, minimally compromised patient, when peritonitis is macroscopically minimal, when the bowel appears of "good quality," when the patient's serum albumin levels are reasonable, we would resect the involved segment of small bowel and reanastomose. Such a sequence of events is possible only when the leak presents within a day or two after the operation (usually caused by a technical mishap). An immediate reoperation before local and systemic adverse repercussions develop may thus provide definitive cure. Other circumstances when attempts at repair of the leak are reasonable would be during early reoperations for upper gastrointestinal leaks (e.g., following bariatric operations; › Chap. 31) for which exteriorization of the leaking part is impossible. So, one tries to patch, and one leaves a drain: if the leak redevelops—and usually it does—one hopes at least to establish a controlled fistula.

In all other circumstances, the less-heroic but logical and lifesaving option of exteriorization—if technically possible—of the leaking point as an enterostomy should be carried out, and at **any level**.

### *Conservative Management*

The principles of conservative management are few and simple:

- **Provide aggressive supportive care.**
- **Restore fluid and electrolyte balance.** All the fistula's losses should be measured and replaced.
- **Exclude and treat associated infection**. This has been mentioned and is repeated here only to emphasize that when your fistula patient dies it is usually because you were not aggressive enough in pursuing our advice. Try to do it PC. If an operation is needed, try to drain the abscess through a direct, local approach, avoiding the risks of a "complete" laparotomy (› Chap. 49).
- **Protect the skin** around the fistula from the corrosive intestinal juice. A well-fitting colostomy bag around the fistula often does the trick. Otherwise, place a tube connected to a continuous suction source adjacent to the fistula, place stom-adhesive sheaths around the defect, and cover the entire field with an adhesive transparent dressing (similar to the "sandwich" described in › Chap. 52.2). Make generous use of Karaya or zinc paste to protect the skin around difficult-to-manage, complex fistulas. Although such wounds require lots of effort and dedication, they are almost always manageable—but only if you care. The way the abdominal wall of your fistula patient looks is how you look!
- **Provide nutrition**. Proximal gastrointestinal fistulas require total parenteral nutrition (TPN) initially until a nasal feeding tube is inserted beyond the leak level. Distal small bowel and colonic fistulas will close spontaneously whether the patient is fed orally or not. As emphasized in › Chap. 46, using the intestine for feeding, if possible, is better. In proximal fistulas, it is often possible, and beneficial, to collect the fistula's output and reinfuse it, together with the enteral diet, into the bowel below the fistula.
- **Delineate anatomy**. This is best done with a sinogram, injecting water-soluble contrast into the fistula tract. This will document the level of the bowel defect and, it is hoped, the absence of distal obstruction or loss of continuity—prerequisites for successful conservative management.
- **Strive to achieve spontaneous closure**, the likelihood of which depends on the site and anatomy of the fistula; this should be possible in approximately one-third of patients.
- **Proceed with surgical closure when indicated**, but delay it when the patient and the patient's abdominal wall and peritoneal cavity are not ready, usually not within 6 weeks.
- **Refer the patient to a specialized** center if your own setup is unable to cope with the demanding care of fistula patients.

### *Gimmicks*

The initial output of a fistula has few prognostic implications. A fistula that drains 1,000 ml/day during the first week has the same chance of spontaneously

sealing as one with an output of 500 ml/day. Artificially decreasing a fistula's output with total starvation and administration of a somatostatin analogue is cosmetically appealing but not proven to be beneficial.

In patients with a well-established (and long) fistula tract (which takes a few weeks to develop), it is possible to hasten the resolution of the fistula by **blocking the tract.** Many "innovative" methods have been reported as successful (usually in small series of patients), ranging from the injection of fibrin glue (through a fiberscope) deep into the tract, to plugging the tract's orifice with chewing gum (chewed by the patient, not by you…).

### *Fistula Associated with a Large Abdominal Wall Defect*

Not uncommonly, the end result of intestinal leaks and reoperative surgery is an abdominal wall defect with multiple intestinal fistulas in its base. **This so-called complex or type IV fistula is a catastrophe that carries a very high mortality rate.** (According to our classification [Schein and Decker 1991], type I are foregut fistulas; type II, small bowel; and type III, colonic.) With growing popularity of open abdomen management techniques, these types of fistula are observed with increasing frequency. The distance of the fistulous opening in the intestine from the surface of the defect and the condition of the peritoneal cavity have crucial bearing on the treatment of this condition. It is practical to distinguish between two situations (Fig. 50.1):

- **Type IV-A fistulas**. This is a scenario occurring early after the development of the intestinal leak. Here, the fistula is located in the depths of the abdominal wall defect, resulting in prolonged contact of large peritoneal surfaces with gastrointestinal contents, allowing increased absorption of toxic products, thus perpetuating local and systemic inflammatory responses and organ dysfunction.

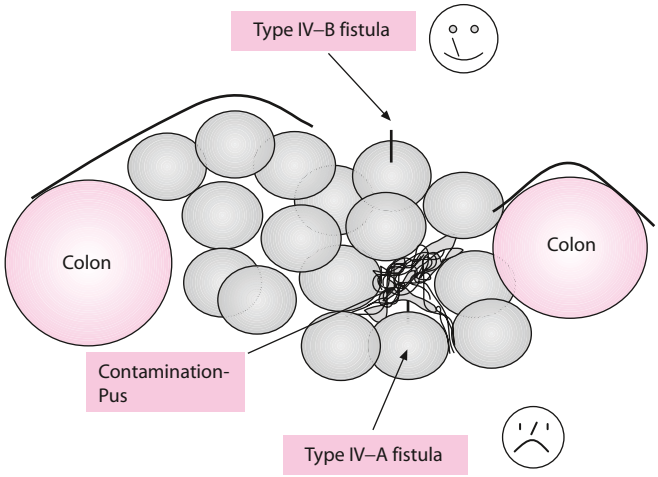


**Fig. 50.1.** Type IV-A fistulas versus type IV-B fistulas

In such instances, reoperation is necessary to exteriorize or divert the intestinal leak away from the defect. Otherwise, the patient is doomed as more than half of the patients with this type of postoperative fistula die!

- **Type IV-B fistulas.** This is a late phenomenon in the natural history of leakage. These are "exposed" fistulas near the surface of the defect. Also called "bud" or "exposed" fistulas, they result from damage to matted intestine that forms the "bed" of the defect (the so-called central visceral block). Because the peritoneal cavity is usually clean and sealed away from intestinal contents, the patient is free of SIRS or "sepsis," but the management of such fistulas needs a lot of your ingenuity.

#### Management

- The aim in general is to *convert* the life-threatening type IV-A fistula to a problematic but not immediately life-threatening type IV-B fistula by diversion or exteriorization. Once the intestinal effluent stops pouring into the general peritoneal cavity, one is left only with the prolonged management of the "exposed" fistula.
- With type IV-B fistula, the immediate task is to control the output of the fistula. Use your creative skills to construct a sealed vacuum dressing of your choice to cover the whole defect—sucking out the fistula's effluent. We use a modification of our sandwich (Chap. 52.2). Others would suture a large colostomy bag all around the rim of the fistula (bag enterostomy), placing a suction tube into the bag.
- A tiny "exposed-bud" fistula may be dealt with temporarily (until definitive reconstruction) using the following technique: define the mucosal and submucosal layers of the pouting intestinal hole; close it with a fine monofilament suture. Immediately cover the repaired bowel and the surrounding abdominal wall defect with a split-thickness skin graft. This should be successful in half of your attempts. Some would apply fibrin glue between the sutured intestine and skin graft. Other surgeons[1] try to patch such holes with human acellular dermal matrix (Alloderm) and fibrin glue.
- Such simple measures are impossible when the exposed fistula is large (as large as a colostomy). Such fistulas will not heal if not covered with well-vascularized tissue, whether skin flaps or musculofascial flaps (for a good discussion about "fancy" options in the management of "exposed" fistulas, look at Jamshidi and Schecter, 2007).
- In most such patients, however, you will have to control the fistula, support the patient, wait for the abdominal wall defect to contract, wait for resolution of the peritoneal inflammation, wait for maturation of intra-abdominal adhesions, and only then—after at least 6 months and usually more than that—consider "take down" of the fistulas and abdominal wall reconstruction. A simple rule of thumb is that the condition of the abdominal wall defect reflects the condition of the peritoneal cavity. A well-contracted abdominal wall defect and fistulas that look like surgical stomas are indicators that an elective intervention is possible and safe (Chap. 52).

**Remember: the key term in such patients is WWW—wait, wait, and wait!**

### Scenario 2: You Suspect a Leak But Do Not See One

Your patient is now a week after an uneventful right hemicolectomy for a carcinoma of the cecum. She is already at home, and eating, when a new pain develops on the right side of her abdomen, accompanied by vomiting. The patient returns to the emergency room. She is febrile, her right abdomen is tender with a questionable mass, the abdominal X-ray suggests an ileus or partial small bowel obstruction (Chap. 48), and the white cell count is elevated. You suspect an anastomotic leak.

**From a clinical standpoint, there are three types of intestinal leaks that "you cannot see":**

- **Free leak**. The anastomosis is disrupted and the leak is *not* contained by adjacent structures. The patients usually appear "sick," exhibiting signs of diffuse peritonitis. An immediate laparotomy is indicated.
- **Contained leak**. The leak is partially contained by perianastomotic adhesions to the omentum and adjacent viscera. The clinical abdominal manifestations are **localized**. A perianastomotic abscess is a natural sequela.
- **A mini-leak**. This is a "minute" anastomotic leak, usually occurring late after the operation when the anastomosis is well sealed off. Abdominal manifestations are localized, and the patient is not "toxic." A mini-leak is actually a **"perianastomositis"**—an inflammatory phlegmon around the anastomosis. Usually, it is *not* associated with a drainable pus-containing abscess.

In the absence of diffuse peritonitis, you should document the leak and grade it. Colonic anastomoses are best visualized with a Gastrografin enema. For upper gastrointestinal and small bowel anastomoses, give Gastrografin from above. We usually combine the contrast study with a CT, searching for free intraperitoneal contrast or abscesses. There are a few possibilities:

- Free leak of contrast into the peritoneal cavity (a lot of free contrast and fluid on CT). You have to reoperate. We previously discussed what to do: it is best to take down the anastomosis if technically feasible.
- Contained localized leak (a local collection or abscess on CT). The rest of the peritoneal cavity is "dry." This is initially treated with antibiotics and PC drainage (Chap. 49).
- No leak on contrast study (a perianastomotic phlegmon on CT). This represents minileak or perianastomositis and usually resolves after a few days of antibiotic therapy.

Note that a contained leak or a minileak may be associated with an obstruction at the anastomosis—a result of the local inflammation. Such obstruction

usually resolves spontaneously (within a week or so) after the pus has been drained and the inflammation has subsided (Chap. 48).

## Conclusion

We have tried to persuade you that an anastomotic leak is not one disease but a variety of conditions requiring customized approaches. To keep morbidity at bay, tailor your treatment to the specific leak, its severity, and the condition of the affected patient. Above all, remember that nondrained intraperitoneal bowel contents and pus are **killers**—often silent ones.

> We tend to remember best those patients we almost killed; we never forget those we actually managed to kill.
>
> Good surgeons operate well; great surgeons know how to manage their own complications.

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# 51 Leaks Following Colonic or Rectal Anastomoses

Jonathan E. Efron

In the previous chapter we have focused mainly on leaks originating from the small bowel. But, for the majority of surgeons, the most common "leaks" are those following elective or emergency colorectal surgery. We have asked Dr. Efron to elaborate further on this topic.—The Editors

A colonic or colorectal anastomotic leak can be a devastating complication for both the patient and the surgeon. They occur in approximately 3% of all patients

Surgeons are judged by the way they manage their own complications.

undergoing colonic or rectal resections. The rate is double that in patients undergoing extraperitoneal resection of the rectum (risk factors for leakage are listed in Table 51.1). Diagnosis and management of a leak can be very difficult as patients vary dramatically in their clinical presentation, and the natural state of mind of the surgeon is one of denial. Nevertheless, making accurate and early diagnosis of anastomotic leaks is essential if morbidity and mortality are to be minimized.

## Timing, Signs, and Symptoms of an Anastomotic Leak

- **Timing**: leaks occur as early as 24 hrs and as late as 3 weeks after surgery. The majority, however, occur within a 5- to 10-day time period after the operation, most often on day 7. There is no easy way to suspect an anastomotic leak, and for this reason any complication that occurs in a patient after colonic resection should be viewed with suspicion.
- **Symptoms** vary from a low-grade fever with tachycardia to circulatory and respiratory collapse. The only clue may be no other signs except for symptoms of a cardiac complication such as chest pain or an arrhythmia. The patients may or

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**Table 51.1.** Risk factors for colorectal anastomotic leaks

| |
|---|
| High blood loss (>500 ml) |
| Presence of peritonitis/sepsis |
| Malnutrition: significant weight loss; albumin less than 3.0 dg/l |
| Low anastomosis: 5 cm or less from the dentate line (some studies define as any extraperitoneal anastomosis) |
| Use of steroids |
| History of radiation therapy to resected area |
| Smoking |
| Diabetes mellitus |
| Obesity: body mass index (BMI) > 30 |
| History of Crohn's disease |
| Use of immunomodulators other than steroids |
| Recent history of chemotherapy (administered <1 month from surgery date) |

may not have localized or diffuse abdominal pain, and this pain may or may not be associated with tenderness. Pain and tenderness may be very difficult to assess after a major laparotomy with a large incision. For this reason, the assessment of pain and tenderness needs to be repeated and not a single evaluation. A patient who appeared well on postoperative day 3 with tenderness around his or her incision and who then develops diffuse abdominal pain is worrisome and requires further investigation. This is particularly pertinent when dealing with patients who have undergone a laparoscopic colectomy. These patients tend to have minimal pain and tenderness, so pain or tenderness that surpass what the surgeon would expect should be taken seriously and investigated. Nausea and vomiting are often present, indicating an ileus, but the patient may very well be passing flatus or having bowel movements. Bowel activity *does not* rule out the presence of an anastomotic leak. And indeed, the presence of fresh blood in the stools, combined with other features of local and systemic inflammation, makes leakage highly probable, with the blood originating from the edges of the dehisced bowel. Leaks may present with the signs of either cardiac or pulmonary events such as myocardial infarction or pulmonary embolism. Chest pain, pleuritic pain, tachycardia or other arrhythmias, and oxygen desaturation may all result from a leak.

- **Laboratory**: patients will usually become dehydrated from the induced sepsis and possible ileus. This usually manifests as a decrease in urine output and a rising ratio of serum urea nitrogen (BUN) to creatinine. Other laboratory

changes that are concerning are either a rise or significant drop below normal limits of the patient's white blood cell (WBC) count. A falling WBC count is more worrisome than a rising one. X-rays demonstrating significant free air under the diaphragm more than a few days postoperatively are also concerning but by no means diagnostic. Also, a lack of free air certainly does not rule out a leak.

Given the wide range of symptoms that occur in patients with an anastomotic leak, the best way to ensure detection is to view any clinical mishap as indicative of a possible leak. With this low threshold, the surgeon can then proceed to the next step, which is performing the appropriate diagnostic study. The overall condition of the patient dictates whether further workup or immediate intervention is appropriate.

## Diagnosing a Leak

If the patient is unstable with decreasing respiratory or cardiac function, then re-exploration is required after a brief period of resuscitation. Systemic sepsis induced by peritonitis from an anastomotic leak requires urgent attention and may not allow for radiological tests to be performed to confirm or rule out a leak. The difficulty here, of course, is that a nontherapeutic laparotomy is unlikely to help a patient whose condition is due to the acute coronary syndrome or pulmonary embolism alone. If, however, the patient is stable, evaluation of the anastomosis should be undertaken.

The gold standard for diagnosing colonic or colorectal anastomotic leaks has been the Gastrografin enema. Gastrografin is favored as an intraluminal agent over barium for evaluating a bowel anastomosis because barium, when leaked into the peritoneum, causes a significant inflammatory reaction in the peritoneal cavity (see also Chap. 4). Retrograde studies are sometime feared by surgeons who claim that the tests themselves may cause a leak, but these fears are unfounded. It is helpful for the surgeon to be present when the studies are performed to help the radiologist delineate postoperative anatomy and to insert the enema catheter to avoid injury, especially in a low colorectal anastomosis. When evaluating a low colorectal anastomosis, the balloon on the enema catheter should not be inflated as this may obscure the leak by obstructing the anastomosis.

Increasingly, *a computed tomographic (CT) scan of the abdomen and pelvis with intravenous and rectal contrast has supplanted the Gastrografin enema as the test of choice for identifying anastomotic leaks.* The accuracy of detecting anastomotic leaks using a Gastrografin enema is not well documented, but recent studies have shown that a CT scan with rectal contrast has a significantly higher sensitivity than Gastrografin enema. Another benefit of CT is better delineation of the surrounding anatomy; it can identify any associated abscess, and this possibly may permit definitive therapy with percutaneous drainage of the

abscess. Both CT with rectal contrast and Gastrografin enema are acceptable for identifying leaks, and the choice of which test to use should be decided by the local availability of expertise and facilities.

## What to Do When a Leak Is Identified (see Fig. 51.1 and 51.2)

What to do when a leak is identified depends on the severity of the leak and patient's response to it. In this context, it is useful to think of leaks as asymptomatic or symptomatic, contained or with free perforation, or as minimal, minor, or major. This allows us to allocate them to the various therapeutic options. Minimal or "minileaks" are discussed in Chap. 50, but both minor and major leaks produce significant morbidity, whereas major leaks also have a significant increase in mortality. Whether the leak is symptomatic or not or minimal or major, may be influenced by the presence or not of a proximal diverting stoma as discussed below.

**Asymptomatic leaks** may occur in up to 10% of colorectal anastomoses and are detected on routine diagnostic enemas. They are seen primarily in patients

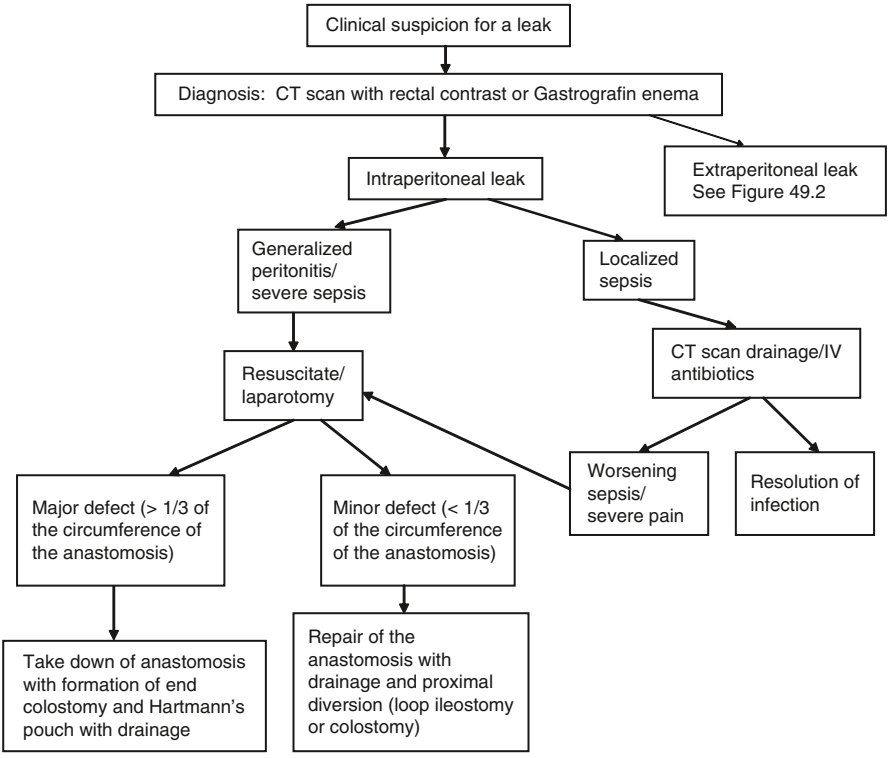


**Fig. 51.1.** Algorithm for management of intraperitoneal colorectal or colocolonic anastomotic leaks. Adapted from: Phitayakorn et al. World J Surg (2008) 32:1147–1156

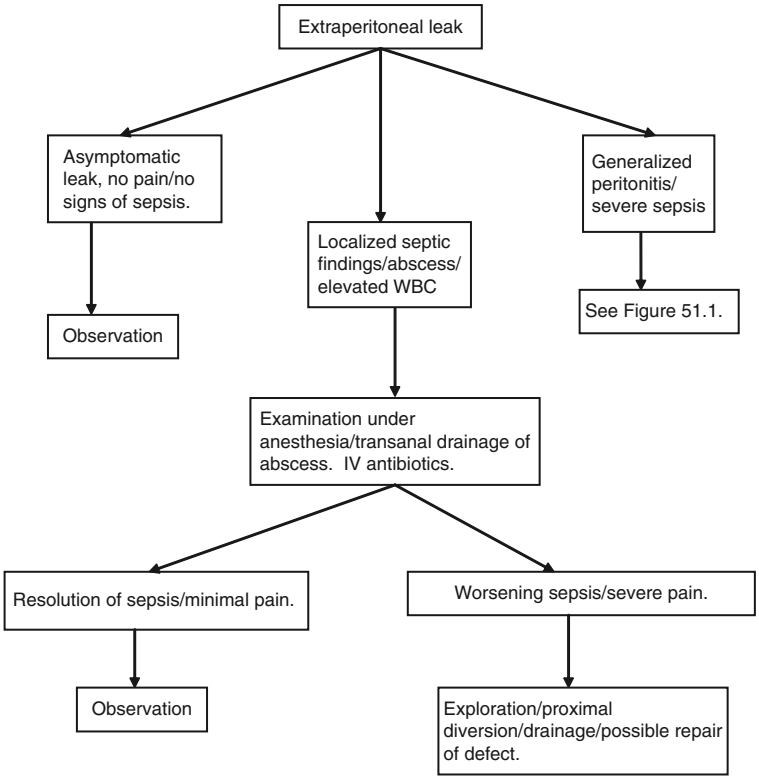


**Fig. 51.2.** Algorithm for management of extraperitoneal colorectal anastomotic leaks. Adapted from: Phitayakorn et al. World J Surg (2008) 32:1147–1156

undergoing low colorectal resection with a covering proximal stoma as these are patients who have routine enema studies prior to closure of their stomas. They need not be treated if there is neither pain nor sepsis, and almost all such asymptomatic or minimal leaks heal without the need for surgical intervention. However, delayed sequelae may include stricturing at the anastomosis, which is amenable to dilatation.

