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Community Medicine

A Student's Manual

Parikshit Sanyal

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The Health Sciences Publisher
New Delhi | London | Philadelphia | Panama

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Website: www.jaypeebrothers.com
Website: www.jaypeedigital.com

Community Medicine: A Students Manual

First Edition: **2015**

ISBN 978-93-5152-779-4

Printed at

Preface

Community medicine is not something medical students (at least the majority of them) enjoy studying very much. Of all medical specialties, it has the distinction of being most deglamorized. However, in this age of increasingly super specialized medicine, community medicine is the sole voice speaking for primary health care, holistic approach to medicine and the role of preventive medicine. It is essential that in their disregard for the subject, present day medicos do not completely ignore such fundamental and brutally important aspects of medicine.

It is with this aim in sight that I had set out to write this book, essentially a compendium of my class notes. As the title implies, I have tried to maintain a student's perspective throughout. But as much as I have tried to be succinct, the very informative nature of the subject, along with ongoing advances have resulted in a slightly larger volume than my expectations.

I hope students enjoy the book, and appreciate the concepts herein.

Parikshit Sanyal

Acknowledgments

- To the members of Department of Community Medicine, Radha Gobinda Kar Medical College, Kolkata, India (2005–2007); for I have borrowed their methods of teaching, and often their material. Specifically, Dr DK Das, Professor AB Biswas, Dr GN Sarkar, Dr RN Roy, Dr Sujishnu Mukherjee, Dr Prabha Srivastava, Dr Sarbajit Roy.
- To Professor Arnab Sengupta, for inculcating the ideas of social medicine into me.
- To wikipedia (en.wikipedia.org), the National Library of Medicine (nlm.nih. gov), and the Centers for Disease Control and Prevention Public Health Image Library (phil.cdc.gov) from where I have borrowed several images and references.
- To Professor Sujit Kumar Chowdhury, DR Lawrence, PN Bennett and MJ Brown, whose methods of writing I follow rigorously.
- To Mr Bankim Chandra Chatterjee, my philosopher.

The Brave New World

> Never have people lived so long, or been so healthy, and never have been medical achievements so great, as the 21st century. Yet paradoxically, rarely has medicine drawn such intense doubts and disapproval as today.
>
> *—Cambridge Illustrated History of Medicine*

Today, of all times, it seems that we have grown 'used to' the spectacular advances in medicine. We believe, we are the inhabitants of a free world, and our right to a long, healthy life is at par with our rights to education, democracy and free speech. Let us, if only for a few paragraphs, try to retrace our steps.

As all of you should be well aware by this age, this planet is not the right place for a gentleman (or woman) to be. Our existence is plagued continually by bacteria, diabetes, super-cyclones, famines, pollution, global warming, heart disease, viruses, militants and warmongers. Its no short of a miracle that our species has still managed to survive the time it has here. Times were not always so good as it is today. Most of human history is smeared with blood, sputum, phlegm and pus. Epidemics and outbreaks have periodically swept entire continents, men have succumbed to boils and carbuncles far too often, been hapless victims of calamities countless times, and killed each other over land far too frequently. It was only in the last century that we reflected upon the progress of our species, for the first time, and decided it was time to take control.

The first frontier to conquer was of course, germs. At the outbreak of the Second World War, penicillin was still at the laboratory stage and remained rationed for several years. Before the advent of 'antimicrobials', pneumonia, meningitis and similar infections were still frequently fatal. In combination, the development of vaccine initiated a two pronged attack on infections. Tuberculosis, the 'white plague' of Europe, was given the coupe de grace by the introduction of Bacillus Calmette-Guérin (BCG) vaccine and streptomycin in the 1940s. The first vaccines for poliomyelitis appeared in the next decade. Together, vaccines and antimicrobials proved to be an effective, if only temporary solution, to the infection nuisance.

This 'pharmacological revolution' was extended on to a broad front in 1950s. The new biological drugs beat the bacteria, improved the control of deficiency diseases, and produced effective medications for many emerging noncommunicable disease (i.e. chlorpromazine, diuretics, digitalis). The new fields of plastic and transplant surgery were opened by steroids who tackled the rejection problem.

Meanwhile, research in basic science transformed our understanding of battle with disease. Notably two inventions, the discovery of deoxyribonucleic acid (DNA) structure (Franklin, Watson and Crick) and the cracking of genetic code (Har Govind Khurana), not only helped scientific progress but also left a lasting impression on the public imagination. Finally, it seemed that we have the key to a healthy life, eradication of all diseases and even immortality. Things were looking good.

The ushering of the medical market

It was about this time since medical 'specialties' started to grow and form a lasting image over public imagination. Cardiology made its first breakthrough by surgical

intervention of congenital cyanotic heart disease in 1944. Open heart surgery dates from 1950; bypass surgery, from 1967. The rising trend of diabetes mellitus in urban population made 'endocrinology' emerge as a clinical discipline, and suddenly everyone was rushing for a postgraduate degree. By the 1990s, everyone was referring to doctors as 'specialists' in one or other field, and the statement "I am a doctor" was to be retaliated immediately by "Of what?"

The conversion of medicine from a social service to a 'performing art' was exemplified no better the development of surgery. By 1970s, surgery was beginning to resemble space travel—It seemed to know no bounds. Organ replacement developed first with kidneys; and then transplantation became a banner headline in 1967 when Christiaan Barnard sewed a woman's heart into Louis Washkansky who lived for 18 days. By the mid 1980s, hundreds of heart transplants were being conducted each year in the US alone. Gone were the days of the good ol' family physician, enter the new breed of superhealers. From a low profile, humble service-medicine was transformed into a headline generator and revenue service. Corporates, media and lawyers jumped into the medical melting pot, health insurance companies grew obscenely fat, hospitals grew shinier and expensive; physicians, once the friends of people, turned into solicitors and 'consultants'. Every diagnosis, for them, is just a scan away, and every cure, a pill or an incision away.

It has not helped wither that technological breakthroughs as electron microscopes, endoscopes, computerized tomography (CT), positron emission tomography (PET), magnetic resonance imaging (MRI), lasers, tracers and ultrasonograms (USG) have created a revolution in medicine's diagnostic capability, but only at the price of increasing cost. Modern medicine has effectively banned people with a cash flow problem. What has further aggravated the problems are the litigations that now smear medicine, and the fear of being sued has lead to the practice of 'defensive medicine', that is to go through a battery of investigations (usually called 'the workup' in physician circles) rather than use of the clinical acumen, skyrocketing the cost of care even higher. Effectively, health itself has become the new disease. The creation of these health commodities allows them to be bought and sold like carrots and iphones. Future risks, for example, of diabetes, cancer, heart disease or mental illness can then be traded as derivatives, with the disease risk traded against the possibility of a new or cheaper treatment in the future.

> Markets depend on units that can be bought and sold, so markets in health depend on breaking down healthcare services (investigations, procedures and prescriptions), health (e.g. biological values such as blood pressure and cholesterol) and illness (diabetes or cancer) into unit parts that are measurable and saleable. The challenge, according to market advocates, is technical. All they have to do is define and value those parts in order that they become commodities.
>
> *—A petition on "Keep the NHS public" website*

So this is where we stand today: In spite of all the tremendous advances, an atmosphere of disquiet and doubt now pervades medicine. We find ourselves increasingly entangled in dystopia, no longer do we expect to live our dreams to the fullest, no longer do we expect a healthy and long life. No longer do we

expect that our successors will have enough of food and fuel to support the entire population. No longer can we expect to return home safely when we go out to work. Our only feeble wish is to die a less painful death than those at US military prisons, Afghanistan, Somalia, Cambodia, Vietnam ... you could go on forever. Euphoria bubbled over penicillin, over heart transplants, and over the first test-tube baby—But no amount of scientific achievement has overshadowed greed, corruption and short sightedness of our species.

> Only two things are infinite: The Universe and human stupidity. And I am not so sure of the first one.
>
> *—Albert Einstein*

The prospects of medicine

> I never think of the future; it comes soon enough.
>
> *—Albert Einstein*

Now that the big battles against disease have been won, medicine is also more open to criticism. Worldwide, socialized medicine faces grave political risk; in the USA, insurance and litigation scandals dog the profession. In rich countries, the poor are often excluded from health services. In the developing world, for lack of international will, malaria and diarrheal diseases remain rampant. Diphtheria and tuberculosis are resurgent in the Union of Soviet Socialist Republics (USSR). Not least, the pandemic of AIDS destroyed any native faith that disease has been conquered.

There is a second, much larger issue looming over, much larger than the commercialization of medicine. After we have won over all the scourge and pestilence, what is now the aim of medicine? Where to stop? Is its prime duty to keep people alive as long as possible? Is its charge to make people lead healthy lives or just to cure them when they are ill? Or is it but a service industry, to fulfil whatever fantasies its clients may frame for their bodies for instance, a facelift or cosmetic remodeling? Is developing an HIV vaccine the best thing we could put our funds into? Is the rate of human population growth healthy for this planet? Medicine has reached the point where Hippocrates, William Harvey and Lord Lister could only have dreamt of. What next?

> To put developments in a nutshell, two facts give powerful (if conflicting) evidence of the growing significance of medicine. First, the trebling of world population in the last 60 years no small percentage of which is caused by new medical interventions and preventions. Second, the introduction of the contraceptive pill, which, in theory at least, paved a simple and effective means to control that population.
>
> *—Cambridge Illustrated History of Medicine*

The great divide of medicine presents another pressing problem. On one hand, you have the well-to-do classes, who live within an expanding medical establishment faced with a healthy population of its own creation. Thus medicine is directed to medicating normal physiology (such as menopause), to convert risks to diseases, to treat trivial complaints with fancy procedures. Patients want it, they want some intervention, 'something to be done' to them, they simply do not agree that the mole on their left shoulder is simply a mole and just that. In this age of litigations, doctors dutifully, and often wilfully comply to order that costly biopsy and

staining procedure. The motto: Everyone has something wrong, everyone must be cured. On the flip side, you have millions rotting in poverty, hunger, illiteracy and social injustice. More and more doctors push each other for a berth in the urban money-mad-medical-machine, and the millions in dark in the depths of our country are tormented into second hell as ever, to be flocked into polling booths into the election season.

So comes social (community) medicine

Surely, modern medicine has all the right tools and methods, but the wrong policy. Could not it be rectified? People in Britain and France have done it. It is called socialized medicine. It is the notion that the health of oneself is not one's own responsibility but the shared burden of the community, and when one gets sick others help him out (through taxes), so that he gets the best medical care. All this without a buck spent on treatment.

But the idea of social medicine is far more revolutionary that socialized medicine. The principles of social medicine are:

- To reorient the public mind and medical education on thinking about health rather than thinking in terms of diseases.
- To prevent, by community effort, the emergence of disease through simple interventions, lifestyle and community behavior.
- To free medicine from the shackles of big corporates and push it into every home, every kitchen, every toilet; to educate people on healthful behavior, to establish a bottom up health system through primary health care; to orient people that illness is not to be reported in a big five star hospital, but to your local health center.
- To socialize prevention, diagnosis and cure of diseases.

So how does all this jargon relate to me?

> If I am not for myself, who will be for me?
>
> If I am only for myself, what am I?
>
> If not now, when? —*Hillel*

I know all this feels a little tiresome to read. But you, of all people, must initiate the change. You will be great clinicians no doubt, and great surgeons galore, but picture in your mind, if you will, the poorest of the poor you have seen, the sickest of cripples, the nastiest of lepers, the creepiest of AIDS patients, who will never be able to afford you on a pay per visit basis. It is a decision you have to make early—Do you want to exercise your great skills over the ivory tower off corporate medicine, or do you want to come down, if only once in a while, to those outside your clinic.

I do not really like quoting clichés, but I really cannot afford to miss out these famous lines—

> ... He who uses these (skill, knowledge, human understanding) with courage, humility and wisdom will provide an unique service for his fellow man and will build an enduring edifice of character within himself. The physician should ask of his destiny no more than this, he should be content with no less.
>
> —*Tinsley R Harrison*

How to read this book

> "and what is the use of a book," thought Alice "without pictures or conversation?"
>
> *—Lewis Carroll, 'Alice's Adventures in Wonderland'*

No book is complete, and this one even more so. However, you should have a comprehensive, if not detailed understanding of community medicine, after going through this one. It would suffice to say that God is in the details, but we really do not need to know God, do we? Try to identify keywords which I have emphasized. Try to make lists, as I have made, from clauses of definitions. Focus more on the diagrams than text, focus more on the flowcharts than details of individual steps. Ensure that you would be able to utter at least two or three sentences of any topic from this book, but cover everything. Try to make mind maps of your own, remember with pictures, not words. Ignore all the numbers and detailed statistics, they are here just for formality. This is the approach which has benefitted me most as a student, and I believe it will make you enjoy (a keyword) this subject as much as I have.

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Health and Disease

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INTRODUCTION

> "The time has come", said the walrus, "to talk of many things".
>
> —*Lewis Carroll, Alice in Wonderland*

The genesis of the medical revolution started in a clinic. Thus the architecture of medicine inclines progressively towards the clinic.

But medicine is not as much a trade as it is a social doctrine. And this notion took ages to develop into a tangible idea. That medicine is not just another profession, and health is no commodity has only gained foothold since the middle of last century. It is worthwhile to take a look at major landmarks that have imprinted upon our world view.

The rise of public health

Systems of medicine have been in practice since antiquity, but typically they achieved little. In absence of knowledge of anatomy, physiology and pathology, the practice of medicine carved its niche as a mystic specialty that a few shamans and priests were capable of. This kind of medicine was learnt from experiences, kept minor ailments under check, and was generally accepted by people. Nothing fancy was expected from the doc, just a few herbs, a little bloodletting, and there you go. The idea of hygiene and community sense, once reaching its pinnacle in Roman and Indian civilization, achieved an all time low in the middle ages. No wonder plagues swept across Europe that time.

The Renaissance changed everything. The newfound awareness, the virile curiosity, and the outlook to analyse things, to believe in 'humanity' rather than the clergy-the trends were clearly here to stay. The period following 1500 AD was the

'age of revolutions' – not only in the arts, industry and geographical expanses – but also medicine.

Revival of medicine

The father of scientific medicine was (probably) Paracelsus, who publicly burnt the works of Galen and went out to preach that medicine was governed by the same principles of the natural philosophy, the same rules that rotated the planets in their courses. Andreas Vesalius raised anatomy to a science, and so did Ambroise Pare for surgery. Thomas Sydenham established the clinical methods, William Harvey pioneered the circulatory system – and Antoni van Leeuwenhoek came up with his microscope. The change had set in.

The great sanitary awakening

Unforeseen to the harbingers of the industrial revolution, the after effects were already beginning to take a toll on the mean life expectancy of England's laboring class. The cholera epidemic in London of 1832 led Erwin Chadwick to investigate the health of the inhabitants of a large town in England. His report stated, albeit revolutionary for his time, that filth is man's greatest enemy, and the style of documentation is still a lesson.

Cholera appeared time and again in the western world until John Snow studied the epidemiology of cholera in 1854 and established the role of drinking water. The great sanitary awakening followed – that is people began to pay attention to what they release as much as to what they take in. Then they began demanding clean water, well lit houses, cleaner air, and good sanitation systems – all from the state. It was becoming clear that the state must share some of the responsibilities of the health of its citizens.

The phases of Public Health

Public health is the

- Science and art of
  - Preventing disease
  - Prolonging life
  - Promoting health and efficiency of each individual through
  - An organized community effort.
    (CEA Winslow, 1920)

Disease control (1880–1920)

Following the 'Great Sanitary Awakening' in England (inspired by the works of Erwin Chadwick), the first Public Health Act was passed in 1848. It emphasized mostly on sanitation and community hygiene, and was implemented from 1880–1920. These 40 years were aimed at control of physical environment (sanitation). The points stressed upon were improvement of sanitation, water supply and sewage disposal. The modern concepts of septic tanks and protected urinals and air-conditioned meat vendors was born in this era.

Health promotion (1920–1960)

This period focussed on the quality of life (i.e. improving the host factor in the epidemiologic triangle). The stress was upon nutrition, education and general well-being of the community. It was this phase that mental disorders came into limelight. In total, the phase was aimed at improving the herd immunity (the resistance of a community to a disease).

Social engineering (1960–1980)

As the communicable diseases were conquered (at least, in the developed world), the noncommunicable diseases began to steal the show. Research in the later half of twentieth century emphasized on social and behavioral aspect of diseases. Much ado was placed over the social harmful factors (smoking, sedentary lifestyle, malnutrition, alcohol, etc).

Also in this phase – It was noted that some social practices like artificial feeding and home child delivery can affect long-term community health.

Health for all (1978–now)

In spite of medical advances

1. Large no. of population (almost half the world) can't access health care
2. Glaring contrast remains between service to rich and poor
3. Dissatisfaction grows over health service.

Beginning from 1978 at the Alma Ata Conference, the idea of "health for all" began to take shape and became the WHO slogan for the year 2000.

Preventive medicine

Preventive medicine is the art and science of

- Health promotion
- Disease prevention
- Disability limitation
- Rehabilitation.

The first doctrine of preventive medicine, fresh fruits and vegetables to prevent scurvy, came from **James Lind** (1716–1794), a naval surgeon (the navy suffered from scurvy too frequently, during the long voyages in sea without fruits). The real revolution was brought about, however, by **Edward Jenner** and his pox **vaccine** (latin 'vacca' = cow, animals from which Jenner derived his vaccine), in 1796.[1] Following his discovery, the later part of the nineteenth century is marked by Pasteur's antirabies treatment, cholera vaccine, diphtheria antitoxin, typhoid vaccine, antiseptics and disinfectants.

Two more breakthroughs elucidated the bizarre modes of diseases transmission: one, the transmission of sleeping sickness by Tsetse fly, demonstrated by Bruce, a British army surgeon (of Brucella fame) and second, the experimental proof of mosquito-borne transmission of malaria by **Ronald Ross** (1898). Thus the field of medical entomology was born.

Today, preventive encompasses a huge array of medical, social and political issues. This encumbers from the fact that disease is not only a disgrace for a nation, but also a significant retardation to development.

Social medicine

Social medicine is to study a man as a social being in his total environment with special reference to social factors which have direct/indirect relation to human health

—Jules Guerin (1848)

Social medicine is a natural successor to social anatomy (demography), social physiology (well-being of the community) and social pathology (dowry, early marriage, malnutrition, prostitution, etc.).

A definition of community medicine

It is the study of:

- Health and disease in the
- Healthy and sick subjects
- Of a population of defined community or group
- With an aim to identify their health problems and needs (**community diagnosis**)
- And suggest appropriate remedial measures (**community therapy**).

Making a community diagnosis

1. Demography and vital statistics (birth rate, death rate, marriage rate) of the community
2. Morbidity and mortality data
3. Talk with leaders (formal and nonformal) – to know what are their 'felt needs'
4. Formal surveys in the community
5. Visits and transect walks through the area
6. Individual interviews and KAP (knowledge-attitude-practice studies)
7. Survey of available resources, including health services.

Health

"A state of complete physical, mental and social well being and not merely the absence of disease or infirmity"

—WHO, 1984

This is the utopian definition of health, "complete physical, mental and social well-being" is neither practical nor feasible. According to this definition, health is no static entity, but it resembles a pendulum oscillating between positive health and death.

Operational definition

Health is a condition or quality of humans expressing **adequate functioning** in a given condition – genetic or environmental.

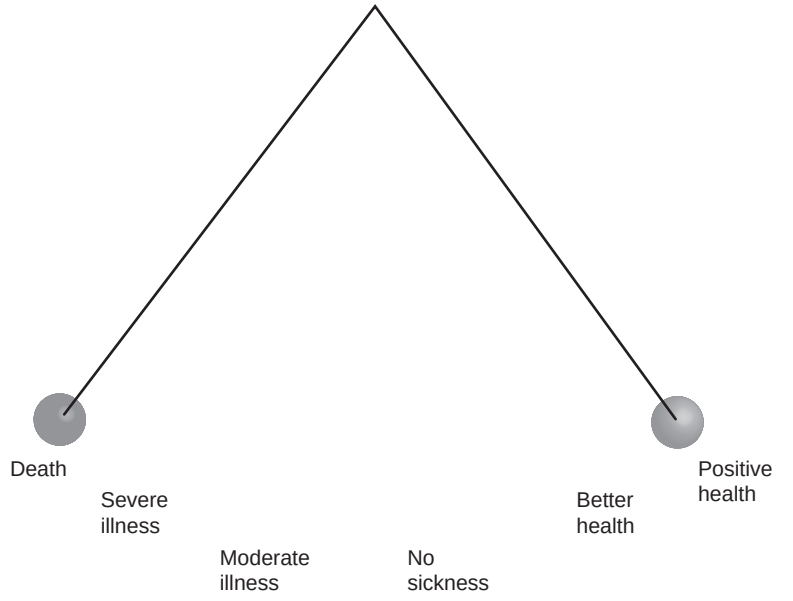


Figure 1.1. Pendulum of health

Positive health

It is the +ve end of the health pendulum (Fig. 1.1)—A perfect functioning of humans in terms of biological, social and psychic components.

Changing concepts of health

Over the ages, what people have thought of 'health' has undergone several changes.

Biomedical. "Health is an absence of germs in the body"—A definition which ignores environmental, psychosocial and cultural factors. The definition gained momentum in late eighteeth and nineteeth century, with the advent of germ theory of disease.

Ecological. "Health is dynamic equilibrium between man and his environment". The changing environmental and economical scenario in the early twentieth century gave rise to this perspective.

Psychosocial. As most of infectious diseases had been conquered by the late twentieth century, health professionals began to discover that 'health' has a lot more to it than bodily well-being, and psychological, social, economic and political factors affect health profoundly.

Holistic. Synthesis of all above, the working concept nowadays.

Whose headache is health?

Individual

Self care (as you know – the best care) is all those practices which keep the individual healthy (hygiene, sleep, good food, no smoking/drinking, active life).

It is unaided by professionals and unchallenged by health insurance companies. Every individual takes care of himself/herself determined by the degree of his/her enlightenment.

Our age is gradually seeing the burden of the medical professional shifting to his patients. Urban patients are now taught to record their BP and blood sugar. Over the counter (nonprescription) sale of drugs has also been an alarmingly rising trend.

Could medicine be practised on an individual level?

NO. "Physical, mental and social well being" cannot be achieved by any individual simply by himself, whatever his socioeconomic status is. No system of internal medicine (centered around a single patient) can provide total health. The poor, being the ones with the cash flow, are less dependent on individual health care and more on state health services. But the rich too, can NOT simply **buy** health, and have to depend upon community health sometime or the other. For example, nosocomial infections are a problem of super specialty hospitals that predominantly affect the rich. These highly resistant organisms have evolved over decades of rueful antimicrobial practices, and such practices can never be stopped until the state comes up with a definitive 'antimicrobial policy' which will be based on regular sampling of organisms from environment, testing their resistance patterns and recommendation of a select set of antimicrobials for each category of organisms, beginning with the one with the narrowest spectrum. This is only one of several instances of how community health affects the rich people and super specialty 'five star' hospitals.

Community

There are three levels of community activity in health care

1. The people can provide health workers with resource (money, men, and materials), logistics and shaping the plan. (SUPPORT)
2. They can utilising and join the health service. (PARTICIPATION)
3. They can be more than mere consumers, i.e. they can take part in planning, implementation and evaluation of service. (INVOLVEMENT).

Until quite recently—The public was viewed as sources of pathology and targets of pharmacology. The viewpoint is self-occlusive. There are matters beyond the doctor that will eventually affect community health like environmental sanitation, pollution, transport facility and many such factors. The wise decision (and a little clever too): "People's health in people's hand" and "Health care **by** the people" (not just **for** them).

Community participation has been difficult to obtain in India. It is a country where caste and creeds inhibit community from unifying at all, and where people still boycott pulse polio on supernatural/communal grounds.

State[2]

The paradigm of socialized medicine is that the **state** is responsible for health of its citizens, not merely doctors and hospitals. Indian constitution recognises the responsibility of the state for the health of its citizens. Also, National Health Policy 1983 indicates commitment to health for all.

United Nations

Programs have been taken in international level concerning health. This is necessary because, especially communicable diseases are a threat to the whole human population. The UN, Technical Cooperation in Developing Countries (TCDC), Association of South East Asian Nations (ASEAN) and South Asian Association for Regional Cooperation (SAARC) are important mechanisms for international cooperation.

The eradication of smallpox, the pursuit for 'health for all' and the campaign against AIDS are typical examples of international collaboration.

Determinants of health

Health is a combined property of man and his environment. These are all the factors, ultimately, who determine the health of the individual.

Genes

It is not where community medicine can interfere much. Genes will continue to produce ailments and we can at most go for genetic screening and counseling of high-risk parents.

Environment

Internal environment. The cause of majority of noncommunicable disease like ischemic heart diseases, diabetes, etc.

External environment. Most communicable diseases/"environmental" diseases are due to the external environment. The external environment can be divided into physical, biological and psychosocial components—each of which affects health.

Lifestyle

It is 'the way people live'. It includes culture, behavior, and personal habits (smoking, etc.). Lifestyle develops from a combined influence of peers, parents, siblings, teachers and media (television, internet).

Health requires a healthy lifestyle. The 1960–1980s saw the social engineering phase of public health – when lifestyle was the most stressed upon factor.

Socioeconomy

Economic status. It determines the purchasing power, standard of living, quality of life and disease pattern. The per capita GNP is usually the important factor if the wealth is distributed evenly (which eventually is a far dream in most of the countries in the world). Ironically – poverty and affluence can both be curses over health.

Education. Particularly, education of **women** is important, as the mother is in charge of the household. The world map of poverty, unemployment, communicable diseases closely coincides with that of illiteracy. Studies indicate that education, upto an extent, can compensate for the degradation of other determinants of health like economy and health facilities.

Occupation. Unemployment leads to high morbidity and mortality, psychic and social disorders (like eveteasing). Again, working in a physically/mentally

hostile environment (acid factory/dye industry/grumpy boss/hostile colleagues) will affect the health of the individual.

Politics

Politicians decide health policies, choice of technology, resource allocation and manpower policy. Often the main obstacles to implementing a health service are not the technology but a forthcoming change in government. The percentage of GDP spent on health (should be 5% according to WHO) is a quantitative indicator of the government's dedication to the health of its public.

Others

Food and agriculture, education, industry, communications especially, via electronic means, social welfare, rural development indirectly affect the health. This is no medical zone. Doctors can't create employment, distribute free manure and seeds to farmers, cook mid-day meals or anything like that. They are called only **after** children are poisoned by a mid-day meal for damage control.

Health Systems

It is the service given to individual or community for prevention of disease, promotion of health and therapy. Any organization which is primarily involved in health care comes under health system.

1. Health services: Services provided by the government
2. Private services.

Educational institutions do not come under health service because their primary goal is not health. Whereas health care = preventive + therapeutic medicine for both sick and normal people of a community, medical care is only therapeutic.

Indicators of health

Indicators are variables who help measure a change. An **index** is an amalgamation of indicators. The health indicators are used to measure health status of community, compare health status of two communities, assessment of health care need and identify priority (i.e. whether you need programs for tuberculosis rather than scrub typhus in India), allocate resources (which are, as always, limited) and monitor/evaluate health programs.

A health indicator should be

- Valid (i.e. actually measures what it is supposed to measure)
- Reliable (should reproduce the same results over and again)
- Sensitive (able to reflect a small change)
- Specific (reflects changes only in the situation concerned)
- Relevant (should actually show something).

Measuring health (which is rather a subjective phenomenon) is not easy. Multiple categories have been sought upto group these indicators.

Mortality

Table 1.1. Mortality indicators

Crude death rate	$\frac{\textbf{Number of deaths in a year}}{\textbf{Mid year population (1st July population)}}$ It is a fair indicator of the comparative health of people. It is influenced by the age composition of the population. Thus although not a perfect indicator – improving cDR is of course one of the goals
Infant mortality rate	$\frac{\textbf{Infant deaths}}{\textbf{Live births}} \times \textbf{1000}$ in that year. It is affected by the socio-economy of the population. In addition—It is a sensitive index of health care availability to mother and child
Child (1–4) mortality rate	It correlates with maternal and child health (MCH) services also indicates nutrition, immunization. India contributes to about 5.6 million child deaths every year, more than half the world's total[a]
Under 5 proportional mortality	$\frac{\textbf{Deaths under 5}}{\textbf{Total deaths in a year}}$ It determines community hygiene, immunity and care
Disease specific mortality	$\frac{\textbf{Number of deaths from a particular disease}}{\textbf{Mid year population}}$ Shows the prevalent disease pattern of the country
Proportional mortality	$\frac{\textbf{Deaths caused by a particular disease}}{\textbf{All deaths in a year}}$ For communicable diseases – they represent the fraction deaths that could have been prevented if that disease is controlled
Life expectancy	**The expected (in the statistical sense) number of years of life remaining at a given age**

[a] "Hunger critical in South Asia", BBC 2006, http://news.bbc.co.uk/2/hi/south_asia/6046718.stm

Morbidity

Table 1.2. Morbidity indicators

Incidence and prevalence	**Incidence** is new cases/unit time and **prevalence** is (existing + new cases)/unit time
Notification rate	The percentage of cases reported to health service
Sickness absenteeism	It refers to the number of days one stays absent because of sickness. The commonest cause of absenteeism in our country is, however, social occasions and strikes

Disability

Table 1.3. Disability indicators

Event type No of days of restricted activity Days spent in bedrest Work days lost for illness	Indicates how long someone stays ill
Person type Degree of mobility limitation, activity restriction	Indicates to what extent someone stays ill
Disability adjusted life years (DALY)	One DALY = 1 lost year of healthy life. DALY = years lived with disability + years lived shorter than life expectancy

Sullivan's index

It is the expectation of life free of disability, i.e. Life expectancy – (days spent in bedrest + days spent with restricted activity). It is considered to be the most advanced health index.

Nutrition

Anthropometry. Usually done in preschool (<5 years) children usually to see nutritional status. 46% of children in India are underweight, one of the highest rates in the world and nearly the same as subSaharan Africa.[3]

Women's nutrition. Because women bear and rear children, and are usually the ethical driving force behind any civilized society, the social and nutritional status of women is a good indicator of progress. Most Indian women are malnourished. The average female life expectancy today in India is low compared to many countries, but it has shown gradual improvement over the years. In many families, especially rural ones, the girls and women face nutritional discrimination within the family, and are anemic and malnourished.[4]

Prevalence of low birth weight. It is a general indicator of antenatal care.

Indicators of health care delivery

These indicate the quality (how good the health care facility is), quantity (how many of hospitals, doctors and health care workers are there) and distribution (how evenly the facilities are spread across the country) of health services.