- **Symptomatic leaks** that are contained may be treated by antibiotics only or by minimal access intervention. CT-guided percutaneous drainage is usually possible, especially in low colorectal anastomoses, often through a gluteal approach. Once the abscess is drained and sepsis has resolved, then a fistulagram either under fluoroscopy or under CT will help delineate any continued connection between the anastomosis and the cavity. If a fistula persists, long-term drainage and gradual removal of the drain catheter may allow healing without having to reoperate. After 2–3 weeks with the drain in place, often the cavity will collapse around the drain, and if no connection with the bowel is demonstrated, then the drain can be removed. Similarly, stricture is a long-term complication that will require monitoring.
- **Low colorectal anastomotic leaks that are contained** can often be watched without intervention, especially if small. Contained abscesses in the lower pelvis

resulting from leaks can often be drained under direct vision in the operating room through the anus. If the opening is large enough, these cavities will shrink in size and close down or may just become incorporated into the anastomosis, eventually undergoing epithelialization. If this occurs, most require no further intervention. Some patients with chronic posterior cavities arising from an anastomosis will develop recurrent abscesses that require drainage. In these patients, inserting and firing a linear stapler along the tract of the abscess cavity will sufficiently open the cavity so that it may heal in conjunction with the bowel. Some of these patients will have chronic pain requiring redo colorectal or coloanal anastomoses, often with a hand-sewn anastomosis performed through the abdomen and anus (coloanal anastomosis) with proximal diversion.

- **Major, large leaks that result in peritonitis** almost always require relaparotomy. As mentioned, if a patient is not doing well, early laparotomy is better then waiting. If on re-exploration the site of the leak is small—less than a quarter of the circumference of the anastomosis—and the anastomosis appears viable, then suture repair and proximal diversion with either a loop ileostomy or loop colostomy is an option. Of course, thorough peritoneal toilet is essential (Chap. 12). I would drain high-risk anastomoses and the evacuated abscess cavity, but not all surgeons would agree with this (Chap. 42). Closure of the abdomen may be difficult if the peritonitis has been present for a period of time prior to reoperation (Chaps. 43 and 52). If the anastomosis has a large defect, does not appear viable, or is not reparable, then take down of the anastomosis with closure of the distal stump and formation of an end colostomy is required. If there is a large amount of stool present in the distal colon or rectum, then irrigation of the rectal stump is recommended to remove any solid stool. Again, I would drain the pelvis if this was the site of the anastomosis, and drains should be left over a Hartmann's stump located in the pelvis, but I know that this may be controversial (Chap. 42).

**A course of wide-spectrum intravenous antibiotics is always provided irrespective of whether the leak is minor or major as an adjunct to its operative, percutaneous, or "conservative" management.**

### Proximal Diversion: Does It Matter?

Proximal diversion is a consideration at the time of the initial elective operation and therefore largely outside the scope of this book. Proximal diversion or the formation of a "protective" stoma during the elective operation (preceding the leak) is controversial as no study has clearly demonstrated that formation of such a stoma decreases the anastomotic leak rate. However, proximal diversion does significantly decrease the morbidity and possibly the mortality that may occur from an anastomotic disruption. Anastomotic leaks have also been shown to be an independent predictor of worse cancer-specific survival, and those colorectal cancer patients who do leak have a higher recurrence rate. A diverting stoma is not a free ride, however,

as the complications that can occur from either an ileostomy or a colostomy are significant. These include dehydration, peristomal skin complications, and obstruction. In addition, significant problems can occur when these stomas are closed, including anastomotic leaks, bleeding, wound infections, formation of enterocutaneous fistulas, and abdominal wall hernias (see also Chap. 14).

Many of the stomas formed *after* a leak has taken place are "permanent" in that they are never reversed. Pelvic sepsis that occurs after an anastomotic leak from a low anterior resection also causes significant fibrosis to occur. This scarring will often adversely affect the patient's bowel function by inhibiting distention and reservoir capacity of the neorectum. These patients often suffer from significant anterior resection syndrome with clustering of bowel movements, incontinence, urgency, and frequency. Once this scarring has occurred in the pelvis, little can be done to correct it.

In view of these problems, some believe that a good rule of thumb with respect to proximal stoma formation is that if you think about diverting a patient, no matter how fleeting that thought, then divert the patient.

## Special Considerations

### *Laparoscopic Surgery*

Laparoscopic colectomies are now routinely performed for all forms of colorectal pathology. As is the case with laparoscopic bariatric surgery (Chap. 31), laparoscopic colectomy patients may have only subtle findings to indicate a possible leak. Tachycardia or fever in the acute postoperative setting can be concerning, and those patients should be closely watched. Excessive pain and tenderness is also concerning as these patients should not suffer from the same pain that occurs in those with a large midline incision.

In our hands, most laparoscopic segmental colectomies can easily be discharged on postoperative days 2–4, which creates a dilemma in monitoring patients for anastomotic leaks as these leaks usually occur between postoperative day 5 and day 10. As I mentioned, patients may be passing flatus and having bowel movements when a leak occurs. While most surgeons do not require bowel activity prior to discharge from the hospital, patients should not be bloated, should be free from nausea, and should tolerate a diet prior to discharge; they should also be relatively pain free with minimal tenderness. In brief, do not discharge patients with soft signs of developing leak. It is important to counsel patients on the signs and symptoms of anastomotic leak and have them contact you or return to the emergency room if they start experiencing these symptoms. Any complaint in the first week after discharge in these patients should be taken seriously. Seeing patients early (within a week) in the postoperative period after a laparoscopic colectomy provides comfort not only for the patient but also for

the surgeon. No studies to date have documented adverse outcomes occurring from anastomotic leaks that are diagnosed as an outpatient as opposed to an inpatient. Otherwise, the therapeutic approach to leaks developing after laparosopcic procedures should not differ from those after the open ones.

### *Obese Patients*

Obese patients make abdominal surgery challenging in many aspects (see Chap. 31). They often have multiple medical comorbidities related to their obesity (diabetes, hypertension, sleep apnea) that contribute to postoperative complications. Obesity has been shown to increase the risk of anastomotic leaks. Assessing a postoperative obese abdomen is perhaps the greatest clinical dilemma. It is often quite difficult to determine if these individuals have peritonitis from a possible leak. Often, they may have pain but show no signs of tenderness, or they may have no pain at all. Sometimes, the only early signs may be a change in the WBC count or slight tachycardia or fever. These patients therefore require close monitoring, and I would consider early radiological evaluation of the anastomosis if they show any concerning signs. Similarly, a very low threshold should be maintained for returning to the operating room because it can be so difficult to clearly define even diffuse peritonitis in these patients, and significant delays in the diagnosis can lead to catastrophic outcomes.

## Conclusions

Prevention is always the best treatment; however, this is not always possible. If one even remotely contemplates proximal diversion for a colonic or colorectal anastomosis, a stoma should be constructed at the initial operation. Keep a high awareness for the presence of an anastomotic leak and have a low threshold for performing radiological investigations and returning to the operating room. Finally, if a leak is detected, ensure adequate drainage of infection and diversion of the fecal stream if required.

> If you do a colostomy, there will be always someone to tell you why not primary anastomosis; if you do a primary anastomosis, there will be always someone to tell you why not colostomy.

# 52 Re-laparotomies and Laparostomy for Infection

Moshe Schein · Roger Saadia · Danny Rosin

*When is a surgeon (not a new, but an experienced one) nervous? Not during operations. But basically a surgeon's nervousness begins after the operations, when for some reason the patient's temperature refuses to drop or a stomach remains bloated and one has to open it not with a knife, but in one's mind, to see what had happened, to understand and put it right. When time is slipping away, you have to grab it by the tail.* (Alexander Solzhenitsyn)

Remember, we discussed the principles of management of *intra-abdominal infection (IAI)* (› Chap. 12)? We told you that to improve survival in some patients, *source control* and *peritoneal toilet* must be pushed a little further; some patients need a relaparotomy, and in many of these the abdomen is left open (laparostomy). These modalities are now discussed in greater detail. At the end of the chapter, we present an invited commentary about laparoscopic abdominal re-exploration after open surgery.

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# 52.1 Re-laparotomy

Moshe Schein · Roger Saadia

## Definitions

Before we continue you should be reintroduced to some definitions.

**"On-demand" versus "planned" relaparotomy**

- **On demand**: in the aftermath of an initial laparotomy, clinical or radiological evidence of an intra-abdominal complication forces the surgeon to reoperate.
- **Planned** (or "electively staged"): at the initial laparotomy, the surgeon makes the decision to reoperate within 1–3 days, irrespective of the patient's immediate postoperative course.

Both types of relaparotomy have a place in the postoperative management of the patient following a laparotomy, but they apply in different clinical contexts.

## Re-laparotomy on Demand

The unexpected development of intra-abdominal infection (IAI) after the initial, "index" laparotomy constitutes the indication for re-exploration. The two postoperative complications that may require a relook are *generalized peritonitis* and *intra-abdominal abscess*. A postoperative suture line or anastomotic dehiscence may manifest itself either as an external fistula, with no peritoneal contamination, or as IAI, whether generalized peritonitis or a localized abscess. Leaks take place typically between the fifth and eighth postoperative days but may occur earlier or later (Chaps. 49 and 50).

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## Postoperative Peritonitis

Peritonitis complicating a laparotomy is termed *postoperative peritonitis*. This is one of the most lethal types of peritonitis—killing between one-third and one-half of the patients—for the following three reasons:

- Its diagnosis is usually delayed because the abdominal signs (tenderness, distension) are initially masked by the expected similar signs of the normal postoperative abdomen.
- It occurs in the postoperative phase, when the patient is catabolic, with associated inflammation (systemic inflammatory response syndrome, SIRS) and immunodepression (compensatory anti-inflammatory response syndrome, CARS) (› Chap. 54).
- It is a case of *nosocomial* secondary peritonitis for which the microbiology is less predictable and more noxious due to previous antibiotic administration and prevailing hospital flora.

There are several possible clinical presentations developing within days of a laparotomy:

- *Generalized peritonitis.* The abdominal findings are out of proportion to the normal postoperative state (severe abdominal pain and tenderness, massive or prolonged ileus). There may be associated systemic repercussions (fever, leukocytosis) that are uncharacteristic of the expected postoperative recovery. Sometimes, the diagnosis is made easier by the additional presence of an enterocutaneous fistula (› Chap. 50), deep wound infection (› Chap. 55), or abdominal wall dehiscence (› Chap. 53).
- *Organ dysfunction.* Renal failure or incipient acute respiratory distress syndrome (ARDS) manifesting itself as atelectasis or pneumonia. Not infrequently, the surgeon seeks expert advice from medical colleagues (nephrologist, chest physician, infection disease specialist, intensivist). Of course, renal failure or pneumonia may well occur in a postoperative patient for a variety of reasons that are unrelated to an intra-abdominal complication. However, persistent or recurrent IAI may present initially as a single system dysfunction and progress, in time, to multiple-organ failure. It is essential first to be aware of the relationship between IAI and organ dysfunction (› Chap. 54) and second to be humble enough to consider the possibility of a surgical complication in one's patient (› Chap. 45). The diagnosis is established by careful clinical evaluation of the abdomen, usually supplemented with abdominal imaging, mainly computed tomography (CT).
- *The intensive care setting.* The possibility of IAI is raised because of the need for prolonged ventilation or aggravation of multiple-organ dysfunction in a critically ill postoperative patient, for example, after massive trauma or major abdominal surgery. Intensivists are usually quick to point to the abdomen as the culprit and eager to spur the surgeon to re-explore. In a ventilated, paralyzed patient, the abdomen cannot be evaluated clinically. There is therefore a real dilemma in

differentiating between, on the one hand, the presence of an abdominal focus of infection and, on the other hand, SIRS without infection (Chap. 54) or an infection elsewhere. Abdominal CT scanning is very useful but, unfortunately, less so *in the first 5–7 postoperative days*. After any laparotomy, tissue planes are distorted and potential spaces may contain fluid; even the best radiologist cannot tell you whether the fluid is blood, serous fluid, leaking bowel contents, or pus. In addition, transporting a critically ill patient on maximal organ support to the CT suite is not an innocuous undertaking. Thus, the decision to reoperate during the first postoperative week can be extremely vexing and requires good co-operation between surgeons, intensivists, and radiologists.

- *Intra-abdominal abscess* (Chap. 49).

**Remember:** the diagnosis of postoperative intra-abdominal "septic" complications is extremely difficult. *Denial* is a major culprit. Surgeons hate to admit to their own failures and confront them. Consider, for a moment, your past experience: haven't you seen patients fading away while their deterioration is blamed on a "bad bout of pneumonia"? Autopsy would have uncovered unsuspected intra-abdominal complications in a good proportion of them.

Mark M. Ravitch reminded us wisely: "The last man to see the necessity for reoperation is the man who performed the operation."

The following admonition should be deeply imprinted in your surgical soul: **look for pneumonia inside the abdomen.**

### Planned (Electively Staged) Re-laparotomy

A negative relaparotomy is better than a positive autopsy but is not, nevertheless, a benign procedure.

The policy of *planned relaparotomies* is decided on during, or immediately after, the initial, index, operation for peritonitis, when the surgeon decides to reoperate within 1–3 days, irrespective of the patient's immediate postoperative course. The decision to re-explore the abdomen is part of the initial management plan. Historically, mesenteric ischemia (Chap. 23) was probably the first instance for which a planned relook laparotomy was advocated. In the context of IAI, the main justification for a relook is to deal with persisting infected collections or to anticipate the formation of new ones before they have had the time to amplify the existing SIRS and to tip the patient into irreversible multiple-organ failure (Chap. 54).

## Indications for Planned Re-laparotomies

Planned relaparotomies need to be better defined and restricted to well-selected patients. A relaparotomy is best undertaken during the **first postoperative week**—a period when abdominal CT findings are "nonspecific" and CT-directed percutaneous or open procedures are not an option.

- **The most appropriate indication is failure to obtain adequate source control during the initial operation.** A classic example is infected pancreatic necrosis (› Chap. 19). Another example is an intestinal leak that cannot be safely repaired or exteriorized (e.g., a neglected leak from the retroperitoneal duodenum)—a scenario commonly associated with postoperative peritonitis.
- The necessity to **redebride or redrain poorly localized, "stubborn" infected tissues,** for example, in diffuse retroperitoneal fasciitis due to retroperitoneal perforation of the duodenum or colon.
- Instability of the patient during the initial operation may occasionally lead to an abbreviated **"damage control"** procedure, with an obligatory subsequent planned relaparotomy to complete source control and peritoneal toilet. Obviously, when hemostatic packs have to be left in situ, a relaparotomy is needed to remove them.
- In the past, **diffuse fecal peritonitis** was considered a relative indication, with the rationale that in the face of massive fecal contamination another laparotomy is necessary to achieve an adequate peritoneal toilet. Now, most such patients can be treated with a "single" operation, supplemented if necessary with percutaneous drainage or an "on demand" reoperation.
- The need to reassess high-risk anastomoses. This highly controversial objective is being promoted as part of the "staged abdominal repair" or STAR (D. H. Wittmann) combining an obsessive policy of planned relaparotomies with laparostomy followed by a complicated method of abdominal closure. Dr. Wittmann assures us: "Leaks can be resutured … they all heal … with STAR." We urge you strongly to share our skepticism.

## The Conduct of a Re-laparotomy

The key piece of advice for the surgeon who plans to re-enter a recently opened abdomen is to **be gentle!** The peritoneal surfaces are edematous, friable, and vascular and so is the bowel. Reoperative abdominal surgery is a situation for which the dictum "first do no harm" has particular relevance. Do not perforate the bowel, do not cause bleeding—such mishaps in an already compromised patient are often a death sentence.

Another important tip: **know your way around.** Ideally, the surgeon who has performed the original procedure should be either the one to reoperate or at least a member of the reoperating team. Think about the infected postoperative abdomen as a thick jungle; a previous journey through it renders a return easier.

You will remember, for example, that the colon was "stuck" to the lower end of the incision; your partner who did not accompany you on your first trip may instead enter the lumen of the colon, with horrendous consequences.

The abdominal relook itself aims at draining all infected collections and controlling, if necessary, persistent sources of contamination. How thorough the exploration depends on the individual case. Sometimes, there are several interloop abscesses that need to be drained, and the whole bowel must be carefully unraveled. Particularly later in the natural course of peritonitis, the intestines are matted together, forming a "central visceral block"; it is then prudent to explore the spaces *around* the matted bowel (subphrenic spaces, paracolic gutters, pelvis). The decision about the extent of exploration is important because the more extensive it is, the more dangerous it is to the viscera. As you have been told here again and again, the more you do, the more local and systemic inflammation you trigger. **The extent of the exploration depends not only on whether your operation is "directed" or "nondirected" but also on its timing.**

### "Directed" Versus "Nondirected" Re-look

**Let the CT scan be your guide**. A "directed" reoperation implies that you know exactly where you want to go. The CT scan identifies a right subhepatic collection, with the rest of the abdomen appearing "clean." You can proceed directly to where the trouble is, sparing the rest of the abdomen the potentially damaging effects of your manipulations. Conversely, a "nondirected" relook is a blind re-exploration when you are not sure where the problem lies, for example, when the CT scan shows a diffuse collection; in this instance, a thorough search is required.

### Timing of the Re-look

When you re-explore the abdomen 24–72 hrs after the index operation, the adhesions between viscera and peritoneal surfaces give way easily; you can enter any space with atraumatic dissection. At this stage, total abdominal exploration is readily feasible. However, as time goes on, the intra-abdominal structures become progressively cemented to each other with dense, vascular, immature adhesions that are troublesome to divide. Clearly, abdominal reentry between 1 and 4 weeks after the index operation may be hazardous and will remain so until the eventual maturation of the adhesions several weeks later.

Consequently, during an early relook operation, you may unravel the whole bowel and drain all interloop collections. In contrast, you will find, at "late" reoperations, a **central mass of matted small bowel.** Leave it alone! Dissection of the individual loops at this stage is dangerous and nonproductive because significant

collections are to be found only at the periphery—**above** (under the diaphragms or under the liver), **below** (in the pelvis), and on the **sides** (in the gutters).

During re-exploration sharp tools are rarely needed. Your fingers are the safest dissecting instrument. Remember: where tissue planes are fused, forbidding admission to your gently pinching fingers, nothing is to be found. **So, follow your fingers to where the pus lies.**

### The Leaking Intestine

Dehisced suture lines and anastomoses must be defunctioned, ideally by the fashioning of appropriate stomas or, if this is not possible, by tube drainage. Resuturing leaking bowel in an infected peritoneal cavity is doomed to failure and carries a prohibitive mortality (Chap. 50).

### Drains

The use of intraperitoneal drains is controversial in this setting. They are certainly not required as long as planned relaparotomies continue. The placement of a drain at the *final laparotomy* is another matter; the advantages need to be weighed against the risk of damage to viscera that are extremely friable as a result of recent re-explorations. The use of drains in our practice is strictly limited to specific situations as discussed elsewhere (Chap. 42).

### When to Stop?

As in most vital aspects of life, too much of anything is not good, and too many planned relaparotomies are harmful. When to stop? In such a management program, the decision to quit must be based on the finding of a macroscopically clean peritoneal cavity and evidence that sources of contamination have been controlled definitively. Whether the source is controlled or not is obvious, but estimation of whether the peritoneal cavity is "clean" or not requires experience and judgment. Thus, do not send your junior partner or senior resident to re-explore it alone.

A frequent dilemma is whether you "take your spouse for dinner or the patient back to the operating room (OR)" (you may lose even if you make the correct choice).

When peritonitis persists despite apparently adequate source control and repeated reoperations—think about **tertiary peritonitis** (Chap. 54).

### Are Planned Re-laparotomies Beneficial?

What is the verdict? Do planned relaparotomies reverse, prevent, or aggravate SIRS and multiorgan dysfunction? Is the benefit-risk ratio favorable?

Any surgical maneuver that successfully eliminates the source of infection and evacuates contaminants and pus has to be beneficial; this is axiomatic. The problem is that planned relaparotomies are a double-edged sword: they may achieve the goal but may also injure the host. Indeed, strict adherence to the policy of planned relaparotomies is definite overkill. If one operates until the abdomen is clean, then—in retrospect—the last operation was unnecessary. Multiple relaparotomies are attended by a high morbidity accounted for by destabilizing an intensive care unit (ICU) patient during repeated trips to the OR, iatrogenic bowel injuries, and possibly the stimulation of an exaggerated inflammatory response. We believe that, in the long run, we serve our patients better with a **low-threshold** policy of postoperative *on demand* percutaneous CT-guided drainage procedures or *on demand* CT-directed laparotomies. This will appease the advocates of planned relaparotomies whose main fear is to miss the boat. One or two planned relaparotomies may still have a place in the indications listed and only in the first postoperative week when both the imaging is less reliable and the abdomen safer to re-enter. It is our opinion that at a later stage of a critically ill patient's course, on demand is the way to go based on the patient's clinical condition and convincing imaging. Common sense and experience must prevail when level I evidence is lacking.