- Doctor: Population ratio—As of 2009, there is one doctor for every 1588 Indians. The 289 medical colleges in the country add 33,382 new doctors each year.[5]
- Doctor: Nurse ratio—The doctor nurse ratio in India was almost 1:1.5.[6] Indian nurses are in great demand in the middle east and Europe. Add to that the career development facility for nursing personnel is very minimal in India. This is evident from a survey in 1993, which revealed that very few nurses in India get three promotions in their whole service career. And finally, they have this 12 hour a day schedules all through their career. No wonder nursing is not a popular profession in India. All this have contributed to the fact that in 2004, there was not even **one** nurse per thousand population (0.8/1000).

- Bed: Population ratio—With a world average of 3.96 hospital beds per 1000 population. India stands just a little over 0.7 hospital beds per 1000 population.
- Population: Health centers—The target in India is to establish one PHC for every 30000 population.
- Population: Trained birth assistants.
- Competence of doctors—In a 2005 World Bank study, World Bank reported that "a detailed survey of the knowledge of medical practitioners for treating five common conditions in Delhi found that the average doctor in a public primary health center has around a 50–50 chance of recommending a harmful treatment".
- Attendance of health care workers—Random visits by government inspectors showed that 40% of public sector medical workers were not found at the workplace.[7]

Indicators of utilization of health services

- Proportion of infants 'fully immunized' against vaccine preventable diseases
- Proportion of mothers getting antenatal care
- Proportion of deliveries conducted by professionals
- Couple protection rate (percentage of eligible couples using family planning methods)
- Bed occupancy and turnover rate in hospitals (indicates average length of hospital stay).

Indicators of social and mental health

The incidence of homicides, suicides, crime, juvenile delinquency, drug abuse, domestic violence, accidents, smoking and alcoholism indicate the health status of the society as a whole.

Global piece index

In the face of rising conflict all over the world, a global piece index (GPI) has been constructed an alternative to GDP (which indicates monetary well-being). It is the amalgamation of 23 indicators regarding militant activity in a country, deaths from war and internal conflicts, spending on military budgets and relationship with neighboring countries. **New Zealand** seems to be the most peaceful country, while India ranks 122nd.

Health policy indicators

Proportion of GDP spent on health and primary care. Indicates how dedicated the state is to providing 'health for all' for its citizens. India spent a healthy 6% of GDP towards healthcare in the mid 1990s, but the amount has declined since and by 2007, has declined to 2% only,[8] which translates to only **Rs 37/month/Indian** on health care.[9]

Equity of distribution. The problem of maldistribution of health care has taken ugly shape in India. Essentially, we have very little health infrastructure in our villages, and that is the reason majority of doctors dislike a rural posting. Again the very fact doctors are unwilling to serve in the villages prohibits the development of health infrastructure. Meanwhile, occult practitioners and quacks exploit the situation to its full advantage.

Degree of decentralization. The primary care approach recommends that every sick person should first report to his primary health center, from where he is escalated through a chain of referrals to the large hospitals, if necessary. However, this decentralized approach has not really gained momentum in India. Most PHCs lack the facilities even for emergency care, 40% of them are understaffed, and most peripheral doctors are habituated to blindly refer any patient to tertiary care centers in the cities. In effect, 700 million people have no access to specialist care and 80% of specialists live in urban areas.[10]

Proportion of GDP spent on health related resources (water, housing, environment). Indicates the breadth of health care perceived by the state.

Socioeconomic indicators

Per capita[11] income. India boasts of some of the richest men in the world, but on an average, an Indian man earns $1070 per year only (ranked 143nd in the world), and 75.6% of the population live on less than $2 per day (purchasing power parity), which is equivalent to 20 rupees per day in nominal terms.[12] Poverty forces poor people to work unhealthy professions, get into crime, neglect their children and die young. Because infants of poor parents die more frequently, and children of poor parents are expected to work from their very toddler age, poor people have the most fertility (which, in turn, aggravates the poverty even further).

Measuring income disparity: Gini coefficient

This index measures the degree of inequality in the distribution of family income in a country. The cumulative family income is plotted against the number of families arranged from the poorest to the richest. If income were distributed with **perfect inequality**, the index would be 1. In no disparity at all, the index remains 0.

Gross domestic product and its fractionation. Although the GDP in India is a healthy $1.209 trillion (with a 6.7% growth rate in 2007/2008 fiscal year), economy in India is at best in a confused state. Most of the GDP is spent in agriculture (17.2%), industry (29.1%) and services (53.7%), leaving very little to health and education. **41.6%** of population live below new **international poverty line (i.e. < 1.25 US$/day purchasing power parity)**,[13] which is an, however, improvement from 60% in 1981.

Dependency ratio. Indicates the load on the working population of the country; for every 100 working persons, there are 62 dependents in India (most of them children).

Family size. Indicates the preconceived notion people have about how large their family should be; the average family size in India is **2.8**,[14] which has reduced slowly but steadily over the years, but still too high.

Unemployment. By 2008 estimates, 6.8% of adult people of productive age group are unemployed in India. Unemployment is a social pathology that leads to increase in crime, sexual harassment and unemployed men fall easily to communal and political provocations.

Literacy. By 2007 estimates, India has acheived 66% literacy,[15] i.e. 66% of Indians "aged seven years or above can read and write any one language with understanding" [National Literacy Mission].

Per capita calorie availability. Indicates equity of distribution of food. Although restaurants, coffee shops and MacDonalds' are growing everyday, India still has to feed 46% of its children to adequate nutrition.

Per capita floor space. Indicates availability of housing facility. The figures in India are startling. One in three Indians live in less personal space than a prisoner in the US, that is < 60 square feet/ person.[16] The average space per person is about 103 square feet in rural areas and 117 square feet in urban areas (if you find the figures a little abstract, any toilet in middle class family is about a 100 square feet; try measuring your room yourself).

Environment

Population having access to safe water. While the share of those with access to an improved water source[17] is 86%, the quality of service is poor and most users that are counted as having access receive water of dubious quality and only on an intermittent basis. In rural areas, the figure is 83%.

Percentage of houses with safe water supply at home or 15 minutes distance. The definition of "improved water source" in India is within 1.6 km of housing, and I doubt whether 1.6 km could be considered a 15 minutes walking distance (especially while carrying a pot of water).

Percentage of houses with sanitary facility. Only one in three Indians (33%) in all of India (and only 22% in rural areas) has access to improved sanitary facilities. Open defecation is still a common practice, and the streets are everybody's urinal.

Other indicators regarding air pollution, water pollution, soil, noise and radiation are also pertinent to health.

Quality of life

PQLI – Physical quality of life index - money can't buy happiness. It is determined by an average of

1. Infant mortality
2. Life expectancy at 1 year (because life expectancy *before* 1 year has already been included as infant mortality rate)
3. Literacy above 15 years.

All three have same weight. India scores a PQLI of 43.

Health for all (WHO)—Indicators

- Health policy—Commitment to health for all, equitability of distribution, community involvement, framework.
- Socioeconomy—Economic growth rate, GNP/GDP, income distribution, work condition, adult literacy, housing, food availability.
- Health care provision—Availability, accessibility, quality, utilization.
- Health status—Low birth weight, nutritional status of children, psychosocial development, infant mortality, 1–4 year mortality, maternal mortality, life expectancy at birth, disease specific mortality, morbidity, disability.

Well-being

The feel good factor, if analysed, has two components.

- **Standard of living**. This is a generic term that is rough indicator of our capacity of expenditure. There are vast discrepancies about standards of living all over the world. Actually it is an index comprising of income, standards of housing, sanitation, nutrition, occupation, education, recreation and health services.
- **Quality of life.** It is a very subjective feeling arising from the health status, happiness, education, social security, intellectual attainments, freedom of expression and social justice.

Health and development

Health is an integral part of economic development of a country. Health and its maintenance is a major social investment. However, it has been shown conclusively that simple health measures, use of primary health care, aseptic delivery practices, improvements in education can improve health status of the community without any extra monetary input. A healthy population will, eventually, serve the country better and accelerate development.

Human development index

The HDI indicates quality of human condition based on **life expectancy at birth, per capita annual income** (in US$ purchasing power parity[18]), **educational attainment** (adult literacy and mean school years). It varies between 0–1. In HDI rank, India is **134th** in the world.[19]

The Human Development Index has been criticized on a number of grounds, including failure to include any ecological considerations, focusing exclusively on national performance and ranking, and not paying much attention to development from a global perspective.

India—Indicators of human development [UN Human Development Report, 2009]

- Probability of not surviving upto age 40—15.5%
- Adult literacy—66%
- Population using improved water source—89%
- Children underweight for age—46%
- Population earning below international poverty line (less than $1.25/day purchasing power parity)—41.6%.

Gender related development index

To accomodate varying male: Female ratio in countries, and emphasize the differences in gender specific literacy, the UN has devised the **Gender related development index**. The principal difference from HDI is UN uses a different standard for male and female life expectancy, basically assuming that it is natural that women should live about 5 years longer than men.

Three tier health care

The health system that most of the nations, including India, have set up after 1980 consists of three tiers, as recommended by "health for all" policy.

Primary care

This is the bare necessity—Essential health care every citizen needs. It is the first level of contact between public and the health system. Most of common health problems should be served at this very level. This includes primary health center/ subcenters. The nature of service is curative + preventive.

Table 1.4. Differences between primary health center and hospital

	Primary health center	Hospital
National norm	1 PHC/30000 population	No such
Catchment	Definite	None
Type of care	Curative + preventive	Curative

Secondary care

Diseases beyond the primary level are referred to secondary level which provides mainly curative service. It includes district hospitals, subdivisional hospitals, state hospitals, community health center (CHC—'1st referral unit')/rural hospitals. The level is called the '1st referral level' because ideally, it should receive only those patients who have been referred from primary level.

Tertiary care

It is superspecialty care. It includes medical colleges, regional hospitals and medical institutes (i.e. school of tropical medicine). This tire is also concerned with training, administration, planning and management of the whole system.

Disease

In the absence of a WHO definition, I will only cite those definitions of 'disease' which other people have mentioned.

1. Ecologist—"A maladjustment of the human organism to his environment"
2. Physician—"Any deviation from normal functioning or state of physical and mental well being"
3. Microbiologist—"Disruption of equilibrium in the epidemiological triad of host, agent and environment".
4. Nutritionist—"Deficiency/excess/disbalance of bodily components"

None of these are complete on their own. The true definition of disease would have to incorporate *all* of them.

Two similar terms must now precisely be understood. *Illness* is the subjective *feeling* of being unwell. A man can be diseased but not ill. *Sickness* is the role that the individual assumes when ill ("Oh I'm sick to my stomach, I cannot attend office today!" or "You're really sick! You need help."). A man can be sick without a disease.

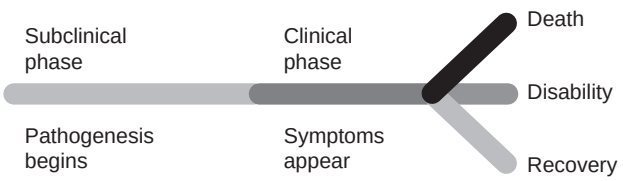


Figure 1.2. Disease stages

Each disease usually begins with entry of the agent → subclinical phase → mild/moderate/severe clinical phase → recovery, death or disability (organ amputation/prosthetics/carrier states in case of communicable diseases, etc.) (Fig. 1.2). This is usually the *spectrum* of a disease.

Iceberg of disease

Actually, there are four kinds of people in this world (Fig. 1.3).

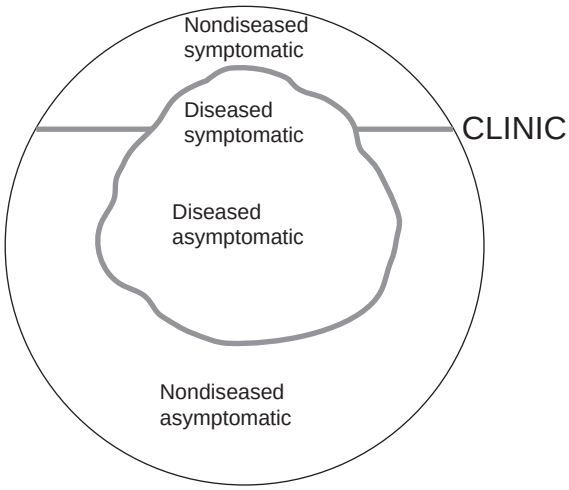


Figure 1.3. Iceberg of disease

Most communicable diseases have an asymptomatic period, during which the person spreads the disease without his/her knowledge. Noncommunicable disease, such as cancers, may have a long latent period before presentation and detection. **Screening tests** are the rapidly applied tests in apparently healthy individuals of a community to detect cases without symptoms.

Perceptions of disease

Over the ages, our ideas of disease causation has gone a few paradigm shifts.

Supernatural. Although enlightened thoughts were not uncommon in ancient civilisations, the belief that diseases were 'curses' from above dominated the middle ages.

Germ theory. This was the first scientific insight into causation of disease. It embodies a one to one relationship between microbes and disease, and especially gained momentum in the 19th century. But it was grossly inadequate because microbes represent only a fraction of disease causation.

Epidemiological triad. Everyone exposed to a microbe does not get the disease. A tuberculosis Bacilli is particularly prone to infect a malnourished/immunocompromised individual than a healthy one, who live in a filthy environment. This demanded a broader concept of diseases the epidemiological triad of host, agent and environment. Mind the term 'agent' here. An agent is any entity that is usually prelude to a disease. It may be a bacterial toxin/cholesterol/carbon monoxide and anything that is the root cause of the disease.

The multifactorial causation theory. The preacher of multifactorial causation was probably Pettenkofer, but in an age when his words were lost amidst fanatics of germ theory. What he proposed was no less than revolutionary, and has shaped our modern perception of health more than anything. **An interaction between various factors ultimately produces a disease often called the risk factors**. Relative importance of these factors can be quantified and arranged in order of priority. These factors are only suggestive and not absolute causative agents. Analytical epidemiological studies are used to identify these risk factors.

Web of causes. As the tide of infectious diseases receded, the burden of disease in developed countries shifted to noncommunicable diseases, and we realised that a number of factors operate in collaboration with each other to produce heart disease or diabetes. Not all of them are to be controlled as they have their relative importances. A few important interventions will usually suffice. Example: Male sex and increasing age are nonmodifiable risk factors for ischemic heart disease, but cessation of smoking, exercise and dietary changes offer an affordable protection.

Epidemiologic basis of disease

Consider the statement "*Mycobacterium tuberculosis* causes tuberculosis". Hardly surprising. You have been aware of it since your ninth standard. Except that not everyone infected with *M. tuberculosis* has tuberculosis. Almost everyone of us is infected, but not ill. You'll insist that the disease affects predominantly people of a certain economic class. So you must rephrase "*Mycobacterium tuberculosis* causes tuberculosis in lower socioeconomic groups". And yet its incomplete, because as well as in lower economic classes, tuberculosis is gaining foothold in the middle class, especially those on a tight schedule and health care professionals. Thus you have to rephrase again, and it could go on.

The fallacy is that "*M.tb* causes tuberculosis" is an *epidemiologic truth*, which is a concise statement of the epidemiologic triangle of tuberculosis (Fig. 1.4).

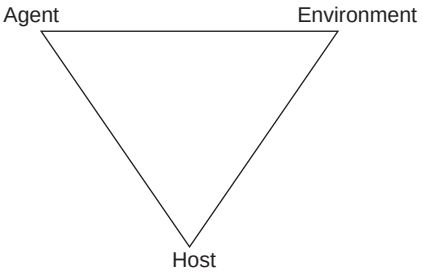


Figure 1.4. Epidemiologic triangle

Neither one, in isolation, can produce disease.

Natural history of disease/clinical course

The natural history of disease is the evolution of course of a disease overtime uninterrupted by any treatment.

Prepathogenesis. It is the phase before onset of disease but the epidemiological triad is ready. The situation is usually referred to 'man in midst of disease'.

Pathogenesis/clinical phase. It is the phase from onset of pathogenesis to termination. It begins with exposure to the agent and the epidemiological triad becomes active. It has two phases—Subclinical (no symptoms) and clinical (symptoms appear).

Physical basis of disease

It is very embarrassing to classify diseases. Most names have been given irrationally by looking at a single sign or symptom (i.e. 'diabetes mellitus' meaning 'frequent sweet urine') or when more details were known, an underlying pathology (i.e. 'myocardial infarction'). There is no consistency in the concept of what you call the disease. For example, a man with long standing diabetes mellitus developed hyperlipidemia, which accumulated as an atherosclerotic plaque in his left coronary artery, and culminated in thrombosis of that artery resulting in a myocardial infarction, ending up with left heart failure and pulmonary edema, ultimately the man dying of apnea. So what was his disease?

In this case, his disease should really be called 'myocardial infarction', and 'diabetes mellitus' an association. The 'hyperlipidemia', 'atherosclerosis' and 'thrombosis' are pathogenic mechanisms in this case, although they may oddly become diseases themselves (it is customary to include anything as 'disease' which has some 'definitive treatment'. Thus hyperlipidemia is a disease but nobody calls 'hypercreatinemia' a disease, they simply refer to as 'renal failure').

The definitive classification of diseases is published by WHO every 10 years in a book called **International Classification of Diseases, Injuries, and Causes of Death**. It assigns a three character alphanumeric code to every major condition. Often a fourth character is added for more exact specification: For example, ICD C92 is myeloid leukemia", which may additionally be specified as C92.0 ("acute") or C92.1 ("chronic"). Broader groupings are readily formed—For example, ICD C81– C96 consists of all malignant neoplasms of lymphatic and hematopoietic tissue. This system is used for coding death certificates. It determines the presentation of results in the registrar general's reports and in the diagnostic registers of most hospitals. The system has to be revised periodically to keep pace with medical usage. The ninth revision came into general use in 1979, and has now been superseded by the 10th revision for many applications. The ICD 10 (1993) has a whopping 21 major chapters.

Without delving into such complexity, I have tried to classify diseases from the deepest roots as I can. It will seem odd at first but it is the only way to bring some rationality into it. However, these types are always bound to overlap.

Genetic diseases

Defects in genome may lead to diseases, via enzyme deficiency and altered metabolism. Genetic diseases are on the rise, chiefly due to more exposure to radiation.

Congenital genetic diseases

- Hereditary, i.e. carried over from an ancestor (hemophilia, autoimmune diseases and probably diabetes).
- Mutations—Due to gross mutation during intrauterine life (drug exposure, radiation, congenital anomalies).

Acquired genetic diseases

- Neoplasia, i.e. ill-regulated growth of tissue due to an acquired mutation in some growth regulating gene.
- Aplasia—The reverse of neoplasia.

Infections

Infections are the major chunk in developing countries. Agents which infect man are viruses, bacteria, protozoan, helminthes, fungi, arthropods and some algae. Infections may cause complications belonging to many other categories in this list.

Diseases of imbalance

Deficiency anemias, malnutrition, vitamin deficiencies, etc. fall in this category. Again, obesity be considered a diseases of excess.

Degenerative diseases

Alzheimer disease, cirrhosis fall into the category of degenerative disease, i.e. replacement of tissue parenchyma by fibers/glia, etc.

Trauma

Accidents and war are man-made epidemics.

Psychiatric diseases

The classification of psychiatric diseases has produced an entire offshoot from ICD, called the Diagnostic and Statistical Manual (DSM).

Risk factors

The gods are just, and of our pleasant vices
Make instruments to plague us

—King Lear, V.iii.193 William Shakespeare

An attribute or exposure that is significantly associated with development of a disease. They are only suggestive and not absolute proof of disease occurrence. Also, they must be identified prior to disease occurrence. Risk factors are suspected from *descriptive epidemiologic studies* and established by *analytic studies*.

Actually, risk factors represent unclarified 'agents'—This means the cause effect relationship is usually lacking in them.

Types

1. **Additive**: Smoking + dyes cause additive effect on bladder CA
2. **Synergistic**: Hypertension and hypercholesterolemia potentate each other in development of CHD
3. **Modifiable**: Smoking, hypertension, increased cholesterol in CHD
4. **Nonmodifiable**: Age, sex and genes. This is the risk for which risk groups are mostly defined.

Community risk

Air pollution, water contamination, traffic problem, urban congestion, etc. are risk factors to the health of the entire community.

Risk groups

Presence of risk factors in an individual/group make them more vulnerable to certain diseases. They can be identified by certain definite criteria and more attention is paid to them (risk approach).

Surveillance

It is the

- Ongoing systematic collection, analysis, interpretation of health data
- Essential to planning, implementation and evaluation of public health services
- Closely integrated with the timely dissemination of these data for action.

Simply put – Surveillance is keeping a vigil over the health status of a community and taking necessary steps every moment. It may be said to be the adaptation of health service to changing health status.

Purpose

1. To provide information on new/changing trends in health status.
2. Timely warning on public health disasters/epidemics.
3. Feedback to modify policy or action.
4. It helps to plan and set program priority and also to evaluate public health programs (i.e. how good they fare).

Procedure

Collection of data (mainly health indicators) → Consolidation and interpretation of data (i.e. making out information from data – what are the facts behind the figures?) → Dissemination of appropriate plan in demand of changing health status → Action for control and prevention.

Types

1. **Individual**: It is the surveillance of a healthy person/patient while he has a disease or may carry the disease.[20]
2. **Local/National/International**: Public health problems are kept under close vigil by the nation. Disease like malaria, yellow fever, rabies and relapsing fever are under international surveillance.
3. **Sentinel**: Not all cases report to the government hospitals. Sentinel surveillance aims at identifying missing cases through specialized institutions (like Beleghata ID hospital) or competent individuals (physicians). It is only supplementary to national surveillance but gives more unbiased and detailed data on the cases of a disease and helps find missing cases. A typical example is HIV surveillance which is jointly done by national and sentinel surveillance.

Monitoring

Monitoring may be said to be a small fraction of surveillance. It is the *performance and analysis of routine measurements aimed at detecting changes in the*

environment/health status of population. **Thus we have monitoring on air pollution**, growth and nutritional status. In most cases, monitoring is not a medical affair – technicians and instruments can very well measure dust in the air or MAC of a child. To interpret this data (as in surveillance) however, needs professionals.

Three levels of struggle against diseases

In addition to providing socialized health care to its citizens, the responsibilities of the state also include programs and actions against **public health problems**—A disease that affects a large number of people, killing/disabling a lot of people and decreasing the productivity of the nation (tuberculosis, malaria, leprosy in India, to name a few). There are three steps, to be followed one after another, to win over a disease (Fig. 1.5; A = agent, H = host, E = environment).

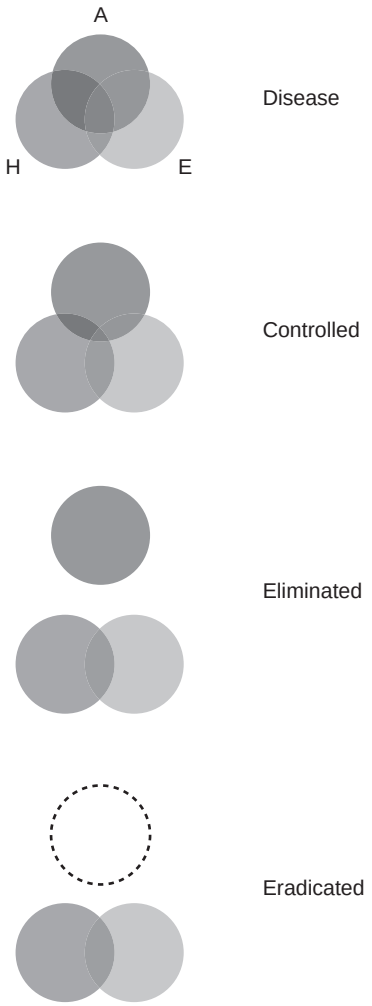


Figure 1.5. Three levels of struggle

Disease control

The 'agent' remains in environment but to such a low level that it ceases to be a public health problem. A state of unstable equilibrium is maintained in the epidemiological triad. It is an ongoing operation and functions by primary and secondary prevention. Its objectives are:

1. Reduce number of disease occurrence in community.
2. Reduce duration of diseased states.
3. Reduce the associated financial burden to the individual and the nation.

Elimination

This means **interruption of transmission** of disease – by any means. Even after elimination is achieved, apparently, hidden foci of infection may persist and unrecognized methods of transmission may also exist.

The leprosy elimination program was taken up in 1983 and the aim was set at <1 case/10000 population.

Eradication

It means the wiping out of the agent from the environment. It is an absolute goal and have been achieved only for smallpox (May 1980) and Dracunculiasis (2000). Poliomyelitis and measles are serious contenders in line. The criteria for a disease to be eradicated are as follows:

1. Should not have an extra human reservoir (in which case that reservoir become a headache, and often the solution becomes to eradicate the reservoir too!).
2. Should not have a long carrier state (in the 180 days that require to show up a Hepatitis B infection, you will eventually lose patience to know the results of the postexposure prophylaxis).
3. Should have good tools to fight against (AIDS can't be eradicated until better antiretroviral drugs, and at least a vaccine are in).
4. Cases should be easily detectable.
5. Should not have subclinical cases (patients who don't come to doctor make the health status worse and actually help the disease to survive).
6. International cooperation is necessary to eradicate a disease.

Prevention

Recall the definition of preventive medicine: *Preventive medicine is the art and science of health promotion, disease prevention, disability limitation and rehabilitation (Fig. 1.6)*.

Primary prevention

These are the activities directed to prevent the occurrence of disease in a human population. The aim is to prevent disease and prolong life.[21]

Health promotion. "The process of enabling people to increase control over their health and its determinants, and thereby improve their health".[22] Health promotion consists of all the activities which are not aimed at any specific diseases but serve to improve the host factor in epidemiologic triangle.

1. Health education.
2. Environmental modification (reducing air pollution, safe water, sanitary latrines, control of insects and rodents, improving housing).

3. Engineering lifestyle (antismoking campaign, condom promotion, etc.).
4. Genetic and marriage counseling (to prevent congenital diseases, i.e. Thalassemia).
5. Increasing the standard of living (i.e. the income, education and occupational status).
6. Health legislation, i.e. forming rigid standards of health care, sanitation and issues relating to health.

Specific protection. The measures which target *particular* diseases. The idea of specific protection, especially that killer diseases could be stopped by simple interventions such as 'vitamin oils' and 'shots' gained mass acceptance in between 1970–1990s.

1. Immunization.
2. Nutrient supplementation (vitamin A, iodine).
3. Chemoprophylaxis (prior medication to at risk population).
4. Protection against occupational hazards (masks and sound mufflers for workers).
5. Avoiding allergens (for asthmatics).
6. Quality control of consumer products (salt—For iodine deficiency diseases, drugs—To avoid adverse drug reactions, cosmetics—To avoid allergy).

Secondary prevention

Those actions which halts the progress of a disease in an individual at the incipient stage and prevents further complications. It is indeed prevention because it prevents further spread of that disease from that individual.

- **Early diagnosis**
  1. **Screening tests** are done in healthy population of a community (i.e. PAP smear).
  2. **Case finding** means diagnosing something else in patient other than his chief complaint.
  3. Special medical examination of risk groups.
- **Prompt treatment**: A quick cure, helps the patient as well as stops further spread of disease.

Secondary prevention has the disadvantages of being more expensive, less effective in prevention/relief and it fail to prevent loss of productivity to community – as the individual is already diseased.

Tertiary prevention

All measures available to

- Reduce or limit impairment and disabilities
- Minimize suffering caused by existing disease
- Promote the patients adjustments to irremediable conditions.

Tertiary prevention alleviates the pain of the patient who has already been scarred by a disease.

Disability limitation

Disease (leprosy)
↓
Impairment (anatomical/psychic concept – like destruction of eyes in leprosy)
↓
Disability (physiological concept – like loss of vision due to eye damage)
↓
Handicap (social concept – blind because of leprosy)

Disability can be limited by proper treatment – in this case with MDT.

Types of handicap

- Physical—Poliomyelitis
- Mental—Autism
- Social—Orphans.

Rehabilitation: *Combined use of*:

- Medical, social, educational and vocational measures
- For training and *retraining* the disabled individual
- To the highest possible level of function.

There are four dimensions of rehabilitation

1. *Medical*: If possible restoration of function (physiotherapy/gadgets, etc.)
2. *Vocational*: Restoration of capacity to earn a livelihood (training and creating jobs)
3. *Social*: Reintroduction into family, kins and society as a whole and involving everyone to maintain the same relationship with this person.
4. *Psychic*: Restoration of self-esteem and confidence.

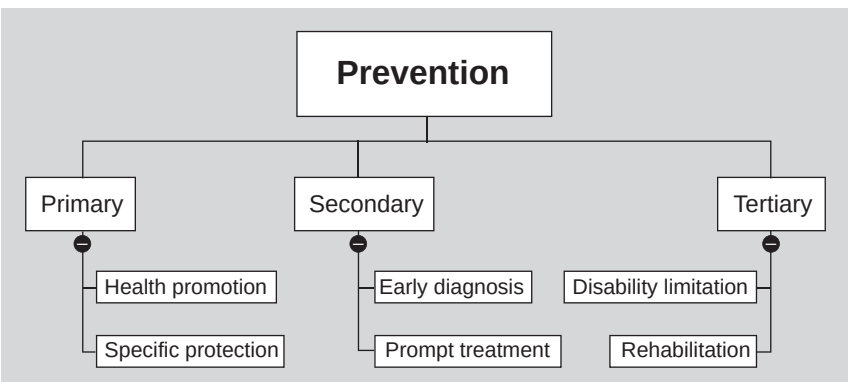


Figure 1.6. Three levels of prevention

STATISTICS

There are three kinds of lies: lies, damn lies, and statistics.

—Benjamin Disraeli

The mathematicians dilemma

Science believes in generalizing the special, to make general rules that apply to everybody, from a limited number of observations. This is the only way research is possible, because it is not humanly possible to examine every human on this planet to verify if he has a set of femurs. When Vesalius cut up his very first corpse, he instantly made the decision that the human species, not just the specimen on his table, but the entirety of the human species, must have two femurs. Herein lies the mathematician's dilemma. A *formal proof*, as iterated in pure mathematics, should not be based on induction (that is, no one specimen should be held representative of whole of the population). A proof must establish, by mathematical procedures, an *identity* between two sides of an equation. This kind of reasoning, however smart it may sound, is absurd in most kinds of research except pure mathematics. For the more mundane kind of research, we cannot examine whole populations and we *have to* deal with samples. A few questions immediately spring up—

- What should be the sample size to make a reasonable conclusion (i.e. can I make a statement "every human has two femurs" by examining only *one* corpse, or do I need more samples).
- What should be the benchmark of 'statistical significance' (i.e. how can I decide if between two events, one is being caused by another, or if its just by chance—There is no real relationship between them).
- What will be the amount of error (how valid will be our research) when we deviate from the mathematical 'formal proof' method (i.e. by doing research on samples).
- How much will the research be biased by a distorted sample (i.e. a study done on shoe size on a sample of acromegalics).