# Laparostomy

52.2

Moshe Schein · Roger Saadia

P. Fagniez of Paris coined the term *laparostomie* (laparostomy), which entails leaving the abdomen open. Open management of the infected abdomen was instituted in the belief that the peritoneal cavity should be treated open like an abscess cavity. It soon became clear, however, that sometimes there was still a need for thorough abdominal re-exploration in search of deep pockets of infection. Laparostomy has become an *adjunct* to the policy of repeated laparotomies; indeed, if the abdomen is to be reopened 48 hrs later, why close it at all?

The notion that peritonitis and its operative treatment often result in increased intra-abdominal pressure (IAP) has been raised sporadically throughout the twentieth century. However, only recently have clinicians accepted the concept that the prevention or treatment of intra-abdominal hypertension (IAHT) with laparostomy is beneficial. The potential advantages of laparostomy are substantial. Necrosis of the macerated abdominal midline incision closed forcefully and repeatedly in the presence of an edematous and distended bowel is avoided, better diaphragmatic excursion may be expected, and the abdominal compartment syndrome with its renal, respiratory, and hemodynamic repercussions is prevented (Chap. 40).

## Indications

For practical purposes, consider laparostomy when the abdomen either cannot be closed or should not be closed (Fig. 52.2.1).

**The abdomen cannot be closed:**

- After major loss of abdominal wall tissue following trauma or debridement for necrotizing fasciitis
- Extreme visceral or retroperitoneal swelling after major trauma, resuscitation, or major surgery (e.g., ruptured abdominal aortic aneurysm)
- Poor condition of fascia after multiple laparotomies

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**The abdomen should not be closed:**

- Planned reoperation within a day or two—why lock the gate through which you are to re-enter very soon?
- Closure possible only under extreme tension, compromising the fascia and creating intra-abdominal hypertension (IAHT).

## Technical Considerations of Laparostomy

Now that you have decided not to close the abdomen, how should you manage it? The option of simply covering the exposed viscera with moist gauze packs has been practiced for generations but is inadvisable: intestine, if not matted together, can eviscerate; it is also messy, requiring intensive work to keep the patient and the patient's bed clean and dry. More important, it carries a significant risk of creating spontaneous, "exposed," intestinal fistulas (Chap. 50). A friable, dilated bowel wall does not weather well the trauma of exposure and repeated dressing change and is likely at some point to break down. Temporary abdominal closure (TAC) devices to cover the laparostomy wound are therefore highly recommended.

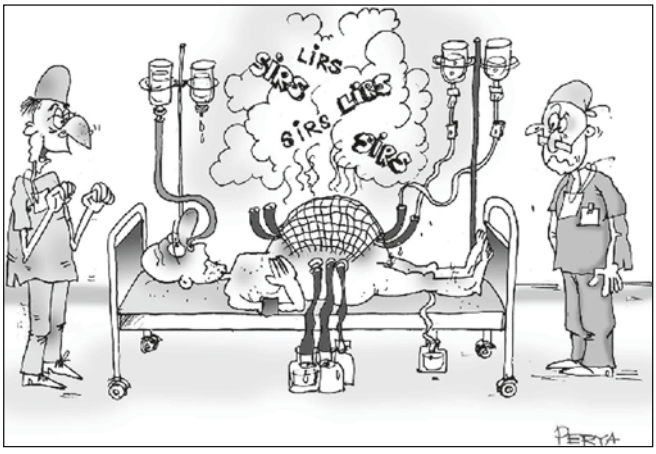


**Fig. 52.2.1.** "I told you that laparostomy would make him better: it lets the SIRS out…"

### Temporary Abdominal Closure

The ideal method of TAC has to:

- **Allow re-exploration**, offering easy access for relaparotomies, if needed.
- **Offer drainage** to the peritoneal exudate and later for possible fistulas.
- **Preserve fascia** for future abdominal closure.
- **Avoid "loss of domain":** when the fascial edges retract, the viscera bulge out, and the viscera cannot be returned to the peritoneal cavity.

Your local guru has probably a preferred method of TAC, be it a "Bogota bag" made of a large sterile intravenous fluid bag, a ready-to-use transparent "bowel bag", a synthetic mesh (absorbable or nonabsorbable), or a Velcro-type material, which can be tightened like your tennis shoe (Wittmann patch). We even know a guy in South America who uses discarded nylon hose for this purpose. Currently, there is a whole line of homemade or commercial products based on the vacuum concept.

In fact, we were the first authors to recommend the use of vacuum suction for TAC (*British Journal of Surgery*, 1986). We dubbed our system the "sandwich technique." Current commercial "VAC" (Vacuum Assisted Closure) products are a refinement of this original concept. Our sandwich technique has somewhat evolved over the years. It now consists of:

- An absorbable, permeable, synthetic mesh sutured to the fascial edges
- Two suction drains placed over the mesh in the edges of the abdominal defect and brought out through the skin to collect the abdominal effluent
- Sheets of Stomahesive applied to the healthy skin bordering the defect
- A large, adhesive, transparent sheet (Steridrape or Opsite) stuck on top to cover the entire abdomen

This arrangement is beneficial since the viscera are protected, the laparostomy's output is measurable, the patient remains clean and dry, and the demands on nursing are minimized (Fig. 52.2.2).

The sandwich can be modified depending on the circumstances. If, for example, an early relaparotomy or closure is contemplated, the mesh is not used (Fig. 52.2.3). Instead, a perforated plastic bowel bag (much wider than the abdominal defect) is applied over the viscera; its edges are tucked in under the abdominal wall bordering the defect. Early adhesions between intestine and abdominal wall are thus prevented. A protective, absorbent, layer of gauze (or a "green towel") is held in place over this plastic sheet by a large Opsite. Suction is unnecessary if the patient is returned to the operating room (OR) the next day.

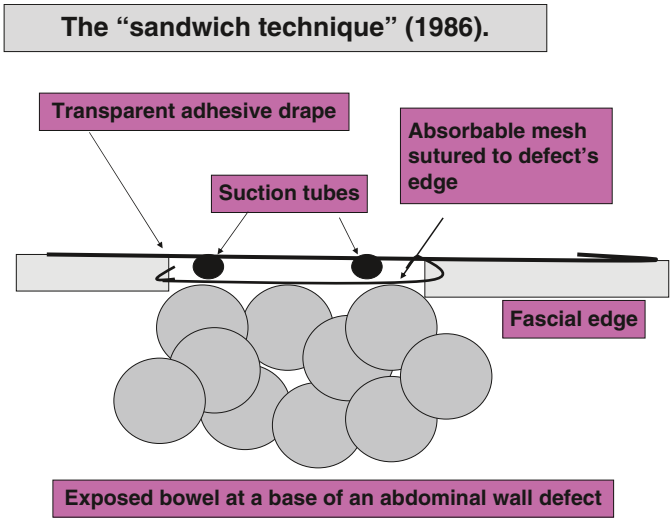


**Fig. 52.2.2.** The “sandwich technique” in the management of laparostomy (when neither further laparotomy nor “early” abdominal closure is planned)

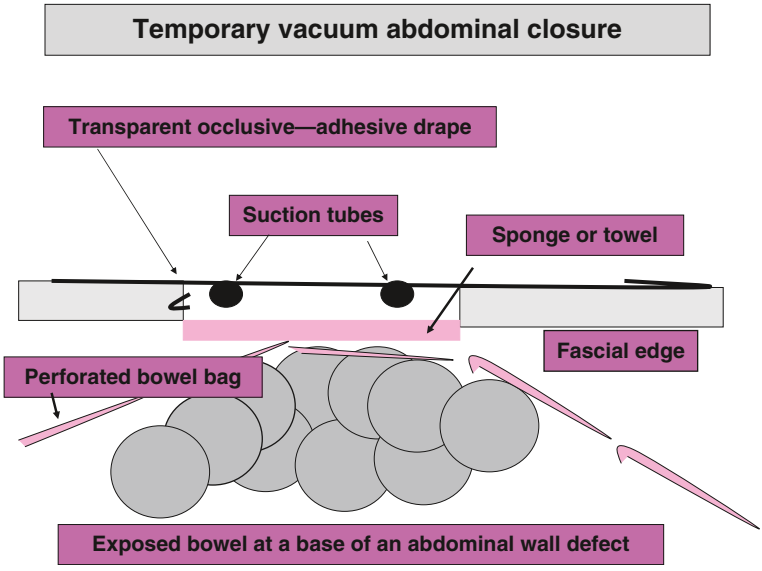


**Fig. 52.2.3.** Temporary vacuum abdominal closure (when additional reoperations or “early” abdominal closure are planned)

The most dreaded complication of TAC devices is the development of spontaneous intestinal fistulas resulting from the intimate contact between the artificial cover and the intestinal wall. The best prevention is to interpose, whenever possible, the omentum between TAC prostheses and intestine.

## Terminating the Laparostomy

Once the laparostomy has outlived its usefulness, it is the time to plan for abdominal closure. Two options exist, depending on both the surgeon's preference and local abdominal conditions: early abdominal closure or delayed abdominal wall reconstruction.

### *Early Abdominal Closure*

The optimal time window for early abdominal closure is quite narrow, about a week from the last abdominal exploration. Beyond that, the fascia retracts laterally, and adhesions form between intestine and abdominal wall. You will find it then impossible to mobilize and push the bulging viscera back into the abdominal cavity (loss of domain), to say nothing of the stubborn refusal of the fascial edges to meet in the midline. Even within the first week, the longer the delay, the more difficult and risky this endeavor becomes. It goes without saying that feasibility of early closure is predicated on a number of factors, including defect size, resolution of the ileus, and absence of fistulas (Chapt. 50). In rare cases, the defect is so small that the fascial edges lend themselves to midline suturing without tension (one wonders, in such cases, whether a laparostomy was indicated in the first place!). More commonly, in small-size defects, the fascia is left open, but primary cutaneous closure is possible after undermining the skin edges. The patient is left with a ventral hernia, but skin cover is superior to any prosthetic material (Chap. 43).

Most laparostomy wounds in the aftermath of a *real* abdominal disaster are large and present with fixed, retracted edges and with loss of domain for the abdominal viscera. Recently developed biomaterials are being aggressively marketed for this setting. They are claimed to be superior to synthetic meshes in resisting infection in these frequently heavily contaminated wounds. It turns out that they are not totally immune to infection. While providing a temporary bridge, their other claim to fame is their purported ability to stimulate growth of site-specific cells to replace the prosthesis with new fascia (not scar). In practice, the majority of patients undergoing early abdominal closure with these "wunderbioprostheses" are found, on brief follow-up, to have large ventral hernias. It seems therefore that, in many instances, the biopatch is no more than a tremendously expensive TAC.

Some surgeons advocate early reconstruction using "component separation techniques" to bridge the fascia (see Fig. 52.2.4), occasionally buttressing the unapproximated midline with an underlay of bioprosthesis. Experimentation with these novel techniques is inadvisable for the uninitiated. In our practice, most patients are treated with **delayed abdominal reconstruction**.

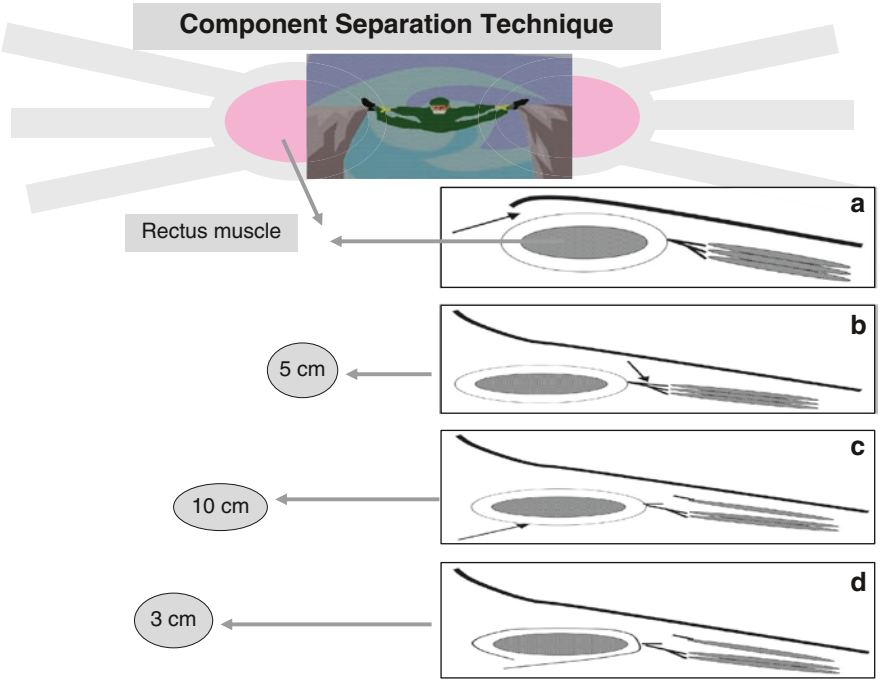


**Fig. 52.2.4.** Component separation technique: same maneuvers are performed on both sides. (**a**) *Arrow*—skin-flap elevation off the anterior abdominal wall. (**b**) *Arrow*—incision at the junction of external oblique fascia with the rectus abdominis. The external oblique is then dissected off the internal oblique laterally. These two steps (when performed on both sides) would allow the myofascial unit to slide medially for distances of 5 and 10 cm, respectively. (**c**) *Arrow*—incision of the posterior rectus sheath, which is then dissected off the rectus muscle to allow additional medial mobilization for 3 cm. This step is optional. (**d**) The completed procedure.

[Laparoscopic modifications of the procedure have been described and used for abdominal wall decompression in abdominal compartment syndrome without violating the peritoneal cavity.]

However you manage the abdominal wall defect, remember that your patient has just recovered from the immense stress of severe peritonitis and multiple operations—he cannot take much more at this stage.

### *Delayed Abdominal Wall Reconstruction*

Consider the following scenario: an obese patient develops a breakdown of his colorectal anastomosis with severe fecal peritonitis. The patient is now grossly distended, "septic," and in respiratory failure. He undergoes a Hartmann procedure, and obviously, his abdomen cannot be closed (Chap. 40), so he is managed

with a laparostomy. In your judgment, he does not require planned reoperations. Early closure is not a realistic option. How to proceed?

At this juncture, we would have used our sandwich technique. A fancier (but more expensive) VAC system over the mesh also works well at this stage. A couple of weeks later, a healthy layer of granulation tissue appears over the disintegrating absorbable mesh. A split-skin graft can now be applied onto the defect. The resulting ventral hernia is usually wide necked and well tolerated except for its cosmetic appearance. Many patients feel fortunate enough to have survived their "surgical saga" and find the end result acceptable with the added support from an abdominal Velcro binder.

A detailed discussion of the delayed *elective* abdominal reconstruction of the laparostomy defect is beyond the scope of this book. However, here are the principles involved:

- Delay reconstruction for up to 12 months or more until the abdomen looks and feels like "jelly": the skin graft is "loose" and "pinchable" away from underlying structures, the scar is soft, and the stomas or fistulas, if present, are prolapsing.
- At operation, excise the skin graft, lyse all adhesions, and use the component separation technique to bridge the fascial defect, combined, if necessary, with synthetic mesh. Avoid the use of synthetic mesh in contaminated fields; for example, when the operation involves the resection of an intestinal fistula or take down of a stoma.

### Antibiotics

Prolonged courses of postoperative antibiotics may be justified in patients with severe intra-abdominal infection who require reoperations or laparostomy for additional source control and peritoneal toilet (Chap. 47). Antibiotics should be continued as long as the source and residual infection are "active." Recent evidence suggests that, in this subgroup of patients (postoperative peritonitis), antifungal prophylaxis with fluconazole may decrease the incidence of intra-abdominal superinfection with *Candida* species.

### Is Laparostomy Beneficial?

Complications do occur with laparostomy, the most morbid being spontaneous enteric fistulas (Chap. 50), and there is always the need for subsequent reconstruction of the abdominal wall. How favorable is the risk-benefit ratio of laparostomy in these patients?

The physiological benefits of a decompressing laparostomy for significant IAHT/abdominal compartment syndrome are well proven in trauma and

general surgical patients (› Chap. 40). There is also a large body of experimental evidence suggesting that elevated IAP promotes systemic absorption/translocation of peritoneal endotoxin and bacteria, thus increasing the mortality rate of peritonitis in small and large animals. Although the issue of raised IAP and its treatment with laparostomy has not been studied specifically in the setting of peritonitis, it is probably true that treating IAHT is beneficial. The risk-benefit ratio of *prophylactic* laparostomy in borderline IAHT is not clear as yet. In our practice, therefore, we reserve laparostomy for patients with severe IAHT, those who "cannot be closed," and those we plan to re-explore.

## Conclusions

*Relaparotomy* and *laparostomy* are therapeutic measures that are indicated in a small minority of patients. They represent, for the time being, the heaviest weaponry in the surgeon's mechanical armamentarium for the treatment of severe intra-abdominal infection and other postlaparotomy abdominal catastrophes. Remember that unnecessary relaparotomies carry significant morbidity in these patients. An aggressive but selective policy of directed, "on demand" relooks, supplemented sparingly by laparostomy, is probably superior to the indiscriminate use of "blind" planned relaparotomies with routine laparostomy.

> He who operates and runs away may get to reoperate on the same patient another day.

# Laparoscopic Abdominal Re-exploration[1]

Danny Rosin

No surgeon likes to face a postoperative complication, but the need to treat such a complication by repeated surgery is even more distressing. Such complications include intestinal obstruction, intra-abdominal bleeding, hollow viscus perforation, and inadvertent bowel injury resulting in intra-abdominal infection. In some cases, such as mesenteric ischemia, a repeat operation is a planned "second-look" procedure (Chap. 23).

The presence of a fresh abdominal wound makes it rational to re-explore through this same incision. However, reopening of a recent incision and re-exploration by laparotomy may increase short- and long-term morbidity. Relaparotomy is associated with pain, ileus, and increased risk of abdominal infection. It may increase the risk of wound infection and eventual wound dehiscence or later development of an incisional hernia. Overall, it may extend the recovery period of the patient, on top of the condition that prompted it, serving as a "second hit."

Treatment of complications after laparoscopic surgery is frequently attempted by a repeat laparoscopy in an attempt to avoid a formal laparotomy (see also Chap. 58). Indeed, complications such as bleeding or bile leak after laparoscopic cholecystectomy can be successfully approached by a second laparoscopy (Chap. 20.1). Laparoscopy is also a valid treatment option in various acute surgical conditions (Chap. 57). Laparoscopy is frequently performed in the presence of abdominal scars and previous operations, and adhesions and moderately distended bowel are no longer considered to be contraindications for laparoscopic intervention. Given the morbidity associated with relaparotomy and the ability of trained laparoscopic surgeons to deal with acute abdominal conditions,

[1] We invited Dr. Danny Rosin to tell us how laparoscopy could be used for abdominal re-exploration—even following open procedures (and have added our own comments at the end of his section).—The Editors

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it naturally follows that acute surgical complications may be optimally handled by a minimal-access approach.

## Postoperative Conditions Treated by Laparoscopy

- **Mesenteric ischemia** (Chap. 23). One of the earliest applications of laparoscopy after a recent laparotomy was to perform a second-look operation after treating acute mesenteric ischemia. The purpose of this procedure is to ascertain the viability of potentially ischemic segments of bowel, for example, around the anastomosis after resection of gangrenous bowel. As the secondary intervention is a simple diagnostic procedure (unless further resection is necessary), it can readily be accomplished via laparoscopy. It has even been suggested that laparoscopic ports should be left in place at the end of the first operation to facilitate access at the second look, but we consider this approach unnecessary and risky. Possible injury can be caused by the port itself, which may also serve as a port of entry to bacteria, and reinsertion of new ports is simple enough.
- **Early postoperative small bowel obstruction** (Chap. 48) is a relatively infrequent condition as opposed to the more common postoperative ileus. At times, it will require a second intervention. Laparoscopic management of bowel obstruction is an established procedure, and we have successfully applied this approach in several cases of early postoperative obstruction after appendectomy, colectomy, and laparotomy for trauma.
- **Peptic ulcer perforation** (Chap. 18) is another rare postoperative complication not directly related to the specific procedure performed but possibly related to postoperative stress response or to ulcerogenic medications. We have treated such a case by laparoscopic omentopexy, just as in our standard approach to "primary" duodenal peptic perforations.
- **Intra-abdominal infections** (Chaps. 12, 49, and 52.1) may include established abdominal abscesses and septic conditions associated with recent anastomoses. Most of the postoperative abscesses are amenable to percutaneous drainage guided by computed tomography (CT), but a few are not accessible and mandate surgical drainage. Unless treating a patient in extreme conditions of septic shock, laparoscopy can be used to access the abscess cavity, drain and irrigate it, and leave suction drainage in the area.
- **Anastomotic leak** (Chaps. 50, 51) is another dreaded postoperative complication. It may manifest as a free intestinal leak or as an inflammatory condition ("perianastomositis"). Exteriorization and stoma creation are the usual treatment of the first condition, but the peritonitis leads to a high rate of wound infection, abdominal wall edema, and a risk of increased intra-abdominal pressure. The need for temporary abdominal closure is frequent. Laparoscopy may permit bowel exteriorization and abdominal toilet without disturbing the original laparotomy wound. In addition, perianastomositis, although usually responsive

to antibiotic treatment, may be associated with the presence of free abdominal gas but without actual spillage of bowel contents. This is frequently treated by anastomotic takedown or a proximal diversion. We have a limited experience with several patients in whom laparoscopy revealed a localized inflammatory process, without actual spillage or generalized peritonitis, and despite the presence of free gas, drainage alone led to full recovery.