Statistics is the answer to these questions.

Data

1. *Data* are the figures you derive directly from the source. Primary data is obtained directly from the population (as in census), and secondary data from a record. Obviously, primary data is always more reliable than secondary.
   Data has two parts, i.e. a *variable* (like age, gender, income, number of spouses, etc. denoted by x) and a *value* (i.e. 40 years, male, Rs. 3500, 3).
   A *variable* may be qualitative (Boolean data, subjective data) or *quantitative* (numbers). Quantitative data may be further classified as *continuous* (i.e. height, weight which may have any value) or discrete (size of shoes which can have only a fixed set of values).
2. Universe is the extent of a statistical survey being undertaken, also called the population. The numerical size of the population is denoted by η. Subgroups in this universe are called *samples*.
3. An *event* is just that, an event that has or will occur.

Representation of data

The meaning of statistics can ultimately be altered by very easy steps. The simplest is to represent data in such a manner that your boss will find it to easy to comprehend and give you a raise.

Tables

1. **Simple tables**: It is the first thing you have to do with any data. Be sure that everything is readable, the entries have some order (i.e. alphabetical listing) in them, and the table has a title.
2. **Frequency distribution tables**: It describes the frequency of an event within class intervals of a universe. Mind that the class intervals (the domains) do not overlap and are of equal width. A title should unambiguously show what the table contains.

Table 1.5. Frequency distribution table of shoe size in a class

Size	Number of students
7	12
8	15
9	18
10	11
11	6

Diagrams

There are only a few fundamental types of diagrams.

1. **Bar diagram**: Prepare the bars with same width and also same gaps between them.

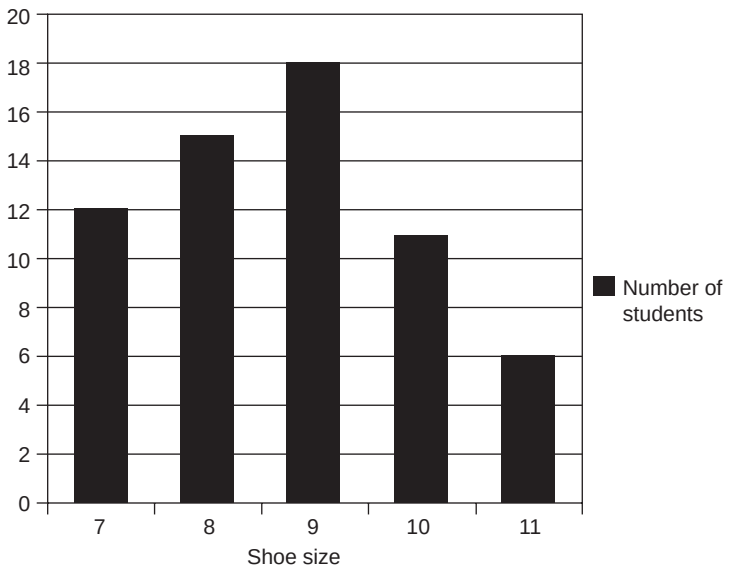


Figure 1.7. A bar chart of distribution shoe sizes in a class

2. **Histogram**: It is similar to a bar diagram except that the *area* of the blocks count. It is the representation of an FD table, so the bars are adjacent (to emphasize the *continuous* nature of the variable being measured, in this case, shoe size, which can be any value between 7–11). By adding the midpoint of top of these bars – you can get a *frequency polygon*.

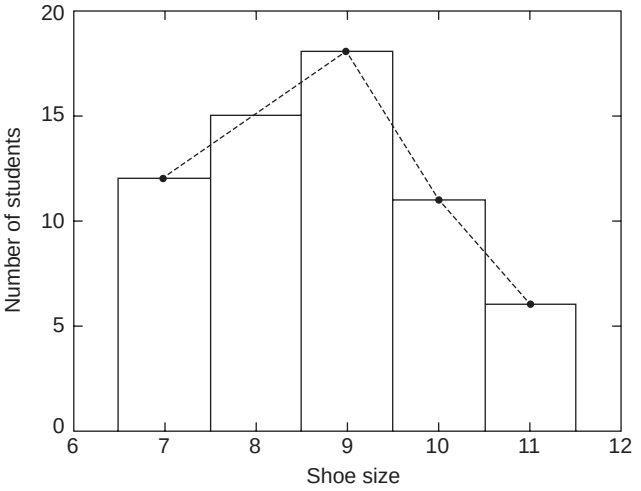


Figure 1.8. A histogram showing distribution of shoe sizes in a class

3. **Pie charts**: It represents categories of data as percentage of total and makes a circle of it. Use specific symbols for each category and make a legend outside. Write only the percentages outside the circle.

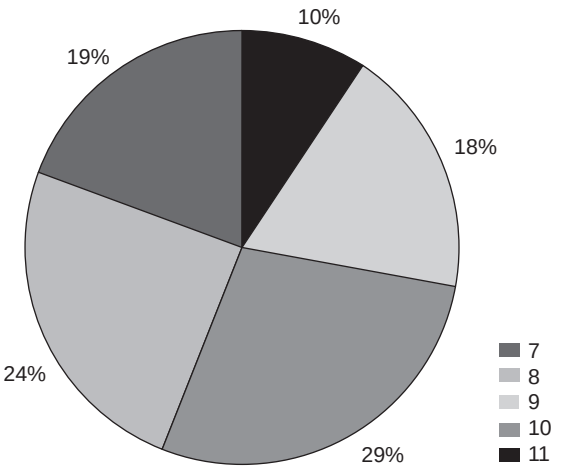


Figure 1.9. A pie chart showing distribution of shoe size in a class

Measurements

By definition, any set of rules for assigning numbers to attributes of objects is *measurement*. Not all measurement techniques are equally useful in dealing with the world, however, and it is the function of the scientist to select those that are more useful. The physical and biological scientists generally have well-established, standardized, systems of measurement, unlike social scientists.

The issue of measurements were discussed in great detail by SS Stevens in an article in 1951.

Properties of measurement scales

Magnitude

The property of magnitude exists when an object that has more of the attribute than another object, is given a bigger number by the rule system, i.e. If A is heavier than B then *weight* of A is more than weight of B.

Intervals

The property of intervals is concerned with the relationship of differences between objects. If a measurement system possesses the property of intervals it means that the unit of measurement means the same thing throughout the scale of numbers. That is, an inch is an inch is an inch, no matter were it falls immediately ahead or a mile down the road.

Rational Zero

A measurement system possesses a rational zero if an object that has none of the attribute in question is assigned the number zero by the system of rules. The object does not need to really exist in the "real world", as it is somewhat difficult to visualize a "man with no height". The requirement for a rational zero is this: If objects with none of the attribute did exist would they be given the value zero.

Scale types

In the same article in which he proposed the properties of measurement systems, SS Stevens (1951) proposed four scale types. These scale types were nominal, ordinal, interval, and ratio. Each possessed different properties of measurement systems.

Nominal Scales

Nominal scales are measurement systems that possess none of the three properties discussed earlier. **Nominal renaming** scales apply random numbers (or words) to objects (i.e. social security numbers, classification of diseases). **Nominal categorical** scales apply a different number to each category of objects (i.e. Belgians = 1, Indians = 2, Irish = 3).

Ordinal Scales

Ordinal scales are measurement systems that possess the property of magnitude, but not the property of intervals. The property of rational zero is not important if the property of intervals is not satisfied. Anytime ordering, ranking, or rank ordering is involved, the possibility of an ordinal scale should be examined. As with

a nominal scale, computation of most of the statistics described in the rest of the book is not appropriate when the scale type is ordinal. Rank ordering people in a classroom according to height and assigning the shortest person the number "1", the next shortest person the number "2", etc. is an example of an ordinal scale.

Interval Scales

Interval scales are measurement systems that possess the properties of magnitude and intervals, but not the property of rational zero (i.e. the height of a person). It is appropriate to compute the statistics described in the rest of the book when the scale type is interval.

Ratio Scales

Ratio scales are measurement systems that possess all three properties: Magnitude, intervals, and rational zero. The added power of a rational zero allows ratios of numbers to be meaningfully interpreted, i.e. the ratio of John's height to Mary's height is 1.32, whereas this is not possible with interval scales.

Sampling

The use of sampling has already been underlined.

Random sampling

Random sampling is left entirely to nature's own laws of entropy, and everyone has equal probability of being selected. It provides the greatest number of possible samples, but it is also most prone to produce a distorted sample. For example, out of fifty girls and fifty boys, a random sample of 20 has to be chosen. Now it is perfectly possible that the sample of 20 that we choose has exactly 10 girls and 10 boys. It is, however, much more probable for our sample to be distorted either way, i.e. we could select more girls than boys or vice versa. It is, by sheer chance, also possible that we select only girls, and that would be an embarrassingly distorted sample, not even close to representating the population.

Matched random sampling

If we really want to do a scientific study, let's say about the pattern recognition skill differences between boys and girls, we must select pairs of a boy and a girl, who are identical in all aspects (age, mental growth, family background, etc.) *except* that one is a boy and other a girl. We could make a comparison only if such *matching* has been done.

Systematic sampling

Suppose we pick every 10th person from our hundred (i.e. 1, 11, 21, 31 … or 4, 14, 24, etc.), which reduces the number of possible samples (in our particular case, only 10 samples can now be chosen, beginning from 1, 11, 21 to 9, 19, 29). However, if the boys and girls are so arranged that every 10th person is a girl (i.e. if there is *periodicity* in the population and we resonate with that periodicity), we would end up with 10 girls again. This is the drawback of systematic sampling.

Stratified sampling

We could divide the population into reasonable groups (strata) and then takes samples from each group so that no group gets predilected (we could split our hundred into 'boys' stratum and 'girls' stratum, and then go on random/systematic sampling within each stratum; this way ensure that we *do not* end up with only girls or only boys in our sample). Stratification can be done on the basis of age, sex, religion or any other attribute.

Cluster sampling

We could also set up groups of 10 among our hundred (each group may include both boys and girls) and select one from each group. This kind of sampling is used in immunization survey among children.

Errors in sampling

Sampling error

Each sample of a universe differs from another sample, and this is unavoidable, omnipresent whim of nature.

Nonsampling errors

1. **Overcoverage**: Inclusion of data from outside of the population.
2. **Undercoverage**: Sampling frame does not include elements in the population.
3. **Measurement error**: The respondent misunderstand the question.
4. **Processing error**: Mistakes in data coding.
5. **Nonresponse**: People unwilling to take part in a survey may get included in the sample.

Calculation of sample size

Where the sample and population are identical in characteristic, statistical theory yields exact recommendations on sample size (the formulae are, however, not exactly nice looking, and perhaps best to avoid in this textbook). However, where it is not straightforward to define a sample representative of the population, it is more important to understand the cause system of which the population are outcomes and to ensure that all sources of variation are embraced in the sample. Large number of observations are of no value if major sources of variation are neglected in the study.

Patterns of data

Consider the shoe sizes in a class. There will be few short ones, a few large ones, but most of the children will fall in between. Every data observed from nature has two opposing characteristics.

- The tend to accumulate in and about a central figure (**central tendency**)
- But nevertheless, some of them stand out far from this figure (**dispersion**).

Measures of central tendency

1. The **mean** is the arithmetic average, denoted by an $\bar{x}$ or μ. It is not, however, a very good indicator of the actual distribution of the variable. Suppose you admit a dinosaur (or any fellow with really big feet) in your class, then the

mean shoe size will be dramatically altered even though only one member has been added. *Mean* is affected severly by the values at the end.

2. The **median** is the midline value of a distribution arranged in ascending order, or the average of two midline values (if number of data is even). The median is *not* affected by terminal values.
3. The **mode** is the most commonly occurring value in a distribution.

Measures of dispersion

Range

It defines the normal limits of a variable. Range of a biologic variable is worked out only after measuring the characteristic in large number of healthy persons of the same age, sex, class, etc. Range gives an idea of how big is the universe, but not anymore about the details.

Mean deviation

A particular value x of a variable is said to be deviating from the mean $\bar{x}$ by an amount $x - \bar{x}$. This is the *deviation* of x from mean. The **mean deviation** is the average of all such deviations.

$$\text{Mean deviation} = \frac{\sum|(x - \bar{x})|}{\eta}$$

The mean deviation gives an idea on how widely the data varies. Mind the absolute value sign '|' around the deviations. If we do not ignore the sign of deviations, positive and negative variations tend to cancel each other out. Say the shoe sizes in a class of 11 (in ascending order) are 6,6,7,7,7,8,8,9,9,9,10. The mean, in this case, is $\sum x/\eta = 7.818$. Lets calculate the mean deviation.

Student number	Shoe size (x)	Mean ($\bar{x}$)	Deviation ($x - \bar{x}$)	$\lvert x - \bar{x} \rvert$	Mean deviation ($\lvert x - \bar{x} \rvert/\eta$)
1	6		–1.818	1.818	
2	6		–1.818	1.818	
3	7		–0.818	0.818	
4	7		–0.818	0.818	
5	7		–0.818	0.818	
6	8	7.818	0.182	0.182	12.182/11= 1.107
7	8		0.182	0.182	
8	9		1.182	1.182	
9	9		1.182	1.182	
10	9		1.182	1.182	
11	10		2.182	2.182	
	86			12.182	

Variance

It is the $\sum(x - \bar{x})^2/\eta$. The variance of a population shows how widely it is distributed. Suppose, in two different classes, the mean shoe size is same. In this

case, the class with the more *variance* has a more varying set of students (i.e. there are more number of students who have a very small or very large shoe size) than the class with less variance. Variance is only a number, and its unit is the square of the unit of the thing we want to measure.

Standard deviation

This is important. Standard deviation is the square root of variance.

$$\text{Standard deviation } \sigma = \sqrt{\frac{\Sigma(x - \bar{x})^2}{\eta}}$$

when dealing with the whole universe, or

$\sigma = \sqrt{[\ \sum (x - \bar{x})^2 / \eta - 1\ ]}$, when in a sample (Bessel's correction)

Student number	Shoe size (x)	Mean ($\bar{x}$)	Deviation ($x - \bar{x}$)	$(x - \bar{x})^2$	Variance $\sum (x - \bar{x})^2/\eta)$
1	6		–1.818	3.305	
2	6		–1.818	3.305	
3	7		–0.818	0.669	
4	7		–0.818	0.669	
5	7	86/11 =	–0.818	0.669	
6	8	7.818	0.182	0.033	17.635/11= 1.603
7	8		0.182	0.033	
8	9		1.182	1.397	
9	9		1.182	1.397	
10	9		1.182	1.397	
11	10		2.182	4.761	
	86		12.182	17.635	1.603

The standard deviation = √variance = √1.603 = 1.266. After Bessel's correction, variance = 17.635/(11 – 1) = 1.763 and SD= √1.763 = 1.327.

Properties of standard deviation

For constant c and random variable x,

$\sigma(x + c) = \sigma(x)$ (the SD is not changed if each value is incremented by same amount)

$\sigma(cX) = |c| \times \sigma(x)$ (i.e. if each value of a population gets multiplied, the SD is also multiplied).

Importance of standard deviation

The standard deviation serves as the 'unit' of variability. We speak 'the shoe size of this student is 3 standard deviations (i.e. 3 × 1.266) more than the mean, i.e. the shoe size is mean + 3 × standard deviation = 7.818 + 3 × 1.266 = 11.616.

Probability

As much as we are fond of data and patterns of data, there is always a limit to how much data we can collect, and at some point of time, we have to stop collecting data and do some hypothesizing. It would have been very fortunate if we could

measure all the data about every aspect of everybody, but until that happens, we have to indulge in speculation and forecasting. The beauty of inferential statistics is that, not only does it allow you to make a reasonable prediction, but also allows you to specify the possible amount of error (i.e. "I am 93% sure that there is 13% chance of rain today"). *Probability* is a theory of uncertainty which deals with these speculations. It is a necessary concept because the world according to the scientist[23] is unknowable in its entirety. However, prediction and decisions are obviously possible. As such, probability theory is a rational means of dealing with an uncertain world.

Probabilities are numbers associated with events that range from zero to one (0–1). A probability of zero means that the event is impossible. For example, if I were to flip a coin, the probability of a leg is zero, due to the fact that a coin may have a head or tail, but not a leg. Given a probability of one, however, the event is certain. For example, if I flip a coin the probability of heads, tails, or an edge is one, because the coin must take one of these possibilities.

In real life, most events have probabilities between these two extremes. For instance, the probability of rain tonight is 0.40; tomorrow night the probability is 0.10. Thus, it can be said that rain is more likely tonight than tomorrow.

The 'odds' of an event. The probability of an event happening/the probability of it not happening. If the probability of rain tonight is 0.3, the odds of rain tonight are 0.3/(1–0.7) or 0.3/0.7.

The meaning of the term probability depends upon one's philosophical orientation. In the CLASSICAL approach, probabilities refer to the relative frequency of an event, given the experiment was repeated an infinite number of times. For example, the .40 probability of rain tonight means that if the exact conditions of this evening were repeated an infinite number of times, it would rain 40% of the time.

In the SUBJECTIVE approach, however, the term probability refers to a "degree of belief/ confidence." That is, the individual assigning the number 0.40 to the probability of rain tonight believes that, on a scale from 0–1, the likelihood of rain is 0.40. This leads to a branch of statistics called "Bayesian statistics." This has lead to the term confidence intervals for the zones in the normal curve (i.e. that a 95% of values lie within 2 standard deviation means that I have confidence that 95 of 100 values will be in this zone).

No matter what theoretical position is taken, all probabilities must conform to certain rules. Some of the rules are concerned with how probabilities combine with one another to form new probabilities.

- For example, when events are independent, that is, one doesn't effect the other, the probabilities may be multiplied together to find the probability of the joint event.

  $P(A \text{ and } B) = P(A) \times P(B)$ when A and B are independent events

  The probability of rain today AND the probability of getting a head when flipping a coin is the product of the two individual probabilities.
- When two events are **mutually exclusive**, i.e. only one of them is possible at a time, then $P(A \text{ or } B) = P(A) + P(B)$

- For two independent events, the probability of either one of them or both occurring at a time (i.e. at least one of them occurs) is
  P(A or B or both) = P(A) + P(B) – P(A and B).

Testing for truth

Truth must ultimately be tested. The material sciences (physics, chemistry) usually provide us with the tools to conduct the tests, but it is statistics which tells how to interpret the test. Common to all tests is to find *association between two events*, i.e.

- To test whether the finding of bronchial sounds is diagnostic of pneumonia (a diagnostic test).
- To test whether smoking is associated with lung cancer (a study to discover risk factors).
- To test whether radiation is effective in lung cancer (a *therapeutic* test).

All tests must be

- **Reproducible**, i.e. gives the same result over and over even if different samples are tested, by different observers with different skill levels.
- **Accurate**, i.e. yields result close to the GOLD STANDARD test, and reflects the true progression of the disease to that level which the test values suggests.
- **Valid**, i.e. can distinguish between a positive and a negative result (i.e. between diseased and nondiseased) to a satisfactory degree.

Every diagnosis or decision is based on a test, be it the history, a symptom or sign, or some laboratory routines, or the history of an exposure to a risk factor. Such a diagnostic test must bear a few accreditations if it has to qualify for being used in clinical reasoning.

Reproducibility

It is the ability of a test to yield the same results over and over, irrespective of variations in basis of test, method or skill. No test is wholly reproducible. Suppose you have diagnosed a man to have pulmonary tuberculosis by examining sputum smears. A fellow physician may wholly disagree with you, because, when he did the examination, either

1. Patient became sputum negative (variation in the basis of the test).
2. The physician used a different stain (variation of method).
3. The physician made an error in spotting the AFB (variation of skill).

Because no test is wholly reproducible, the whole medical business continues to run on uncertainty.

Accuracy

A test is accurate when

1. It yields results equal, on the average, to a GOLD STANDARD test.
2. The test result indicates the true progression of the disease.

Suppose you devise method X to determine blood glucose. To qualify as accurate, this test has to yield results consistent with that of the Glucose oxidase reaction; in addition, it must truly reflect the progression of diabetes mellitus.

Validity

This is the challenge: To distinguish truth from just another chance finding.

Put simply, let's say you attend some kids with complaint of cough, fever and mild dyspnea. You did some auscultation (the test) and find bronchial sounds in some of these kids, and label them as pneumonia. Next, your boss carries out a culture on their lung aspirate (the Gold Standard Test) and disproves your findings. The final scenario is this.

	Diseased Culture +ve	Nondiseased Culture –ve
+ve test Bronchial sounds	a	b
-ve test No sound	c	d

The **sensitivity** of the test is the probability of diseased people yielding positive result,= a/a+c. The **specificity** is the reverse, the probability of healthy people to give negative result = d/b+d.

A test which is very nonspecific will yield too many false-positive results; on the other hand, an insensitive test gives too many false-negative results. The importance we attach to a positive or negative result is thus a function of the cut off value of the test, i.e. after which level we consider the results positive. Selecting a low cut off reduces the specificity of the test, and a high cut off dampens sensitivity.[24] In fact, the curve of sensitivity vs specificity looks like this (Fig. 1.10).

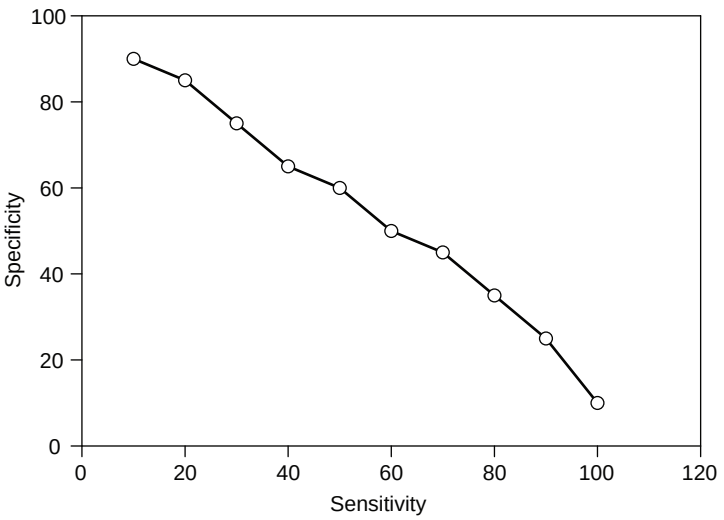


Figure 1.10. Sensitivity versus specificity curve

The perils of using nonqualified tests are many. Too many false +ve results are a burden on the health infrastructure, cause useless anxiety to the victim, and once labeled – it's hard to get rid of the 'feeling' of the disease (consider what goes

though anyone after being diagnosed HIV +ve). Again, too many false –ve results fail the entire purpose of clinical medicine and screening.

Predictive value

This is the most important aspect of a test from a clinical viewpoint. A **positive predictive value** is the probability of someone testing positive actually having the disease, i.e. a/(a + b). Similarly, a **negative predictive value** is the probability of not having the disease in someone testing negative = d/(c + d).

The positive predictive value is affected by the

- Prevalence of the disease—In areas of high prevalence of some disease, tests for the disease are more valid.
- Specificity of the test.

The usual challenge: Determine posttest probability from pretest knowledge

Bayesian statistics states that the positive predictive value is much higher if the disease in question is very prevalent. In another words, **the usefulness of a test, apart from an inherent quality of itself (the sensitivity and specificity), is also dependant upon how common the disease is**. Suppose the prevalence of a disease in a certain area is P in a population of η; this means, the chance of any individual of that area of having the disease is P (the **pretest probability**). Now we do the test and get a positive result. Given the specificity and sensitivity of the test, what is the probability that the individual is truly diseased?

	Diseased	Nondiseased	Total
+ve test	a	b	a + b
–ve test	c	d	c + d
Total	a + c	b + d	

Now, obviously, the number of total diseased people a + c = Pη and nondiseased people b + d = (1 – P)η. Again, the positive predictive value is (number of diseased people who tested +ve/total number of people who tested +ve) = a/a + b.

Given the sensitivity of the test is *SN* and specificity is *SP*, we know that

$$SN = a/(a + c) \text{ and } SP = d/(b + d)$$

From these equations, calculate your heart out for the value of positive predictive value a/a+b; you will find it to be

$$\frac{P \times SN}{(P \times SN) + (1 - SP)(1 - P)}$$

This is the positive predictive value of a test if the sensitivity, specificity and prevalence is given. There is, however, a more subtle way to achieve the same result. The odds of an individual having the disease before the test is, obviously, P/1 – P (see definition of Odds). Now, Bayesian statistics states that the odds (r) of having the disease *after* a positive test is

$$r = \frac{P}{1 - P} \times \frac{SN}{1 - SP}$$

Suppose posttest probability (or positive predictive value) is x; then by definition of odds

$$\frac{x}{1-x} = r$$
$$\text{or, } x = r - rx$$
$$\text{or, } x + rx = r$$
$$\text{or, } x = \frac{r}{1+r}$$

This x is the posttest probability or the positive preditive value (do the actual calculation on a real problem and you will find the result from the two methods to be identical). The factor **sensitivity/1-specificity** is called the **likelihood ratio** of the test.

Hypothesis testing

I mentioned earlier that inferential statistics allows you to predict **both** the probability of an event and the amount of **error** of that prediction. This section is to determine that amount of error.

The usual hypotheses

The null hypothesis (H_0). It states that there is *no relation* between the two events tested.

The alternate hypothesis (H_a). It states that the two events are related.

Obviously, both cannot be true simultaneously.

Errors

Type I error (α). The error that happens when *null hypothesis is rejected in spite of it being true* (i.e. you wrongly diagnose an association between traveling to Goa and ulcerative colitis, or something equally bizzare). The probability of a Type I error happening in a test is called **p value** or α limit of the test.

Type II error (β). The error that happens when the *null hypothesis is accepted in spite of it being false* (you miss a true association)

The **power** of a test, i.e. the probability that a test detects any difference that *actually* exist, is $1 - \beta$.

Tests for statistical significance (Fig. 1.11)

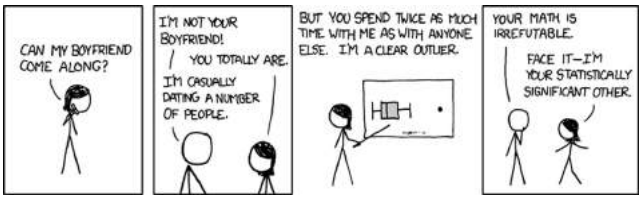


Figure 1.11. You can't deny mathematics[25]

The normal distribution

The idea of the normal distribution is primary to hypothesis testing. In collecting data, if our sample is large enough, we tend to have a distribution much like that of our shoe size example (Fig. 1.12).

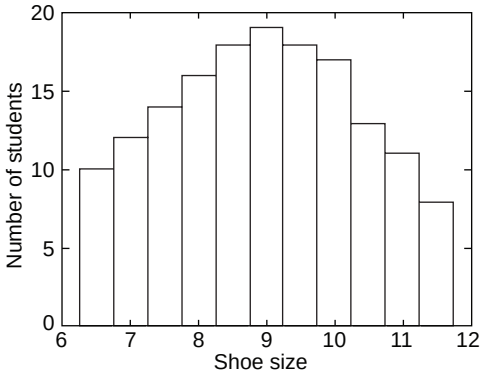


Figure 1.12. The shoe size histograms after more data collection in 0.5 size intervals

When we connect the top of the bars, we get a curve like this (Fig. 1.13).

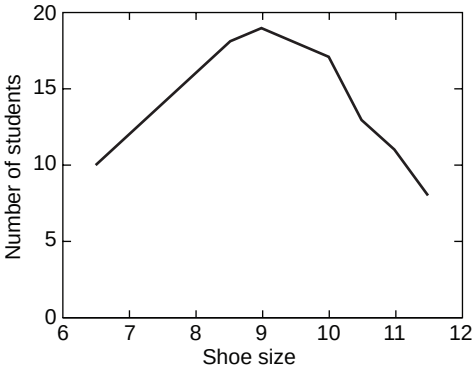


Figure 1.13. Line graph showing distribution of shoe sizes

If we plot the y-axis from 6 instead of 0, as to emphasise the variations, not actual values, then we get (Fig. 1.14).

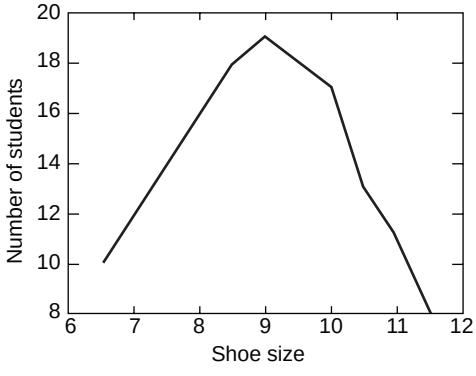


Figure 1.14. Line graph showing distribution of shoe sizes, y-axis corrected

If we could go on, and collect show size data of an *infinite* number of students, and if shoe sizes would vary *continuously* (that is, the **class intervals**, would be adjacent—There was no discrete jumps from size 6 to 6.5 but 6.1, 6.11 and so on), we would actually produce a **normal curve**. It is a curve which has been sketched after **infinite number of observations and with no gaps in between class intervals**. Because the curve is absolutely hypothetical, it is a **bell-shaped**, **symmetrical curve with absolute continuity** (Fig. 1.15).