### Technique

Access to the abdominal cavity must be established by the open technique, using a Hasson cannula, as the bowel may be distended and adherent to the abdominal wall. The port is placed away from the previous incision, usually laterally in the abdominal wall, to avoid the inevitable adhesions to the fresh scar. Some of the adhesions can be separated bluntly by careful movements of the camera as the bowel may be edematous and friable. Further trocars are placed as necessary, when enough space is established, to complete the space creation and permit abdominal exploration. Nontraumatic instruments should be used, and bowel handling should be kept to a minimum, preferably manipulating the bowel by grasping its mesentery to avoid serosal tears and perforations. Although at times the pathology is evident, it is frequently hidden by adhesions of omentum and bowel loops. The abdomen may initially appear "benign," but a thorough search in spaces such as the pelvis, subphrenic areas, or retroperitoneum may reveal a compartmentalized process. Previous data obtained by a CT scan may help direct the exploration and prevent false-negative explorations and missed pathologies.

**I believe that laparoscopic abdominal reexploration has a definitive role in:**

- Persistent early postoperative intestinal obstruction
- "Second look" for mesenteric ischemia
- Perforated peptic ulcers
- Drainage of abscesses and collections (when percutaneous attempts fail)
- Drainage (with or without exteriorization) for anastomotic leaks

### Editorial Comment

We agree with Dr. Rosin that laparoscopic re-exploration in the hands of well-trained and experienced laparoscopic surgeons may be advantageous compared with relaparotomy. The patients must be well selected in terms of their physiology (you do not want to pump lots of gas into the distended belly of a moribund patient) and intra-abdominal pathology. In fact, in most instances the procedure would be CT-guided laparoscopy to compensate for the lack of manual

exploration of blind spots. And, what is true with any laparoscopic procedure should be *crucial* here: "Do not f**k around, do not damage anything—and for God's sake—know when to stop and open up!" [By the way: did you know that Hemingway's publishers forced him to change the term f**k to "muck"? Well, we have to thank our own publishers for being more permissive! ☺ The Editors]

> "A surgeon … is like the skipper of an ocean-going racing yacht. He knows the port he must make, but he cannot foresee the course of the journey. At every stage he must have a plan, based on a working knowledge of his present position, that will allow him to make for the best of several available harbours should things go wrong, or if none is suitable he must know where to find temporary refuge under the lee of the land till he can resume his journey." (William Heneage Ogilvie, 1887–1971)

# 53 Abdominal Wall Dehiscence

Moshe Schein

*The gut bursts out either because you did not close the tummy properly or it has no room inside…*

When performing rounds on your patient, who 5 days ago had a laparotomy for intestinal obstruction, you find his wound dressings soaked in some clear pinkish fluid. "Change the dressings more frequently," you mutter to the intern. A day later, during lunch, you are paged by the head nurse on the floor: "Doctor, Mr. Hirsch's intestines are sprawled all around his bed. Please come and help!" How embarrassing.

## Definitions

*Abdominal dehiscence* is either complete or partial, the latter being much more common.

- **Partial** (covert, latent) dehiscence is a separation of the fascial edges of the wound without evisceration or full exposure of the underlying viscera. It presents usually a few days after the operation with some serosanguinous peritoneal fluid seeping through the wound. When the skin edges are separated or if, as commonly occurs, wound infection is present, you may see the exposed fascia, loose fascial sutures, and occasionally a fibrin-covered loop of intestine.
- **Complete** dehiscence is full a separation of the fascia and skin. Loops of intestine—if not glued in place by adhesions—eviscerate "all over the place."

## Etiology

Multiple mechanical, local, and systemic factors contribute to abdominal wound dehiscence: ileus, distension, deep wound infection, pulmonary disease, hemodynamic instability, stomas in the wound, age >65, hypoalbuminemia, systemic infection, obesity, uremia, uncontrolled diabetes, malignancy, ascites, corticosteroid use, and hypertension. These are factors that cause *poor tissue*

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*healing* or increased *intra-abdominal pressure*, and you will find a few of these in any patient who suffers a dehiscence. Dehiscence, be it complete or partial, is associated with a significant mortality rate. The prevailing perception is that dehiscence is only a marker for these underlying local and systemic factors and thus is not directly responsible for the associated morbidity and mortality. However, the way dehiscence is managed also affects the outcome, as you'll see below.

## How to Prevent Dehiscence?

You can prevent dehiscence by:

- Choosing a "correct" incision (› Chap. 10)
- "Correctly" closing the abdomen (› Chap. 43)
- Not closing abdomens that should be left open (› Chaps. 40 and 52)

Generally, it appears that vertical incisions—especially the midline—are associated with a greater incidence of dehiscence than transverse incisions. In mechanical terms, **three main causes for dehiscence exist: the suture breaks, the knot slips, or the tissue breaks** (i.e., the suture cuts through the tissues). The last mentioned is the dominant one. Please reread › Chap. 43 to ingrain in your brain how dehiscence can be prevented by correct abdominal closure. And, remember that abdomens that are very likely to burst could be left open as discussed elsewhere in this book (› Chaps. 40 and › Chap. 52.2).

### *Additional Points*

- **Closing the skin only!** To avoid intra-abdominal hypertension and subsequent fascial dehiscence, you can leave the fascia unsutured but close the skin. This is what we do occasionally in high-risk situations, after, for example, laparotomies for mesenteric ischemia or intestinal obstruction within a complex incisional hernia, when and where the intestinal distention precludes tension-free closure. Particularly in elderly patients, the skin is lax and mobile, thus easily approximated to cover the resulting fascial defect. We suture the subcutaneous layer with heavy absorbable suture and the skin with nylon 2–0, which is left in situ for at least 2 weeks. (Do not let any nurse or intern come near these sutures!). Patients are easily mobilized wearing a Velcro abdominal binder to support their wound. Of course, the morbidity of this "planned hernia" is much less than that of formal "laparostomy" (› Chap. 52). **A planned hernia is much better tolerated than fascial dehiscence!**
- We use abdominal Velcro binders after major laparotomies (e.g., significant abdominal wall incisions) on a regular basis. We do not allow a patient out of bed without wearing a binder to support the abdominal girth. No, we do not have a randomized double-blind study to show that this prevents dehiscence, but it makes us feel better.

When a patient develops early signs of fascial dehiscence by draining serosanguinous fluid from the wound, a common error committed by junior surgeons is to rush and treat it like a "wound infection" by removing the skin sutures. This may, however, convert a minor, partial dehiscence, which could be treated conservatively, to a complete dehiscence needing an immediate reoperation. Make it a rule: do not let anyone remove sutures (or staples, drains, or tubes) from your postoperative patients without your consent. Unfortunately, in most hospitals the lash is no longer allowed, but you should still punish harshly those who commit such crimes; they will not do it again.

## Treatment

"Traditional" surgical texts advocate an immediate surgical closure of the dehiscence. For example, Schwartz's textbook recommended that, "If the patient can tolerate the procedure, a secondary operative procedure is indicated." But, what kind of patient "cannot tolerate the procedure" is not stated. The guidelines published by the American College of Surgeons state that if "dehiscence is significant, an immediate operative reclosure is preferred." A text devoted to complications in surgery suggests that "when a dressing is found soaked in salmon-pink fluid … a fascial defect or a loop of bowel palpated just below the skin … a binder must be applied and the patient sent promptly to the operating room." In addition, "failure to repair dehiscence results in evisceration in most cases … reclosure, in contrast is strikingly successful." Another recent text on reoperative general surgery emphasizes that "abdominal wound dehiscence is clearly a surgical emergency" requiring fascial reclosure (Fig. 53.1).

Managed according to these recommendations, the patient is taken to the operating room, where the abdomen is resutured with "retention sutures" (see Chap. 43). So, why is the mortality so high? Many still think that "most deaths associated with dehiscence today are the result of ongoing primary disease rather than being a direct result of this complication." There is a large body of data, however, to suggest that such a hypothesis is not true. Instead, it appears that the "recommended" treatment of the dehiscence by "reclosure" plays a significant role in the associated morbidity and mortality.

We believe that that forcing the distended intestines back into a cavity of limited size may kill the patient. The **fatal factor** leading to the high mortality rate associated with abdominal wound dehiscence is not the dehiscence itself but the emergency procedure to correct it, which produces intra-abdominal hypertension, which in turn adversely affects cardiovascular, respiratory, renal, and intestinal function, leading to multiorgan dysfunction and eventually to death (Chap. 40).

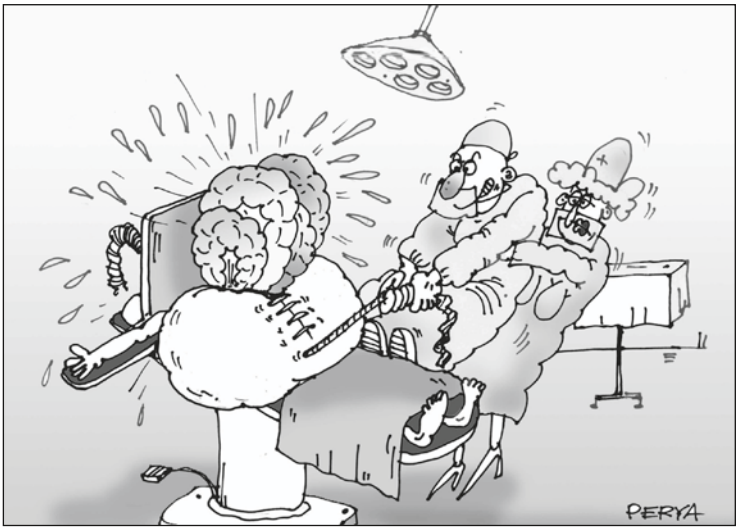


**Fig. 53.1.** "Doc, pull harder!"

## Recommended Approach to Dehiscence

Instead of routinely "pushing back" the bulging viscera into the limited space of the peritoneal cavity, be selective, using the following rationale:

- **Complete dehiscence mandates** an operation to reduce the eviscerated abdominal contents. You cannot leave the intestine hanging outside in the bed. You may attempt a reclosure of the fascia when a faulty closure technique or a broken suture is the cause of the dehiscence and local circumstances permit, but only if the facial edges can be approximated without excessive tension. If this is not the case, you should leave the abdomen temporarily open, using one of the temporary abdominal closure (TAC) methods described in Chap. 52.2 (Skin closure only is another option; see preceding discussion). We avoid reclosure also when the abdominal wall is frail or if the cause of the evisceration—persistent intra-abdominal infection—is still present. **What is the use of resuturing the abdomen if the factors causing the evisceration in the first place are still present?**
- **Partial dehiscence may be managed conservatively.** Many surgeons feel compelled to take the patient to the operating room and resuture the fascia. But, what is the rush? In our experience, this not only is unnecessary but also may complicate matters. The natural course of a partially dehisced wound is to heal by granulation and scarring with or without the formation of an incisional hernia. Resuturing such a friable wound in a compromised patient entails the additive risks of anesthesia and abdominal re-entry while not preventing the eventual hernia. The hernia, if symptomatic, can be repaired electively at a later stage. If the bowel is partially exposed, we would approximate the skin to cover it. Otherwise, the wound is managed as any open wound (Chap. 55) until healed.

**In summary:** regard dehiscence as a **symptom rather than a disease.** Operate for complete dehiscence with evisceration; resuture fascia or use a TAC device selectively. Most cases of partial dehiscence are best treated conservatively.

Commonly, dehiscence of the abdominal wound represents a spontaneous decompression of intra-abdominal hypertension and thus could be defined as a "beneficial" complication.

# 54

# LIRS, SIRS, Sepsis, MODS, and Tertiary Peritonitis

Moshe Schein

*The larger the operation—the greater the trauma*
*The greater the trauma—the stronger the SIRS*
*The stronger the SIRS—the sicker the patient*
*The sicker the patient—the higher the M & M*

## Local and Systemic Inflammation and Its Consequences

In the first chapter of this book, we talk about your patient being locally and systemically inflamed by his surgical disease, your treatment, and the complications of both. In almost every subsequent chapter, you have been reminded that the magnitude of the inflammation correlates with that of the disease process and the operation. You were told that the more inflammation there is—or that you create—the more likely is your patient to develop organ dysfunction or failure and to die. In this chapter, we concentrate on the inflammation—both local and systemic—and its consequences. The biological events involved are immense and chaotic, but let us maintain a simplistic attitude—you did not buy this book to read about cytokines, right?

### *Background*

Matters were much simpler for us surgeons only a few years ago. Postoperative or post-traumatic fever, raised white cell count, deteriorating organ system function, with or without shock, meant for us only one thing: "sepsis." And, sepsis meant "infection," usually bacterial in nature, necessitating antibiotic therapy. So, we administered the "strongest," ever-changing antimicrobial agents available on the market; we looked for pus, draining it whenever present, and we prayed for the infection to subside. Some of our patients, however, continued to deteriorate, dying slowly from respiratory or renal failure. We buried them, blaming the death on an "intractable sepsis," which in our minds always signified an infection "somewhere" in their blood, abdomen, urine, or lungs. Look around you: isn't this the way many of your senior colleagues, mentors, or teachers still think and practice?

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Then, in the early 1980s, when our supportive care and reoperative efforts became more aggressive and our patients lingered in the intensive care units (ICUs), resulting in prolonged survival, we began to note that many of our patients were dying a septic death in the absence of infection; we did not understand why. In the late 1980s, the rapidly developing field of molecular biology produced a huge amount of data to explain that a lot of what we see in clinical practice is not sepsis or infection but **inflammation**, which in turn is fueled by proinflammatory mediators such as **cytokines**. This has totally changed the way we look at the surgical patient. We see this patient as **inflamed** by the disease and the operative trauma together with the postoperative complications and associated therapies. In fact, most of our postoperative patients who die today do so from inflammation or infection—alone or in combination. But, before we go further, we need to clarify a few issues in terminology.

### *Terminology*

Take a knife and cut your finger; sooner or later, your finger will manifest the classic signs of inflammation—redness, swelling, warmth, and pain—produced by locally generated inflammatory mediators. This is *LIRS or local inflammatory response syndrome.*

Now, take a patient and apply multiple, and deeper, knife wounds to the soft tissues. In addition to the local inflammation, the patient will experience signs of systemic inflammation: fever, tachycardia, and even elevation of white cell count. This is *SIRS or systemic inflammatory response syndrome.* SIRS occurs when the local proinflammatory mediators of LIRS spill over into the systemic circulation, affecting the entire organism. In surgical practice, most instances of SIRS are secondary to LIRS. Think of acute pancreatitis, retroperitoneal hemorrhage, and acute cholecystitis. *Note that proinflammatory cascades leading to SIRS are initially, at least, well compartmentalized locally, with the SIRS representing only the tip of the iceberg.*

LIRS and SIRS can be generated by *sterile*, noninfective causes (tissue trauma, necrosis, burn) as well as infective causes (e.g., acute appendicitis). The ensuing clinical manifestation are, however, indistinguishable.

- **Infection** is a microbiological phenomenon characterized by the invasion of normally sterile tissue by microorganisms. The host's local response to the infection is LIRS; the systemic response is SIRS. And, here we arrive at the term *sepsis*.
- **Sepsis** is currently defined as the systemic response to *infection* consisting of systemic inflammation (*SIRS)* with microbiological evidence of infection. (Sepsis = SIRS + Infection). In other words, *SIRS* and *sepsis* represent an identical host-determined response, the former in culture-negative patients and the latter when infection is documented. Both manifest a continuum of clinical and pathophysiologic severity.

According to current consensus, SIRS may be diagnosed in any patient who manifests two or more of the following: temperature > 38°C (100.4°F), heart rate >90/min, respiratory rate >20/min, white cell count > 12,000 cells/mm$^3$. With such a low inclusion threshold, it appears that most of your emergency abdominal postoperative patients, and all your surgical intensive care unit patients, experience a degree of SIRS. (In fact, there was someone who said that even engaging in vigorous sex produces clinical SIRS—"To SIRS with love." ☺)

The noxious stimuli, which incite *proinflammatory* mediators leading to LIRS and SIRS, induce in parallel potent anti-inflammatory mediators to produce what the late Roger Bone (1943–1996, the "father" of SIRS) termed CARS or compensatory anti-inflammatory syndrome. CARS manifests clinically as immunodepression and an increased susceptibility to infection, so typical in the aftermath of major surgery and trauma. Conceptually, the balance between SIRS and CARS determines outcome. When CARS equalizes SIRS, homeostasis results. When SIRS is unopposed, organ dysfunction develops. When CARS is the winner, primary or secondary infections may remain as the only manifestation of the entire process.

As with many other essential things in life, too much may be harmful and too little may be unsatisfactory. The same is probably true for the inflammatory and anti-inflammatory responses, which in a certain phase and magnitude are beneficial but when out of control are harmful. Understand, however, that these events are extremely complex, chaotic, nonlinear, and unpredictable; some severely traumatized patients do not progress from SIRS to organ failure, and some do. Your grandmother may be right: *genes* play a role in everything.

This is, of course, a highly simplistic version of the reality, much of which we still do not understand, but as Ralph Waldo Emerson (1803–1882) said: **"It is proof of high culture to say the greatest matters in the simplest way."**

### *From SIRS to Multiorgan Dysfunction Syndrome (MODS)*

The same proinflammatory mediators that locally possess salutary actions, when overproduced and systemically spread, eventually damage the microcirculation, resulting in progressive damage to vital organs. The inflammatory mediators released by the circulating macrophages, which are activated by the disease or injury, result in widespread endothelial damage, causing capillary leak and coagulation and resulting in cellular damage and then organ dysfunction (lungs, kidneys, liver, gut, etc.). Cytokines such as interleukin 6 (IL-6) not only promote local coagulation but also suppress local fibrinolysis, a compensatory mechanism that attempts to lyse the forming clot.

Thus, your SIRS patient swells, gains weight, his or her lungs become wet, the gastric mucosa bleeds, liver enzymes rise, the kidneys fail, and so it goes. He becomes autointoxicated with inflammatory mediators. The more severe the

damage to the organs, the greater the number of organs involved and for a longer duration and the less likely is your patient to recover. When three organ systems fail the prognosis is grim; when the fourth joins in, the die is cast.

### *The Second-Hit Phenomenon*

Imagine a boxer in a ring. Having just received a major blow, he lifts himself back up to his feet, where almost erect, he receives a second hit, which is softer than the first one but enough to send him back onto the floor—a fatal knockout. Similarly, your SIRS patient is susceptible to a second hit; the inflammatory response, switched on by the primary hit, is easily amplified by additional, albeit relatively minor, hits. Think of your patient as an aging boxer. The abdominal emergency plus your operation represent the first hit. From that point, any additional procedure (or complication) constitutes a potential second hit, which greatly increases the magnitude of the inflammation.

## Treatment of SIRS and MODS

The search for the magic bullet to arrest the cascades of LIRS and SIRS and to modulate CARS continues, but meanwhile is there anything we can do for these patients?

- First, we need to use terms accurately, distinguishing between local inflammation and infection, between SIRS and systemic sepsis. We must understand that LIRS and SIRS do not always mean infection and thus may not be an indication to administer antibiotics (Chap. 7 and 47).
- Second, we must restore and maintain perfusion of end organs to prevent additional ischemic injury, which will contribute to the inflammation (Chap. 6).
- Third, we must avoid adding fuel to the inflammatory fire, appreciating that what we do, and how we do it, does matter. A prolonged operation and rough handling of tissues means more inflammation, more LIRS and SIRS. Unnecessary and poorly timed reinterventions may produce a second hit in a previously primed host.
- Fourth, we should deal promptly with ongoing infective (e.g., abscess) and noninfective (e.g., necrotic tissue) sources of LIRS and SIRS.
- Fifth, we should attempt to preserve the integrity of the mucosal layer of the gut (through early enteral feeding) to prevent translocation of bacteria and endotoxin, which may contribute to SIRS, sepsis, and MODS (Chap. 46).
- Sixth, we should minimize iatrogenic contributors to LIRS and SIRS. The patient must not be continuously injured and crucified in bed with indiscriminate insertion of catheters, tubes, and pipes. Blood products may be harmful and should be used judiciously (Chap. 45). Antibiotics are a double-edged sword and may in fact increase SIRS by various mechanisms.

It is impossible to prove that each of these measures decreases SIRS and MODS, but proper management as a whole is the mainstay of prevention of this "horror autotoxicus."

## Tertiary Peritonitis

In Chap. 12, you were introduced to the concepts of peritoneal contamination and infection and the terms **primary and secondary peritonitis.** In Chap. 52.1, you read: "When peritonitis persists despite apparently adequate source control and repeated reoperations, think about *tertiary peritonitis.*" So, what exactly is this entity?

The aggressive supportive and operative measures discussed in the previous chapters allowed for the initial salvage of patients who previously would have succumbed early to uncontrolled secondary peritonitis. This success, however, created a new subgroup of patients. Let us take one as an example:

A 75-year-old male underwent an emergency subtotal colectomy with an ileorectal anastomosis for an obstructing carcinoma of the sigmoid colon (Chap. 25). Six days later, he was rushed for a re-laparotomy because of diffuse peritonitis and a documented free anastomotic leak. At operation, his abdomen was found to be full of fecal material. It was cleansed, and the anastomosis was dismantled; the rectum was closed as in a Hartmann procedure and the ileum exteriorized as an end ileostomy. The abdomen was left open as a "laparostomy" (Chap. 52.2). During a planned re-laparotomy 48 hrs later, residual collections of "thin" pus were evacuated. The patient continued to be "septic" and developed MODS. Computed tomography (CT) of the abdomen showed fluid in the pelvis and gutters; diagnostic aspiration revealed the presence of fungi. An antifungal agent was added to the wide-spectrum antibiotics the patient was already receiving. He continued to deteriorate; a relaparotomy disclosed a bit of murky peritoneal fluid, which grew *Candida* and *Staphylococcus epidermidis*. The antibiotic regimen was changed. MODS worsened, leading to the patient's demise 5 weeks after the first operation. The hospital bill was $250,000.