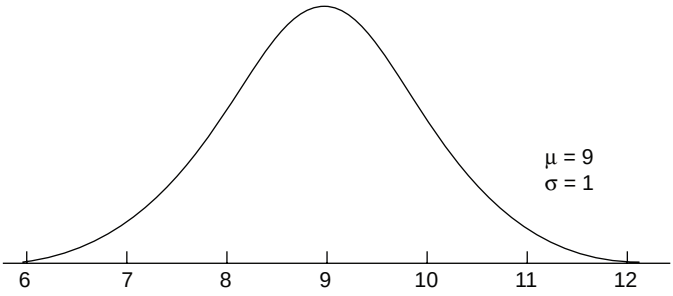
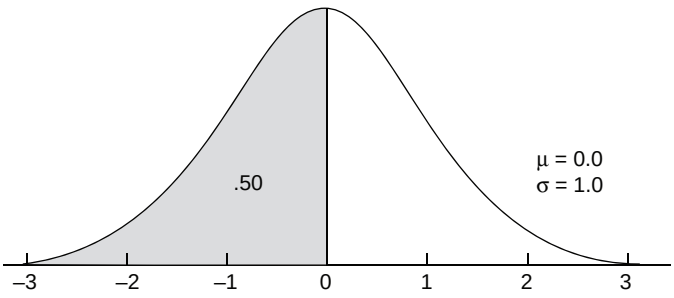


Figure 1.15. Normal curve of shoe size distribution

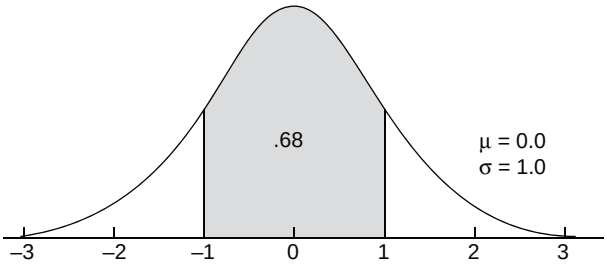
The standard normal curve

The standard normal curve is a member of the family of normal curves with μ = 0.0 and σ = 1.0. The value of 0.0 was selected because the normal curve is symmetrical around μ and the number system is symmetrical around 0.0. The value of 1.0 for σ is simply a unit value. The x-axis on a standard normal curve is often relabeled with multiples of σ and called z-scores.

There are three areas on a standard normal curve that all introductory statistics students should know. The first is that the total area below 0.0 is .50, as the standard normal curve is symmetrical like all normal curves. This result generalizes to all normal curves in that the total area below the value of μ is .50 on any member of the family of normal curves.

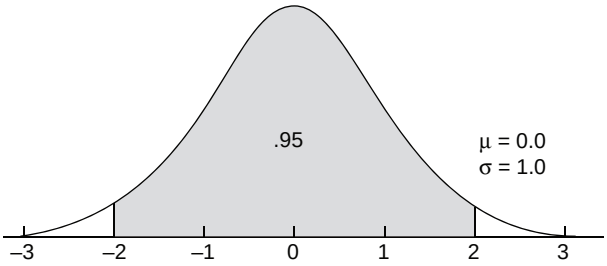


The second area that should be memorized is between z-scores of –1.00 and +1.00. It is .68 or 68%.



The total area between plus and minus one sigma unit on any member of the family of normal curves is also .68.

The third area is between z-scores of –2.00 and +2.00 and is .95 or 95%.



This area (.95) also generalizes to plus and minus two sigma units on any normal curve.

Conversion of a normal curve to a standard normal curve

Suppose a variable x has a mean $\bar{x}$ and a standard deviation σ. It will produce a normal curve, no doubt. But it is easier, if we want to emphasize the *variation* rather than the actual values, to plot it in a standard normal curve by making the mean 0 and standard deviation 1 (Fig. 1.16).

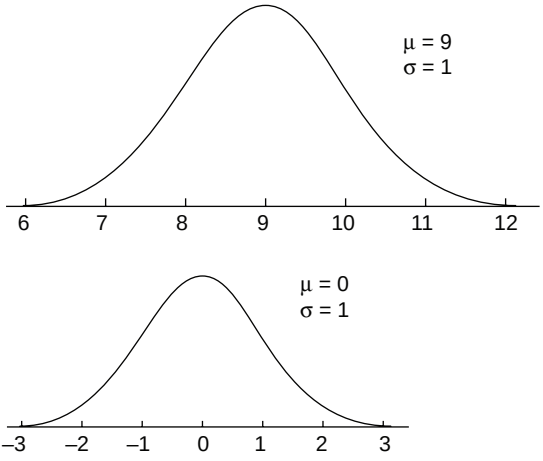


Figure 1.16. Making a standard normal curve

Think of a **relative deviate** z which is the reduced form of x for a standard normal curve.

$$z = (x - \bar{x})/\sigma$$

Now plot the curve of z. The curve of z is shifted parallel to x and is reduced in size, but the fractional areas under confidence intervals remain the same.

Skewed distributions (Fig. 1.17)

1. Positively skewed—Mode < median < mean
2. Negatively skewed—Mode > median > mean

Remember that mean is always dragged towards the tail of the distribution.

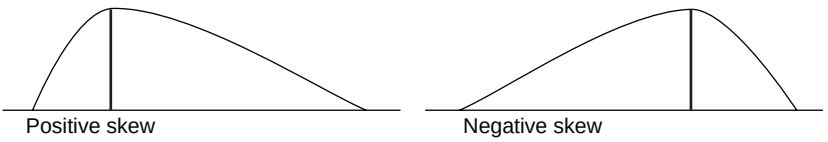


Figure 1.17. Skew

Probability and the normal curve

Each particular area bound between any two z-scores (any two vertical lines over the x-axis) is associated with a certain degree of probability, and all such areas are called **confidence intervals**. In continuum with our example of shoe sizes, what of those students who have a size of 11.5 or more? Could they be considered acromegalics, or perfectly 'normal' people? Look at the normal curve of shoe sizes again, the size 11.5 lies outside two standard deviations (11 in our curve). The area inside two standard deviations in a normal curve, as I have already illustrated, is 0.95. Thus the area outside 2 standard deviations is obviously, 0.05. This means that **the person with a shoe size of 11.5 has a 0.05 probability (or 5%) chance of being normal**.

Levels of significance and p-values

We have mentioned that 95% of values lie within 2 standard deviations around the mean. This means, 5% of values lie outside (thus called 'outliers'). This is called the **alpha limit** of two standard deviation. A p-value or probability value can be calculated for each interval in the normal curve (recall that the p-value is the probability of a Type I error, i.e. a false association is diagnosed). The p-value indicates the **probability of a member from that interval being selected in a random sample**. A p-value outside this limit (< 0.05) is **statistically significant**, i.e. if the members of this interval (the last 5% area under the normal curve) appear in a random sample, that sample has 5% chance of being representative of the population. Similarly, p-value may be deduced for any confidence interval.

Binomial distribution

Not every variable (in fact, none) is distributed normally. For the real world with its variations, inequalities and heterogeneities, statisticians have found certain patterns of how certain events tend to happen. These 'models' of reality are known as 'statistical distributions'.

Let's say that a certain event has only two outcomes, such as tossing a coin. It has two possible outcomes head (H) and tail (T). Let p be the probability of head, q be the probability of tails. Clearly, from an abstract, neutral viewpoint, both p and q equal 0.5.

Now we do the tossing twice. The possible number of outcomes now become four (HH, HT, TH, TT). What is the probability of each of these occurrences? Remember that the probabilities of two independent events get *multiplied* if that are to occur together. The outcomes of the two tosses are independent (i.e. the results of the first toss does, in no way, affect the second), so probabilities must be multiplied.

Outcome	Probability
HH	$p \times p = p^2$
HT	$p \times q = pq$
TH	$q \times p = pq$
TT	$q \times q = q^2$

The total of all probabilities, i.e. summation of all possible outcomes of a binary event (as in a coin toss), is the **binomial distribution**. When tossing only twice (n=2), the summation is $p^2 + 2pq + q^2$. For tossing n times, we can generalize the formula by mathematical induction

$$(p+q)^n$$

which results in the expansion (following Newton's binomial theorem; ${}^nC_r$ denotes possible combinations of r things among n slots)

$$p^n + {}^nC_1p^{n-1}q + {}^nC_2p^{n-2}q^2 + \ldots {}^nC_rp^{n-r}q^r + \ldots q^n$$

or, to put it concisely (review the formula of combinations from your class XII algebra book)

$$\sum_{r=0}^{n} \frac{n!}{r!(n-r)!}\, p^r q^{n-r} \text{ (put values of r from 0 to n)}$$

where r is number of times we get a 'head' out of those *n* number of tosses, and we denote a 'head' as our success. Now let's put Aside all this coin business and do some real research. Suppose Aspirin cures, and has been curing, most headaches (80% of the times) since time immemorial. Now you introduce a new drug 'headgone' that boasts 98 cures out of 100 patients who have tried it. Is it really any significant improvement over Aspirin?

Apart from the pharmacological issues (its pharmacokinetics and safety profile), let us be purely statisticial. The time tested Aspirin cures headaches 80% of the time, so that probability of cure by aspirin *p* is 0.8; obviously, probability of no relief with Aspirin q is 0.2. With the new drug, we have had 100 patients and tried our luck to see whether they are relieved. Its exactly the same as tossing a coin (the new drug) 100 times (on 100 patients) and assesing cure or no cure (just like heads or tails). 98 of them were cured, so our 'success' r is 98. If we *assume* the null hypothesis H_0 is true, then it follows that this new drug is no different from

Aspirin, and the number of times it cures is no different from Aspirin. So we can, in effect, apply the results of this drug to Aspirin and see what is the probability that Aspirin will cure 98 out of 100. We choose, from the many terms in the binomial expansion of $(p + q)n$ (see earlier), that particular term which matches our particular r which is 98.

$$^{100}C_{98}p^{98}q^{100-98}$$

which equals to

$$100! / 98!(100\text{-}98)! \times (0.8)^{98}(0.2)^2 = 6.302 \times 10^{-8}$$

What this means is that the probability of 98 out of 100 cures by Aspirin is so low that it cannot be even be expressed properly in decimals (and we have to resort to scientific notation), but this new drug has already done it. So we conclude that 'headgone' must actually be better.

Student's[26] test and the t distribution

Suppose in the mean of a population (or universe, as we'll call it) is μ. Each sample from this population has a different mean ($\bar{x}$) value. These means, if plotted are found to be normally distributed around the universal mean μ. If we take any number of samples (each containing *n* members[27]), then each sample will have its own mean $\bar{x}$. It is perfectly possible that we choose a sample from the lower end of the universe, i.e. only the shoe sizes between 6–8, so that we get a sample mean = 7. It is also possible that we get, in our sample, only the largest shoe sizes, giving a sample mean = 11. But if we go on taking random samples, chances are that we would encounter every shoe size in most of our samples. So the means of our samples could be any or all of 6, 6.5, 8, 8.5, 11.5 or any plausible value. If we go on taking infinite number of samples, we would, obviously, get an infinite number of sample means. The mean of these infinite number of means, as we will find out, will be equal or very close to the universal mean μ, and the sample means themselves will form a normal curve. This 'normal curve of the sample means' will, again, have the universal mean μ as its mean. This is the **central limit theorem**.

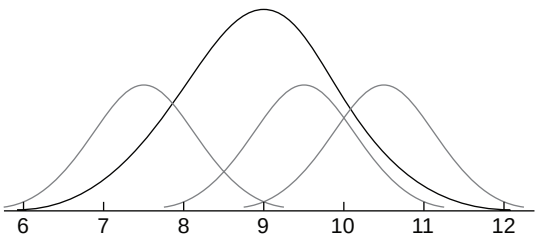


Figure 1.18. Normal curves of small samples taken from the population shown along with the universal curve

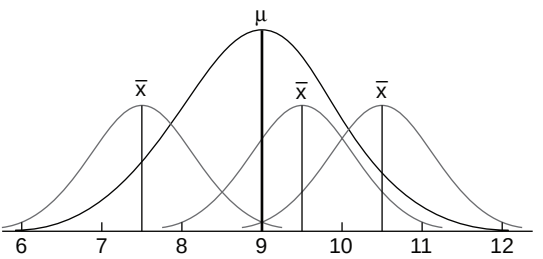


Figure 1.19. The means of the samples form a normal distribution around the universal mean

The same rules of normal curve apply these sample means also, i.e. if a sample has a mean outside, say 2 standard deviation about the universal mean $\bar{x}$, it has little (5%) chance of being selected (i.e. it is not a representative sample of the population).

The standard deviation of these sample means is called the **standard error of the mean**. This is equal to, as you see, SD of the original distribution/root of number of members in the sample ($\sigma/\sqrt{n}$). Thus there is also a relative deviate of these sample means (analogous to the z-score of a normal curve), which we denote by t.

t = (Mean of a particular sample—Mean of all sample means)/Standard error of the mean = $\frac{(\bar{x}-\mu)}{\frac{\sigma}{\sqrt{n}}}$

To determine whether a sample is representative, we need to know

1. The t-value of the sample.
2. The *degree of freedom*, which is one less than the number of members in the sample ($n - 1$).
3. Standard t distribution table.

For examples, lets say that the mean (μ) of shoe sizes in the entire class (universe) is 9, and standard deviation (σ) is 1. We take a sample of 21 (n) students and measure their shoe size, and the mean of this sample ($\bar{x}$) is 10.12. Now, the t-value of this sample is = $(\bar{x} - \mu)/(\sigma/\sqrt{n}) = (10.12 - 9)/(1/\sqrt{20}) = (1.12) / (1/4.472) = 5.006$

Table 1.6. The standard t distribution table in a very concise form

Degree of freedom	p = 0.5	p = 0.1	p = 0.05	p = 0.01
1	1.000	6.37	12.71	63.66
5	0.727	2.02	2.57	4.03
10	0.7	1.81	2.23	3.17
20	0.687	1.71	2.06	2.79
25	0.684	1.71	2.06	2.79
> 25	0.674	1.64	1.96	2.58

Find the t-value corresponding to the degree of freedom in this table, which matches **less than or equal to the value in our result** (i.e. 5.7). That particular t-value in this table is 2.79, which indicates a p-value of < 0.05 (see the top row of the table). Thus, the difference of shoe size in this particular sample from the average of the class is statistically significant.

Note that in this case, we take the whole population (universe) to compare with our sample. But the test could well be used to compare *two* samples, and the universe itself could serve as a sample. In fact, the **unpaired** t-test is most useful for comparing two samples. A second kind of t-test, the **paired** t-test, is useful for comparison of the *same* sample before and after an intervention.

The paired t-test

Suppose the mean shoe size in a class of 30 is 9.5. For a month, we apply growth hormone injections in each of the students and then measure their shoe sizes again.

This time, we find the mean show size to be 10.3. Does this indicate a statistical difference?

Like all statistical tests, we assume that there is, actually, no differences and the children grew up due to their natural growth spurt, not our hormone injections. To test this hypothesis, we must find the difference in shoe size of each individual student before and after injection, which we call *d*. Obviously, for 30 students, we will get 30 differences (d_1, d_2, d_3 ... d_{30}). The summation of these differences (Σd) divided by number of students (η) in this case 30, is the average difference or $\bar{d}$. The null hypothesis, states that the value of $\bar{d} = 0$ (i.e. there should no difference).

Now comes a crucial step. If these differences (d_1, d_2 are plotted, they will form a distribution among themselves. Some students will have grown much more than others (high difference) and some not much (low difference). We could, in theory, deduce of **standard deviation of differences**, which is

$$\sigma_d = \sqrt{\frac{\Sigma(d - \bar{d})^2}{\eta - 1}}$$

The t-value, in the paired t-test is

$$t = \frac{\bar{d}}{(\sigma_d / \sqrt{\eta})}$$

This t-value is checked, in the standard t table, for a degree of freedom $\eta - 1$, and a p-value could be deduced. Note the central theme in paired t testing is that we use the mean and standard error of *differences* before and after intervention, rather than using actual values.

The unpaired (independent) t-test

While comparing the shoe sizes of two classes, we assume that there is no difference in shoe sizes at all between the two classes (the null hypothesis). Because the two classes are nor paired (the same student cannot read in two classes) neither are the number of students the same in two classes, we cannot measure the standard deviation of differences directly, as we did in the paired t-test. Thus find the *pooled* standard deviation of two classes, which is

$$\sigma = \sqrt{\frac{\eta_1\sigma_1^2 + \eta_2\sigma_2^2}{\eta_1 - 1 + \eta_2 - 1}}$$

where η_1, η_2 are the number of students in two classes, respectively, and σ_1 and σ_2 are the standard deviations of shoe sizes in those classes. The t-value, in the unpaired t test, is

$$t = \frac{\bar{x}_1 - \bar{x}_2}{\sigma\sqrt{\frac{1}{\eta_1} + \frac{1}{\eta_2}}}$$

χ^2 test

It is the test applied where two groups of subjects, with differing characteristics, have to be tested for a common variable. They are first arranged in **Table 1.7**.

Table 1.7. Contingency table

	Acromegaly	Normal	Total
Shoe size > 11	a	b	a+b
Shoe size < 11	c	d	c+d
Total	a + c	b + d	

We assume that shoe size is no indicator of acromegaly (the **null hypothesis**). Then the ones with small shoes are expected to have the same rate of acromegaly as those with large shoes (and that should be the prevalence of acromegaly in whole population in general). The incidence of acromegaly in whole population is a+c/(a + b + c + d). Thus expected numbers in the four cells are calculated likewise, i.e. expected number in cell 'a' is total number of students with shoe size > 11 × incidence of acromegaly in whole population = (a + b) × (a + c)/(a + b + c + d). Similarly, expected number in cell b = Total number of students with shoe size < 11 × incidence of 'no acromegaly' (normal children) in community = (c + d)×(b + d)/(a + b + c + d).

χ^2 for each cell = (observed number - *expected number)*^{2}/*expected number*.

$$\chi^2 = \sum \frac{(0-E)^2}{E}$$

The summation of values of χ^2 is calculated. Now the *degree of freedom* of a table is (rows-1)(columns-1). The probability that matches the χ^2 value we just got and the degree of freedom in the χ^2 table, if less than 0.05, rejects the null hypothesis.

Table 1.8. The standard χ^2 table in a very very concise form

Degree of freedom	p = 0.99	p = 0.95	p = 0.05	p = 0.01	p = 0.001
1	0.000157	0.00393	3.841	6.63	10.83
2	0.0201	0.103	5.991	9.21	13.82
3	0.115	0.352	7.815	11.34	16.27

Analysis of variance and the F test

Similar to the t-test, the F test or ANOVA compares three or more samples. It is most useful when three or more groups have variable characteristics (like children with blood groups A, B, AB and O) and need to be compared over a common variable (whether blood group affects shoe size).

Pearson's correlation

The **correlation coefficient** is the degree of association between two variables, i.e. if number of observations is n,

$$r = \frac{1}{n} \frac{\Sigma(x-\bar{x})(y-\bar{y})}{\sigma_x \sigma_y} \quad (\sigma \text{ is standard deviation})$$

A positive r indicated that y increases with x, and the more nearer r becomes to 1, the relationship becomes almost linear. For example, suppose we measure

the shoe sizes and height of the students in a class and get the following graph (Fig. 1.20).

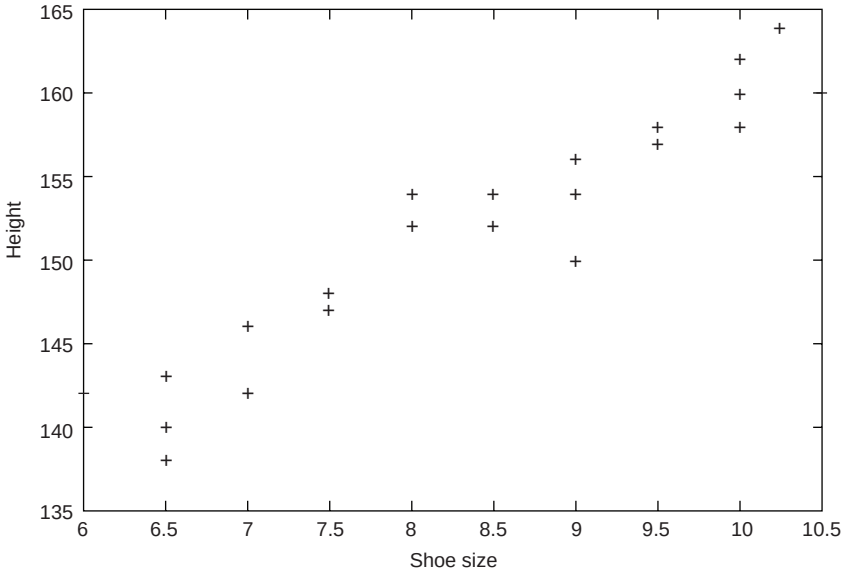


Figure 1.20. Scatter diagram showing relationship of shoe size and height in a class

Obviously, this indicates positive correlation (positive r) between shoe size and height. It is possible, if enough data is available, to predict the height from the shoe size of a student, a process known as **simple linear regression**. The **regression coefficient** b is the amount of change of y per unit change of x ($\delta y/\delta x$) which can be calculated from the correlation coefficient *r*

$$b = r\,(\sigma_y/\sigma_x)$$

The value of y for a certain x then becomes a matter of equation

$$y = a + bx$$

where a is the value of y when $x = 0$ (the intercept of the correlation line on the y-axis).

Spearman's rank correlation

If data is not measurable in an interval scale (as we are used to) but in an ordinal scale ('1st boy','2nd boy' or 'Grade I cancer','Grade II cancer', etc.), then the degree of correlation between two such variables (i.e. ranking of boys in a class and ranking of the same boys in annual sports—as to see whether boys good in studies are also good in athletes) is measured by Spearman's rank correlation coefficient ρ.

Poisson distribution

The Poisson distribution describes events that are *very* unlikely, or *very* random (like rate of disintegration of radioactive atoms). There are many other kinds of distributions, and more details of each, which are more appropriate in a textbook of statistics rather than this one.

2

Epidemiology

KEY FEATURES

- THE TWO FACES OF MEDICINE
  - Three common components
  - Aims of epidemiology
  - Sources of data
  - What epidemiologists measure
  - Descriptive epidemiology
  - Analytic epidemiology
  - Experimental epidemiology
  - Association
  - Uses of epidemiology
  - Epidemiologic triangle
- SCREENING
  - Types of screening

THE TWO FACES OF MEDICINE

But absence of evidence is not the same as evidence of absence

—Anonymous

There are two faces of medicine

- **Internal** medicine is confined to a single person at a time;it describes four attributes of a disease—Its cause, clinical course, methods of diagnosis and treatment.
- **Community** medicine describes the same diseases from the perspective of *many* people, or society; unlike internal medicine, it describes only a single key attribute of a disease—Its epidemiology, i.e. to whom, where and when the disease happens.

Epidemiology (Greek 'epi' – over, 'demos' – people) has been defined as

The study of *distribution* and *determinants* of health related states and events in specified populations and *application* of this study to control health problems.

—John M Last

Mind Mr Last here. He says *distribution*, which means to who get the disease, and where and when they get it. He says *determinants*, i.e. what made the victims susceptible to a disease. He says *health related states and events*, as not all events qualify for a disease (i.e. a change in average height of men in an area due to better nutrition). And lastly, he wants to *apply* his knowledge so that diseases can be prevented.

Three common components (Fig. 2.1)

1. Study of disease frequency – Rates ratio, proportions
2. Study of distribution of disease – Time, place, person
3. Study of determinants of disease – Exposures.

Aims of epidemiology

1. To describe the distribution and magnitude of health and disease problems in human population.
2. To identify etiological factors in the pathogenesis of disease (does).
3. To provide the data essential to the planning, implementation and evaluation of health services and to set up priorities between services (i.e. you don't want immunization for Rocky Mountain Spotted Fever in India).

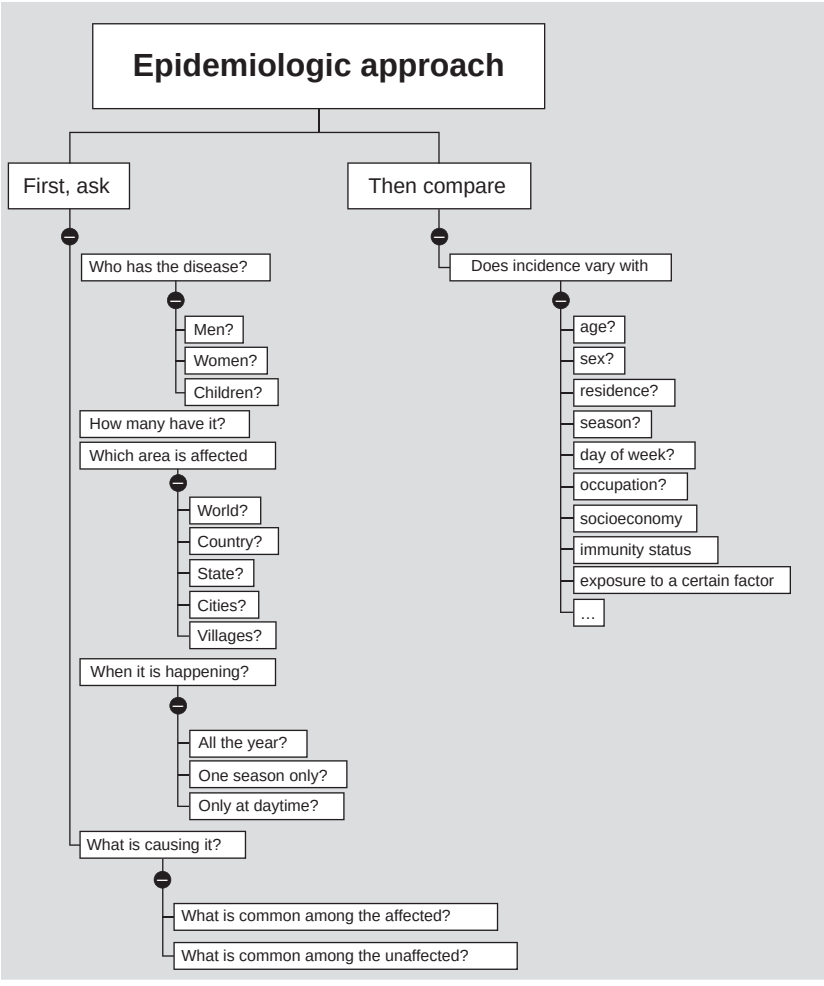


Figure 2.1. The epidemiologic approach

Sources of data

1. A **census** is collection of data from every member of a population; theoretically it should provide the most reliable data. In plainspeak, by 'census' we refer to the demographic data collection that occur every 10 years in India.
2. Registration of vital events—Birth, death and marriage.
3. Sample registration system—See the chapter on health information.
4. National sample survey.
5. Hospital/health center records.
6. Disease registers, i.e. The Revised National Tuberculosis Control Program maintains a Tuberculosis Register in each DOTS center.
7. Epidemiologic studies.

What epidemiologists measure

Tools

Rate ('/')

It is a fraction of two different variables where the numerator is a subset of the denominator (i.e. crude death rate = total deaths/mid year population; total deaths are a subset of mid year opulation, only those people who have *lived* this year can *die* this year). The importance of rate is that it gives how fast an event is happening.

Ratio (':')

It is the fraction of two different and independent variables, i.e. no one is a subset of the other). When we say the ratio of deaths due to TB: leprosy = 2:1, we assume that the person who has died of leprosy *cannot* die again of tuberculosis.

Proportion ('%')

It is a fraction of two variables, both of which measure the *same physical quantity*, and the numerator is a subset of the denominator. Think about it very carefully. When we say proportional mortality due to tuberculosis = 1%, we mean deaths due to tuberculosis/total deaths = 1/100. Note that *both the numerator and the denominator in this equation measure the same thing, i.e. death.* In case of rate, however, the numerator and denominator usually measure different things. In crude death rate, the numerator measures *deaths*, while the denominator measures *population.*

Suitable denominators

All the three above tools require a suitable denominator. Following is a list of standard denominators to be used.

1. **Mid year population** = population on 1st July; this is taken as a 'mean' population of the year, because the actual population will be increasing throughout the year.
2. **Population at risk** = people who are exposed to a factor, i.e. those who have ate from same kitchen, and are still susceptible to a disease; if a person has prior measles, he can't be included in the population at risk for measles.

3. **Person-time** = ∑ the time each person has attended a study. If 2 persons attend a study for 3 years and 3 persons for 4 years, the total person-time is = 2 × 3 + 3 × 4 = 18 person years. But there is a GREAT DISADVANTAGE in this method – 10 person × 10 years = 100 person years = 100 person × 1 year. This means, a study of 10 people followed up for 10 years equates, in person time, a study on hundred people followed up for one year. Obviously, many chronic diseases will not show up in this one year so in spite of studying 100 person years, you may not be able to find any disease.
4. **Event related denominators** are picked suitable for the situation, i.e. infant mortality rate should be expressed as infant deaths/live born infants, not infant deaths/whole population.

Death

Importance of death rate

1. Death has an unambiguous definition, diagnosis is seldom wrong.
2. For very active and **rapidly fatal** diseases**, disease specific death rate ≅ incidence of disease.** Here death rate is a **surrogate indicator** of incidence.

WHO protocol for death certificate

It should state,

1. Immediate cause of death (but not the mode of death, i.e. heart failure, asphyxia, coma, etc.) – like hypoglycemic shock.
2. Underlying cause if any, like diabetes.
3. Associated significant disease, if any.

Crude death rate (cDR)

cDR = (number of deaths during the year/mid year population) × 1000. It is important to note that

1. cDR does not reflect variations due to composition of population. In a community with mostly older people, the cDR will be high.
2. Many diseases do not result in death. In Hiroshima, many children are still born with absence of limbs, etc. cDR will not reflect this.
3. In our country, many deaths go unregistered.

Specific death rates

If there is a 'crude' death rate, like crude oil, it follows that there must also be some 'refined' death rates. **Specific death rate** cover the shortcomings of cDR. They may be

1. Cause specific, i.e. tuberculosis specific death rate = deaths due to tuberculosis/mid year population.
2. Attribute or variable specific (deaths in particular age group age, in either sex, in a particular stratum of income, etc.).

The *cause specific* death rate has one disadvantage. The 'cause' of death is very frequently misinterpreted, or the mode of death is written instead (i.e. 'heart failure' instead of 'myocardial infarction').

Case fatality proportion

It is the total cases of a disease who have died/total cases of the disease, i.e. the 'killing power' of the disease.

Proportional mortality

1. Proportional mortality from a disease = (deaths due to a disease/total deaths) × 100
2. Age specific proportional mortality = (deaths in that age group/total deaths) × 100.

Survival proportion

It is the proportion of survivors in a group (suffering from a disease) over a period of time. It is used especially in prognosis of cancers.

Standardized death rates

Because death rates can vary greatly due to composition of population, it is customary to use 'standardized' death rate while comparing two communities. The 'standard' here is arbitrary, selected only for comparison.

Direct standardization

1. Measure death rates in two populations.
2. Select a 'standard' population (i.e. a population which you know details of composition, like the population of India)
3. Apply the death rates of the 'real' population (that which you are studying) to this 'standard' population to get the 'expected deaths' in the 'standard population' (i.e. what would have been the number of deaths in the standard population if it suffered from the same rate of mortality as your study population).

Age	Mid year popl in study	Mid year popl in 'standard'	Deaths in year	Rate/1000	Expected deaths (i.e. standard population × death rate)
0–12	4500	9600	20	4.4	42.24
13–30	4000	19000	12	3.0	57
31–60	5000	9000	15	3.0	27
>60	7000	8000	150	21.4	171.2
TOTAL	25000	45600	197	7.8	297.44

So the standardized death rate = 'expected' death/1000 population = 297.44/45600 × 1000 = 6.52, which is a lot less than the 'real' death rate 7.8. This is due to the prevalent >60 population in the study population, which contribute to the substantially high death rate. After standardization, the figure is closer to real, and comparable to other such standardized rates.

Fallacy. Direct standardization needs the age specific death rates of the study population. But if we knew that, we would not be doing standardization in the first place.

Indirect standardization

The **Standardized Mortality Ratio (SMR)** is the ratio of 'real' deaths in a community/'expected' deaths derived from death rates a standard, larger community (i.e. the whole country). Suppose a village is affected seriously by arsenic in water, and the following are the figures.

Age	National death rate/1000	Population in village	Deaths in village/1000	Expected deaths (village popl. X national death rate)
0–12	3	300	Unknown	0.9
13–30	5	400	Unknown	2
31–60	8	200	Unknown	1.6
>60	25	100	Unknown	2.5
TOTAL		1000	9	7

The SMR is 9/7 × 100 =129. This SMR >100 means that this village has more mortality than the nation. The advantage of indirect standardization is that you do not have to know the age specific deaths in the community.

Morbidity

What is a 'case'?

Measuring disease frequency in populations requires the stipulation of **precise** diagnostic criteria. Doctors generally assume that for any disease, people can be classfied as 'those who have it' and 'those who donot have it'. However, today we recognize the diagnostic importance of subclinical infections, carrier states, premalignant dysplasias, in situ carcinoma, mild hypertension, and presymptomatic airways obstruction. It appears that disease in populations exists as a *continuum* (recall the pendulum from the first chapter) of severity rather than as an 'all or none' phenomenon. The rare exceptions are mainly genetic disorders like Gaucher disease, one either has it or one does not (however, there are also carrier states for genetic diseases like hemophilia).

Clinicians' definition of a case is wide and often, blurry, because of this reason. The definitions in textbooks of medicine often use subjective terms like 'very severe disease','rat tail appearance','mutton fat keratic precipitates'. These have no place in a textbook of epidemiology. The epidemiologist needs **exact definitions.** There are three methods to devise exact case definitions.

1. **Statistical**—"Normal" may be defined as being within two standard deviations of the age specific mean. However, 5% of values will always fall outside two standard deviations and special statistical test need to be used to see whether they are 'really' abnormal. More importantly, what is usual is not necessarily good.
2. **Clinical**— Clinical importance may be defined by the level of a variable above which symptoms and complications become more frequent (i.e. a researcher may find that atherosclerosis accelerates if serum cholesterol > 200 mg/dl, and term the case as "hypercholesterolemia").

3. **Prognostic**—Some clinical findings such as high systolic blood pressure or poor glucose tolerance may be symptomless and yet carry an adverse prognosis. Thus impaired glucose tolerance itself is a disease, even if it is not causing any symptoms.

To diagnose a person as a *case*, an ordinary interview is not enough. Two persons with rheumatoid arthritis might have very different feelings about 'morning stiffness', and one could deny having it at all, depending on the interviewing technique. A standardized **questionnaire** is an essential element in the epidemiologist's toolbox.

Indicators of morbidity (illness) fall in three groups,

1. Frequency of disease—Incidence and prevalence.
2. Duration of disease—Mean duration.
3. Severity of disease/killing power—Case fatality.

Incidence rate

It is the **number of NEW cases during a given period/POPULATION AT RISK during that period.**

Incidence is inclusive, i.e. one person suffering from two bouts of cold in the same year constitutes two 'incidences'. Also, if the population at risk has changed during the period, go for a 'mean' population.

Sometimes measurement of incidence is complicated by changes in the population at risk during the period when cases are ascertained, for example, through births, deaths, or migrations. To circumvent this problem, add the period of time each person has spent being one of population at risk. The summation of these person-times then becomes the denominator for incidence.

$$\text{Incidence} = \text{number of new cases} / \Sigma(\text{person} \times \text{time})$$

Incidence density is the rate of new cases appearing. It uses person-time as denominator.

Attack rate. It is the same as incidence in a limited period, used to describe an epidemic. It is expressed as %.

Secondary attack rate = *number of cases who develop disease within one maximum incubation period of the disease/number of persons actually at risk and exposed to the initial case* × 100. The SAR is the determinant of the communicability of a disease, and also shows effectiveness of a health program.

Incidence of a disease may be lowered by

1. Loss of virulence of agent.
2. Enhanced host resistance.
3. Control programs.

Incidence comes of use in

1. Epidemiologic studies—INCIDENCE is more related to etiology than prevalence; if suddenly the HIV mutates to a benign form, the prevalence will be unchanged until existing HIV patients die, but the incidence will be zero immediately.
2. Incidence of a disease is the probability that a person of that community will have that disease, i.e. incidence is the **absolute risk** of falling ill (c.f. relative risk – see later).

3. Evaluating health service (if incidence is increasing, services are failing).
4. Sudden rise of incidence may mean CHANGE OF EPIDEMIOLOGIC TRIANGLE (either the agent has become more malignant, or there has been a sudden environmental change that is facilitating the agent, or the hosts have lost resistance) and an epidemic is likely to follow.

Prevalence ratio

Prevalence is the (total number of cases (old and new) at a point of time or during a period/population at risk (point population or mid interval population), usually expressed as %. Prevalence is related both to ETIOLOGY and PROGNOSIS.

Even in a chronic disease, the manifestations are often intermittent. In consequence, a "point" prevalence, based on a single examination, at one point in time, tends to underestimate the condition's total frequency. If repeated or continuous assessments of the same individuals are possible, a better measure is the period prevalence defined as the proportion of a population that are cases at anytime within a stated period.[28]

From common sense, if the incidence and population is stable, PREVALENCE = INCIDENCE × MEAN DURATION OF ILLNESS.

Prevalence is increased by:
1. Increase in incidence.
2. Increased in duration of illness (i.e. any treatment that only prolongs life but does not cure will only increase prevalence) – thus prevalence is also high in chronic diseases.

Prevalence is lowered in:
1. Rapidly fatal diseases (accidents, homicides and food poisoning, etc. have no prevalence).
2. Diseases which are cured fast.

Importance of prevalence. Prevalence is a snapshot of a disease in a community and gives
1. Magnitude of the disease.
2. How much of health service is needed (i.e. how many hospital beds needed in a particular area).
3. Prevalence is the probability that a person in an area has a disease (i.e. if prevalence of tuberculosis in Kolkata is 15%, the probability that anyone person has tuberculosis is 0.15).
4. If that person tests for tuberculosis and is found positive, what is the probability that the test is correct? In this case, the probability that the person has the disease is determined not only by the sensitivity and specificity of the test, *but also the prevalence* of tuberculosis. Testing for a certain disease is *more reliable* if the prevalence of the disease is high.
5. Prevalence is often used as an alternative to incidence in the study of rarer chronic diseases such as multiple sclerosis, where it would be difficult to accumulate large numbers of incident cases.

Descriptive epidemiology

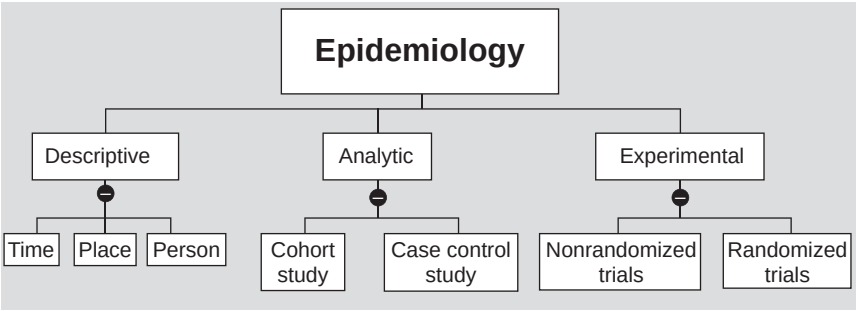


Figure 2.2. Types of epidemiology

The *descriptive* part of epidemiology describes the state of a disease in terms of time, place and person and does not ask *why*.

1. **Cross sectional studies** give a still photograph at a particular moment. It gives only prevalence of a disease, not incidence.
2. **Longitudinal studies** (through follow-up of study population overtime) shows a incidence, but needs more resources to be carried out.

Time distribution

Short-term fluctuations.

Epidemics

An **epidemic** is occurrence of a disease/health related behavior/event in a community or a region clearly above *expectancy*. The *expectancy* of a disease (i.e. those number of cases we *expect* the disease to produce each year) is defined as the mean incidence in last three years plus twice its standard error.

1. **Point source (common source) epidemics (Fig. 2.3)**—An explosive outburst of a disease within a very short period, and the source is usually a single, identifiable entity. Examples are chickenpox in a hostel (source = a boarder), food poisoning at a dinner (source = a contaminated item), toxic shock syndrome

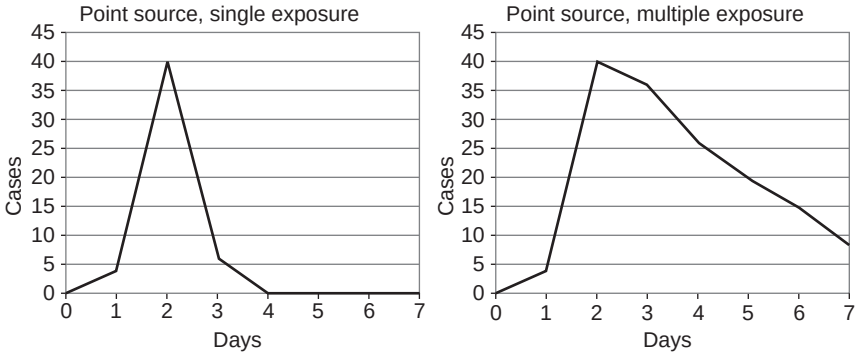


Figure 2.3. Point source epidemic

in many children after vaccination (source = a vial of contaminated measles vaccine).

2. **Propagated epidemic (Fig. 2.4)**—It is propagated from person to person, thus the initial rise in number of cases is not very high, but cases build up gradually over long time and tail off slowly. Examples are tuberculosis and typhoid fever.

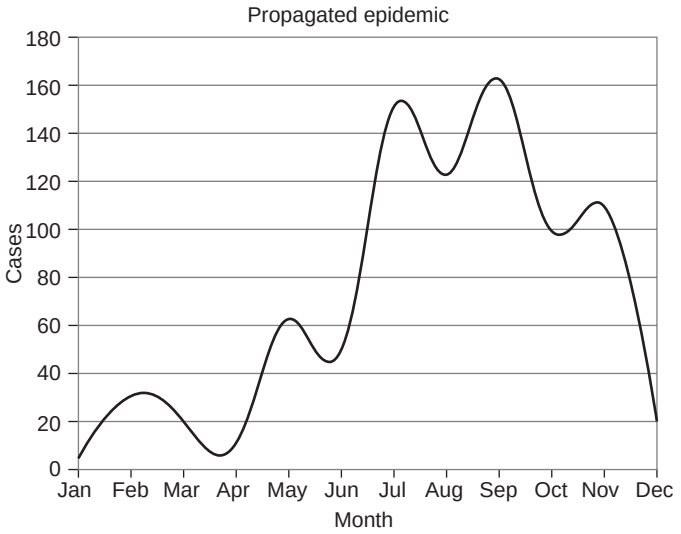


Figure 2.4. Propagated epidemic

Endemic

An **endemic** is a *habitual* presence of a disease in a given geographical area without flux of new cases from outside. Syphilis, cholera, leprosy, tuberculosis are some of the many endemic diseases in India. A *hyperendemic* is one which is prevalent in all age groups and a *holoendemic* is prevalent only in children.

Pandemics

A pandemic is an epidemic affecting large population in a wide geographical area (i.e. the H_1N_1 influenza).

Periodic fluctuations

1. **Seasonal trends**—Summer: diarrhea, rain: poliomyelitis, fall (just after monsoon): cholera, winter: acute respiratory infections, spring: measles and mumps.
2. **Cyclic trends**—A few diseases come back with new vengeance every once in a while (measles: every 2–3 years, rubella: 6–9 years, influenza pandemic: 7–10 years, accidents: every weekend).

Long-term Fluctuations

Slow epidemics do not spread from person to person, but changes in socioeconomic status and lifestyle causes rising incidence of these diseases (diabetes, ischemic heart diseases, drug abuse).

Place distribution

International

People living in different places suffer from different ailments. The African continent houses the largest number of HIV patients. There is no rabies in island countries (UK, Australia) for the simple reason that dogs can't swim. The commonest cancer in women worldwide is breast cancer, but in India cervical cancer tops.

National

Parts of the same country have diseases that differ. Filariasis and vitamin deficiency is very common in south India, kala-azar and malaria in eastern India. Variations due to place of residence can usually be attributed to some other root cause, i.e. the dominance of malaria in north-eastern states due to very high rainfall and growth of mosquitoes.

Rural/urban variations

1. Predominantly 'urban' diseases—COPD, accidents, carcinoma lung, mental illnesses
2. Predominantly 'rural' disease—Helminth infestations, organophosphate poisoning.

Local

Spot maps show distribution of a disease in a locality (Fig. 2.5).

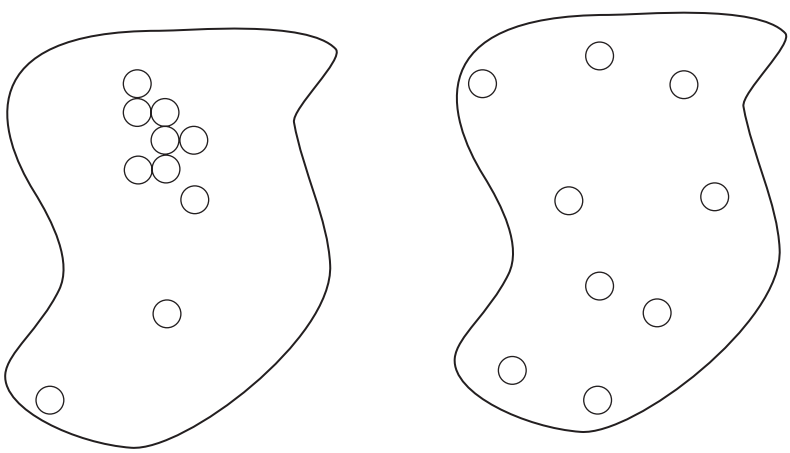


Figure 2.5. Spot maps

Person distribution

People with certain characteristics are prone to some diseases, and resistant to others. The *characteristics* are of two kinds:

1. An **attribute** is a quality of a person which is constant, i.e. sex, race. The attribute of having sickle cell disease gives protection from malaria.
2. A **variable** is a quality of a person which may change, i.e. age, behavior, martial status, migration, etc. Increasing age is a risk factor for cancer, ischemic heart disease and dementia.

Analytic epidemiology

Analytical studies are used to determine whether or not a statistical association exists between a disease and a suspected factor, and if it exists, the *strength* of the association.

Table 2.1. The 2 x 2 table

	Diseased	Healthy
Factor present	a	b
Factor absent	c	d

1. Retrospective/Case control studies begin with a group of cases (patients) and controls (normal) and finds the incidence of exposure in them in past.
2. Prospective/Cohort studies begin with a cohort (group of individuals who share common characteristics and experience within a defined period), classifies them according to exposure and finds out the incidence of disease in both groups (Table 2.1).

Case control study

Objective

To determine the cause retrospectively from effect (Cause ← Effect)

Steps

Hypothesis (formed from descriptive study)

↓

Selection of case and controls

Cases should be selected according to fixed diagnostic and eligibility criteria. Cases and controls have to be matched

↓

Data collection

The proforma for collection should be pretested for errors. Also, the procedure should be standardized and unbiased

↓

Analysis and Interpretation

Exposure rates among cases and controls are to be found and from that, disease risk is to be derived.

Variables, attributes and matching

Suppose we have a hypothesis "OCPs cause cervical cancer." To test it, we choose a case group of 20 women of 50 years age, and a control group of 20 women 30

years of age. What will we find? Evidently, at 50 years, many women will develop cervical cancer irrespective of intake of OCPs – while at 30 years, most women don't develop cervical cancer. We will find an association *stronger* than the true situation between OCPs and cervical cancer.

What is the problem here? *Age* is a variable that is affecting both the intake of OCP (because only those aged 50 are taking OCPs in our study), and cervical cancer, i.e. it is influencing both exposure and effect. Such a variable is called a **confounding** variable, and to eliminate it we have to go for matching.

Matching. Matching is a process in which controls are selected in such a way that they are more or less *identical with the cases* in all respects, except that they do not have the diseases. It is never possible to get a full match, but matching gives more objectivity to the study.

1. Group matching: Two groups with identical characteristics are chosen.
2. Pair matching: For each case selected, a control is chosen with same attributes and variables.

Some variables affect only exposure, i.e. Muslims usually do not use OCPs. Here, the variable 'religion' is influencing intake of OCP. To take a control group of Muslims in the previous study will be a misjudgement. Again, some variables/attributes affect only the *occurrence* of a disease, not the exposure (i.e. obesity is an independent risk factor for IHD; it can create problems in a cohort study on 'effect of smoking on CHD'; those who are smokers + obese will develop more IHD than only smokers).

Too much matching

1. Makes difficult to find controls
2. Reduces Odds' ratio.

Bias

A bias is "any systematic *nonrandom* error in the design, conduct, or analysis of a study that results in a mistaken estimate of an exposure's of subject on the risk of disease." Mind the terms well. Bias is *systematic*, i.e. not due an error in data collection. It is also *nonrandom*, i.e. not a sampling error (see the chapter on statistics). It *could* have been avoided by good study design (but sampling errors can't be avoided by design, because no sample is identical to the population.

Confounding bias. This, as already mentioned, can be eliminated by matching or using standardized parameters (like standardized mortality ratio).

Selection bias. Selection bias hits you if you have chosen a distorted sample. For example, if you choose a slum to study the prevalence of tuberculosis, you'll come to the conclusion that tuberculosis is only a disease of poor people, which it is actually not.

- Admission rate (Berksonian) bias—In a hospital based study, you tend to get cases with the risk factors rather than controls with risk factor; again, people with a disease are less likely to get admission than those with a comorbid illness
- Lead time bias—If a modern test is able to detect a cancer very early in its course (time A) and the clinical signs appear much later (time B), then B-A is

said to be the **lead time**. Because the cancer was detected a lot earlier, we may falsely assume the patient to be surviving longer than usual.

Information bias

- Recall bias: People who have suffered more of something recall it more easily.
- Interviewer bias: This in plain terms means asking more questions to cases than controls. This can be eliminated by blinding method (single/double/triple).
- Unacceptibility bias: Subjects will often give 'desirable' answers to please the interviewer.

Other subtle kinds of bias. More problematic are those kind of bias that are 'cognitive'. A gastroenterologist who has a special interest in Crohn's disease may be referred patients whose cases are unusual or difficult, the clinical course and complications of which are atypical of the disease. If he choses to do a study on Crohn's disease, his results are likely to be distorted. Such systematic errors cannot usually be measured, and assessment therefore becomes a matter for subjective judgement.

Bias cannot usually be totally eliminated from epidemiological studies. The aim, therefore, must be to keep it to a minimum, and assess their potential effect on the results. The motto of the epidemiologist: "dirty hands but a clean mind" (*manus sordidae, mens pura*).

Calculation of exposure rates

Rate of exposure in cases and controls, as you have learnt from the previous table, is respectively $a/(a+c)$ and $b/(b+d)$. IF $a/(a+c) > b/(b+d)$ then a statistical association may be suspected. The strength of this suspicion can be calculated by a χ^2 test.

Estimation of risk

1. **Relative risk**: It is (incidence of disease in exposed/incidence of disease in nonexposed). It denotes the *strength* of the association. Because we actually have no estimate of incidence in a case control study (we do not know if these cases are new cases, or for how long they have been diseased), **relative risk can NOT be calculated from case control studies.**
2. **Odds' ratio**: (see also the chapter on statistics) It is ad/bc, and gives the strength of association between exposure and disease in a case control study. It is the closest estimation of relative risk. The numerator is persons who have developed lung carcinoma for smoking × persons who have not developed lung carcinoma due to avoidance of smoking. The numerator is essentially an advertise to stop smoking. The denominator assembles all the opposite things, i.e. persons who have not developed lung carcinoma even on smoking × persons who have developed lung carcinoma in spite of avoidance to smoking. It is the ratio of two 'odds', i.e. the odds of developing lung carcinoma due to smoking, and the odds of not developing such.

Cohort study

Objective

Cause → Effect

Steps

Hypothesis
↓
Selection of study and comparison group
The same processes of diagnostics, eligibility and matching are required
↓
Follow up and data collection
The cohort should be stable, accessible and cooperative
↓
Analysis and interpretation
Incidence of outcome in study and comparison group, also risk is to be estimated

Estimation of risk

1. **Relative risk**: It is $\{a/(a+b)\} / \{c/(c+d)\}$. It gives the strength of association and needed for etiological enquiries. This is evident, because RR = 1 means essentially no risk, RR > 1 is some risk and RR < 1 is reverse risk (i.e. you should then *start* smoking to avoid lung cancer) A relative risk = 2 means smokers are actually twice as likely to develop lung cancer as nonsmokers.
2. **Attributable risk**: It is $\{a/(a+b) - c/(c+d)\}/ a/(a+b)$.
   It is the **amount of risk among the exposed that can be erased by removal of factor**. It is of potential public health importance. The reason is clear: suppose c persons have developed lung cancer in due course, without smoking. So we may conclude that $c/(c+d)$ is the natural incidence of disease. This has to be subtracted from $a/(a+b)$ to get the incidence of cancer exclusively due to smoking (because not all smokers develop lung carcinoma due to smoking, but other causes too!). It gives the potential *impact* of a factor.

$$\frac{\text{Incidence in exposed} - \text{Incidence in nonexposed}}{\text{Incidence in exposed}} = 1 - \frac{1}{RR}$$

We measure attributable risk, not relative risk, in our daily lives. For example, in deciding whether or not to indulge in a dangerous sport such as rock climbing, it is the attributable risk of injury which must be weighed against the pleasures of participation. It is not necessary that the 'non exposed' be exposed to no risk factor. We could deduce attributable risk of smoking 20 cigarettes a day against 10 a day. Here, we assume 10 cigarettes a day to be 'no exposure'. Relative risk is less relevant to making decisions in risk management than is attributable risk. For example, given a choice between a doubling in their risk of death from bronchial carcinoma and a doubling in their risk of death from oral cancer (i.e. which one they feel safer, smoking or chewing tobacco), most informed people would opt for the latter. The relative risk is the same (two), but the corresponding attributable risk is lower because oral cancer is a rarer disease.

3. **Population attributable risk**: It is the excess risk in population solely attributable to a particular risk factor. It is given by

   PAR = (Incidence in total population – incidence in nonexposed)/Incidence in total population

   Suppose, in any population of size x, the percentage of smokers is 60%. We conduct a cohort study in a sample taken from this population. Suppose also, that in our study, incidence in exposed = m and incidence in nonexposed = n; thus we may extend these results to the whole population as follows.

   Incidence in whole population = 0.6m + 0.4n = Z (say)

   The PAR, in this case = (Z – 0.4n) / Z

Comparison of Case Control and Cohort Studies

	Advantage	Disadvantage
Case control	Inexpensive (you just need a calculator, no need to chase your cases for prolonged period) Quick results (no follow up over years required) Suitable for rare diseases (if you do a cohort study on xeroderma pigmentosum, wonder how many of your cohort will actually develop the disease? It is better to start with cases themselves. Multiple risk factor may be studied (Cohort studies do not allow this; if you start with multiple risk factors in a Cohort study, you'll be left wondering which one has finally caused the disease)	Incomplete information (because Case Control studies are retrospective, you won't know when and at what rate these cases developed the diseases, i.e. **incidence** is not deduced) Recall bias Problem of matching (matching is much easier in Cohort studies, when you have the selection in your hands; but in case control studies, you need *cases* and they are often very dissimilar with control, but you can't exclude them because your study population will then become smaller; its hard to find good cases anyway) Relative risk not found (because incidence can't be deduced)
Cohort study	No bias of recall/interview (actually, there is very little requirement for an interview; only thing to note is the development of the disease) Yields both relative and attributable risk Can yield association with other diseases as by product (i.e. you start with smokers to study lung cancer and they start to develop bladder cancer instead; it will still make a good study)	Large no, of subjects required (you do not know how many of them are actually going to develop the disease, so you must include as many as possible) Long follow-up Attrition of population (loss of subjects) Change of criteria overtime (i.e. if during your follow-up years, the very definition of lung cancer changes, you are sure to have a mix-up) Expensive (due to the follow-up)

Retrospective Cohort studies. Cohort studies are difficult to conduct for chronic diseases. One approach that can help to counter this problem is to carry out the follow-up **retrospectively**. In developing ideas about the fetal origins of coronary heart disease, it was possible to find groups of men and women born in the country of Hertfordshire before 1930 whose fetal and infant growth had been documented. These people were traced, and the cause of death was ascertained for those who had died. Death rates from coronary heart disease could thus be related to weight at birth and at one year old.[29]

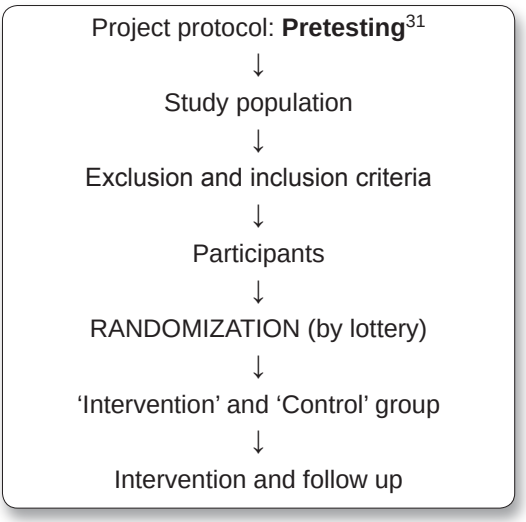
Experimental epidemiology

Experimental studies try to *intervene* in the natural course of things and observe the change, if any. Experiments are carried out by every person, sometimes subconsciously, every moment of life. The progression of human civilisation (as we chose to call it) has been paved by experiments, both successful and failed.[30] Let us spare a few moments on the *design* of experiments. Because experiments are only second nature to humans (we are insanely curious) and will be the groundstone for scientific development for many years to come, it is imperative that we have a framework for conducting *good* experiments that will yield valid results. Grossly there are two kinds of experiments.

- Those experiments where there is only an 'intervention' group and no 'control' group. Suppose I feed a few rats day and night with pizza and observe that they are growing obese. Should I conclude that pizza makes rats obese? It is equally possible that the very rats I am studying are genetically predisposed to being fat, and that they are on all pizza diet a sheer coincidence. Such studies are not useful for epidemiologic purposes. They are, however, useful in the basic sciences (physics, chemistry). If I synthesize a new fertilizer from reaction of two earlier chemical compounds, I do not need to show any 'control' compound where the reaction was *not* done. The discovery will speak for itself.
- Those experiments where there are two groups, 'intervention' and 'control'. The control group is given no intervention. These studies are useful in epidemiology, but *selection of controls* is pivotally important. Suppose I feed a group of rats with pizza (the 'intervention' group) and give another group vegetables to eat (the 'control' group). It may so happen that the control group is genetically prone to be fat, and the intervention group has malabsorption disease (it is a wild proposition, but real life situations can get close). Then I will find that eating pizza makes rats *thinner*. To avoid such deductions, the cases and controls must be identical in all respect *except* that one group will have been intervened, and other not. Because prior knowledge of *every* attribute and variable of each member is impossible, we have depend upon chance and draw lotteries (literally) to place a member into one of the two groups. This way we avoid clustering of any attribute in either group. Now, rats which have malabsorption have equal chance to be picked in either the intervention or control group, so the net effect due to the attribute 'malabsorption' gets nullified. This process is known as **randomization.**

The randomized control trial

The key concept in such a trial is that after you have got your participants, you have to *randomize* them, i.e. distribute them in such a way that everyone has equal chance of being either in treatment or control group.



Sources of bias and how to cancel them

It is human nature to favor the 'intervention' group rathern than the 'control' group if there is prior knowledge. Quite unaware of ourselves, we feel better on taking drugs, even if it was no drug at all. Not only the subjects, but even the investigators frequently fall prey to this kind of misjudgement. In a subconscious effort to justify the intervention, the investigator will usually put an extra tick mark or give a few more points to those who have received the intervention. This kind of bias can be eliminated by the following methods.

1. **Single blinding**—Participants must not know what group they belong to (i.e. they are unaware of what is being given to them, a drug or plain saline)
2. **Double blinding**—Investigator should also be unaware of that
3. **Triple blinding**—Lastly, the one who analyses the data should also be unaware; he only gets aggregate figures (i.e. "247 rats were given pizzas and 121 of them showed weight gain") and not raw data of individual members (which should remain with the investigator).

Types of randomized control trials

1. Drug trials, which are necessary to introduce a new drug
2. Preventive trials for new vaccines
3. Risk factor trials
4. Cessation experiments—Whether health status improves after 'cessation' of a particularly bad habit (like smoking)
5. Trial of a suspected agent
6. Evaluation of health services—Whether a new intervention improves the health status of the community.

Nonrandomized trials

1. Uncontrolled trials—Where there is no control group; no control group was ever used for testing the diagnosis of cervical cancer but PAP smear (i.e. it was never checked whether omission of PAP smear changed the survival of a group of patients), but it has proved to be effective.
2. Disasters, war and emergencies provide a scope for natural experiments. The London Cholera epidemic allowed John Snow to identify water as a vector of cholera. The effects of the nuclear bombs in Japan allows studies on late effects of radiation.
3. Before and after studies are best known with the Sabin vaccine.

Association

An association between two events may be

- Spurious—Often arising due to bias; any study anywhere will find that more than 95% of people die while lying on a bed; it will be very wrong to conclude that lying down on a bed is fatal
- Indirect—A study in any village of India will show that the height of a person is inversely related to the length of hair; this is an indirect association because women are usually shorter and they have longer hair in general
- Direct.

Direct association may be

1. One to one
2. Multifactorial.

Association is not causation. To establish causation, one must prove the following (Bradford Hill criteria)

1. The cause actually precedes the effect (**temporal** association)
2. An relative risk > 1, a significant Odds' ratio or a dose-response relationship (**strength** of association)
3. To show that the effect is almost missing where the cause is absent or is caused by other factors (**specificity** of association)
4. To show that the cause-effect course takes place time and again (**consistency**)
5. If possible, to show some scientific reasoning behind it (biological plausibility)
6. It should (not *must*) conform to common sense (**coherence**).