You have seen similar patients, eh? Probably one of them is now fading away in your ICU. The term *tertiary peritonitis* was coined to describe this situation, which develops late in the postoperative phase, manifests clinically as SIRS with MODS, and is associated with a peculiar peritoneal microbiology consisting of yeasts and other weird commensals. These organisms, normally of low virulence, probably act as a *marker* of tertiary peritonitis and not its *cause*. Their presence also reflects the global immunodepression of the affected patient, allowing superinfection of the re-explored abdomen with organisms resistant to the antibiotic regimen the patient is receiving. Further antimicrobial administration and operative interventions are futile and may contribute to the peritoneal

superinfection. The usually fatal outcome of tertiary peritonitis, which conceptually falls within the SIRS-MODS complex, indicates that current antibiotic-assisted, mechanical answers to severe peritonitis have about reached their limits, and the patient is unsalvageable.

> "Our ingenuity in developing terminology exceeds our abilities to take care of these patients once they have developed the syndrome of MOF. The solution to MOF or MODS or SIRS is prevention." (Arthur E. Baue)

[We asked John Marshall of Toronto, who originated many of the terms described, to tell us more on how to prevent and treat SIRS, MODS, and tertiary peritonitis.—The Editors]

## Invited Commentary

John Marshall

The world of the critically ill surgical patient is a strange one. Its genesis lies in the performance of feats of surgical daring that were almost unimaginable even half a century ago, and its progress reflects the expression of processes that have no precedent in evolutionary biology. Could Halsted or Kocher have anticipated an age when surgeons would sew the liver of a cadaver into a patient dying of cirrhosis or salvage a patient who presents in cardiac arrest from a gunshot wound to the heart? The leading surgical minds of their era spoke of "shock" because they believed that wounded patients died of an overwhelming sense of fear, and it was not until the early years of this century that Alfred Blalock (1899–1964) refined this view and showed that shock arose not from the brain but from a lack of circulating volume within the vascular tree. He set the stage for a bold and unprecedented conceit—that the clinician, through the correction of acute physiologic derangements and the support of fundamental physiologic functions—could prevent, or at least forestall, the inevitability of death from acute life-threatening illness.

The late John Border (1926–1996), a trauma surgeon who contributed so much to contemporary views of the pathogenesis of critical illness, captured this conceptual advance by allusion to a classical motif from American cinema. The scene is a battlefield during an unnamed war. Surgeons are operating desperately to save the life of the shy and handsome, but somehow anonymous, soldier who has been wounded. The urgency of their mission is underlined by rapid cinematic cuts between the surgeons and the rubber bag that moves in and out as

the patient inhales the ether that provides him with pain relief. The situation becomes desperate. Beads of sweat appear on the brows of the operating surgeons, and the movement of the anesthetic bag becomes shallow … then stops. The surgeons bow their heads, and the camera pulls back to show a silent medical team, lost against the sullen sky of the enveloping evening. And Border opined: "They didn't realize that all you have to do is to squeeze the bag."

We have squeezed the bag, and much more, and the author of this chapter has beautifully articulated the consequences of that squeezing. It is both incomprehensibly complex and very simple; let me just underline a few of the principles that I hope you will retain from these discussions.

First, patients no longer die of their primary diseases; rather, they die of their response to that disease. Shock kills not because of a deficit of circulating intravascular volume (a state that we can readily correct with intravenous fluids) but because of the biologic processes that are activated during reperfusion of ischemic tissues. Infection kills not because of uncontrolled proliferation of microorganisms (a process we can easily avert with source control measures and systemic antibiotics) but because the host responds to the infecting microorganism. This concept was beautifully demonstrated in an animal study performed more than two decades ago by Michalek et al. (1980). Two strains of mice, one known to be sensitive to endotoxin and the other resistant because of a point mutation in a single gene, were irradiated and then given bone marrow transplants from the other strain. The lethality of endotoxin, a bacterial product, was transferred to the resistant-strain animals who received bone marrow cells from their sensitive relatives. In other words, the lethality of bacterial endotoxin is not an intrinsic property of the molecule but rather a function of the fact that the host responds. It is not uncommon to see a critically ill, immunosuppressed patient who survives a life-threatening infection only to become gravely ill as the immunosuppression abates, and he or she is then able to respond to the infection.

An important corollary of this principle is that interventions against infection will not alter the course of a disease process whose pathophysiology reflects the *response* to infection. Stated differently, surgical source control and systemic antibiotics are anti-infective measures whose objective is to reduce the size of the microbial inoculum with which the host must contend. Their utility is critically dependent on establishing a diagnosis by demonstrating that a focus of infection, or uncontrolled microbial proliferation, is present, and it is incumbent on the surgeon to demonstrate conclusively that such is the case for antibiotics kill not only the organisms responsible for the infection but also the normal colonizing flora of the host. In doing the latter, they facilitate colonization, and ultimately superinfection, by antibiotic-resistant organisms, a state that is epitomized by the phenomenon of tertiary peritonitis, described in this chapter.

Equally, the injury experienced by the critically ill surgical patient reflects not only what happened to him or her prior to arrival at the hospital but also the intervention of the surgeon and other clinicians who provided care. Contemporary critical illness is an intrinsically iatrogenic disorder for it only arises in patients who in the absence of medical intervention would have died, but its evolution reflects the inadvertent consequences of the interventions used to resuscitate the patient and to sustain life. The challenge we face as clinicians is to apply new technologies, but even more importantly, to recognize the potential adverse consequences of these and to know when to back off.

Yet another concept intrinsic to this discussion is that the "syndromes" of critical illness are not well-defined pathologic entities but rather metaphors for a process that we only dimly understand. For example, more than a decade ago a group of intensivists met to try to achieve consensus on the definition of *sepsis* (Bone et al. 1992). They coined the phrase *systemic inflammatory response syndrome* out of a desire to assert that the clinical syndrome of sepsis can arise in patients who are not infected and to recognize that we did not have terminology to describe such a state. However, this concept does not necessarily define a syndrome if by a syndrome we are referring to a constellation of signs and symptoms caused by a discrete pathologic process (Marshall 1999) and the criteria proposed to delineate that supposed syndrome were both arbitrary and highly nonspecific (Vincent 1997). SIRS implies a response, and a relatively significant one at that, but its diagnostic import is nothing more than that the clinician should consider looking for a cause of that response (Marshall and Baue 2000). The notion that there are other syndromes designated as CARS or MARS (mixed acute response syndrome) (Bone 1996) similarly overstates our basic understanding and descriptive capacity. It is a biological truism that an acute inflammatory response entails the release of both pro- and anti-inflammatory mediators (and even this distinction is simply a matter of conceptual convenience for a human intellect that insists on categorizing), but it far oversteps current understanding to suggest that we can identify discrete syndromes or clinical manifestations that point to a particular pattern of mediator response. SIRS and CARS are useful as concepts but entirely unhelpful as patterns of clinical manifestations that might guide the care of a particular patient or even shape the design of a clinical trial.

Finally, despite Dr. Schein's admonition that "you did not buy this book to read about cytokines," let me try to convince you that, although the inflammatory response is complex (and sufficiently complex that no one really understands it in a comprehensive way), its basic principles are not only straightforward but also seductively appealing. Inflammation is mediated primarily by the *innate* immune system, in contradistinction to the *adaptive* immune system that includes T cells and B cells. Innate immunity is highly conserved through evolution; the same principles

that regulate innate immunity in the pope, also regulate innate immunity in fruit flies and sea slugs, so they have to be simple. The innate immune system evolved to recognize danger both from microorganisms in the environment and from injured tissues in the host. Cells of the innate immune system—principally neutrophils and macrophages—recognize molecular patterns that signify danger (e.g., complex lipids and carbohydrates that are found in bacterial, but not mammalian, cells, or molecules such as heat shock proteins or RNA that are normally found within the cell). Recognition occurs through a family of ten receptors called toll-like receptors ("toll" is the German word for "cool"—nothing sophisticated here) that bind these substances and, in doing so, activate a series of intracellular cascades that lead the cell to express genes that encode inflammatory mediators, two of the most important being tumor necrosis factor (TNF) and IL-1. These mediators also can activate cells, leading to the release of a complex mélange of cytokines, prostaglandins, and reactive intermediates of oxygen and nitrogen and triggering the coagulation cascade.

But, let us return to the world of clinical reality. We do not need fully to understand the inflammatory process to recognize that we need to minimize exposure of the innate immune system to danger signals, whether by draining an abscess to reduce the bacterial load, providing rapid resuscitation to prevent tissue ischemic injury, or taking steps to limit iatrogenesis through keeping ventilatory volumes low and minimizing unnecessary exposure to vasoactive drugs and antibiotics. Good clinical care is grounded in common sense and carefully considered intervention, not in esoteric renderings of biology.

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# 55 Wound Management

Moshe Schein

*The fate of the surgical wound is sealed during the operation; almost nothing can be done after the operation to modify the wound's outcome.*

*A minor complication is one that happens to somebody else.*

All that is visible to the patient of your wonderful, lifesaving, emergency abdominal operation is the surgical wound (Fig. 55.1. ). Wound complications, although not life threatening, are an irritating source of painful, and often prolonged, morbidity, which bothers the patient and his or her surgeon alike. It is no wonder, then, that throughout generations, surgeons developed elaborate rituals to prevent and treat wound complications. Now that you are reading one of the last chapters of this book, it is hoped you are sufficiently brainwashed to deplore elaborate gimmicks and to demand pragmatic solutions instead.

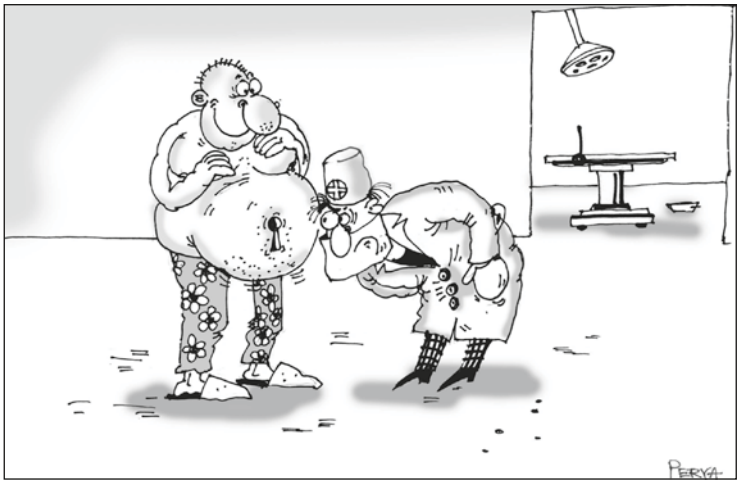

**Fig. 55.1.** "I hope you are satisfied with the beautiful wound, eh?"

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## Definitions and the Spectrum

For practical purposes, you do not need complicated definitions used by epidemiologists or infection control nurses—the (usually humorless) creatures who tell you not to walk out of the operating room with your scrubs on…

- An **uncomplicated** wound is a sutured wound that heals uneventfully by primary intention. Note that following emergency abdominal surgery, an entirely uncomplicated wound is an exception. You don't believe us? Start to document from now on all your wounds and see for yourself the number of weeping or red and swollen wounds your patients have.
- **Complicated** wounds are extremely common after emergency surgery when prospectively assessed by *independent* observers. Conversely, when "reported" by surgeons, they become "rare" or "minor" due to our natural tendency to suppress or ignore adverse outcomes.

**The spectrum of wound complications is wide and encompasses infective and noninfective complications, minor and major.**

- **Minor complications** are those irritating aberrations in the process of healing that, however, do not impede primary healing of the wound: a small hematoma, a little erythema, some serous discharge. The distinction between an infectious and noninfectious process is difficult and also unnecessary. Why take swab cultures from such a wound if it will not affect therapy?
- **Major complications** are those that interfere with the process of primary healing and require your intervention: a large hematoma or a wound abscess in need of drainage.
- **Wound infection** for practical purposes is a wound that contains pus and requires drainage. Usually, such an infection represents a "walled-off" wound abscess, with minimal involvement of adjacent soft tissues or underlying fascia. Rarely, surrounding cellulitis is significant, or the deep fascia is involved, denoting a (deep) **invasive** infection.

## Prevention

Surgical technique and overall patient care are of great importance in minimizing the incidence of wound infection. Rarely is one aspect of management of singular importance, but it is the sum of the parts that yields favorable results. Emergency surgery is particularly associated with wound problems for several reasons. Contamination of the wound may arise from intestinal bacteria released at the time of bowel resection or from the organisms present in the established infection that the surgery was performed to treat (Chap. 12). In addition, there is insufficient time preoperatively to reverse all conditions that may adversely affect wound healing, such as shock, diabetes, and malnutrition (Chap. 6).

Evidence suggests that tissue hypoxia, hypothermia, and poorly controlled blood sugar predispose to wound complications. Thus, try—the best you can in the few hours you have (if any at all) before operation—to oxygenate the patient better (yes, give him that oxygen mask!), warm him up and administer insulin if necessary.

When you deal with complicated wounds, you get wound complications.

Yes, this aphorism is true, and a certain rate of wound complications is obligatory and inherent in the nature of this type of surgery. Nevertheless, you should strive to keep it as low as possible. How?

Let us reiterate here the above-mentioned aphorism: **"The fate of the surgical wound is sealed during the operation; almost nothing can be done after the operation to modify the wound's outcome."** Whether your patient develops a wound hematoma or infection depends on your patient and on you and is determined during the operation—not afterward. We quote Mark Ravitch again: **"The likelihood of wound infections has been determined by the time the last stitch is inserted in the wound."**

**Meticulous technique** as described in Chap. 43 is paramount. Here, a few preventive points are re-emphasized:

- Operate efficiently and carefully; avoid "masturbating" the tissues
- Do not strangulate the fascia with interrupted figure-of-eight sutures of wire, Ethibond, or vicryl; instead, use low-tension continuous springlike monofilament closure—letting the abdominal wall breathe (Chap. 43)
- Do not barbecue the skin and underlying tissues with excessive use of diathermy
- Do not bury tons of highly irritating chromic (or anything else) in the subcutaneous fat
- Do not close the skin with the even more noxious silk
- Do not place contaminating colostomies in the main abdominal wound
- Do not leave useless drains in the wound (or anywhere else). Do not forget that drains increase the risk of wound infections

Transfer your meticulous technique to the ward also. Nosocomial (hospital-acquired) infection is a menace to our patients. We have already mentioned the contribution that indiscriminate use of nonindicated antibiotics makes to the emergence of resistant organisms. The prevalence of these germs as colonizers of our patients is increasing, and spread from patient to patient is a major problem. Doctors are a major vector in this spread. Wash your hands every time you touch a patient. It seems astonishing that this message has to be repeated now, but studies have shown time and again that nurses are much more meticulous in their approach to this issue than MDs. This act of handwashing after each patient contact should be so ingrained that you have a sense of incompleteness until it is performed.

## Antibiotics

Antibiotic prophylaxis reduces the wound infection rate; its anti-infective effects are in fact more pronounced in the surgical wound than within the peritoneal cavity (› Chap. 7). Intraincisional antibiotics have an additional preventive role (› Chap. 43); this makes sense if you consider that the wound's defense mechanisms are much weaker than those of the peritoneal cavity. Many years ago, it was shown that systemic antibiotics are effective in preventing wound infections only if given within 3 hrs of bacterial contamination—the "effective period." **Postoperative antibiotics cannot change the fate of the wound** as they will not penetrate the area. Despite what you have been told hitherto by your local infectious disease specialists or surgical "gurus," brief *perioperative* antibiotic coverage is as effective in preventing wound infection as 7 days of post-op administration (› Chap. 47).

## Non-closure or Delayed Closure of the Wound

Leaving the skin and subcutis completely or partially open following contaminated or "dirty" procedures is still advocated by some "authorities." True, it may prevent wound infection in the minority of patients who are bound to develop one. At the same time, leaving these wounds open condemns the majority, whose wounds are destined to heal more or less uneventfully, to the morbidity of open wounds, the associated problems of management, and the risk of superinfection. Look at › Chap. 43 for more details on this controversial issue.

## Management

### *The Uncomplicated Wound*

Throughout history, surgeons were fascinated with the treatment of wounds because all they could do was to manage external post-traumatic wounds. For hundreds of years, surgical leaders advocated simplicity in the management of wounds.

Felix Wurtz (1518–1574) wrote: **"Keep them as neat and clean as possible, and disturb them as little as you can; so far as may be practicable, exclude the air; favor healing under the scab; and … feed it as you would a women recovering from her confinement."**

The great Joseph Lister (1827–1912) said: **"Skin is the best dressing."** The renowned physician William Osler (1849–1919) maintained: **"Soap and water and common sense are the best disinfectants."**

But, most surgeons took literally the famous adage by Ambroise Paré (1510–1590): **"I dressed him and God healed him"** and practiced unnecessarily elaborate wound management policies.

The uncomplicated primarily closed surgical wound needs almost no care. A day after the operation, it is well sealed away from the external environment by a layer of fibrin. It can be left exposed. Isn't it ridiculous to see gloved and masked nurses changing sterile dressings on routine surgical wounds? Some patients demand their wounds be covered; cheap dry gauze is more than adequate for this purpose. The chief aim of elaborate "modern" dressing material impregnated in antibiotics, silver, or whatever is to enrich the medical-industrial complex. We do not use them. Patients with uncomplicated wounds can shower or bathe any time.

### *The Complicated Wound*

For the complicated wound, the punishment should fit the crime. Minor nonspecific complications should be observed; the majority will resolve spontaneously. Again, starting antibiotics because a wound weeps a little serous discharge is not going to change anything; if the wound is destined to develop an infection, it will—with or without antibiotics. Major wound hematomas require evacuation, but this is extremely rare following abdominal surgery.

### *Wound Infections*

Wound infection following an emergency abdominal operation is usually caused by endogenous bacteria—the resident bacteria of the abdominal organs breached during the operation or the bacteria that caused the intra-abdominal infection in the first place. Following noncontaminated operations (e.g., blunt splenic trauma), the bugs causing wound infections are exogenous skin residents, usually a *Staphylococcus*.

A *streptococcal* wound cellulitis may develop a day after the operation with pain, swelling, erythema, and elevated temperature. This is mentioned in all textbooks, but we have never seen one; we have also never met anyone who observed such an early *Strep* wound infection. Wound infections also may present in your private office even weeks after the operation, skewing—underestimating—your hospital infection control data (which are collected only to pay lip service to the administration's need to produce statistics).

When in doubt, do not rush to poke in or open the wound—creating complications in wounds that would otherwise heal. Instead, be patient, wait a day or two, let the infection mature and declare itself.

Remember: a "hot red" surgical wound with surrounding erythema does not mean "cellulitis." It means that there is pus within the wound that has to be drained. As a rule, removing a few skin sutures and draining the pus treats most wound infections. There is no need to lay the whole wound open if only part of it is infected. You do not need a computed tomographic (CT) scan to diagnose a wound infection (this is not a joke; this is what "modern medicine" is educating people to do). All you need do is to remove a few sutures or staples and probe the wound.

## Aftercare

Aftercare should be simple. Open shallow wounds are covered with dry gauze and cleaned once or twice daily with water and soap. There is nothing better for an open wound than a shower or bath. Deeper wounds are **loosely** packed with gauze to afford drainage and prevent premature closure of the superficial layers. Antibiotics are not necessary. Do you give antibiotics after the incision and drainage of a perianal abscess? Of course not. So, why treat wound infections with antibiotics? A short course of antimicrobials is indicated when severe cellulitis is present or the abdominal fascia is involved, indicating invasive infection.

**Wound swabs? Wound cultures? Gram stains?** What for? As you know by now, the causative bacteria are mostly predictable (Chap. 12), and besides, how could the microbiological results change the therapy outlined in this chapter? The answer of course is that they do not. But, some wounds will become problematic, and it is then valuable to know the nature of the organism involved. The correct antibiotic can be administered without having to guess sensitivities or wait for the result of cultures. MRSA (methicillin-resistant *Staphylococcus aureus*) is currently endemic in the United States and elsewhere in the world and is increasingly responsible for our postoperative wound infections. Early treatment of complications from these wound infections is obviously desirable. Early cultures from leaking wounds do therefore have some role to play, but be sure to prevent your junior colleagues from prescribing antibiotics just because a positive culture appears.

Nurses and for-profit home care agencies push elaborate and expensive wound care methods to justify their continued involvement. Local application of solutions or ointments of antiseptics or antibiotics destroy microorganisms and human cells alike, induce allergy, and encourage bacterial resistance. **Expensive forms of wound coverage are a gimmick**. The industry is aggressively promoting various devices for **"vacuum wound therapy,"** claiming that application of negative pressure has beneficial effects on the healing of wounds. To the best of our knowledge, such claims are scientifically unfounded. Obviously, vacuum devices

offer the best solution for "productive" wounds (e.g., intestinal fistula in the middle of an abdominal wall defect;—Chaps. 50 and 52.2), but applying an expensive vacuum device on nonproductive wounds seems ridiculous to us.

**Simple is beautiful. Use soap and water; for our problematic wounds, we are enthusiastic users of honey. Try it!**

"I describe to my students what an injured animal does: it lies under a shady bush (rest, splint) by a water source (fluids, nutrition), licks the wound frequently (dressing changes) until it is clean and healing (time and patience)—and hope it makes them think past the gorgeous dressing promoted by manufacturers' reps." (Barry Alexander)

"Dressings on undrained wounds serve only to hide the wound, interfere with examination, and to invite adhesive tape dermatitis." (Mark M. Ravitch, 1910–1989)

"A surgeon should not wear a long tie that could dangle embarrassingly and dangerously down into a wound or incision while he leans over the patient." (Francis D. Moore, 1913–2001)

# 56 Postoperative Bleeding[1]

Samir Johna

*The wounded surgeon plies the steel*
*That questions the distempered part;*
*Beneath the bleeding hands we feel*
*The sharp compassion of the healer's art …*
(*East Coker*, T.S. Eliot, 1888–1965)

Every stroke of the scalpel opens capillaries or larger vessels, shedding precious blood. Blood—the iconic image of surgery—is a sign of the surgical sacrifice made by the patient through the ministrations of the surgeon. This sacrifice has an inverse benefit: the greater the bloodshed, the worse will be the outcome. The scalpel's bloody harvest must be limited by the joint actions of the surgeon's technique and the patient's natural hemostasis. This interplay of patient factors and surgical technique determines the amount of bleeding during and after surgery. If the patient's hemostasis is weak, then the surgical control of bleeding must be "strong" and complete. The attitude of a wise surgeon should endorse a "perfect hemostasis" rather than an "acceptable blood loss."