Uses of epidemiology

1. To describe magnitudes of disease
2. To know causation of disease
3. To know natural history of a disease
4. Description of health status in population
5. Health planning and identifying priorities
6. Evaluation of intervention.

Epidemiologic triangle

A disease is a culmination of a joint venture by three entities – agent, host and environment (the Epidemiologic triangle). The following section describes each in detail.

Agent

Agents may be

1. Living beings (virus, bacteria, protozoa, fungi, helminthes, algae…) – or more precisely, the proteins that they bear
2. Molecules (drugs/poisons – like aspirin, OCPs, snake venom, nerve gases, alcohol , lipids, autoantibodies, etc.)
3. Waves (UV-ray, γ -ray, sound, X-ray, etc.).

Host

A host is an organism that affords substinance of an agent.

Hosts for living agents

1. **Definitive host**: It is the host which harbors the adult stage of the parasite, i.e. the period of sexual reproduction. Man is the definitive host in many cases, except for malaria and hydatid disease.
2. **Intermediate host**: It harbors the young/larval form of the parasite. A sexual reproduction takes place within this host.
3. **Reservoir host**: These are infested animals that make parasites available for transmission to other hosts. Man is a reservoir host of *Leishmania donovani* (Kala-azar) and *Entamoeba histolytica* (Amebiasis).
4. **Natural host**: Humans are natural hosts for *Balantidium coli*, but it is a rare infection.
5. **Accidental host**: Humans may sometimes be accidental hosts for hydatid disease.
6. **Paratenic hosts**: A host that harbors a parasite but shows no development of the parasite is called a paratenic host.

Environment

The issue of environment and health has been dealt in a later chapter.

SCREENING

Screening is the examination of people who are asymptomatic in order to classify them as likely or unlikely to have a disease. Screening gives the diagnosis an early edge, i.e. gains a **lead time** before a clinical diagnosis is made. This is especially important for diseases whose response to treatment is best before a certain critical point in their natural history.

Offhand, screening has four purposes

1. Early diagnosis (and prompt treatment) of cases and importantly, carriers.
2. Prospective diagnosis (i.e. screening during immigration/emigration).

3. Detection of natural history (i.e. finding more details about 'preclinical' stages of diseases).
4. To educate people about self-diagnosis of disease.

Types of screening

1. **Mass**: It is actually a large scale early diagnosis and prompt treatment, needs a lot of resources.
2. **Screening high risk population**: This, as we will see, is more fruitful than mass screening.
3. **Multiphasic**: A battery of tests employed instead of a single screening test. However, because of prohibitive costs, such method can't be used *en masse*.

Screenable diseases

- Large number of asymptomatic cases
- Long preclinical phase (i.e. people tend to live with it for long time before reporting)
- Not rare (a high prevalence increases success of screening)
- Well-known natural history
- Good tests are available to detect the disease
- Treatment available, especially early treatment is advantageous for the diseases (otherwise, why do screening?).

3

Health and the Individual

KEY FEATURES

- GENES
  - Chromosomal diseases
  - Single gene (Mendelian) diseases
  - Multifactorial diseases
  - Prevention of genetic diseases
- NUTRITION
  - Energy
  - The proteins
  - Lipids
  - Carbohydrates
  - Vitamins
  - Minerals
  - Antioxidants
  - Water
  - Nutritional assessment of community
  - Food hygiene
  - Foodborne diseases
  - Nutritional problems in public health
  - Protein energy malnutrition
  - Nutritional anemia
  - Vitamin A deficiency
  - Iodine deficiency disorders
  - Endemic fluorosis
  - Solutions for nutritional problems
  - Status of principal foods

GENES

This is the area where community medicine can make the least difference. There is little to do about genetic diseases than prevention, and I will be thus quick with this chapter.

Chromosomal diseases

You know of some well-known 'ploidy's – like Klinefelter (XXY, XXXY and so on), Down (Mongolism – trisomy 21) and Turner (XO). The XYY (superman) and XXX… (Superwoman) have recently been identified. Other rare types are loss of entire chromosomes, mosaics, etc.

Single gene (Mendelian) diseases

Autosomal dominant	Achondroplasia Huntington's chorea Neurofibromatosis Polyposis coli Brachydactyly Marfan syndrome

Contd...

Contd...

	Retinoblastoma Blood group disorders Hyperlipoproteinemias I–IV Polycystic kidney Polydactyly Spherocytosis
Autosomal recessive	Fibrocystic pancreas Phenylketonuria Albinism Tay Sachs Agammaglobulinemia (Swiss) Alkaptonuria Cystic fibrosis Galactosemia Hemoglobinopathies Maple syrup urine disease Hirschsprung disease (megacolon)
X-lined dominant	Vit D resistant rickets Familial hypophosphatemia Blood group Xg
X-linked recessive	Hemophilia Duchenne's muscular dystrophy Color blindness G6PD deficiency Hydrocephalus Retinitis pigmentosa Bruton's disease

Multifactorial diseases

These diseases can't be only attributed to genes like diabetes, CHD, HTN, schizophrenia, duodenal ulcer, heart malformations, etc.

Prevention of genetic diseases

Health promotion

Genetic counseling

It is a communication process of apprising an individual/ family about genetic diseases, its management and prevention in a nondirective manner, with due attention to psychosocial aspects. It covers the risk assessment for a disorder to occur, education on genetic diseases and psychosocial counseling. The counselor must remember that

1. The family/parents are the decision makers
2. They have right to full information
3. Everything between him and the family is confidential.

Types

1. **Prospective**—After identifying at risk individual by screening (i.e. thalassemia) and preventing marriages between two carriers.
2. **Retrospective**—How to live on after your child is born with a genetic disease.

Steps

1. Detailed family history
2. Educate about risk of genetic disease
3. Discuss nongenetic factors that may have effects
4. Explain role of genetic testing, risks, benefits and limitations
5. Management.

Indications

1. Mother > 35 or father > 50
2. Consanguineous marriage
3. Previous history of birth defect or genetic disease
4. High-risk ethnic groups
5. USG/prenatal test suggesting a defect.

Eugenics

This is aimed at improving the 'genetic environment', i.e. the artificial selection of only the 'good genes' to remain in the community, and eliminating the 'bad genes' by preventing reproduction of people carrying bad genes and abortion of fetuses with the bad genes. The subject is so debated that 'eugenicists' have often been vindicated as 'racists' or 'fascists', etc. People certainly devour the right of free reproduction.

Specific protection

Protection from X-rays during pregnancy, and gonads should always be covered.

Early diagnosis

1. To screen carriers by routine laboratory methods other than chromosomal analysis (i.e. Duchenne's muscular dystrophy can be identified by serum creatinine).
2. Prenatal diagnosis – Amniocentesis of at risk parents; chromosomal disorders may be identified form fetal cells in the amniotic fluid; also, some biochemical test may be useful (high α – feto protein in amniotic fluid indicates neural tube defects).
3. Screening of newborns.
4. Diagnosis at preclinical stages (thalassemia is readily seen from Hb electrophoresis, sickle cell by oxygen exposure test) of at risk babies.
5. Treatment have limited success, like giving Phenylalanine deficient diet to phenylketonuria patients.

Rehabilitation

Of course, this is the most important part, and often the therapeutic target, for genetic diseases.

NUTRITION

An early achievement of medical science was the recognition that good health called for a suitable kind of diet. Not only moderation of diet, good health required certain key ingredients in the food we take. The human body is rather specialized in its dietary needs. A plant can live on just carbon dioxide, water and just that. Microorganisms, likewise, can synthesize organic molecules like glucose and amino acids from inorganic sources. As evolution proceeded, there is a gradual dependency of the organism on external sources of glucose, amino acids, lipids and vitamins.

The very scum of the human society is that we have to eat. How nice people would have been to each other if they could have just made their own food out of sunlight, like plants! But nobody likes the life of a plant either. Food is the spice of our life. In the sorry state of affair that the world is in, 36 million people die of starvation, directly or indirectly, each year;[32] however, in this same world, obesity has reached epidemic proportions in many developed and developing countries.

Nutrition is the study of food and its relationship with health. *Food* is something that we eat (very easy to remember). *Nutrients* are specific molecules within food which are required for the body and have a specific dietary constituency.

The English physician Prout (who was a century ahead of time in suggesting that all elements were made of hydrogen) made the classification carbohydrates, proteins and fats. Of these, in theoretical basis – glucose, eight particular amino acids and three particular fatty acids, eleven vitamins and legion minerals were indispensable.

Food standards

The Codex Alimentarius, which means "food law" or "code" in Latin, is a comprehensive collection of internationally adopted and uniformly presented food standards and related texts (including guidelines), produced by the WHO and the Food and Agriculture Organization (FAO). The Codex texts address a wide range of general matters that apply to all processed, semi-processed and raw foods distributed to consumers, such as food hygiene, food additives, pesticide residues, contaminants, labeling and presentation, and methods of analysis and sampling. The complete Codex Alimentarius is available, via the Codex website (www.codexalimentarius.net).

In India, food standards are determined and cerified by the Bureau of Indian Standards (the ISI mark) and Prevention of Food Adulteration Act.

Energy

The unit of energy is a Joule; however, for our purpose we will use the much old-fashioned calorie = 4.184 J. The average calorific values of proteins, fats and carbohydrates stand at 4, 9, and 4 kcal/g respectively.

Energy requirement

Categories of requirement

1. Basal (to carry out resting metabolism)—1 kcal/hr/kg

2. Energy required for daily activity
3. Energy required for occupation.

Indian reference for energy (Table 3.1)

Table 3.1. The Indian reference person

	Man	Woman
Age	20–39	20–39
Activity time	At least 8 hrs	At least 8 hrs
Sleep	Adequate	Adequate
Weight	60 kg	50 kg
Cocurricular activity	4–6 hrs	4–6 hrs
Walking	2 hrs	2 hrs
Energy need	2875 kcal/day	2225 kcal/day

Energy requirement is defined as the energy level most likely to prolong life and least likely to cause obesity (Tables 3.2 and 3.3).

Table 3.2. Daily energy requirements in kcal [Indian Council of Medical Research]

	Male	Female
Infants	108–118/kg	Same
1–3 years	1240	1240
4– 6 years	1690	1690
7–9 years	1950	1950
10 –12 years	2190	1970
13–15 years	2450	2060
16 –18 years	2640	2060
Reference adult sedentary	2425	1875
Reference adult moderate worker	2875	2225
Reference adult heavy worker	3800	2925
Pregnant woman		+300
Lactation		
1st 6 months		+550
2nd 6 months		+400

Table 3.3. An easier chart to determine energy requirement for children

Weight	Energy requirement (kcal/day)
<10 kg	100x
10 + x kg	1000 + 50x
20 + x kg	1500 + 20x

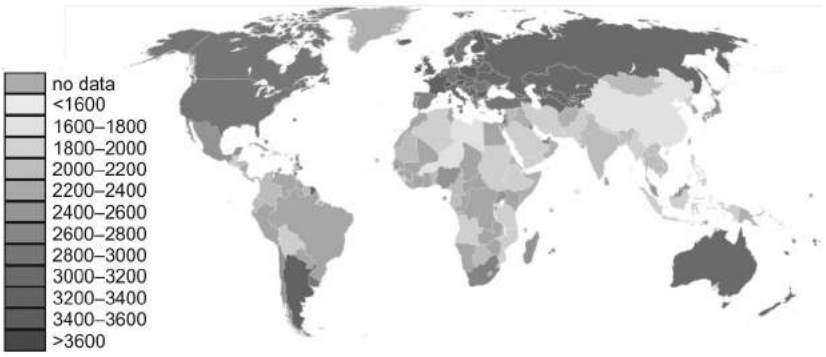


Figure 3.1. Per capita energy consumption in the world[33]

Unit of energy consumption

The energy need of an adult sedentary man/day is called 1 consumption unit (2425 kcal).

Balanced diet

It is the diet which has nutrients in adequate amount for bodily function with small provision for illness. Before prescribing a balanced diet, you must note the local climate and food habit. Also, the energy distribution between carbohydrates, fats and proteins (60:20:20) should be maintained.

Dietary Goals/Prudent diet [WHO]

1. 20–30% energy from fat
2. Saturated fatty acids <10% of energy consumption
3. Increase in fiber intake and reduction of refined carbohydrates (flour, bread, suji, sugar)
4. Restriction of energy rich food like alcohol
5. Salt <5g/day
6. 15–20% of energy intake from proteins
7. To avoid junk food
8. Cholesterol < 100 mg/1000 kcal.

The proteins

Proteins (from a Greek word meaning 'of first importance') are of first importance. They constitute 20% of the body weight by structure, and most of the 'functions' of the body, from lifting a bucket to writing a thesis, are carried out by proteins.

Quick facts

1. *Biologically complete* proteins are egg and milk, i.e. contain the entire gamut of essential amino acids. It is possible to combine two incomplete protein sources (e.g. rice and pulse together as a khichdi) to make a complete protein source, and such combinations are the basis of distinct cultural cooking traditions.

2. The **essential amino acids** are Lys, Leu, Val, Met, Trp, Phe, Ile, Thr[34]; Arg and His are semiessential amino acids. For premature babies Tau (taurine) and Cys are also essential (luckily, they are found in breast milk). These are called *essential* because the body cannot synthesize them in necessary amount to meet the needs.
3. Egg is held as the reference protein (NPU=100). It has high digestibility coefficient and biological value.
4. Most plant proteins lack 1–2 essential amino acids. **Cereals lack Lys and Thr, pulses lack Met and Cys**. These amino acids limit the food from being complete. Thus these are called the Limiting amino acids. When we mix cereal and pulse, by the supplementary action – the khichdi becomes complete.
5. Imbalanced proteins: Excessive consumption of Leu (as in maize) inhibits formation of Tryptophan to niacin, resulting in Pellagra (common in Andhra Pradesh).

Evaluation of protein nutritional status

Anthropometry

Weight, height, MAC and skin fold thickness (see later, 'PEM').

Biochemical assessments

Serum **albumin** is the best marker of protein nutritional status. Serum **transferrin** is not very specific (it may be depressed in many systemic diseases). An albumin level> 3.5 g/dl indicates satisfactory nutrition, 3–3.5 g/dl moderate malnutrition and <3 g/dl indicates severe malnutrition.

Evaluation of a protein

Digestibility coefficient (DC)

Percentage of ingested protein absorbed after complete digestion.

Biological value (BV)

Percentage of nitrogen retained from the total nitrogen absorbed from a protein. 1 gram of protein gives almost 6.25 mg of nitrogen to be assimilated in the body tissues such as muscles, connective tissue and blood.

Net protein utilization (NPU)

Proportion of ingested protein retained in body = (DC × BV)/100. It accounts for the total *pharmacokinetics* of a protein (absorption, distribution, metabolism, excretion).

Protein efficiency ratio

Gain in weight of young laboratory animals per unit of protein consumed.

Amino acid score

Ratio between amino acids content of a protein with a reference protein (egg).

Evaluation of amount of protein intake

The **protein energy ratio** is the (energy obtained from proteins/total energy from diet) × 100.

Protein requirement (g/kgBW/day) – ICMR (Table 3.4)

The chart assumes an average NPU = 65 in Indian diet. Note that children upto 5 years require the most protein/kg/day, which is again maximum in infants. Also, this intake is sufficient only when *energy* requirements are met. If energy requirement is not met, ingested protein will be used up for energy production.

Table 3.4. Protein requirement (g/kg)

Adult	1
Pregnancy	1 + 15
1st six months of lactation	1 + 25
2nd six months of lactation	1 + 18
Preschool children (maximum in infants)	1.2–2.3
Adolescents	1.2–1.4

Lipids

Lipids are concentrated source of energy. They constitute 10–15% of body weight. Of these, 99% is present in adipose tissue.

Types of lipids

Lipids are usually classified into fatty acids, triglycerides, steroids and phospholipids. **Fatty acids** may be further classified as

- **Saturated** fatty acids have all of the carbon atoms in their fatty acid chains bonded to hydrogen atoms
- **Unsaturated** fats have some of these carbon atoms double-bonded. Unsaturated fats may be further classified as *mono*-unsaturated (one double-bond) or *poly*-unsaturated fatty acids (PUFA, having many double-bonds). Furthermore, depending on the location of the double-bond in the fatty acid chain, unsaturated fatty acids are classified as ω3 or ω6 fatty acids.

What is this ω business

The IUPAC recommends nomenclature of organic compounds in a numerical fashion. The principal function group in a compound is termed carbon number 1 (or the α carbon), and the rest follows. It was customary to label the COOH group number 1 carbon in organic acids. Thus

$$CH_2—CH_2—CH = CH—CH_2—CH_2—COOH$$

is buta-4-enoic acid (the double bond is on the 4th carbon beginning from COOH). In an alternative convention, we may begin numbering from the last carbon, and call it ω1 (ω is the last letter of the Greek alphabet). Thus the same compound is an ω3 fatty acid, because double-bond is over the third carbon beginning from the end.

An appropriately balanced intake of ω3 and ω6 acids partly determines the relative production of different prostaglandins: one reason a balance between ω3

and ω6 is believed important for cardiovascular health. In industrialized societies, people typically consume large amounts of processed vegetable oils, which have reduced amounts of the essential fatty acids along with too much of ω6 fatty acids relative to ω3 fatty acids.

Trans fats

All the double bonds in a natural fatty acid are cis isomers. **Trans fats** are a type of unsaturated fat with *trans*-isomer bonds; these are rare in nature and typically created in an industrial process called (partial) hydrogenation. Trans fats are one of the causes for accelerated atherogenesis. They are favored by the industry only because they are resistant to rancidity (but that does not serve as a good excuse to manufacture them).

Essential fatty acids

They are 'essential' in the sense that the body is unable to synthesize them – linolenic, linoleic and oleic acid. Of these, **linoleic acid** is most important (the rest can be synthesized from linoleic acid). These PUFA reduce serum LDL (by forming esters of cholesterol), and also form the prostaglandins. The essential fatty acids are found in all vegetable oils (except coconut oil), eggs, milk fat, fish oil.[35]

Source of lipids

1. *Animal fats* usually have good amount of saturated fatty acids , except fish oils. Examples include ghee, butter, milk, cheese, eggs and meat.
2. *Plant oils* are rich in PUFA except palm and coconut oil (which are high in saturated fat). Plant oils are mostly derived from seed storage, i.e. groundnut, mustard, sesame, castor, etc.
3. *Invisible fat* is the lipid which can't be distinguished and removed from a source like cashew, cereals, and pulses. This form is the main source of fat intake. The large rice consumption in south Asians accounts for a huge amount of invisible fat.

Diseases caused by lipids

Hyperlipidemia: Obesity, IHD; breast and colon cancer
Hypolipidemia: Phrenoderma; depigmentation (Kwashiorkor).

Requirement

20–30% of total energy intake, ideally should be from the lipids. Of these, 50% is from plant sources is ideal (for the PUFA).

Hydrogenation of PUFA

Vegetable oil + hydrogen over palladium + Δ > Vanaspati, it increases the shelf life but reduces the PUFA content. It is usually fortified with 2500 IU Vitamin A and 175 IU Vitamin D/100 g.

Refined oil

Oil treated with steam/alkali is clear and palatable and retains the PUFA in it. It is mostly done to remove the free fatty acids (which is potentially toxic to body) and the rancid content, if any.

Carbohydrates

Carbohydrates form the bulk of food. Apart from generating energy, they are required for oxidation of fat and synthesis of amino acids. The carbohydrate reserve in man (glycogen) is about 500 g. Usually most (I mean most) Indians, however poor, are able to meet the carbohydrate demands in someway or the other.

Types of carbohydrates and their importance

Monosaccharides contain one sugar unit, *disaccharides* two, and *polysaccharides* three or more. Polysaccharides are often referred to as **complex** carbohydrates because they are typically long multiple branched chains of sugar units. The difference is that complex carbohydrates take longer to digest and absorb. In contrast, simple carbohydrates are absorbed *very* quickly. The spike in blood glucose levels after ingestion of simple sugars is thought to be related to some of the heart and vascular diseases which have become more frequent in recent times. Simple sugars form a greater part of modern diets than formerly (soft drinks, candys, ice cream), leading to more cardiovascular disease. The degree of causation is still not clear, however.

Dietary fibers

This special class of carbohydrates needs additional mention. Cellulose (found in any vegetable), pectin, inulin and plants gums and mucilages are the chief dietary fibers. They are useful in decreasing colonic cancer; laxative action (osmotic increase of fecal volume) and lowering cholesterol. By reducing the peaks of insulin that occur after a high sugar meal, they also stop progression of Type II diabetes mellitus and prediabetes.

Carbohydrate requirement

50–60% of total energy intake; there must be some dietary fibers.

Vitamins

How much of nutrients do we need?

The Recommended Dietary Allowance (RDA) was developed during World War II by Lydia J Roberts, Hazel Stiebeling and Helen S Mitchell, all part of a committee established by the United States National Academy of Sciences. The Food and Nutrition Board subsequently revised the RDAs every five to ten years.

The current Dietary Reference Intake recommendation is composed of four variables:

1. **Estimated Average Requirements (EAR)**, expected to satisfy the needs of 50% of the people in that age group based on a review of the scientific literature.
2. **Recommended Dietary Allowances (RDA)**, the daily dietary intake level of a nutrient considered sufficient to meet the requirements of nearly all (97–98%) healthy individuals in each life-stage and gender group. The RDA = EAR + 2 standard deviations of EAR (usually approximately 20% higher than the EAR).

3. **Adequate Intake (AI)**, where no RDA has been established, but the amount established is somewhat less firmly believed to be adequate for everyone in the demographic group.
4. **Tolerable Upper Intake Levels (UL)**, to caution against excessive intake of nutrients (like vitamin A) that can be harmful in large amounts.

The Indian Council of Medical Research has developed a list of RDAs for Indians in 1990, and I shall follow those values. Where ICMR has not specified, I have followed the US guidelines.[36]

Vitamin A

Vitamin A (retinol) is needed by the retina of the eye in the form of light-absorbing molecule retinal. As retinoic acid, it is also an important growth factor for epithelial cells.

Vitamin A includes both retinol and a provitamin, β-carotene, which is converted into retinol by intestinal mucosa. Retinol = ¼ of β-carotene content in a food. One international unit of vitamin A is = 0.3 μg retinol.

Source

Vitamin A is abundantly found in,

1. Animal – Halibut liver oil, cod liver oil, eggs, butter
2. Plant – Carrot, spinach, amaranth, green leafy vegetables, mango, papaya.

Vanaspati and margarine are often fortified with vitamin A. The liver stores vitamin A as retinol palmitate for 6–9 months.

Deficiency

Approximately 250,000–500,000 children in developing countries become blind each year owing to vitamin A deficiency, with the highest prevalence in South-east Asia and Africa.[37]

Primary deficiency. Due to inadequate intake of yellow and green vegetables, fruits and liver or early *weaning* from breast.

Secondary deficiency. Chronic malabsorption of lipids, impaired bile production and release, low fat diets, and chronic exposure to oxidants, such as cigarette smoke causes vitamin A deficiency. Vitamin A is a fat soluble vitamin, thus requires presence of fat in diet/adequate bile flow to be absorbed. **Zinc deficiency** can also impair absorption, transport, and metabolism of vitamin A.

Manifestations

1. Ocular (in sequence) night blindness (physiological deficiency appears earlier than anatomic changes) → conjunctival xerosis → Bitot spot → corneal xerosis → keratomalacia
2. Extra ocular—Follicular hyperkeratosis (white lumps of keratin at base of hair follicles), anorexia, growth retardation, and metaplasia of upper respiratory and bladder epithelium to keratinized squamous epithelium.

RDA (retinol in mg) (Table 3.5)

Table 3.5. Vitamin A requirement

Adult (from 7 year onwards)	600
Pregnancy	600 (no increase in demand)
Lactation	950
Child	400

Toxicity

Excess retinol (those who eat intact livers of large animals) causes nausea, vomiting, anorexia, desquamation of skin, enlarged liver, raised ICP and papilledema.

Vitamin D

Vitamin D (calciferol) is a steroid hormone that is converted in liver to 25-hydroxycholecalciferol, and in kidney to 1,25-dihydroxycholecalciferol (DHCC). DHCC helps absorption of calcium from GI tract and kidney, and is necessary for bone mineralization.

Source

Vitamin D is synthesized in skin by action of UV rays of sunlight on 7-dehydrocholestrol (an intermediate in cholesterol biosynthesis). It is also found in fish liver oil, butter, eggs, milk (including human milk). It is stored in adipose tissue.

RDA

Adults 100 IU, children 200 IU, pregnant and lactating mothers 400 IU. (One international unit of vitamin D = 0.025 μg of vitamin D).

Deficiency

Vitamin D deficiency is the result of urbanization, i.e. children beginning to get less sunlight.

1. Rickets is usually observed in children of 6 months–2 years; reduced calcification of bones leading to bow legs, knock knee, deformed pelvis, Harrison sulcus (bony depression over chest), Rickety rosary (prominent costal cartilages); the hypocalcemia may also cause hypotonia, tetany and convulsions.
2. Osteomalacia: Occurs especially during pregnancy and lactation, when demands for calcium and vitamin D are increased.

Prevention

1. Adequate sunlight exposure
2. Vitamin D fortification of food.

Toxicity

Excess of vitamin D can cause hypercalcemia, resulting in anorexia, nausea, vomiting, drowsiness, coma and even cardiac arrhythmias.

Vitamin E

By far the richest sources of vitamin E are vegetable oils, egg yolk and butter. Its role is uncertain, however. It is added to vitamin A in bottles to prevent photo oxidation of vitamin A *in vitro* (it is a natural antioxidant).

Vitamin K

Vitamin K_1 is found in green leafy vegetables, K_2 synthesized by intestinal flora and K_3 in milk. *Deficiency* occurs due to—

- Inadequate green leafy vegetable intake
- Use of broad spectrum antimicrobials (that clear intestinal flora)
- Cholestatic jaundice (vitamin K cannot be absorbed without bile).

Deficiency results in impaired coagulation. Newborns are especially deficient (they have no intestinal flora), and should receive an IM dose of menadione just after birth.

RDA

0.03 mg/kg for adults.

Thiamine

Thiamine was the first water soluble vitamin to be identified.[38] In 1897 Christiaan Eijkman, a military doctor in the Dutch Indies, discovered that birds fed on a diet of cooked, polished rice developed polyneuropathy and paralysis, which could be reversed by discontinuing rice polishing.[39] An associate, Gerrit Grijns concluded that rice contained an essential nutrient in the **outer layers of the grain that was removed by polishing**.[40]

In 1911 **Casimir Funk** isolated an antineuritic substance from rice bran that he called a "vitamine" (on account of its containing an amino group). Dutch chemists, Barend Coenraad Petrus Jansen and his closest collaborator William Frederik Donath, went onto isolate and crystallize the active agent in 1926. The first vitamin was discovered.

Source

Whole grain cereals, wheat, gram, yeast, vegetables, milk; thiamine is lost during milling of rice and toasting of wheat.[41] It may be preserved by parboiling.

Action

Thiamine pyrophosphate is a coenzyme of pyruvate dehydrogenase, α-ketoglutarate dehydrogenase complex, and the transketolase reaction in hexose monophosphate shunt (review some biochemistry at this point, will you). Remember that **pyruvate dehydrogenase** converts pyruvate to acetyl CoA, which then enters into Krebs cycle and goes onto electron transport system. Thus thiamine is essential for oxidative respiration. Tissues like muscles can run on nonoxidative respiration for sometime, but the nerves and the heart cannot. Thus they are most affected by thiamine deficiency.

Deficiency

Beriberi. Beriberi is a disease of nerves and the heart.

- **Dry** beriberi is a symmetric peripheral neuropathy, affecting distal more than proximal limb segments and causing **calf tenderness**.
- **Wet** beriberi is a combination of mental confusion, muscular wasting, edema, tachycardia, cardiomegaly, and **congestive heart failure in addition** to peripheral neuropathy.
- **Infantile** beriberi occurs in infants breast-fed by thiamine deficient mothers (who may show no sign of thiamine deficiency).

Following thiamine treatment, rapid improvement occurs generally within 24 hrs.
Wernicke encephalopathy. It is the most frequently encountered manifestation of thiamine deficiency in developed countries. Often seen in alcoholics, it may also be caused by a gastrointestinal disease, those with HIV, and with the injection of IV glucose or without B-vitamin supplementation. It is characterized by paralysis of eye movements, abnormal stance and gait, and markedly deranged mental function.[42]

Korsakoff Psychosis is a complication of WE, characterized by retrograde and anterograde amnesia, impairment of conceptual functions, and decreased spontaneity.

RDA

0.5 mg/1000 kcal of energy intake.

Riboflavin

In addition to being a vitamin, riboflavin is a food additive (as color) for its bright orange hue.
Industrial use. Because riboflavin is fluorescent under UV light,[43] it is used to detect leaks in any network of fluids (i.e. water supply pipes).

Source

Milk, cheese, leafy green vegetables, liver, kidneys, legumes such as mature soybeans, yeast, mushrooms and almonds are good sources of riboflavin,[44] but exposure to *light* destroys riboflavin. The *milling* of cereals results in considerable loss (upto 60%) of vitamin B_2, which can be avoided if the rice is *steamed* prior to milling.

Neonatal phototherapy. The light used to irradiate the neonates breaks down not only bilirubin, but also the riboflavin; riboflavin supplementation should be given.

Action

Riboflavin is the precursor of flaven adenine dinucleotide (FAD). This molecule, FAD, is cofactor for (time for some biochemistry again)

- Succinate dehydrogenase (in Krebs cycle)
- Complex II of electron transport chain
- Fatty acyl CoA dehydrogenase in β oxidation of fat
- Retinal → retinoic acid

- Folic acid → active form tetrahydrofolate
- Oxidized glutathione → reduced glutathione
- Trp → niacin.

Deficiency

It is particularly common where rice is staple diet. The syndrome consists of angular stomatitis, glossitis, photophobia and itchy eyes, nasolabial dyssebacia and occassionally scrotal dermatitis.

RDA

0.6 mg/1000 kcal of energy intake.

Nicotinic acid/niacin

Niacin was first described by Hugo Weidel in 1873 in his studies of nicotine. Niacin was extracted from livers by Conrad Elvehjem who later identified the active ingredient, then referred to as the "pellagra-preventing factor".[45] It was thought appropriate to choose a name to dissociate it from nicotine, to avoid the perception that vitamins or niacin-rich food contains nicotine, or that cigarettes contain vitamins. The resulting name 'niacin' was derived from nicotinic acid + vitamin.

Sources

Niacin is found in liver, chicken, beef, fish, cereal, peanuts and legumes and is also synthesized from tryptophan *in vivo* (60 mg Tro generates 1 mg of niacin).

RDA

6.6 mg/1000 kcal energy consumed.

Action[46]

- Precursor to NADH, NAD+, NADP+ and NADPH
- Involved in DNA repair mechanisms
- Production of steroid hormones in the adrenal gland
- In pharmacologial doses, it is also a lipid lowering agent.

Deficiency

Dietary niacin deficiency occur where people eat maize as a staple food. Maize is the only grain low in niacin, and **nixtamalization** (cooking maize with alkaline lime) is needed to increase bioavailability. Excess of *Leu*, as in people who eat jowar and maize only, inhibits conversion of Trp to niacin, and may also cause deficiency.

Pellagra

A combination of

- Diarrhea
- Dermatitis—Especially around neck (Cajole necklace)
- Dementia—Irritability, poor concentration, anxiety, fatigue, restlessness, apathy, depression.

Pantothenic acid

It goes onto form **coenzyme A**, which is an important intermediate in many biochemical reactions. Especially, **acetyl coenzyme** A is the chief energy releasing compound and involved in lipid biosynthesis. It is present in all plant food.

Pyridoxin

Sources

It is distributed in all kinds of food, but *cooking*, freezing and canning can reduce the content as much as upto 50%.[47] Usually the plant sources are more stable than milk or animal sources.

Action

The active form of pyridoxine, pyridoxal phosphate, is involved in all transamination recations, ALA synthase (recall synthesis of hemoglobin) and nonoxidative decarboxylation reactions.

Deficiency

A seborrheic dermatitis like eruption, atrophic glossitis with ulceration, angular cheilitis, conjunctivitis, intertrigo, and **neurologic symptoms** of somnolence, confusion, and neuropathy.[48] The antitubercular drug Isoniazid closely resembles Pyridoxine and antagonises its action. Pyridoxine must be supplemented with Isoniazid.

RDA

2 mg.

Miscellaneous uses

- Without enough Pyridoxine, homocysteine builds up in the body which damages vessel linings → atherosclerosis
- Some evidence[49] shows it is useful in **autism**
- A few cases of Carpal Tunnel Syndrome have benefitted from Pyridoxine[50]
- It is occassionally useful in premenstrual syndrome and depression
- At least one study[51] has shown that Pyridoxine increases vividness of dreams.

Folate

Folate is found in all vegetables as well as liver, meat, fruits, cereals and dairy products. Tetrahydrofolate is involved in one carbon transfers; N_5N_{10} methenyl THF is needed in purine synthesis.

RDA

100 mg (400 mg in pregnancy).

Deficiency

Dietary deficiency of folate is rare, but deficiency may be precipitated by folate antagonists (valproate, methotrexate, pyrimethamine, cotrimoxazole and alcohol). The manifestation is a megaloblastic anemia and rarely, infertility; deficiency of

folate in intrauterine life (may be caused by antiepileptic drugs) causes **neural tube defects**.

Ascorbate

Vitamin C has two forms—Ascorbate and dehydroascorbate. This makes it a very good reducing agent. However, it is very sensitive to heat and destroyed by cooking. It helps in the following conversions.

- Proline → hydoxyproline (which goes onto form collagen)
- Phenylalanine → para-hydroxyphenylalanine
- Methemoglobin → hemoglobin
- Folate → tetrahydrofolate
- Tryptophan → serotonin.

It is also a natural antioxidant and plays a role in prevention of cancer.

RDA

60 mg.

Deficiency

The syndrome of bleeding gums, easy bruising, bleeding into joints and delayed would healing, once very common in sailors (they spent long journeys without fresh fruit) known as **scurvy** is rare nowadays.

Methylcobalamin/cyanocobalamin

Sources

Liver, meat, egg and fish; no plant source. 2 mg of this vitamin is stred in liver and stores last for 3–5 years. So if a man becomes vegetarian today, he will develop megaloblastic anemia only after 5 years.

Functions

It is involved in one carbon transfer

- Nucleotide synthesis (in combination with folate)
- Conversion of odd chain fatty acids to even chain (which is required to form *myelin sheath*).

RDA

Adult: 1 μg; pregnancy and lactation: 1.5 μg.

Deficiency

Megaloblastic anemia combined with subacute combined degeneration of the spinal cord. Deficiency is rare, however, methylcobalamine supplementations in the dose of 1500 μg per day, have become rampant in clinical practice for no good reason.

Minerals (Table 3.6)

Minerals (an archaic term, bears no relation with a mine) are all the elements needed for the body except C, H, O and N. The deficiency of minerals in India is

very common, and is largely attributed to presence of oxalates, phytates, tannin and phosphates in diet. Minerals are classified as

- Macrominerals (RDA > 200 mg)—Ca, Cl, Mg, P, K, S
- Trace minerals—Co (for synthesis of vitamin B_{12}, Cu (for cytochrome oxidase), Cr, I (for thyroid hormone synthesis), Fe (for hemoglobin), Mn (for handling oxygen radicles), Mo (for xanthine oxidase), nickel (for the enzyme urease), selenium (present in all peroxidase enzymes), zinc (present in carboxypeptidases and carbonic anhydrase).

Table 3.6. Minerals

Dietary mineral	RDA/AI	Source	Deficiency
Potassium	4700 mg	Legumes, potato skin, tomatoes, and *bananas*	Dietary deficiency does not occur
Chloride	2300 mg	*Table salt* is the main dietary source of chloride	
Sodium	10–15g (hypertensives must restrict to < 2.5g)	*Table salt*, vegetables, milk, and spinach	Dietary deficiency does not occur
Calcium	400 mg, pregnancy—1g, lactation—1.2g	Dairy products (especially milk), animal bones, fish, green leafy vegetables, nuts, millet. Salts dissolved in drinking water; *rice* is very poor in calcium	Deficiency is rare (calcium is present even in water)
Phosphorus	700 mg	Dairy products, bones, all vegetables, animal flesh	Dietary deficiency is rare
Magnesium	200–300 mg	Nuts, soybeans, and cocoa	Alcoholics, patients of chronic liver, disease, preeclampsia may develop deficiency, which manifests as tetany, exaggerated reflexes and irritability
Zinc (component of carbonic anhydrase)	12–15 mg	Widely distributed in food	Growth failure, sexual immaturity, delayed wound healing, dermatitis, congenital malformations

Contd...

Contd...

Iron	28–30 mg	Red meat, leafy green vegetables, fish (tuna, salmon), eggs, dried fruits, beans, whole grains, and enriched grains.	Iron deficiency anemia
Copper (a component of cytochrome oxidase)	2.2 μg	Widely distributed	
Iodine	150 μg	Seafood, fish liver oil	Iodine deficiency disorders
Fluorine	0.5–0.8 mg/l of water	Drinking water, seafood	Deficiency causes dental caries, but excess of fluorine corrodes bones and teeth (fluorosis)
Selenium (cofactor of glutathione peroxidase)	55 μg	Widely distributed	Weight loss, immuno-suppression, cardiomyopathy
Molybdenum (component of xanthine oxidase, aldehyde oxidase, and sulfite oxidase)	45 μg	Widely ditributed	Moth and esophageal cancer

Iron

Although a trace element, iron is required for synthesis of hemoglobin, and that makes it special. The adult human contains 3–4 g of iron (80% in hemoglobin and myoglobin, and 20% storage iron).

Source

Iron comes in two forms

1. **Heme iron** is derived from hemoglobin/myoglobin and found in animal sources (liver, meat, poultry, fish); it is easily absorbed.
2. **Nonheme iron** is found in plant sources (green leafy vegetables, cereals, nuts, oil seed, dry food, jaggery, lemon), is poorly absorbed.

The average Indian diet of vegetables contains many organic acids (phytate, oxalate, phosphates, tannin) that will chelate dietary iron. Also, iron absorption requires vitamin C in abundance in diet.

Deficiency

Iron is physiologically lost through desquamation of epithelial cells. Hemorrhage, either physiologic (menstruation) or pathologic (hookworm, IUDs) increase iron loss. The course of iron deficiency has four stages; anemia does not show up until

stored iron is completely depleted. A ferritin < 15 μg/l indicates iron stores are depleted. With overt deficiency, IDA appears.[52]

RDA

Men 28 mg, women 30 mg, pregnancy 38 mg; about 10% of oral iron is absorbed.

Iodine

The adult human contains about 50 mg of iodine, with 8–12 μg/dl in blood.

Source

Iodine is found abundantly in seafood, milk, meat, vegetables. Natural goitrogens (cauliflower and cabbage) cause an artificial iodine deficiency by interfering with its use in thyroid gland.

RDA

150 μg.

Deficiency

Iodine deficiency is initiated in fetal stage, and results in Iodine Deficiency Disorders (see later), i.e. adverse effects of iodine deficiency on growth and development, which may be prevented.

Antioxidants

As cellular metabolism/energy production requires oxygen, it produces **free radicals**, such as the superoxide ($O_2^{-\bullet}$) as a by product. For normal cellular maintenance, growth, and division, these free radicals must be sufficiently neutralized by antioxidants. Antioxidants may be produced in the body from precursors (glutathione, vitamin C) or may be obtained in the diet (vitamin C, vitamin A, vitamin K). **Phytochemicals** are trace organic compounds found in colorful plants (soya, clover, spices, onion, tea, capsicum, chillies). A major class of them are polyphenol antioxidants, chemicals which are known to provide health benefits to the cardiovascular system and immune system.

Water

People have survived without food forever increasing number of days, and liberalist movements make it a point to highlight *fasting* as a tool of protest. However, even a few days won't pass without water.

RDA

The latest dietary reference intake report by the United States National Research Council recommended, generally, (including food sources) 2.7 liters of water total for women and 3.7 liters for men.[53] Specifically, pregnant and breastfeeding women need additional fluids to stay hydrated.

Nutritional assessment of community

The nutritional assesment is an integral part of making a 'community diagnosis'; it is the clinical equivalent of eliciting the diet history in internal medicine.

Objectives

1. Assess the magnitude of nutritional problems of a community
2. Find geographical distribution of such problems
3. Identify 'population at risk' of a certain disease.

Study method

Nutritional surveys can be cross sectional or longitudinal. Random samples are picked from the community as representatives.

Techniques

1. Clinical examination
2. Anthropometry
3. Diet survey
4. Biochemistry
5. Vital statistics (birth, death)
6. Functional assessment
7. Assessment of environment.

Tools

1. Predesigned format for data collection.
2. Infantometer, scale, weighing machine, Shakir's tape, etc.

Primary steps

1. Pretesting the format
2. Training staff for data collection
3. Supervision of the study.

Clinical examination (Fig. 3.2)

Clinical examination is a simple, direct and inexpensive method, but

- Diagnoses only clinically manifest cases
- Most clinical findings are very nonspecific
- A *doctor* is required to conduct the examination.

Biochemistry

These tests are costly, time consuming and can't be applied on a large scale.

Table 3.7. WHO diagnostics for anemia (cut off Hb values in g/dL)

6 months – 6 years	< 11
6 years – 14 years	< 12
Pregnancy and lactation	< 11
Adult female	< 12
Adult male	< 13

Similarly, vitamin A deficiency (serum vitamin A cut off) is **20 µg/dl**. A variety of tests can be used to detect PEM (serum albumin) and iron deficiency (serum ferritin), etc.

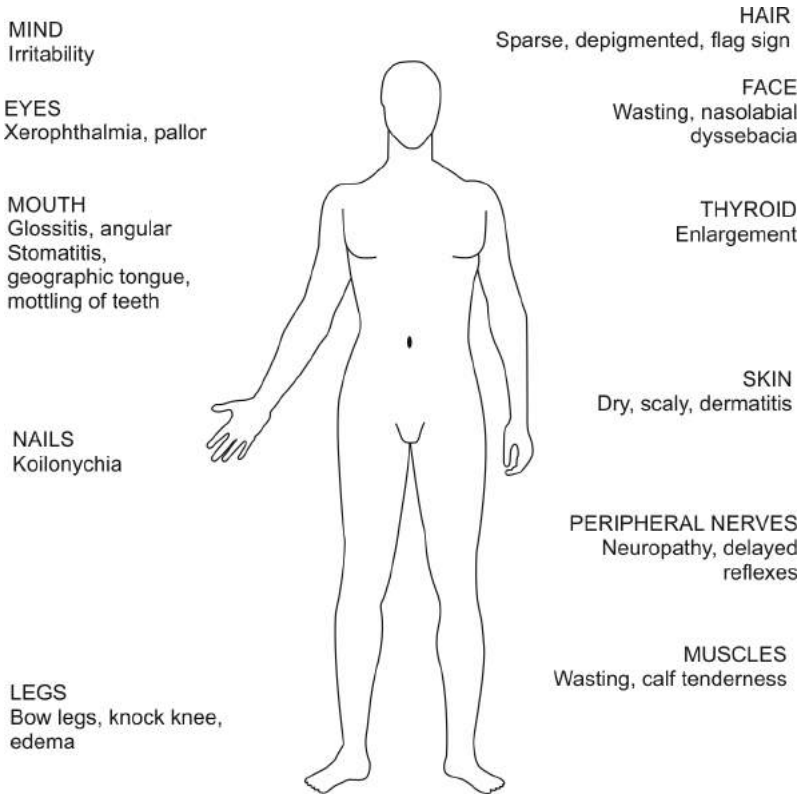


Figure 3.2. Signs of malnutrition

Anthropometry

It is a direct, widely used technique for assesment of under five children. The usefulness of anthropometry depends on accurate assessment of age, standard measurement procedures and reference values. The best thing about anthropometry is that you can enjoy a lazy day while health workers do the work (doctors need not be involved in anthropometry).

Indices

Weight for age. Weight of the observed child compared to that of a median reference child of same age and sex (see growth chart later).

Height for age. Height of a child compared to a reference median child of same age and sex; unlike weight, height does not reduce once it is gained, and any deficit means chronic malnutrition (stunting).

Weight for height. Weight of the child is compared with that of a reference median child of same height. Any deficiency means acute malnutrition (wasting). It is the best estimate of severity of malnutrition, and an age **independent criteria**.

Body mass index. Weight in kg/(height in meter)^2; normally 18.5–25.

Diet survey

It is the direct method to see for yourself what people are actually eating, and to find any inadequacies, and to suggest remedies.

1. To *weight* food, both before and after cooking—Accurate, time consuming, and weighing the cooked food is not acceptable[54] in India.
2. Interviews on what they have eaten—Most common method with reasonably good results.
   - 24 hr diet history
   - Diet cycle: Most families have a system of rotation tin tehir menu, i.e. the same items are rotated over an interval; the duration of the cycle varies (7 days in rural areas and 3 days in urban areas); an interview regarding one cycle will give the general idea on food consumption of the family.
3. Inventory method—Estimate what the family stores in stock for a week; because most families do only buy food on an ad hoc basis, this method is suitable for hostels and institutions, where food is bought on weekly or biweekly basis.

Food hygiene

All the conditions that must be met during *production, processing, storage, distribution* of food so that it remains safe, wholesome and fit for human consumption [WHO].

Household hygiene of cooked food

The UK Food Standards Agency publishes recommendations as part of its Hazard Analysis and Critical Control Points (HACCP) program.[55] Cooking food until the CORE TEMPERATURE is 75 °C or above will ensure that harmful bacteria are destroyed. However, lower cooking temperatures are acceptable provided that the core temperature is maintained for a specified period of time as follows,

- 60°C for a minimum of 45 minutes
- 65°C for a minimum of 10 minutes
- 70°C for a minimum of 2 minutes.

Cooked food should be kept in a shelf (and not covered by thin nets).

Milk hygiene

Sources of infection

Dairy animal, human handler.

Milk borne diseases

1. Zooanthroposes—Brucellosis, tuberculosis, salmonellosis, Q fever, streptococcal infections.
2. Infections from human that are transmitted, via milk—Typhoid, paratyphoid, cholera, viral hepatitis, diphtheria, *E coli* infection.

Prevention

1. Pasteurization of milk
2. Healthy and clean animals
3. Sanitary premises of dairy

4. Sterile vessels to keep milk
5. Safe water supply in the dairy
6. Personal hygiene of milk handlers.

Test for microorganisms in milk

Milk + methylene blue at 37°C → the 'blue' of methylene blue bleaches quickly if the milk is teeming with bacteria.

Pasteurization of milk[56]

It is the heating of milk to such temperatures and for such periods of time as are required to *destroy any pathogens* that may be present while causing *minimal changes in the composition, flavor and nutritive value* (i.e. without coagulating all its proteins). The emphasis on pasteurization is not to kill every bacteria, but provide a milk which should remain pathogen free at least until it reaches the consumer.

The first pasteurization was completed by Louis Pasteur and Claude Bernard on April 20, 1864. The process was originally conceived as a way of preventing wine and beer from souring.[57] Pasteurization kills 90% bacteria except thermoduric ones, and spores; if the milk gets warm again after pasteurization the spores will germinate. Thus milk should ideally be kept at 4°C until it reaches the consumer. HTST pasteurized milk typically has a refrigerated shelf life of two to three weeks, whereas ultra pasteurized milk can last much longer when refrigerated, sometimes two to three months. When UHT treatment is combined with sterile handling and container technology (such as aseptic packaging), it can even be stored unrefrigerated for 3–4 months.

Table 3.8. Pasteurization

Holder (Vat) method	High temp short time (HTST)	Ultra high temp (UHT)
63–66°C for 30 minutes rapidly cooled to 4°C	Rapid heating to 72°C for < 15s, rapidly cooled to 4°C. This is the widely used method	Rapidly heated in 2 stages to 150°C for a few seconds, and rapidly cooled

The term **cold pasteurization** is used sometimes for the use of ionizing radiation to kill bacteria in food.

Tests for pasteurization

1. Phosphatase test—Phosphatase enzyme should be *absent* in pasteurized milk
2. Culture of milk and colony count
3. Coliforms should be absent in pasteurized milk.

Table 3.9. Differences between boiling and pasteurization

	Boiling	Pasteurization
Bacteriological	All pathogens killed but ↑ acid forming bacilli Delays lactose fermentation	All pathogens + acid forming bacilli are destroyed No lactose fermentation

Contd...

Contd...

	Boiling	Pasteurization
Chemical	Insoluble Ca^{++} ↑ 6% ↑ Iodine by 20% No change in taste No scum formation 5% ↓ lactalbumin ↑ Vitamin C	Ca^{++} and Mg^{++} precipitates Total iodine is lost Taste altered (lactose is semi-burnt) Ca and P uptaken in scum Lactalbumin and lactoglobulin coagulates Total vitamin C lost Other vitamins lost to some extent

Foodborne diseases

Table 3.10. Foodborne disease

Intoxicants	**Vegetable toxins**. Lathyrism, Endemic ascites (pyrrolizidine alkaloids), epidemic dropsy (*sanguinariine* in argemone oil) Fungal toxins. Aspergilus, a storage fungus of grain produces aflatoxins which are carcinogenic; to avoid development of this fungus, the moisture of stored should be kept below 10%. Another field fungus, *Claviceps fusiformes* produces Ergotism (nausea, vomiting, giddiness, peripheral vasoconstriction); this toxin can be removed by floating the grain in 20% saline **Bacterial toxins**. Botulinum toxin, staphylococcal enterotoxin Chemicals. Heavy metals (Cd in shellfish, Pb in canned food, Hg in fish), oils, asbestos, pesticides, chemical contaminants from packaging of food
Infections	**Bacterial**—See the chapter on food poisoning Parasites—Intestinal nematodes, amebiasis

Neurolathyrism

It is a crippling disease prevalent in UP and Madhya Pradesh due to consumption of Lathyrus sativus containing β oxalyl amino alanine (which is an analogue of the neurotransmitter glutamic acid). This disease is prevalent in some areas of Bangladesh, Ethiopia, India and Nepal,[58] and affects more men than women. Ingestion of these legume usually occurs due to ignorance of its toxicity and where the despair of poverty and malnutrition leaves few other food options.

Stages

Latent → No stick → One stick → Two stick → Crawler

Control

1. Banning the crop, IEC among people, selective propagation of that strain which produces less toxin.
2. Detoxification: Toxic amino acids are readily soluble in water and can be leached. Bacterial (lactic acid) and fungal fermentation is useful to reduce BOAA content. Parboiling or steeping (soaking in hot water for 2 hrs) dissolves much of the toxin.

3. Recent research suggests that sulfur amino acids have a protective effect against the toxicity of BOAA.[59]

Epidemic dropsy

Epidemic dropsy is an acute onset edema due to intoxication with *Argemone mexicana*,[60] specifically a toxin called *sanguinarine*. Argemone (*sheyalkanta* in bengali and *bharbhanda* in hindi) is frequently used to adulterate mustard oil.

Clinical course

- Proteinuria, with a resultant pitting edema
- Headache, nausea, loose bowels, erythema and breathlessness
- Death may occur due to congestive heart failure.

Detection of Argemone oil adulteration in edible oils[61]

Nitric acid test. 5 ml oil is shaken with an equal volume of nitric acid → acid layer turns yellow, orange-yellow or crimson. The test is sensitive to a concentration of > 0.25%.

Ferric chloride test. 2 ml of oil and 2 ml of concentrated hydrochloric acid are mixed → heated in a water bath at 33.5–35°C for 2 minutes → 8 ml of ethyl alcohol added → heated in the bath for 1 minute → 2 ml of ferric chloride added → heated in the bath for a further 10 minutes. If Argemone oil is present, an orange-red precipitate is formed.

Cupric acetate test. A green color is formed

Paper chromatographic method. The most sensitive method; can detect down to 0.0001% Argemone oil adulteration.

Prevention

- Selective cultivation of yellow-seeded mustard with which neither black-colored Argemone seeds nor dark-brown Argemone oil mixes well so that adulteration can easily be detected even with the naked eye
- A strict ban on the sale of unbranded and unpacked mustard oil
- Educate farmers about of Argemone plants which grow as weeds in mustard fields and may contaminate mustard
- Enforcing the Prevention of Food Adulteration Act.

Nutritional problems in public health

Malnutrition

> Never, whilst memory lasts, can one obliterate the mental picture of those pitiful little bundles of marasmic, apathetic humanity, lying in the arms of gaunt women, on whose faces and forms famine had laid its devastating hands. Their faint, feeble, fretful wails ring still in one's ears today, summoning up visions of wasted stick like limbs, of distended abdomens, of dry, inelastic, scurfy, scaly skins, of hair scanty, brittle and dry, and of sightless dessicated eyes...
>
> *—R H Elliot, Tropical Ophthalmology, Oxford Medical Publications, 1920, chapter 6, page 78*

Malnutrition is a pathological state resulting from a relative or absolute deficiency or excess of one or more nutrients.

Problem

The World Health Organization cites malnutrition as the gravest single threat to the world's public health.

Prevalence. One in every 12 people on this planet is malnourished.[62] Malnutrition is, the biggest contributor to child mortality, present in half of all cases.

Intellectual performance. Malnutrition, in the form of iodine deficiency, is the most common preventable cause of mental impairment.[63] Malnutrition other than iodine deficiency also affects the intellectural performance. Better nourished children perform significantly better in school, partly because they enter school earlier and thus have more time to learn but mostly because of greater learning productivity per year of schooling.[64]

Global hunger index

The International Food Policy Research Institute calculates this index based on
- Proportion of undernourished population
- Prevalence of underweight in children under 5
- Under five mortality.

India ranks 66th in the rankings.

Morbidity. Not only does it cause direct mortality and sickness, it predisposes to several infectious diseases[65] like measles, tuberculosis, infectius diarrheas, which further aggravate the malnourished state.

Cancers. In the developing world, cancers of the liver, stomach and esophagus were more common, often linked to consumption of carcinogenic preserved foods, such as smoked or salted food, and parasitic infections that attack organs.

Metabolic syndrome. Changing food habits in developed and developing countries—A combination of excessive food production and sedentary lifestyles has created the obesity epidemic and rise of 'metabolic syndrome'; Yale psychologist Kelly Brownell calls this a "toxic food environment" where fat and sugar laden foods have taken precedent over healthy nutritious foods; thus obesity is rising in both developed and developing countries where people can afford to spend a little.[66]

Agent

1. Undernutrition
2. Overnutrition
3. Imbalance (i.e. excessive intake of leucine may cause pellagra)
4. Specific deficiencies.

Host

Vulnerable group
1. Pregnant and lactating women
2. Under five children
3. Any person during convalescence.

Cultural influences

Contrary to whatever you may believe, malnutrition is not necessarily due to scarcity of food *per se*. Malnutrition may be present on the face of adequate food supply because of

- Food habits and taboos—Consider ALL the variations people try with breastfeeding, over diluting cows milk, discarding cooking water from cereals; the average asian family still thinks bottle feeding is better than breastfeeding;[67] many mothers discard breastfeeding because they are not aware of the proper method, and breastfeeding is always painful to them.[68]
- Food fads—The single spolit child of rich parents who denies anything but ice cream for breakfast, and is disgusted with vegetables.[69]
- Food hygiene—Keeping the cooked items in the open, keeping water in uncovered buckets.

Life cycle approach

Malnutrition is self-perpetuating. Children born to malnourished mothers are of low birth weight, low immunity and prone to malnutrition. If they are male, they develop to be sickly and often a burden to society. If they are female, however, nature plays its great trick. It is no short of a phenomenon that however malnourished and thinly a woman is, she is still able to perform the basic reproductive function of ovulating once a month. In developing countries, malnourished girls of poor children get married quickly, and are forced into their 'obstetric career', to produce yet another batch of malnourished children.

Sex

Women are physiologically less prone to malnutrition (BMI and energy expenditure is low), but in societies where females eat 'last and least', they are easy victims (Fig. 3.3).

Figure 3.3. How postnatal nutrition differs between sexes: One year old twins in Chittagong, Bangladesh; left—Male, right—Female[70]

Pathologic states (secondary malnutrition)

Anorexia (one reason why pregnant women tend to get malnourished), malabsorption syndromes, liver/pancreas disease, diabetes. *Infections* are worth a special mention. The nemesis of diarrhea, respiratory infections, measles and intestinal worms run a vicious cycle with under nutrition.

Increased food requirement

Infants, toddlers, adolescents, menstruating women and peri-partum women need greater nutrition than usual.

Geography

Climate influences food habits (warm and humid climates makes people it less, digest even lesser, and prone to develop infectious diarrhea) as well as production of food (see climate change later). A high population density is adverse to nutritional satiety of anyone individual.

Socioeconomy

Not only do people starve if they cannot buy food, they begin to eat rubbish and get themselves sick. In recent decades, episodes of famine has always been a problem of food distribution and/or poverty, rathern than shortage of food.[71]

Food production

Food shortages are mostly caused in very under-developed countries by the lack of technology needed for the higher yields found in modern agriculture, such as nitrogen fertilizers, pesticides and irrigation. As a result of widespread poverty, farmers cannot afford or governments cannot provide the technology. It is not, however a big problem in India.

The World Bank and some wealthy countries also press such nations (that depend on aid of World Bank) to stop subsidizing[72] fertilizers, in the name of 'free market' policy (so that all fertilizers companies can do business in those countries). But the United States and Europe extensively subsidized their own farmers.[73,74] Many, if not most, farmers cannot afford fertilizer at market prices, leading to low agricultural production and wages and high, unaffordable food prices.

Climate change. Even small changes in temperatures can lead to increased frequency of extreme weather conditions.[75] Many of these have great impact on agricultural production (i.e. the hurricane katrina, the 'Ayela' in Sunderbans).

India. Scarcity of food as a reason for malnutrition may be true at family level but not when we are speaking of the whole of India. India still produces enough amount of food to feed its millions, but its uneven distribution of food which makes things worse. To add to the misery, Asia shares the largest chunk of human population but the smallest bundle of food.

Consequences (Fig. 3.4)

Prevention. Malnutrition is often neglected over supposedly more 'pressing' problems. The Copenhagen Consensus, ranked micronutrient supplements as number one priority.[76] However, roughly $300 million of aid goes to basic nutrition each year, *less than $2 for each child below two* in the 20 worst affected countries. In

contrast, HIV/AIDS, which causes fewer deaths than child malnutrition, received $2.2 billion = $67 per person with HIV in all countries.[77]

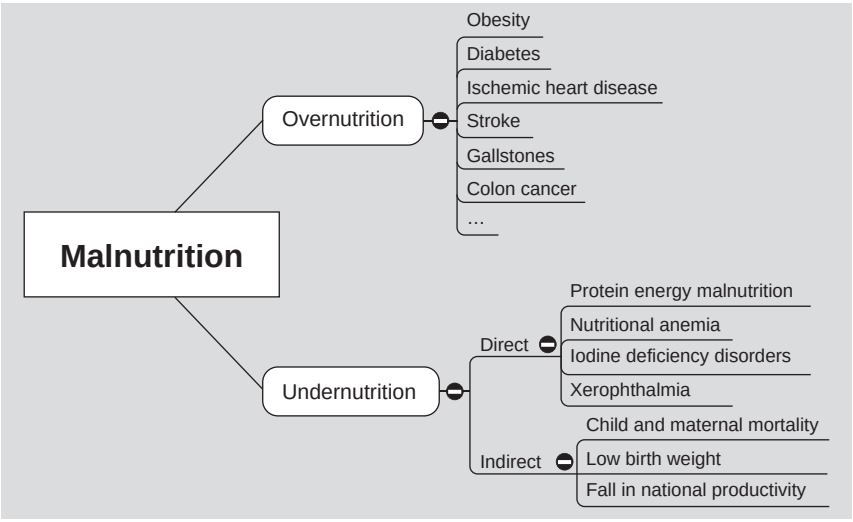


Figure 3.4. Effects of malnutrition

Primary prevention

Health promotion

- Education on nutrition and diet planning (quality + quantity + method of preparation).
- Agricultural development—Many problems now plague the farmers of India. Our population growth has superceded the capabilities of the green revolution. Climate changes make wild more unpredictable. Farmers find more profit in cash crops (coffee, cotton) than cultivating staple food(i.e. wheat). Pulses have to routinely imported. Food prices are skyrocketing, but occassionally farmers have to commit suicide because of excess yield and sudden drop of prices. Farming is losing its prospect and many rural children are abandoning the farms for a city life. It is imperative to put our 'self sufficiency in food' back together quickly.
- Effective **food distribution system**—Most of the famines in the last century in India were caused by a few wholesellers piling up food in their cold storages, and releasing them slowly to skyrocket prices. The rationing system was introduced in India to provide equitable food distribution, but overtime it has weakened and runs in very few places in India.