Bleeding complications are responsible for at least a tenth of surgical deaths. They usually occur in trauma patients, but few types of operations escape the occasional complication due to a postoperative bleed. The bleeding may have started before the operation or during the operation, or it may have commenced following the procedure.

The risks and complications of postoperative hemorrhage can be substantially reduced through measures such as adequate preoperative assessment and the identification and correction of deficiencies of circulating clotting factors, platelet count and function, hematocrit and blood volume, and body temperature. It is imperative, whenever possible, to optimize the condition of the patient prior to surgery. For example, chronically anemic patients can be helped by preoperative synthetic erythropoietin injections to improve their hemoglobin level prior to surgery. Clinical data indicate that the hemoglobin level can be raised by 1 gm/dl in as little as 2 weeks of therapy. Recently, preoperative normovolemic hemodilution has been introduced to minimize the intraoperative loss of whole blood.

[1] Barry Armstrong, MD, contributed to this chapter in the second edition of the book.

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This is achieved by collecting whole blood from the patient immediately before surgery, followed by a quick infusion of isotonic saline solution at a rate of four to one until the hematocrit level is 30% or less, thus inducing oligemia. The collected blood is then retransfused after the completion of the procedure. But, such planned maneuvers are of little use in patients undergoing emergency surgery.

A distinction should be made between technical causes of bleeding and coagulopathy or "surgical" versus "medical" bleeding. While the surgeon is responsible for the former, the latter can be the result of consumptive coagulopathy or congenital coagulation abnormalities, for which preoperative screening is often recommended. As increasingly sophisticated screening tests have entered the market, their effectiveness and utility for routine use have come into question, let alone the cost of sorting out abnormalities of doubtful clinical significance. Patients without a personal or family history of bleeding after dental extraction, previous surgery, or trauma are unlikely to suffer from familial or congenital coagulopathy. By the same token, absence of bruising or other signs of bleeding on routine physical examination predicts a low risk of postoperative bleeding.

Whenever natural hemostasis fails, the surgeon eventually learns about the hematoma, a falling blood count, or unexpected shock. Depending on the size of the bleeding vessel, the quality of the nursing care, and the cooperation of the patient, things might deteriorate slightly or seriously before the surgeon is called. Detecting bleeding and notifying the surgeon are key functions of postoperative nursing care.

Bleeding in the first day or two after surgery is called *reactionary hemorrhage*. If the hemostasis was good when the wound was closed, then this reactionary bleeding is due to a displaced or lysed clot, a failed suture, or a slipped clip. But in truth, in many instances it represents continued oozing that started during the operation. However, did you ever meet a surgeon facing postoperative bleeding in his or her patient who will not say: "It was dry when we closed?"

*Secondary hemorrhage* arises more than a week after surgery. This is usually associated with an infective or inflammatory process. An example would be bleeding from the pancreatic bed after necrosectomy for infected pancreatic necrosis (Chap. 19).

## Preventing Hematomas and Postoperative Bleeding

An ounce of prevention is worth a pound of cure.

– Technical causes of bleeding are least likely to respond to nonoperative intervention, so check the wound hemostasis. Major "pumpers" are controlled as they are encountered. Minor bleeders and ooze should stop spontaneously. Remember that natural hemostasis of minor bleeders ("bleeding time") takes about 5–7 min. Double-check wound hemostasis in midoperation and at closing.

**Table 56.1.** The 12 P's of surgical hemostasis: what to do if the patient is still bleeding (developed by Ahmad Assalia)

| First | Then consider | |
|---|---|---|
| Apply **p**ressure… with **p**acks or **p**ads | Giving **p**latelets, fresh frozen **p**lasma, protamine (to reverse heparin), and **p**acked cells (if still bleeding) | **P**PI (proton pump inhibitor) for bleeding peptic ulceration |
| Have **p**atience | Call **p**rofessor for help | **P**ASG (pneumatic antishock garment) for bleeding from broken pelvis |
| Suture with **P**rolene (or whatever) | If he can't help—**p**ray that you will not meet your patient at a **p**ostmortem | |

Do not let your assistant wipe the wound with a sponge since this may strip the beneficial platelet plugs. Teach him to dab gently at bleeders rather than wipe.

- Patient factors: you surely do not want us to bore you with yet another lecture on hemostasis. So, just remember the 12 P's—a mnemonic that may help your patient clot and prevent the patient from bleeding—presented in Table 56.1.

For details on coagulation testing, access **http://www.anaesthetist.com/icu/organs/blood/test.htm.**

**Many of today's patients present with their abdominal emergency while receiving antiplatelet agents or Coumadin. The following are the commonly ensuing dilemmas:**

- **What should I do if my patient is on antiplatelets or oral anticoagulation?** The management depends on the nature of the surgical disease and the indications for the antiplatelet agents or the anticoagulation. If the surgical disease can be managed nonoperatively, with antibiotics and supportive care (e.g., acute diverticulitis, acute cholecystitis), one should embark on such a strategy while measures are taken to reverse the effects of medication. If successful, surgery can be safely deferred until the antiplatelet drugs have been metabolized or the Coumadin therapy reversed or "bridged" (with subcutaneous low molecular weight heparin) if necessary.
- **How can I reverse antiplatelet effects?** Platelets are irreversibly inhibited by antiplatelet drugs. The serum half-life of Plavix is only 8 hrs, which means that there will be no detectable serum levels after 24 hrs. Therefore, to reverse its effects, platelet transfusions should not be given less than 12 hrs after of the last dose of Plavix. One unit of aphoresis platelets (single donor) should increase the platelet count by 30,000–60,000/μl in a 70-kg adult. Therefore, two units should be adequate for an acceptable level of platelets for surgical hemostasis.

Fact: platelets are ineffective if given within 12 hrs of the last dose of Plavix.

- **What should I do if emergency surgery is needed for a patient on antiplatelet drugs?** Recent data indicate that the hemorrhagic risk varies with the agents used. Intraoperative hemorrhagic risk for aspirin alone is increased by an average factor of 1.5. When clopidogrel (Plavix) is added to aspirin, the bleeding risk is much higher and is associated with higher transfusion rate. Despite this, when an operation is immediately required, one has no choice but to proceed with the Plavix on board as an intraoperative platelet transfusion would be ineffective. However, if the operation can be delayed for more than 12 hrs, one can reverse the Plavix by platelet transfusion. Antiplatelet drugs can be restarted immediately after the operation, particularly in patients at high risk for thromboembolic events (e.g., coronary stent in situ).

- **What should I do if surgery is needed for a patient on oral anticoagulants?** While oral anticoagulation therapy can be continued without increasing the risk of major bleeding for many low-risk procedures such as endoscopy even with biopsy, the situation is different for abdominal procedures. For emergency surgery, fresh frozen plasma (FFP) or prothrombin-complex concentrates (PCCs) should be used intraoperatively to control bleeding by reversing the action of the Coumadin. If time permits, Coumadin can be reversed before the operation as indicated next. Most surgeons do not feel comfortable operating if the INR (international normalized ratio) is higher than 1.5.

- **How can I reverse the anticoagulation effect?** The effects of oral anticoagulants can be reversed by two mechanisms:

1. Stimulating the intrinsic ability of the liver to produce new clotting factors II, VII, IX, and X through the use of vitamin K
2. External replacement through the use of blood products rich in clotting factors such as FFP or PCC

Each option can be used alone or in combination based on the needs of the patient or the urgency of surgery. Vitamin K is usually used orally in a single dose of 2.5 mg. However, this may take 24–36 hrs to work. Vitamin K can be used intravenously in a single dose of 1.5 mg, which is expected to work within 6–8 hrs (be aware of a possible anaphylactic reaction). If the surgery cannot wait that long, then the only alternative is to use FFP or PCC. Each unit of FFP raises the clotting factor level in an adult by 3–5%.The recommended dose is 10–20 ml/kg body weight, which corresponds to 4–6 units in a 70-kg adult.

## Postoperative Wound Hematomas

The most important clotting factor is the surgeon.

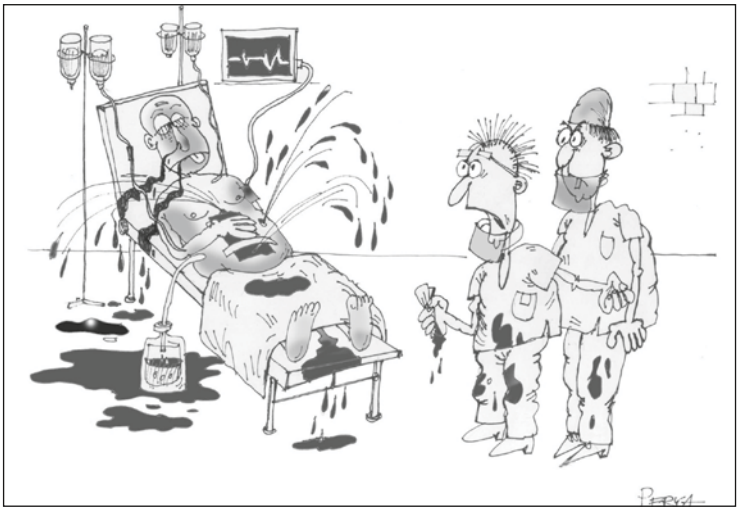

**Fig. 56.1.** "When we closed it was dry…"

**Fallacy 1: "The wound was dry when we closed" (Fig. 56.1).**
**Fact**: careful surgical technique will minimize the risk of post-op bleeding. A single look, as the abdomen is closed, may miss an important bleeder that is temporarily in spasm. Hypotension, surgical retractors, or a pressurized pneumoperitoneum can also mask bleeders. The wise surgeon will check for hemostasis a few times over the last 10–15 min of the operation. The surgeon will relax the pneumoperitoneum or reposition the retractors and sponges to spot hidden bleeders.

If postoperative *wound* bleeding continues despite local pressure, then the wound should be reexplored. Often, this can be done with local anesthetic, evacuating clots and controlling the bleeding points. Give a dose of intravenous prophylactic antibiotic first as reexploration for bleeding boosts the risk of infection. But, if you think that the wound hematoma arises from a major vessel, a return to the operating room will be necessary. For example, a rapidly expanding hematoma at the epigastric trocar site after laparoscopic cholecystectomy typically originates from an injured superior epigastric artery. Awaiting natural hemostasis of the superior epigastric will usually not kill your patient, but it will result in a large, uncomfortable, and ugly hematoma and bruise, which will take weeks to subside.

## Postoperative Abdominal Bleeding

The three words most often associated with reoperation for hemorrhage are: "It will stop."

**Fallacy 2: if the patient is bleeding and hypotensive, then you should start two large-bore intravenous lines and give Ringer's lactate quickly, at least 2 l.**

**Fact**: vigorous fluid resuscitation might restore blood pressure and pulse, but mortality and morbidity are increased. In the presence of uncontrolled bleeding, rapid fluid resuscitation will dilute clotting factors, increase the rate of blood flow from an actively bleeding site, and can "pop the clot" (Ken Mattox), opening new bleeders. Animal and human data show the benefits of restricting intravenous fluids when there is uncontrolled bleeding.

Permissive hypotension and small-volume intravenous therapy are the best strategy for supporting the patient's hemostatic mechanisms.

While any bleeding from or into a superficial surgical wound is obvious to the eye, postoperative bleeding into the abdominal cavity is hidden and thus more difficult to diagnose. Postoperative intra-abdominal bleeding is an *iatrogenic surgical trauma* that presents problems not dissimilar from those arising in the management of penetrating and blunt abdominal trauma (Chaps. 38 and 39, respectively).

- **Is the patient bleeding into the abdomen?** Tachycardia, hypotension, confusion, sweating, increased pain in the incision or the abdomen, abdominal distension, oliguria, dropping hematocrit, or a positive bedside ultrasound scan may all point to the diagnosis. Remember, however, that hypotension after surgery is not always due to blood loss. The persisting effects of anesthetics and narcotics may cause the blood pressure to drop. Postoperative epidural pain relief is a common cause of hypotension but beware of missing hemorrhage in this situation. Fluid resuscitation during the initial operation may have been inadequate to compensate for the fluid losses and "third-space" sequestration. The patient may have lost fluids from diarrhea or vomiting. In the elderly or those chronically taking steroids, an Addisonian crisis may provoke hypotension that responds rapidly to corticosteroids.
- **Should I rush the patient to the operating room?** With profound shock and full-blown abdominal compartment syndrome caused by the expanding hemoperitoneum, you should run to the operating room and open the abdomen. Otherwise, think about the steps mentioned next.
- **Should I image the abdomen?** In a stable patient, CT would confirm the size of the hematoma (e.g., in the gallbladder bed) and help estimate the volume of the hemoperitoneum. As with blunt abdominal trauma, CT diagnosis and follow-up might allow safe nonoperative management. A CT "blush"—extravasating intravenous contrast—may mark the source of active bleeding. In specific situations (e.g., after operations for hepatic trauma), *angiography* could localize and treat the bleeding. Bedside ultrasound is a viable alternative to look for accumulation of free fluid at mainly three locations: the lienorenal recess (around the spleen), the hepatorenal recess (Morrison's pouch), and the pelvis.

**Should I treat the patient nonoperatively?** Today, with most blunt abdominal trauma patients managed successfully without an operation, we tend to apply the same principles to the postoperative abdominal bleeders. Patients who *continue* to exhibit signs of hypovolemia after "gentle" resuscitation should be returned to the operating room. It is worth mentioning here that there is now no place for the old dogma of treating hemoperitoneum by tamponade, waiting for the intraperitoneal pressure to exceed that of the bleeding source. Such outdated practice will only produce abdominal compartment syndrome necessitating abdominal decompression. Stable patients could be placed under close hemodynamic observation and with serial measurement of the hematocrit. The initial need for blood transfusions is not a contraindication to a conservative approach; we seldom know how much of the hemoglobin was shed during the operation and how much after—and how much of the drop is caused by hemodilution. An acceptable rough estimate for hemodilution caused by crystalloid infusion is 2–3% drop in hematocrit for every liter infused.

**Is my conservative approach failing?** Continuing blood loss reflected by the need for more blood would indicate that the conservative approach has failed. *Continued* transfusion is associated with increased mortality, infections, and length of stay—independent of the severity of shock. In patients who cannot be transfused because of religious objections (Jehovah Witnesses) and in patients with low physiological reserve, consider more liberal indications for radiologic or surgical intervention. Also, be quicker to intervene in pregnant patients since even early and mild maternal shock can cause uteroplacental vasoconstriction with serious consequences for the fetus.

**Is it safe to leave a large hematoma or blood clots within the abdomen?** Surely it is better to have a perfectly clean abdomen than blood and its degradation products floating around? On the one hand, blood and its metabolizing hemoglobin offer a perfect breeding ground for abscess-forming bacteria. Moreover, the by-products of old blood have been shown to contribute to the systemic inflammatory response syndrome (SIRS; Chap. 54). Relaparotomy, on the other hand, is associated with its own early and late morbidity (and mortality). While it is the perfect tool to stop the hemorrhage from an actively bleeding artery, it may only increase generalized surface oozing associated with coagulopathy. Remember that large residual clots can be washed and removed by an elective laparoscopy days after the bleeding has stopped.

**Is my patient clotting adequately?** This should be one of your concerns irrespective of whether you decide to wait or to operate. Severe acquired coagulopathy may develop intraoperatively or in the immediate post-op period. The disseminated intravascular coagulation (DIC) syndrome is secondary to a serious insult, such as sepsis; embolism of air, fat, or amniotic fluid; shock; blood transfusion mismatch; extensive cancer; severe trauma; or even when cell-saver blood is used. Recovery requires rapid correction of the primary cause and treatment of the coagulopathy that is consuming both the platelets and the coagulation factors and destroying fibrin and fibrinogen through fibrinolysis. Multiple-component blood therapy will be needed

and possibly specialized treatment such as recombinant activated factor VII. Platelet transfusions may be useful when the absolute platelet count is <50,000 and the patient is bleeding. Alert the blood bank immediately and consider hematology consultation if there is DIC.

- **Consider the specific index operation**. You did the first operation, so you are the one to know best what went—or could go—wrong. Factor it into your decision making.

## Life-Threatening Abdominal Bleeding

> Bleeding started in the rectal area and continued all the way to Los Angeles. (A patient chart, reproduced in Details in Professional Liability, 27 January 1999)

**Fallacy 3: all bleeding eventually stops.**

**Fact***:* [For some this is not "fallacy" because the bleeding will stop when the patient dies.] When a patient is compensating for blood loss, his or her blood pressure may be a third below normal, but central organs remain perfused. The patient is awake and cooperative, is making 0.5 ml/kg urine each hour, and has palpable pulses at the wrists and ankles. However, ongoing hemorrhage or sudden severe bleeding can overwhelm such a steady state. The history (e.g., soaked bedsheets or bandages, a "bloody" primary operation) combined with physical findings will tell you that you must intervene urgently.

*Medical hemostasis* through rapid correction of coagulation abnormalities is useful, but *mechanical hemostasis* is critical in this urgent situation. Reintervention for mechanical hemostasis usually means a relaparotomy but could selectively (in a stable patient) be accomplished laparoscopically, through gastrointestinal endoscopy or by the interventional radiologist.

## Relaparotomy for Hemorrhage

**Fact: in massive hemorrhage, the best place for resuscitation is the operating room.**

In the operating room, you will want as many "aces" in your hand as possible. These multiple options will increase your confidence as you answer the question: "What will stop the hemorrhage?"

Until now, you restricted volume resuscitation and allowed permissive hypotension. Now, immediately before induction of anesthesia, hypovolemia must be aggressively corrected to avoid cardiovascular collapse. Such a collapse is often caused by a sudden decrease in peripheral resistance—a result of muscle-

paralyzing agents and sudden decompression of high intra-abdominal pressure—leading to peripheral pooling and decreased venous return.

- You will want an adequate blood bank, a capable anesthetist, the means to keep the patient warm during surgery, good assistants (including a senior colleague), adequate lighting (consider extra lamps or headlights), good retraction and visualization to allow for rapid exposure of the bleeding site, plus dissection of any major bleeding vessel with proximal and distal control.
- Prepare your equipment. Mechanical hemostasis at reoperation might mean the surgeon's pinching finger, sutures, staples, clips, electrocautery (bipolar or monopolar with or without autologous muscle fragment "welding" for retroperitoneal venous oozing), ultrasonic energy, laser, argon-beam, heat-gun, proximal vessel ligation, injection sclerotherapy, or the application of topical hemostatic agents (gauze packs, sponge balls, gelatin foam, cellulose pads, collagen fleece, topical thrombin, or fibrin sealants). Omentoplasty has been used to cover diffusely oozing surfaces, but topical energy or hemostatic agents can be effective.

If the bleeding has been heavy, you should consider harvesting the shed blood for *autologous autotransfusion*.

Often, the emergency nature of the procedure and the serious state of the patient will have you and the team on edge. The wise surgeon will tell a little humorous personal story or a nonoffensive joke to relax the team. This breaks the emotional ice and will often increase the effectiveness of your team's performance. [Or, more likely in these relaxed days, the surgeon will constantly remind those present that the situation is serious and would they please get a move on.—The Editors].

*Patience* is required in order not to damage adjacent structures and to arrest the hemorrhage. We were educated on the story of a famous British surgeon who was called to operate on a patient who bled after cholecystectomy. At surgery, a large "pumper"—probably the stump of the cystic artery—was visualized in the depths of the triangle of Calot. The surgeon did not rush to apply clamps endangering the nearby bile duct. Instead, he calmly placed a large gauze pack into the gallbladder bed and said: "Chaps, I am leaving for a cup of tea. Call me in 30 min." When he returned everything was dry. [The Editors]

Most probably, the source of blood will be what you expected it to be—something at the site of your previous activity. If this is not the case, search elsewhere; pulling on the omentum during colectomy may have torn the spleen, retracting on the liver to expose the duodenum may have damaged it, eviscerating edematous small bowel may have torn its mesentery, and so forth. It is not

unusual, although some what disconcerting, to find at exploration only blood clots with no evidence of the actual source of bleeding—by now contracted and thrombosed.

Most sources of bleeding will be controlled by the basic P's (see Table 56.1). If not, try one of the hemostatic gimmicks available to you. Make yourself familiar with "specialty maneuvers" (e.g., use of thumbtacks to control presacral bleeding or the Pringle maneuver to temporarily stop profuse bleeding from liver injury while attempting control).

And, do not forget the principles of "damage control" you learned in trauma (Chap. 39): do not hesitate to pack stubborn surface ooze or venous bleeding and come back another day (or after a cup of tea!).

> "The only weapon with which the unconscious patient can immediately retaliate upon the incompetent surgeon is hemorrhage." (William Stewart Halsted, 1852–1922)

# 57 The Role of Laparoscopy

Piotr Gorecki

*The world might look brighter through the (laparoscopic) camera, but not everything bright is gold.*

Laparoscopic options were mentioned (if relevant) *en passant* in the preceding chapters, but a promise was made to elaborate further on the role of laparoscopy in abdominal emergencies. Here it is.

### Key Points

- Laparoscopic evaluation of the peritoneal cavity enables magnified visualization of the peritoneum and intra-abdominal organs with less tissue trauma than with laparotomy.
- Laparoscopy detects the presence of pus, feces, bile, or blood (facilitating the detection of the source of intra-abdominal pathology) and estimates its severity.
- Whether the therapeutic procedure is laparoscopic or conventional depends on the findings, the patient's condition, and the complexity of the planned procedure.
- Advantages of laparoscopy compared to laparotomy are reduced perioperative pain, shorter hospital stay, quicker recovery, and decreased wound complications such as wound infection and incisional hernia. In addition, laparoscopic procedures result in improved cosmesis and greater patient satisfaction.

The key principle of laparoscopy for abdominal emergencies is to limit the incidence of negative or nontherapeutic laparotomy. Should laparotomy follow the initial laparoscopy, a smaller, more strategic laparotomy incision can be placed.

## Overview

Diagnostic laparoscopy (DL) was used for many decades by gynecologists to investigate acute pelvic disorders. In light of the recent boom in basic and advanced laparoscopic techniques, it is no wonder that enthusiasts started to explore

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the role of laparoscopy in the diagnosis and treatment of almost any abdominal emergency. The rationale is simple: laparoscopy may offer an organ-specific diagnosis and, at the same time, provide treatment, thereby avoiding the need for laparotomy. This would minimize morbidity and patient discomfort, shorten the hospital stay, accelerate recovery, and improve patient satisfaction.

Laparoscopy has been used in both acute nontraumatic and traumatic abdominal situations. Master laparoscopists and great aficionados claim to be able to do "anything" through the laparoscope. Dour troglodytes [Dr. Gorecki probably refers to us. M.S., P.N.R.], on the other hand, almost totally reject laparoscopy, except perhaps for very selected indications—such as acute cholecystitis (› Chap. 20.1), acute appendicitis (› Chap. 28), gynecological emergencies (› Chap. 33), and left thoracoabdominal trauma (› Chap. 38). The following, we hope, is an enlightened and modern but balanced view.

› Table 57.1 provides an overview of possible laparoscopic applications in emergency abdominal surgery.

**Table 57.1.** Laparoscopic applications in emergency abdominal surgery

| Clear indications for laparoscopy | Potential and controversial indications for laparoscopy | Contraindications to laparoscopy |
|---|---|---|
| Acute cholecystitis (20.1) | Perforated diverticulitis (26) | Unstable patient |
| Acute appendicitis (28) | Colonoscopic perforation (30) | Presence of abdominal hypertension |
| Perforated ulcer (18) | Intestinal obstruction (21) | Severe established peritonitis |
| Diagnostic laparoscopy (DL) in acute pain of unknown etiology | Intestinal ischemia (23) | Lack of experience |
| Acute gynecological pathology (33) | Acute abdominal pain in a pregnant patient (33) | Elevated ICP (head trauma patient) |
| Thoracoabdominal trauma in stable patient (to evaluate diaphragmatic integrity) (38) | Second-look laparoscopy (52.3) | |
| | Bleeding peptic ulcer (17) | |
| | Drainage of intra-abdominal abscess (49) | |
| | Rule out intra-abdominal source of sepsis in ICU patient (52.1) | |
| | DL in stable trauma patient with no urgent indications for laparotomy (38 and 39) | |

Numbers in parentheses refer to the chapters dealing with the topic
*ICU* intensive care unit; *ICP* intracranial pressure

## Nontraumatic Abdominal Emergencies

Let us start by emphasizing that laparoscopy is absolutely contraindicated in critically ill hemodynamically unstable patients. Simply put, laparoscopy takes more time, and in severely compromised patients you need to find the source of the problem and deal with it immediately. In addition, pneumoperitoneum elevates intra-abdominal pressure, which may be deleterious in unstable, septic, and ill patients, as discussed also in Chap. 40. A sure way to induce a cardiac arrest would be to take a hypovolemic patient, anesthetize the patient, and then pump up the patient's abdomen with gas ($CO_2$).

Laparoscopy can be performed as part of a diagnostic process, as a therapeutic procedure, or both. Its application and availability largely depend on the surgeon's experience and prompt access to laparoscopic instrumentation. DL can be performed expeditiously and even outside the operating room—in the emergency room or surgical intensive care unit—and under local anesthesia. The morbidity from negative DL, as compared to a negative or nontherapeutic laparotomy, is reduced. The use of minilaparoscopy (instruments smaller than 3 mm in diameter) is gaining popularity and may further diminish the morbidity of the procedure.

DL assesses the presence and amount of intraperitoneal blood, bowel contents, or pus and establishes its source. A decision is then made whether control of the source is necessary and, if it is, whether to do so via laparoscopy or laparotomy (see Fig. 57.1).

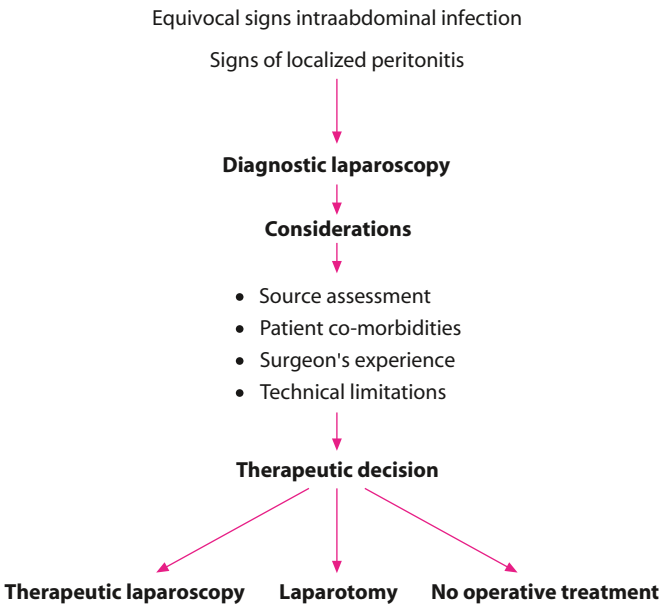


**Fig. 57.1.** Laparoscopy in abdominal emergency: decision-making algorithm

## Editorial Comment

The role of laparoscopy in the diagnosis and treatment of nontraumatic abdominal emergencies is evolving. So far, it has reached wide acceptance in acute cholecystitis and gynecological conditions. There is some rationale to embark on laparoscopy when the source of right lower quadrant pain is questionable—especially in a female patient. However, in most of these patients rational use of abdominal imaging establishes the diagnosis without resorting to laparoscopy, which can be viewed as "controlled penetrating abdominal trauma." Regarding DL under *local anesthesia*—we would wish such an experience only on our enemies.

Many surgeons do favor laparoscopic appendectomy; however, its benefits are marginal. "Lap-appy," though, may be an attractive alternative in the very obese patient, significantly reducing the wound complications. To be able confidently to tackle other conditions through the laparoscope, you must be able to explore laparoscopically the various spaces and corners of the peritoneal cavity. You must be skilled in advanced laparoscopic and intracorporeal suturing techniques if you wish to deal with more complicated situations, such as perforated peptic ulcer.

**Remember:** the acutely ill patient is in desperate need of immediate intervention. The sicker the patient, the more diffuse the patient's peritonitis, the less suitable a candidate the patient is for your magic lenses and trocars. Be selective and use your best judgment.

## Laparoscopy for Abdominal Trauma

You may remember that in Chap. 38 and 39 the author was not too keen on the role of laparoscopy in the trauma patient. Let us hear, however, the siren song of the enthusiast (Fig. 57.2).—The Editors

### *Blunt Trauma*

Figure 57.3 shows the potential applications for laparoscopy in trauma.

Management decisions in blunt abdominal trauma are based on the patient's hemodynamic status and physical findings and the selective and complementary use of diagnostic ultrasonography, computed tomography (CT), and diagnostic peritoneal lavage (DPL). So, where does laparoscopy fit in?

Its main role is to assist in the avoidance of nontherapeutic laparotomy, thereby reducing postoperative morbidity and hospital stay. But first, let us recall


**Fig. 57.2.** "I love to play with it!"

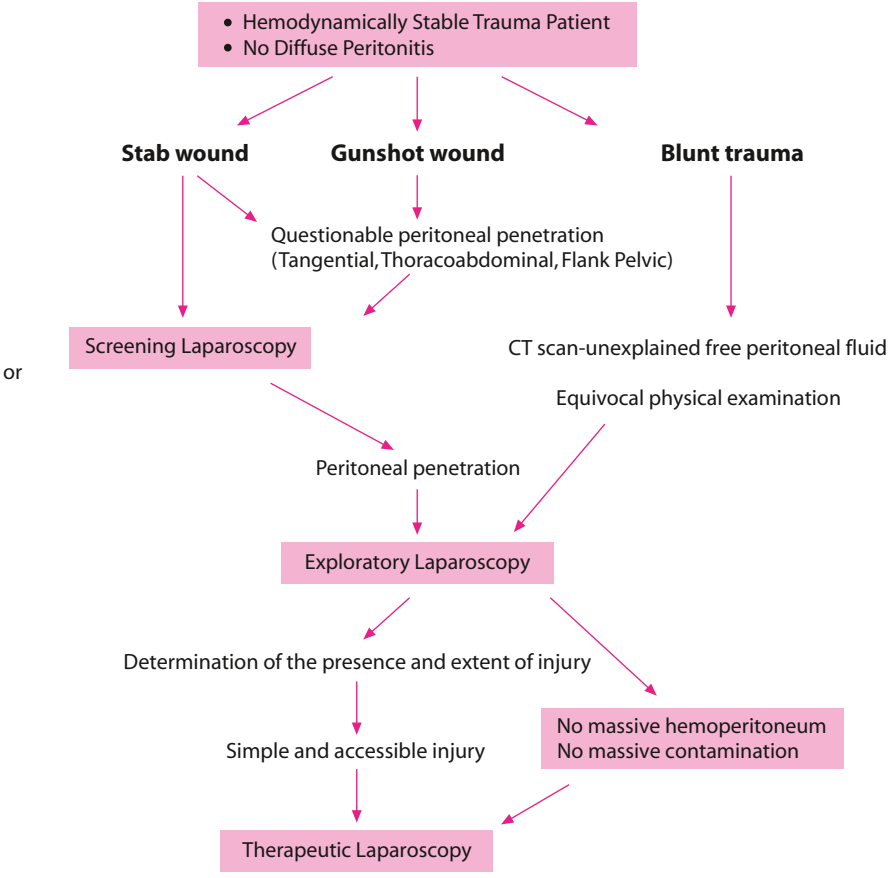


**Fig. 57.3.** Potential applications for laparoscopy in trauma

the preconditions: *laparoscopy should be performed only in the hemodynamically stable patient with no urgent indication for laparotomy.*

The good candidate for DL is a stable patient with equivocal findings on physical examination, CT, or DPL. DL can achieve organ-specific diagnosis; identify and quantify the presence of peritoneal blood, bile, or intestinal content; grade the severity of injury to the liver and spleen; assess whether there is active bleeding and its rate; and rule out diaphragmatic injury. In selected patients with minimal injury, laparoscopy may become therapeutic, for example, evacuating blood or achieving hemostasis of a small hepatic tear.

#### *Penetrating Trauma*

- *Stab wounds*: patients with clinical indications for a laparotomy (e.g., peritonitis or shock) should be managed with immediate laparotomy. DL has a potential role when clinical findings are equivocal, especially in thoracoabdominal wounds, to rule out diaphragmatic penetration. Laparoscopy may become therapeutic when injury is minimal.
- *Gunshot wounds (GSWs)*: the vast majority of the GSWs are managed with immediate laparotomy. However, a few patients with stable vital signs and no peritonitis are candidates for DL to exclude abdominal penetration or prove that the injury is minimal and does not require laparotomy. Again, with thoracoabdominal GSWs, diaphragmatic injury has to be excluded.

### Editorial Comment

"Selective conservatism" based on clinical assessment (Chap. 38) is a well-tested, safe, and cheap approach in patients with stab wounds to the abdomen. The advantages of performing invasive DL in such patients are unsubstantiated and difficult to justify. True, there are instances for which DL is the most sensitive method to diagnose an occult penetration of the left diaphragm, which is commonly associated with left thoracoabdominal wounds, but the natural history of this entity, if left untreated, is unknown. With GSWs, selective conservatism is also possible in the minority of patients but adopted reluctantly by surgeons. In stable patients with borderline abdominal signs, however, DL may prove that the GSW was extraperitoneal tangential; however, this could also be documented on CT.

A crucial limitation of laparoscopy is that it cannot adequately assess retroperitoneal structures such as the colon, duodenum, kidneys, and vessels. It confirms or excludes peritoneal penetration, but in terms of assessing damage a CT is more sensitive and less invasive. Be aware of the risk of tension pneumothorax when performing a DL in patient with diaphragmatic penetration. Deflating the pneumoperitoneum and the insertion of a chest tube can reverse it. Gas embolism is a potential complication when major venous injuries are present, but as our expert pointed out, it has never been reported after thousands of cases. It

appears that the role of laparoscopy in the injured patient is limited, but laparoscopic aficionados claim that growing experience and developing instrumentation will expand its role in the future.

> We are looking at a glass of beer. Open surgery is the beer; laparoscopy is the foam. (Herand Abcarian)

## Technical Considerations

The patient is placed on the operating table in a supine position, and general endotracheal anesthesia is given. If no abdominal distention or previous operation is evident, a Veres needle is inserted in the umbilicus, and pneumoperitoneum is obtained. A 5-mm, 30° angled laparoscope is introduced via a 5-mm umbilical port. An initial brief visualization of the peritoneal cavity is done to rule out massive hemoperitoneum or obvious complex injuries. Two other 5-mm ports are placed in the right upper and left lower paramedian sites as shown (Fig. 57.4).

The surgeon's initial position is on the patient's left side with the patient in the Trendelenburg tilt, which allows inspection of the pelvic structures, rectosigmoid, urinary bladder, both groins, and the iliac regions (Fig. 57.5). Subsequently, the ileocecal junction is identified, and the right colon is inspected. Complete inspection of the small bowel is performed utilizing a "hand-to-hand" technique to run the bowel with a pair of atraumatic bowel graspers from the ileocecal valve to the middle of its length. "Flipping" the bowel back and forth as it is run proximally permits visualization of both mesenteric surfaces of each inspected segment (Fig. 57.6). The surgeon then changes position to the patient's right to facilitate inspection of the small bowel from its middle length to the ligament of Treitz (Fig. 57.7). The descending colon is also inspected from this position. Tilting the table laterally improves visualization of the flanks and mobilization of the colon as needed for complete exploration. Rotating the table into reversed Trendelenburg position allows easy access to the upper abdomen, the diaphragm, the spleen, the stomach, both lobes of the liver, and the transverse colon with its flexures. Inspection of the area of the gastroesophageal junction, posterior wall of the stomach, and the lesser sac including the pancreas requires placement of two additional ports for retraction and grasping by the assistant surgeon (Fig. 57.4). If no complex injuries are found and hemodynamic stability is ensured, a focused therapeutic laparoscopy can follow or a strategically placed incision can be made depending on the nature of the injury and the surgeon's experience in advanced laparoscopic techniques.

Know your limitations and do not compromise the principles of exploration for trauma.

> "If you are too fond of new remedies, first you will not cure your patients; secondly, you will have no patients to cure." (Astley Paston Cooper, 1768–1841)

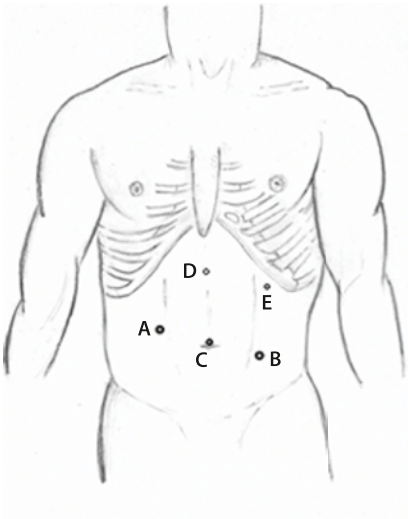


**Fig. 57.4.** Position of trocars during exploratory laparoscopy. *A*, *B* Operative ports, *C* camera, *D*, *E* optional trochars facilitating exploration and therapeutic interventions in the upper abdomen

**Fig. 57.5.** Patient in the Trendelenburg position. Surgeon on the patient's *left*. Exploration of the pelvis, right colon, and small bowel from the ileocecal valve to midjejunum is performed

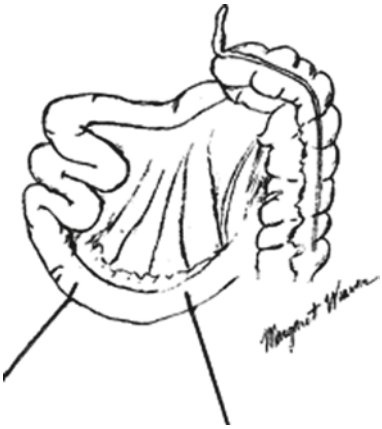

**Fig. 57.6.** Bowel is run in a "hand-to-hand" fashion. "Flipping" the segments of the intestine back and forth as it is run toward the ligament of Treitz allows the inspection of both sides of the bowel with its mesentery

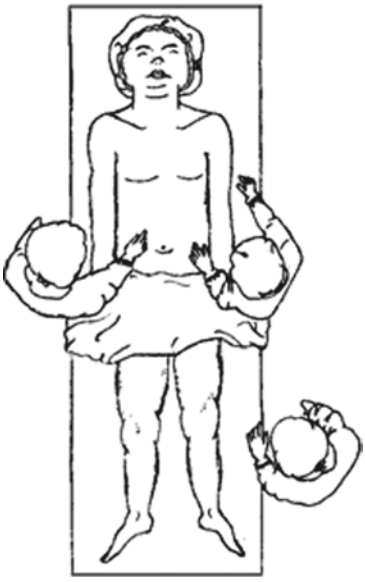

**Fig. 57.7.** Patient in the reversed Trendelenburg position. The surgeon on the patient's *right*. Inspection of the small bowel (from midjejunum to the ligament of Treitz), spleen, liver, stomach, diaphragm, transverse, and left colon. The addition of two other ports in the upper abdomen facilitates exploration of the diaphragm, proximal stomach, and lesser sac

# 58 Complications of Laparoscopic Surgery

Danny Rosin

*Not all laparoscopic operations have to end as laparoscopic operations.*

You should be familiar with complications of laparoscopic surgery for several good reasons:

- As a relatively new technique, it is prone to more complications, some of them new, different, and unexpected. The "learning curve" concept is now well known and frequently quoted—perhaps too much—as an easy excuse. But, the learning curve is real. Its length is different for different surgeons, and it does exist in open surgery as well. Moreover, complications occur to experienced surgeons long after their "completion" of the learning curve.
- Expecting a complication is the first step in preventing it.
- Timely recognition of a complication can be lifesaving. Intraoperative recognition may lead to conversion and prevention of more damage. Early postoperative recognition may lead to early reintervention, whether laparoscopic or open.
- When you perform a laparoscopic operation, more eyes can watch you closely (even the lady who scrubs the floor can watch the monitor through the glass door), and complications will be blamed on the technique, even when they are inherent to the procedure and can happen in open surgery as well (like anastomotic leaks).

In general, complications of laparoscopic surgery are divided into those inherent to the laparoscopic technique and those secondary to the specific surgical undertaking itself (some of which, like biliary injuries, are discussed elsewhere in this book). We hope that by understanding the mechanisms that lead to laparoscopic complications you will be able to minimize them, recognize them early, and manage them appropriately.

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## Laparoscopic Technique—Specific Complications

### *Bleeding*

Bleeding has special importance concerning laparoscopy in two aspects:

- Bleeding control during the procedure itself
- Injury to the abdominal wall vasculature or to intra-abdominal blood vessels during access (discussed below concerning port site complications)

Working in a bloodless field is a prerequisite for successful completion of any surgery, and particularly laparoscopic procedures as the blood obscures anatomy and absorbs some of the laparoscopic illumination; its removal by suction is time consuming and interferes with the pneumoperitoneum.

The actual control of inadvertent bleeding during laparoscopic procedures may prove a demanding task, but most bleeding events can be controlled by carefully identifying the bleeding source and securing it using one of the various techniques available (clip, tie, suture, coagulation, stapler). This is, however, the classical situation in which complications may arise, when the surgeon fails to appreciate personal limits and fails to define the correct point at which conversion is necessary. Injury to surrounding structures (e.g., biliary tract) by thermal energy, inadvertent clipping, or careless transection is frequently preceded by troublesome bleeding. **Thus, if you cannot see exactly what is bleeding and securely control it—convert expeditiously** without hesitation, and please do it as formal open surgery and do not try to complete the procedure using very small incisions without reasonable access and control. Do not ever compromise patient safety just to show that you have done "minimal access" surgery. In such cases, you may find yourself dealing with "maximal" complications!

Vascular injury may be related to abdominal wall vasculature, especially with laterally placed trocars. Using an open access to begin the procedure may give a false sense of security, only to carelessly hit the inferior epigastric artery with the secondary trocar. Attention to the course of the epigastric vessels, visible by laparoscopy, is the way to prevent this complication. If it happens—don't panic; this bleeding may be controlled by several methods:

- Direct pressure from outside (and patience for a minimum of 5 min! Don't peek every 5 seconds)
- Internal pressure using a Foley catheter inserted through the port site, inflated, and pulled back
- Cautery, provided that the bleeding point is well identified and readily grasped
- A transabdominal suture around the artery, passed using a suture passer with laparoscopic guidance

Major vascular injury is a more serious complication; it can occur with primary as well as secondary trocar placement. The primary umbilical port, if forcefully inserted using a sharp trocar, can injure the aorta or the inferior vena cava (IVC). This is especially true in thin patients, in whom the distance between the abdominal wall and the major blood vessels is surprisingly short. Low-lying (secondary) lateral trocars can injure the iliac vessels without even entering the peritoneal cavity, so an open technique and under-vision trocar insertion may not prevent such a disaster. Lightly press on the abdominal wall before inserting the trocar to confirm its correct position and direction into the peritoneal cavity. We suggest that you use blunt/bladeless trocars as it reduces the chance of major vascular or intestinal injury, even in the event of a direct contact with the structure.

### *Bowel Injury*

Even though inadvertent bowel injury may occur during any type of surgery, laparoscopy adds another dimension to this agonizing complication. It may occur during blind as well as open access or during dissection with various energy sources. Gaining access to the abdominal cavity is either by an open technique (usually with the Hasson cannula) or "blindly," by inserting a trocar after inflating the abdomen with gas through a Veress needle. Despite having avid opponents and proponents, no technique is devoid of complications.

When the bowel is adherent to the abdominal wall, usually following previous surgery, blind insertion of the trocar could injure the intestine; this may go unnoticed if the port traverses the bowel loop through and through. We must stress that using an open approach is not a panacea, and intestinal injury may nevertheless occur; for example, during the access by minilaparotomy a bulging loop of bowel may be misidentified as "peritoneum," grasped and entered. However, the chances of missing an intestinal injury are lower with open access. If you were not raised as an "open laparoscopist" for all cases, use it selectively, but with a low threshold in scarred abdomens. The use of "optical" trocars, enabling visualization of the abdominal layers while gradually inserting the port, has some proponents, but it may also show clearly how you enter an adherent bowel loop while it happens!

Although energy sources, mainly electrocautery, are widely used in open surgery, the wide exposure and direct access to the surgical field make the incidence of collateral damage negligible. With laparoscopy, the operating field in view is limited, and electrical currents may travel unexpectedly if the instruments are in touch with an organ (such as bowel) outside the field of view. This is even more dangerous when the electrical insulation along the shaft of the instrument is faulty, especially when reusable instruments are employed. Not only may

the injury be hidden from the surgeon but also its presentation may be delayed as a thermal injury to the bowel tends to manifest late—with perforation and peritonitis developing several days after the procedure. The widespread use of "fancy" energy sources (e.g., harmonic scalpel, LigaSure) increases the chance of mishaps. Remember that not only electrocautery is dangerous, but also ultrasonic shears produce high energy that can cause collateral damage to nearby structures. In addition, they get really hot after application. Be careful not to touch "sensitive" structures such as bowel or blood vessels immediately following the previous action. Take additional care while using energy near vital structures. Always consider the possibility that the patient's postoperative complaints are caused by a hole you have burned in his or her intestine.

### *Suture Line Intestinal Leaks*

Although constructing an anastomosis is rarely a part of emergency laparoscopic surgery, and even in elective surgery many of the anastomoses are completed through a "laparoscopic-assisted" approach, you may occasionally find the need to leave a suture line in the abdomen, in a situation like small bowel resection for obstruction or when repairing a duodenal peptic perforation. Leak rates are usually related to well-established risk factors (Chap. 13), but apart from these general considerations, you should strongly consider whether your technique is mature enough to tackle these cases laparoscopically. Remember, laparoscopy (and you) will be the first suspects to blame… and nobody will condemn you for a timely and safe conversion.

### *Port Site Complications*

Apart from the vascular and intestinal complications of port introduction mentioned above, remember that port sites are surgical wounds, albeit small, and should be regarded as such. **Port site infection**, although rare, may happen, especially if the port is used for extraction of an infected specimen, like an appendix or gallbladder. Use of specimen retrieval bags is the safest and easiest solution. **Herniation** through a laparoscopic port site is infrequently seen but may happen. Small bowel herniation, even partially as a Richter-type hernia, may lead to postoperative bowel obstruction and is frequently difficult to diagnose, especially in obese patients. Be aware of the possibility, and you may even diagnose it before the obligatory computed tomographic (CT) scan (Chap. 48). **Incisional hernias** may also develop in laparoscopic port sites. Infrequent as they are, they can still cause significant inconvenience and even morbidity and spoil the results of minimally

invasive surgery. Fascial closure of 10 mm and larger port sites is recommended. It can be accomplished by a direct suture or with the assistance of a suture passer under laparoscopic guidance. Some "port closure" commercial devices are being marketed.

In obese patients, creating an oblique channel through the abdominal wall while inserting the port may reduce the chance of hernia formation. Some bladeless and expanding trocars are claimed to create less fascial damage and lower the chance of port site hernia.

Suspect an incisional hernia if there is any unexplained pain at the trocar site and do whatever it takes to rule it out! If the presentation is acute, always think about incarcerated bowel (or fat).

### *Pneumoperitoneum-related Complications*

Despite some minor and transient postoperative complications resulting from pneumoperitoneum (e.g., subcutaneous emphysema or hypercapnia), the main problems are encountered intraoperatively. Two factors are involved: the $CO_2$ itself and the increased intra-abdominal pressure (IAP). $CO_2$, despite being preferable to other gases used for abdominal insufflation, is absorbable and therefore may produce *hypercapnia* and acidosis. With concomitant pulmonary disease, the excess $CO_2$ in the blood can be retained and lead to acid-base imbalance and resultant metabolic and hemodynamic problems. Gas embolism has also been described with the use of pneumoperitoneum, especially when large veins are open, as in hepatic surgery. The high solubility of $CO_2$ reduces the overall risk of this condition since it is quite quickly absorbed from the circulation.

**Pneumoperitoneum**-associated elevated IAP per se, regardless of the gas used, may cause hemodynamic and pulmonary adverse effects through physiological mechanisms discussed in Chap. 40. Although numerous experimental studies documented these effects, their clinical significance is much less clear, and except in patients with advanced cardiac or pulmonary dysfunction, one rarely sees any significant adverse effects in daily practice. However, using the lowest insufflation pressure to achieve a working space is recommended. Some studies suggest that postoperative pain is also reduced with the use of lower IAP, perhaps by reducing the peritoneal stretch. Naturally, working with low pressure is easier with a compliant abdominal wall, like that of thin or postpartum patients, and may prove to be more demanding with obese or muscular patients.

Rarely, however, the increased IAP, particularly in lengthy procedures, might cause bowel ischemia. If you do not have a reasonable explanation for the painful abdomen after laparoscopy, suspect bowel ischemia or even portal system thrombosis. Proceed to CT scan of the abdomen to confirm this.

### *Image-related Complications*

The fact that you perform the laparoscopic procedure while viewing an image, and not the actual reality, opens a window for a wide variety of image-related problems:

- **Limited field of view.** The "tubular view" through the endoscope leaves a large portion of the abdominal cavity outside the immediate attention of the surgeon. **Missed pathologies** therefore constitute a potential complication. **Inadvertent injuries**, like the thermal injuries mentioned, may also be the result of the limited view. Instruments passing through "dead spots" within the abdomen, usually during instrument change, may quite easily injure an organ outside the surgical field.
- **Two-dimensional image.** Elements like distance and depth are lost in a two-dimensional (2-D) image and are replaced by estimations based on various visual cues, like size, shade, perspective, and anatomical shape. Some technologies tried to solve this problem by providing a three-dimensional (3-D) image, but none has yet proved successful enough to be adopted for routine use. Failure to translate the 2-D image into 3-D data may result in ineffective maneuvers, prolonged operating time, and even injuries resulting from careless and inaccurate movements.
- **Concepts imposed over the image and optical illusions.** A surgeon who is confronted with an image naturally tries to apply personal knowledge about the relevant anatomy onto the image in front of him or her. Trying to "make sense" out of it is a natural tendency of the brain, and sometimes the expected anatomical image takes precedence over the observed one. Some optical inaccuracies, resulting not only from lack of 3-D information but also from other elements like light and shade, may lead the surgeon to believe in what he or she thinks instead of relying on what he or she sees. Bile duct injury, for example, may result from failure of the surgeon to appreciate that what he or she is dissecting may be the common bile duct. The strong belief that he or she is correct makes the surgeon disregard the fact that the structure is too wide to be a cystic duct. Transecting the "cystic duct" with a stapler because it is too wide is unfortunately a classical demonstration of how common sense is put aside when the surgeon is too confident to stop and re-evaluate the situation.

> The laparoscopic surgeon should believe in what he or she actually sees—not in what he or she thinks the image shows.

## Prevention, Recognition, and Management

With the wide variety of complications described, it is clear that preventing them is of utmost importance. Familiarity with the different mechanisms discussed combined with experience and wisdom should help you to prevent the vast majority of complications.

When an **intraoperative** complication has occurred, you should recognize it immediately and act accordingly. **Converting to an open procedure** is frequently the correct action as minimizing the damage and trying to reverse it are clearly in the best interests of the patient. When the anatomy is not clear or when excessive bleeding is not easily controlled, the decision to convert is better reached earlier than later, before the complication has indeed occurred. **Calling for assistance**, if available, is always a wise move when laparoscopic surgery does not progress as expected. A set of two fresh eyes may see what you have failed to appreciate, and a set of two fresh hands may achieve what you were struggling with for a while. Management of laparoscopic complications depends on the type, severity, and timing of diagnosis. **Intraoperative complications** may be dealt with immediately by laparoscopy (to control a bleeding vessel or repair a small bowel injury) or by converting to an open procedure (to repair a bile duct injury). **Massive bleeding, if not immediately and readily controlled, calls for an immediate conversion.**

Missed injuries not detected during surgery are a major cause of morbidity and even mortality after laparoscopic surgery. In general, the postoperative course after laparoscopy is expected to be smooth and relatively easy. Any problems arising during the recovery period should alert the surgeon that something might have gone wrong. In some cases, only minor signs may be evident; for example, postoperative tachycardia in an obese patient after a bariatric procedure may be the only sign of staple line leak. The surgeon should have a **low threshold for further investigation or even re-exploration**; the sooner the complication is identified and treated, the better the outcome will be. The fact that the procedure was short and minimally invasive and that the expectation is that the patient will recover quickly and smoothly should not deter us from considering the possibility that every complication could occur. Always remember possible missed injuries and be active in seeking the cause for the postoperative trouble. Do not let your "laparoscopic ego" take control of your prudent clinical judgment.

[Anyone who has the agonizing pleasure of reviewing charts of patients who died following laparoscopic procedures (and there are not a few such cases in the real world) is familiar with the pattern: the patient complains, the nurse writes in the report that the patient complains, while the surgeon is looking away—not willing to acknowledge that his or her laparoscopic masterpiece may have gone sour. And, it takes only a few hours to die from a missed duodenal injury! —The Editors]

Keep your good reputation by prevention, early recognition, and correct management of complications. Conversion or open re-exploration should be considered sound surgical judgment and not as a failure.


**Fig. 58.1.** "Hey what is this? Where are we?" "S**T! I did not know she's pregnant..."

# In the Aftermath and the M & M Meeting

Moshe Schein

*A "big" operation in a fit patient may be "small"*
*A "small" operation in a sick patient may be "big"*
*A "big" surgeon knows to tailor the operation and its trauma to the patient and his disease*

*Again and again I find that there are few things so quickly forgotten by the surgical system as a dead patient.* (P.O. Nyström)

Let us hope that your patient survives his or her emergency abdominal operation, and the postoperative course is uneventful. Unfortunately, the overall mortality of such procedures is still far from negligible, and the morbidity rate is generally high. Now, after the storm has abated, is the time to sit down and reflect on what went wrong. As Francis D. Moore (1913–2001) said: **"You want a surgical team that faces each error, each mishap, straight up, names it, and takes steps to prevent its recurrence."**

## The Morbidity and Mortality Meeting

At any place where a group of surgeons is working, it is crucial to conduct a regular morbidity and mortality (M & M) meeting (MMM). This is the venue where you and your colleagues should *objectively* analyze and discuss—in retrospect—all the recent mortalities and complications. You are familiar with the cliché that "some surgeons learn from their own mistakes, some learn from those of others, and some never learn." The aim of the MMM is to abolish the last entity.

Do you have a regular MMM in your department? If you are associated, as a resident or a qualified surgeon, with a teaching department in the United States, you must have a weekly MMM because without a routine MMM the department's residency program cannot be accredited. We know that in many corners around the world MMMs are not conducted; all blunders and failures are swept under the carpet. Elsewhere still, MMMs are conducted in name only, being used to present "interesting cases" or the latest "success stories." This is wrong. The MMM exists to analyze objectively your mistakes and complications, not to

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punish or humiliate anyone, but to educate and improve results. You do not want to repeat the same error twice. So, see to it that proper MMMs are conducted wherever you provide surgical care. And, if you are a "solo" small-town surgeon—conduct your own MMM!

### *Optimal Format for the MMM*

- A routine hour should be dedicated to the MMM each week.
- All interns, residents, and surgeons should attend—regularly.
- All complications and deaths that occurred in any patient treated by any member of the department should be presented.
- "A complication is a complication"—irrespective of whether the outcome was a triumph or tragedy. All must be presented.
- The MMM is a democratic forum. The boss's blunder or that goof by the "local giant" are as "interesting," if not more, as that caused by a junior resident.

The resident team that was involved with the case should present it. They should know all details and rehearse the presentation in advance. The patient's chart and X-rays should be readily available. If you are the presenting resident, be objective and neutral. Your task is to learn and facilitate the learning of others, not to defend or cover up for the involved surgeon; you are not his or her lawyer. Understand that the majority of those who are present are not stupid; they sense immediately when truth is deserted.

### *The Assessment of Complications*

After the case has been presented, the person who presides over the meeting has to initiate and generate a discussion with the intent of arriving at a consensus. An easy way to break the commonly prevailing and embarrassing silence is to point at one of the senior surgeons and ask, "Dr. X, please tell us, had this patient been under *your* care from the beginning, would the outcome be the same?" This technique usually manages to break the ice, prompting a sincere and complete response.

The questions to be answered during the discussion are:

- **Was it a "real complication"?** Some surgeons may argue that blood loss, which required transfusion, is not a complication but a technical mishap, which simply "can happen."
- **Assess the cause:** was it an error of **judgment** or a **technical** error? Operating on a dying terminal cancer patient reflects poor judgment; having to reoperate for hemorrhage from the gallbladder's bed marks a technical error—poor hemostasis at the first operation. **The two types of errors are often combined and inseparable**; the patient with acute bowel ischemia died because his operation was

"too late" (poor judgment) and the stoma, which was fashioned, has retracted, leaking into the peritoneal cavity (poor technique). Often, it is impossible to define whether a technical complication (e.g., anastomotic leak) is caused by poor technique (technical error) or patient-related factors, such as malnutrition or chronic steroid intake.

- Another possibility is to look at the error as an error of either **commission** or **omission**. One either operates too late or not at all (**omission**) or operates too early or unnecessarily (**commission**). One either misses the injury or resects too little (**omission**) or does too much (**commission**). After the operation, one either fails to reoperate for the abscess (**omission**) or operates unnecessarily when percutaneous drainage was possible (**commission**). Note that the surgical community considers errors of omission more gravely than those of commission; the latter are looked at with understanding: "We did all we could, but we failed."
- **Was there negligence? A certain rate of mistakes (it is hoped it is low) is an integral part of any surgical practice as only those who never operate commit no errors—but negligence is deplorable**. The operation was delayed because the responsible surgeon did not want to be disturbed over the weekend or the surgeon operated under influence of alcohol: these are clearly examples of "negligence." When an individual surgeon repeats errors over and over, a *paradigm* is exhibited, which in itself may constitute negligence.
- **Was the complication or death preventable or potentially preventable?** We encourage our residents to report the physiologic score of acute disease, using APACHE II (Acute Physiological and Chronic Health Evaluation II; Chap. 6), of the presented patient. Low preoperative scores (e.g., below 10) mean that the patient's predicted operative mortality was very low, suggesting a preventable death such as anesthetic mishap. A very high score (>20) does not imply, however, that the patient was unsalvageable. High-risk patients are those who require superb judgment and technical skills; these are the patients who do not tolerate even the smallest error.
- **Who was responsible?** The MMM is not a court (Fig. 59.1). Culpability is not the issue, but at the end of the presentation it should be clear to all present how things might have been done better. Blame is to be avoided at all costs (except in the most extreme cases, and then the MMM is not the forum to deal with them) because any system that aims to apportion blame as part of the quality control processes will fail; the truth will be hidden and confrontation avoided. Such is human nature. The sad truth, however, is that in many instances complications and mortality are caused by **"system failure,"** which in purely surgical terms means that the hospital is a cesspit with a malfunctioning chain of command, organization, supervision, education, and morals. For example, the old man was gasping unattended 6 hrs in the emergency room before you were called to assess his acute abdomen. You decided on an emergency laparotomy, but no operating room was available for 2 hrs. Because the orderlies went for dinner, another half hour was

**Fig. 59.1.** "You killed the patient!"

lost until you decided to fetch the patient yourself. Only then did you realize that the antibiotics and intravenous fluids you ordered had not been given. A clueless anesthetist then struggled with the intubation, producing prolonged hypoxia, and on and on. How much damage can an old man take? System failures are much more common than you think; just look around your own environment. (about one of such hospitals you can read here: www.docschein.com/greenwall.pdf.)

- **Was the standard of care met?** As you surely know, the "standard of care" means different things to different people. ("The good thing about standard of care is that there are so many to choose from.") It has a spectrum, which should be well represented and assessed by a group of well-informed practicing surgeons. Take, for example, a case of perforated sigmoid diverticulitis with local peritonitis (› Chap. 26); any operation ranging from a Hartmann procedure (the conservative surgeon) to a sigmoid resection with anastomosis (the modern surgeon) would fall within the accepted standard of care. Primary closure of the perforation would not. This would be easy to assess: "Anyone who would attempt closing the perforation please raise your hand." No hand is raised; the responsible surgeon is left lonely to understand that what he or she did is not acceptable and is outside the practiced standard **in his or her community**. The responsible surgeon may, however, present published literature to support that what he or she did is acceptable elsewhere. And, obviously, local surgeons can be dogmatic and wrong!
- **Evidence-based surgery**. At the end of the presentation, the resident should present literature to pinpoint the "state of the art" and the associated controversies, emphasizing "what could have been done, and should be done when we see a similar case in the future."
- **The surgeon in whose patient the complication arose**. At the end of the discussion, the most senior surgeon involved in the care of the concerned patient

should offer a statement. He or she may chose to present additional evidence from the published literature to show that what was done is acceptable elsewhere. The most graceful way to deal with the situation is to discuss the case scenario frankly and humbly admit any mistakes one may have made. If you had another chance with the same patient, how would you manage the patient? By standing up and confessing, you gain the respect of all present. When you lie, cover up, and refuse to accept the verdict of the gathering, you evoke silent contempt and disdain (or perhaps sympathy from other obsessive liars). So, stand up and 'fess up!

> "One could compare the practice of surgery to an illicit pleasure: you have fun for an hour or two, then you worry, worry and worry; and occasionally you (and your patient) suffer..." (MS)

#### *Conclusions and Corrective Measures*

Finally, the person in the chair has to conclude—was there an error? Was the standard of care met? And, what are the future recommendations and the corrective measures? If you are that chair, and you may be someday, do not be wishy-washy. Be objective and definitive for the audience is not stupid. Essentially, in any department of surgery the face of the MMM, its objectivity and practical value, reflects the face and ethical DNA of the department's chair or director.

> Most "avoidable" surgical mortalities are not caused by one-sentinel—horrendous, clearly evident—error which cries "I am malpractice". Instead, most such "avoidable" deaths result from a chain of allegedly "minor" hesitations, confusions, ignorance, greed, inattention, overconfidence, arrogance, stupidity—which together drive the nails into the coffin. Taken together they may whisper: "We are negligence." (MS)

### Financial Morbidity

In this day and age of growing costs and limited resources, we must not ignore the *financial morbidity*—the excessive spending on unnecessary procedures, even if they were not associated with an immediately visible physical morbidity (Fig. 59.2). When discussing the case, ask the presenter to justify the Swan-Ganz catheter that has been inserted, the reason antibiotics were continued for 7 days, or why the patient was "observed" in the surgical intensive care unit after an uneventful laparotomy? A useful educational exercise is randomly to present a detailed summary of the hospital bill of a presented patient. If you are confronted with what your patient's care, your superfluous acts, and the complications you created actually cost in dollars or euros, you may become a more careful surgeon.

**Fig. 59.2.** "How much money can he bill for sending this guy to his grave?"

## The SURGINET

An ideal and objective MMM as featured in this chapter is not conducted in many places because of local sociopolitical constraints. If this is the case in your neck of the woods, it may be damaging to your own surgical education: how would you know what is right or wrong? Books and journals are useful but cannot replace a thorough analysis of specific cases by a group of learned surgeons. Well, if you have a personal computer and e-mail access, you can subscribe to SURGINET, an international forum of surgeons who would openly and objectively discuss any case or complication you present to them (› Fig. 59.3). Should you want to take part in this "international MMM," send an e-mail message to Dr. Tom Gilas of Toronto, **tgilas@sympatico.ca**, or to one of the editors of this book: **mschein1@mindspring.com.**

## Conclusions

As you know, there are many ways to skin a cat, and it is easy to be a smart-ass looking at things through the "retroscope." Our sick patients and the events leading to the MMM are very complex. But, behind this chaos there is always an instructive truth that should be and can be disclosed and announced. As Winston Churchill said, success is **"the ability to go from failure to failure without losing your enthusiasm."**


**Fig. 59.3.** "SURGINET—please help me!"

"It is usually the second mistake in response to the first mistake that does the patient in." (Clifford K. Meador)

"The two unforgivable sins of surgery. The first great error in surgery is to operate unnecessarily; the second, to undertake an operation for which the surgeon is not sufficiently skilled technically." (Max Thorek, 1880–1960)

**Thanks for reading and farewell.**

We hope you enjoyed our little book. Let us wish you farewell using this memorable quotation from Winston Churchill's 1941 broadcast to the people of conquered Europe:

**"Good night then: sleep to gather strength for the morning. For the morning will come. Brightly will it shine on the brave and the true, kindly on all who suffer for the cause, glorious upon the tombs of heroes. Thus will shine the dawn."**

You—the emergency surgeons—are the heroes of medicine. For you the dawn will shine!

The Editors

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