> ...no matter how a famine is caused, methods of breaking it call for a large supply of food in the public distribution system. This applies not only to organizing rationing and control, but also to undertaking work programmes and other methods of increasing purchasing power for those hit by shifts in exchange entitlements in a general inflationary situation
>
> *—Amartya Sen, nobel laureate economist, in "Poverty and Famines"*

- Socioeconomic development (**income generation**)—A great example has been set by Muhammad Yunus (another nobel laureate) called 'The Grameen Bank'. It provides small loans to help very poor women generate income and those loans can lift women out of poverty, and yet yield nutritional benefits. Some studies (Gardner, et al. see references) show when a woman is provided with an income, she will spend nearly all of it on household needs, especially food. Therefore, by focusing on women empowerment, poverty can be reduced, and also malnutrition, especially hunger can be fought, environmental sanitation; family planning.
- **Improve status of women**—Hunger disproportionately affects females more so than males (Gardner, et al.), due to a myriad of causes (they are 'expected' to eat less than men, but do all the housework and still deliver a baby every year, without respite). If women are able to earn enough money and get education, they can at least have the chance to achieve a more equal status with men, thus reducing a gender bias that 'men require more food than women'.
- **Improve sanitation**—The **sanitation barrier** stops infectious diarrhea, which is a perpetrator of malnutrition.
- Stabilization of **population**—If the population grows out of bounds, as it is today, India (and later the entire human civilisation) is bound to land in a Malthusian[78] trap of diminishing returns.
- International collaboration—The UN World Food Program, and several agenices such as FAO, UNDP and CARE work for improving the nutrition in underdeveloped countries, and to provide emergency nutrition during disasters.

Specific protection

- Nutritional supplementation (*supplementation* implies food that is consumed in addition to the daily meals).

Cash or food? In areas where food is available but unaffordable, giving cash may be more appropriate than food. The UN's World Food Program, the biggest nongovernmental distributor of food, announced that it will begin distributing cash and vouchers instead of food in some areas.[79]

- Immunization—Most infectious diseases of childhood potentiate malnutrition
- Food fortification
- Food surveillance—To make a framework of monitoring quality of food that is distributed to public, and take appropriate action; in India, Food Inspectors are appointed for carrying out surveillance.

Food fortification

Food fortification is

- Deliberate addition of
- One or more micronutrients to particular foods
- So as to increase the intake of these micronutrient(s) in order to
- Correct or prevent a demonstrated deficiency
- Provide a health benefit.[80]

Contd...

Contd...

Examples. Fluoride in water, iodide in salt, vitamin A and D in vanaspati, iron in wheat.

Criteria

- The vehicle must be consumed constantly as part of regular diet.
- The amount of nutrient added must provide an effective supplement even if vehicle is consumed in low amounts, and cause no hazard on high consumption of vehicle.
- The addition should not cause noticeable change in color, taste, smell, appearance or consistency of vehicle.
- The cost of fortification will not raise the price of the vehicle beyond the reach of the population in greatest need.
- Should be consumed *soon* after production.
- The production, distribution and consumption of the food should be possible to monitor.

Advantages

- More effective than intermittent supplements.
- Fortification of widely consumed foods can improve the nutritional status of a large proportion of the population, both poor and wealthy.
- Fortification requires neither changes in existing food patterns (which are notoriously difficult to achieve), nor individual compliance.
- In most settings, the delivery system for fortified foods is already in place.
- Multiple micronutrients can be added in single food.
- Fortification usually does not increase cost of the food.

Disadvantages

- Not a substitute for a good quality diet.
- Everyone in the population is exposed to fortified food, irrespective of whether or not they need it.
- Infants and young children, who consume relatively small amounts of food, do not get their required micronutrients from fortified food alone.
- Fortified foods often fail to reach the poorest people, often due to underdeveloped distribution system; these people often rely on locally produced food.
- The fortificant may affect the acceptability of vehicle; for example, some iron fortificants change the color and flavor of many foods, and can destroy vitamin A and iodine.
- Interactions can occur between two fortificant nutrients; for example, the presence of large amounts of calcium can inhibit the absorption of iron.

Secondary prevention

Early diagnosis

1. Diet survey of the community/sample to detect hidden malnutrition.
2. Recognize early physical signs of malnutrition (by anthropometry and growth monitoring).

Prompt treatment

1. Prescription of improved diet within economic restraints
2. Treating infections/malabsorption syndromes
3. Nutritional supplementation for the malnourished (i.e. ICDS program)
4. Specific therapy (i.e. treatment of xerophthalmic children with oral or parenteral vitamin A in hospital set up).

Tertiary prevention

Disability limitation. Recognize malnutrition early; intensive and immediate therapy before disabilities (bony deformities in rickets, neurological defects in Beriberi, keratomalacia in vitamin deficiencies) set in

Nutritional rehabilitation. JM Bengoa, in his classic paper of 1976[81] > introduced 'Nutritional Rehabilitative Services' as a combination of

- Hospital treatment of severe malnutrition
- Ambulatory (home) treatment by supplementation and educating the mother
- Nutritional rehabilitation centers.

Protein energy malnutrition

It comprises a spectrum of conditions arising from coincident lack of protein and calories, occurring most frequently in young children (1–3 years) and associated with recurrent infection.

Problem

PEM is a major public health problem. Severe forms of the disease are only 1–2% and represent the tip of the iceberg (Marasmus prevailing over Kwashiorkor). Moderate and milder forms occur in many of under five children. 1 case of PEM = 10 hidden cases of borderline malnutrition.

Agent

1. ↓ food – Both in quality and quantity
2. ↓ calorie intake (thus protein gets used upto generate energy).

Host and environment factors

See factors of malnutrition earlier.

Classification

Classification of PEM is based on difference of 'actual' weight of the child and its 'expected' weight, i.e. the 50th percentile[82] from a randomized weight survey. Expected weights have been determined by WHO (from the survey conducted by National Center for Health Statistics) and growth chart, the 'Road to health', was prepared by **David Morley**. Because Asian children are genetrically smaller than the Americans, the Indian academy of Pediatrics assumes that any child who has at least 80% of the weight of the 'expected' weight from NCHS chart, is normal.

Table 3.11. Indian Academy of Pediatrics – Classification based on weight for age

Normal	> 80% of expected
Grade I malnutrition	71–80% of expected
Grade II malnutrition	61–70% of expected
Grade III malnutrition	51–60% of expected
Grade IV malnutrition	< 50% of expected

Another classification, based on how many standard deviations away the child is from the 'expected' was devised by JC Waterlow. This classification gives equal emphasis on both weight and height. A child who was growing normally but acutely malnourished becomes wasted, while chronic malnutrition pulls down the height of the child (stunting). The **height for age** of a child is the ratio of his/her height to the height of a 'normal' child of the same age. Similarly, the **weight for height** is the ratio of the weight of the child to the weight of a 'normal' child of the same *height*.

Table 3.12. Waterlow classification [JC Waterlow, BWHO 1977;55:489]

Height for Age → Weight for height ↓	**Normal (> -2SD)**	**Stunted (< -2SD)**
Normal (> -2SD)	Normal	Stunted
Wasted (< -2SD)	Wasted	Wasted and stunted

A third classification, based on clinical syndromes, was given by the Wellcome trust.

Table 3.13. Wellcome trust classification

Kwashiorkor	60–80% of expected + edema
Marasmus	< 60% of expected – edema
Marasmic kwashiorkor	<60% of expected + edema
Undernutrition	60–80% of expected – edema

Clinical course

Mild to moderate PEM (Grade I and II)

Slow, inactive child with ↓ weight for age and disproportionately large head circumference. Grade I and II PEM may not be apparent clinically and detected only by anthropometry.

Severe malnutrition (Grade III and IV)

Table 3.14. Kwashiorkor and marasmus

Kwashiorkor	Marasmus
The term 'kwashiorkor' was coined by Cecil Williams in 1935, which means 'weaning of first child from breast when second is born'. It is the pathologic adaptation to malnutrition with excessive secretion of *insulin*. A role of *aflatoxins* has also been postulated in kwashiorkor.[a] Uncommon Edema (due to hypoalbuminemia and ↑ vascular permeability) is pathognomonic; edema may give a false sense of build Loss of appetite (thus the child is very difficult to treat) Dry, brittle, lustreless hair, flag sign (alternating bands of gray and black zones in malnutrition, suggesting periods of malnutrition intervened by good nutrition) Dry, hyperkeratotic skin with ecchymoses; alternate hypo/hyperpigmentation ('pavement' dermatosis) Fatty liver Psychic changes (the child becomes *very* irritable) High case fatality	Marasmus is a kind of 'balanced' starvation where the body responds with excess of *cortisol*. It is commoner that Kwashiorkor, and more easy to detect. It occurs only below 2 years of age. Severely wasted (emaciated) and stunted child 'Balanced' starvation 'Old Man' face (loss of buccal fat), wrinkled appearance, sparse hair Dry and inelastic skin No edema or fatty liver Good appetite Better prognosis

[a]"Protein-Energy Malnutrition (PEM) and Undernutrition: Causes, Consequences, Interactions and Global Trends", Johns Hopkins Bloomberg School of Public Health, under Creative Commons Attribution Noncommercial Share Alike license

Diagnostics

Weight for age. This is the principal method to detect PEM. But a single measurement is not enough to tell PEM because it won't give the child's growth history. A constant recording (**growth monitoring**) is needed.

The first curve denotes the 80% of the median proposed by NCHS (which is the standard normal for Indian children). Subsequently, there is 70, 60 and 50 percents of the median. Any child showing a weight below the first curve is malnourished. Indicated in the chart are the grades of malnutrition. Grade I malnutrition is *mild*, Grade II is *moderate*, and Grade III and IV are *severe* malnutrition.

Mid arm Circumference (MAC). It is a very useful reference as it is constant between 1–5 years. It is useful for assessing thinness, used in community nourishment survey. Reduction of MAC is one of the most prominent way the body makes up for malnutrition. Children < 1 have more fat than muscle, and thus MAC is not measured.

Table 3.15. Mid arm circumference

MAC at 1–5 years	Malnutrition
>13.5 cm	Normal
12.5–13.5 cm	Moderate malnutrition
<12.5 cm	Severe malnutrition

Prevention

See prevention of malnutrition (the previous section).

Treatment of PEM. Severe PEM must be hospitalized or admitted in a Nutritional Rehabilitation Center. Mild to moderate PEM may be treated at home, if provisions of a good diet can be made. Included in a good diet are breast milk, liquid feeds of skimmed milk, oil, sugar, cereal gruels with milk, oil, sugar, ripe fruit, cooked vegetables. Establish a daily, graduated intake of [83]

- 3–4 g protein per kg (actual) body weight
- 200 kcal of energy per kg body weight.

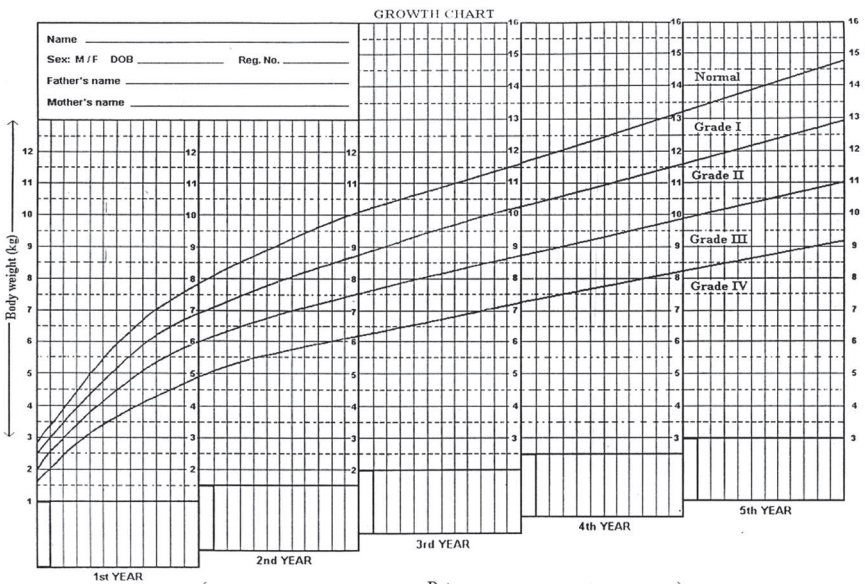


Figure 3.5. This is the graph standardized by the Indian Academy of Pediatrics, and used for growth monitoring in India (by ICDS)

Nutritional anemia

It is subnormal levels of hemoglobin due to deficiency of one or more essential nutrients (iron, B_{12}, folate). Most preschool children and pregnant women in nonindustrialized countries, and at least 30–40% in industrialized countries, are iron deficient.[84] In India, for example, upto 88% of pregnant and 74% of nonpregnant women are affected.[85]

Agent

1. Because animal iron (heme iron) is greater absorbed than plant iron (nonheme iron) – Most Indians who do not have the fortune to consume sufficient animal food suffer from low bioavailability of iron. Again, egg, milk and tea reduce absorption of iron.
2. Chronic blood loss, i.e. Hookworm infestations, malaria, menstruation are major causes of IDA.
3. Deficiency of folate is rare; B_{12} deficiency can occur in those taking only a vegetarian diet.

Host

Age

Full-term infants are normally born with adequate iron stores. Breast milk is relatively low in iron, although the iron in breast milk is much better absorbed than that in cows' milk. Iron deficiency commonly develops after six months of age if complementary foods do not provide sufficient absorbable iron.

Iron requirements are proportional to growth velocity. Accordingly, iron deficiency is most common in the preschool years and during puberty. Another peak may occur in old age. Worldwide, women of reproductive age are prone to IDA because of physiological losses.

Sex

Following menarche, adolescent females often do not consume sufficient iron to offset menstrual losses.

Increased iron requirement

Substantial amounts of iron are deposited in the placenta and fetus during pregnancy. This results in an increased need of about **700–850** mg iron over the whole pregnancy. *Lactation* results in loss of iron, via breast milk. Consequently, for some women a deficiency developed during pregnancy may be perpetuated during lactation. **Repeated close pregnancies** exhaust the iron stores of the mother.

Pathological states

Common infections like malaria (by hemolysis), hookworm, trichuriasis, amoebiasis, and schistosomiasis cause blood loss directly. This blood loss contributes to iron deficiency.

Environment

Low socio-economic status correlates very well with IDA, so does open field defecation (perpetuates hookworm infection). The excess of organic acids in Indian diets (phytates, oxalates) seriously impair iron absorption.

The danger

In pregnancy, anemia is one of the major complicating factors and leads to increased chance of abortions and IUGR, low birth weight, perinatal mortality, maternal morbidity and infections. In children, IDA causes impaired cognitive

function, immunosuppression and ↓ work capacity, decelerates growth, causes abnormal thyroid function and these children are more prone to develop heavy metal toxicity when ingested[86] (because their intestinal capacity for absorbing metals is greatly raised).

Diagnosis of anemia (Hb values in g/dL)[87]

1. Adult male < 13
2. Adult female < 12
3. Adult female pregnant <11
4. 6 months–6 years <11
5. 6–14 years <12
6. MCHC < 34 in any age.

Severe anemia in pregnancy is defined as Hb < 7, and when it goes below 4, it is termed *very* severe. Additionally, stool tests for hookworms is recommended in enemic areas.

Indicators of iron status

- Hemoglobin—To be measured either in the laboratory by cyanmethemoglobin method or in a portable Hemo Cue system
- Serum ferritin < 15 μg/dl indicates a storage iron deficiency
- Serum transferrin saturation < 16% in IDA (normally 33%)
- Serum iron
- Bone marrow iron stain—The definitive test for iron stores.

Prevention

Primary prevention

Health promotion. Nutrition education, **one extra meal** daily in pregnancy; weaning at 'right time, right food, right amount'; family planning; sanitary latrines and use of shoes in the fields (for hookworms); ↑ economic status of people. Promoting the availability of iron-rich foods (meat, fowl, fish, and poultry, legumes and green leafy vegetables), and foods which enhance the absorption of iron (vitamin C). People in lower SES will benefit from developing a kitchen garden or a community farm.

Methods of preparation influence the bioavailability of iron. Cooking, fermentation, or germination can reduce the phytic acid and the hexa- and penta-inositol phosphate content. Iron absorption can vary from 1% to 40%, depending on the mix of enhancers and inhibitors in the meal. **Inhibitors** include

- Phytates, present in cereal bran, cereal grains, high-extraction flour, legumes, nuts, and seeds
- Inositol
- Tannins (tea, coffee, cocoa)
- Calcium, particularly from milk and milk products.

Examples of simple but **effective alterations in meal patterns** that enhance iron absorption

1. Separate tea drinking from mealtime—One or two hrs later
2. Include in the meal some source of vitamin C

3. Consume milk, cheese, and other dairy products as a between meal snack, rather than at mealtime.

Specific protection

Iron and folate supplementation have been taken up by the Government of India since the fourth-fifth year plan for pregnant mothers, lactating mothers and children below 12. The program now also includes adolescents.

Table 3.16. Therapy – Prophylaxis of IDA (Hb 7–11g%)

Subjects	Iron tablets	Dosage
All pregnant women	100 mg iron + 500 μg folate	Prophylaxis: 1 tab × 1 × 100 days Therapeutic: 1 tab × 2 × 100 days
Children under 6 years	20 mg iron + 100 μg folate	Therapeutic: 1 tab (or 1 ml syrup) × 1 × 100 days, preferably in liquid form in 1 ml marked bottles
Children over 6 years	30 mg iron + 250 μg folate	1 tab × 1 × 100 days
Adolescents	Same as adults	

Additionally, the program includes treatment for hookworm infection as Mebendazole 100 mg × 2 × 3 (except in 1st trimester of pregnancy)

Iron fortification. Several food items can be fortified with iron such as rice (Philippines), cereals (Sweden) and wheat (Chile, UK, USA). The National Institute of Nutrition has devised a method of fortifying salt with Fe-orthophosphate, and production has begun since 1985.

Secondary prevention

1. Early diagnosis—Clinical and laboratory examination of women (especially pregnant women) for anemia
2. Treatment.

Vitamin A deficiency

Problem

Vitamin A deficiency is the leading cause of preventable blindness in children.[88] It is especially common in south and east India, where rice predominates in diet (which has almost no vitamin A).

Agent

↓ intake of vitamin A rich food. Remember that in vegetables, β carotene is present and not vitamin A, which is 50% absorbed converted to vitamin A in a ratio of **12:1**. Thus a strictly green vegetable diet is not essential to meet vitamin A requirement.[89]

Host

1. Discarding colostrum (the first source of vitamin A for the newborn)
2. Measles infection
3. Malabsorption and diarrhea
4. Delayed weaning (i.e. starting complementary feeding after 1 year)
5. **Pregnancy** increases vitamin A demand and as much as 7.8% of pregnant women in 'countries at risk' (see later) have night blindness.

Environment

Vitamin A deficiency occurs in an environment of social deprivation, low social status, high population density and inadequate health care facilities. The WHO has defined 'country at risk of vitamin deficiency' as any country where GDP < $15000 (2005). In these countries at risk, the prevalence of night blindness among preschool children is 0.9%, and the prevalence of biochemical vitamin A deficiency (serum retinol < 0.7 μmol/l) is a staggering 33.3%.[90]

Clinical course

The table shows features of vitamin A deficiency in sequence and criteria for accounting a public health problem (prevalence in preschool children of 6). In addition, vitamin A deficiency also causes anemia indirectly and predisposes to recurrent infections.

Table 3.17. Older WHO criteria of Vitamin A deficiency (1982)

Parameter	Value
Night blindness	> 1%
Bitot spot (Triangular, pearly white, foamy spots on bulbar conjunctiva)	> 0.5%
Corneal xerosis/keratomalacia	0.01%
Corneal ulcer	> 0.05%
Plasma vitamin A < 10 μg/dl	> 5%

The recent WHO guidelines recommends a serum retinol < 0.7 μmol/l as vitamin a deficiency and < 0.35 μmol/l as *severe* vitamin A deficiency.[91]

Prevention

Primary prevention

Health promotion. Improving the availability and intake of vitamin A through dietary diversification of food; nutrition education to change dietary habits, as well as providing better access to vitamin A or provitamin A rich foods, such as mangoes, papaya, or **dark green** leafy vegetables; setting up kitchen gardens is a good way for supply of these foods.

Specific protection. Vitamin A is stored in body for nine months, and this is taken advantage of by prophylaxis dosage which is given every 6 months. ALL children of 6 months–3 years must be covered under vitamin A prophylaxis.

The National Institute of Nutrition schedule

Between 9–36 months of age 5 **mega doses of retinol palmitate in arachis oil** are given at 6 months interval, along with vitamin A rich foods. The dose below 12 months of age is 100000 IU and after 12 months, 200000 IU.

For administrative convenience, 1st dose is given with measles vaccine; 2nd dose is given with DPT-B at 18 months.

Because vitamin A is photodegradable, the bottle should not let in light and stored in a cold dark room. Its shelf life is 1 year at room temperature, but has to be used within 6–8 weeks once it has been opened. Vitamin E is often added in the bottle as an antioxidant (Fig. 3.6).

Figure 3.6. The pack of UNICEF vitamin A bottles, and the bottle itself

Many high-risk countries have also adopted the WHO policy of supplementing mothers with a 200000 IU oral dose of vitamin A within six weeks after delivery to enrich their breast milk content of vitamin A, but not yet implemented in India.

Vanaspati ('dalda') is now also **fortified** with vitamin A.

Secondary prevention

Early diagnosis and treatment. For every case diagnosed as vitamin A deficiency, give the 1st dose immediately → Repeat dose after a month (according to the age of the child) → follow up with the regular prophylactic dose schedule 6 weeks later. Along with, provide vitamin A rich food.

For corneal lesions. Treat as an EMERGENCY within 48 hrs with 100000 IU × 2 days + 200000 IU after 2 weeks, or else an ulcer develops. If not treated within 3–4 days, it will cause a permanent scar. If the child cannot eat or drink at all, use a water soluble formulation of vitamin D for injection.

Monitoring. Monitor areas with identified night blindness/measles outbreak.

Iodine deficiency disorders

These include a host of diseases such as goiter, myxedema and cretinism. From being confined to the Himalayan goiter belt – IDD has spread to almost every state of India.

Agent

↓ dietary intake of iodine (RDA 150 μg)

Host

IDD is common in school children and adolescent girls.

Environment

Crops grown in iodine deficient soil are prime cause of iodine deficiency (i.e. where iodine is washed away repeatedly by rainfall, or areas which were once under large glaciers).

Clinical course

1. Fetus—Abortion, stillbirth, congenital anomaly, neurological cretins (spasticity), myxedematous cretins (dwarfism).
2. Neonate—Goiter, hypothyroidism.
3. Child/adolescent—Goiter, hypo/hyper thyroidism, impaired mental functions, hypogonadism.
4. Adult—Goiter, hypothyroidism, impaired mental functions.

Indicators[92]

- Prevalence of goiter—Not very useful, because it only includes the clinical cases; also goiter remains even months after iodine deficiency has been corrected and recovers only slowly.
- Prevalence of cretinism.
- **Urinary iodine excretion**—Most sensitive indicator of recent change in iodine intake.
- T_3, T_4 measurement.
- Prevalence of neonatal hypothyroidism (TSH).

While IDD affects the entire population, a school-based sampling method (children for 6–12 years) is recommended. **Iodine deficiency** is considered to be a public health problem in populations of school-age children where the **median urinary excretion is below 100 μg/l or goiter prevalence is above 5%**.

Goiter

A thyroid glands whose lateral lobes have a volume greater than the terminal phalanx of the thumb of the patient.

Table 3.18. Grading of goiter

Grade 0	Neither palpable nor visible
Grade 1	Only palpable, not visible in normal position of neck
Grade 2	Palpable and visible in normal position
Grade 3	Visible from distance

If > 10% of children (8–12 years)/population are found to be goitrous, it is endemic goiter.

Prevention and control

Primary prevention

No health promotive measures will meet iodine demands. We need specific iodine supplementation.

Specific protection

1. Measures for whole population (iodized salt)
2. Prescriptive measures suitable for children, women of reproductive age, living in insensitive areas (iodized oil).

Iodized salt. Common salt fortified with KIO_3 (less soluble and stable than KI). At production level – iodine is given not less than 30 parts per million during production,[93] and 15 ppm should remain (i.e. should not have sublimated) during consumption (Prevention of Food Adulteration Act). **Salt as a vehicle** is universal, consumed at same level throughout the year, mixing salt and iodine is simple without any adverse reactions, processing can occur in a large scale.

Test for iodized salt. Two solutions, one white for acidic salt and one red to convert alkaline salt to acidic, come in a pack. It gives violet color with iodized salt > 30 ppm.

Iodized oils. Effective for regions where quick iodine is needed (children and women of reproductive age), but iodized salt is not available. Average 1ml injection provides iodine for 4 years.

Secondary prevention

Iodine monitoring. It is important monitor the ecology of iodine, determination of iodine in salt (too much iodine may cause iodine induced hyperthyroidism), and iodine nutritional status in public (prevalence of goiter, neonatal hypothyroidism, radioiodine uptake, urine iodine excretion).

Criteria for monitoring progress towards sustainable IDD elimination

- Salt iodization coverage (proportion of households consuming adequately iodized salts) > 90%
- Proportion of population with urinary iodine levels below 100 μg/l < 50%
- Proportion of population with urinary iodine levels below 50 μg/l < 20% and at least 8 of the 10 below should be present
    1. National body for IDD elimination
    2. Political commitment to salt fortification and elimination of IDD
    3. A responsible executive officer for the IDD elimination program
    4. Legislation of salt fortification
    5. Commitment to regular progress in IDD elimination, with access to laboratories able to provide accurate data on salt and urinary iodine
    6. Program of public education on the importance of IDD and iodized salt
    7. Regular data on iodized salt at the factory, retail and household levels

8. Regular laboratory data on urinary iodine in school-age children
9. Cooperation from the salt industry in maintenance of quality control
10. A database for recording monitoring procedures particularly for salt iodine, urinary iodine and, if available, neonatal TSH.

Endemic fluorosis

A continuous ingestion of water with 3–5 mg/l of fluorides can result in dental fluorosis (mottling of enamel and erosion of teeth), skeletal fluorosis (deposition of fluorine in bones, osteoporosis, genu valgum).

Intervention

An ideal fluoride level is 0.5–0.8 mg/l (any less fluoride may cause dental caries).

1. Change in water source: Surface water contains lower quantities of fluorine (and unfortunately, lower quantities of other minerals too)
2. Defluoridation—The Nalgonda Technique (lime and alum precipitation) is the cheapest method
3. Other measures—Prohibit fluoride fortified water (0.5–0.8 ppm)/toothpaste in endemic areas.

Solutions for nutritional problems

Indirect interventions

1. Nutritional education
2. ↑ Family diet
3. Sanitation
4. Effective food production and distribution system
5. Family planning
6. Health services
7. ↑ SES
8. Education.

Direct interventions

1. Short and medium term measures (supplementation, fortification, etc.).
2. Nutritional programs.

National Program of Nutritional Support to Primary Education, 2006 (Mid-Day Meal Scheme)

The world's largest school feeding program reaching out to about 12 crore children in over 9.50 lakh schools

In contrast to the earlier mid-day meal program, the revised program adds more emphasis on

- Monitoring.
- Subsidizing the food as well as transport and cooking expenses.
- Supplying micronutrients.
- Food supply in draught affected areas.
- More community participation in program management.

- To ensure that the program does not interfere with the purpose of schools (i.e. learning).
- Intersectoral coordination with Ministry of Rural Development, Panchayati Raj for building kitchen cum stores and ensuring good water supply in schools.

Objectives

- Improve nutrition of primary schoolchildren.
- Improve school attendance (specially of poor students) and performance of students.
- Providing nutritional support to children in draught affected areas.

Beneficiaries

Primary schoolchildren (6–11 years).

Components

Mid-day meals *supplement* home diet (not substitute). The earlier recommendations were to provide at least **1/3rd of calorie and ½ of protein req × for at least 200 days per year. The revised program in 2006 plans to provide at least 450 kcal and 8–12 grams of protein + adequate amount of micronutrients daily for every schoolchild**.

Suggestions for food preparation

- Foodgrains must be stored in a place away from **moisture**, in air tight containers/bins.
- Use whole wheat, broken wheat (dalia), parboiled or unpolished rice.
- 'Single dish meals' using combination of wheat/rice, a pulse, a vegetable, and edible oil is both nutritious and time saving (i.e. pulao, khichdi, upma, dal-vegetable bhaat).
- **Cereal: Pulse in 3:1 to 5:1 combination** is necessary to have good quality proteins.
- Use **sprouted** pulses.
- Vegetables should be thoroughly washed **before** cutting.
- Soaking of rice, dal, bengal gram, etc. reduces cooking time.
- **Rice water** (which is rich in B vitamins) left after cooking should be mixed with dal and never be thrown away.
- **Fermentation** improves nutritive value; preparation of idli, dosa, dhokla, etc. may be encouraged.
- Cooking must be done with the **lid** on to avoid loss of nutrients.
- Avoid over cooking.
- Avoid reheating of oil used for frying.
- Leafy tops of carrots, radish, turnips, etc. should not be thrown away but utilized.
- Only iodized salt should be used for cooking mid-day meals.

Expenditures

- Supply of free food grains (wheat/rice) 100 grams per child per school day from the nearest Food Corporation of India godown.

- To ensure that the food gets cooked and not distributed in raw form; the cooking cost of food per child was set at Rs 1.50 to 1.80, and a small percentage of it is to be paid by state government.
- Subsidy for transportation of food grains between Rs 75–100 per quintal.
- Supply of food grains to schoolchildren even during vacations in drought affected areas.
- Provide kitchen cum store and kitchen utensils/devices in schools, in collaboration with Ministry of Rural Development.
- States have to spend 1.8% of total central assistance on monitoring the scheme.

Monitoring

- Local level—Representatives of Gram Panchayats/Gram Sabhas, Parent's associations as well as Mothers' Committees are encouraged to monitor the program.
- Display of information (under Right to Information Act) about quality and quantity of food being used, daily.
- Inspections by state government officers.
- The Food Corporation of India is responsible for the continuous availability of adequate food grains in its depots.
- The state government is also required to submit periodic returns to the Department of School Education and Literacy on running the program.
- Monitoring by institutions of social science research.
- States are required to develop a mechanism for receiving complain from public.

National IDD control program (earlier, Nation Goiter Control Program, 1962)

Figure 3.7. The smiling sun logo for iodized salt

Objectives

Reduce incidence of IDD in endemic districts to < 10% by 2000.

Ensure that salt produced, purchased, sold and consumed is iodized > 30 ppm.[94]

Beneficiaries

Everybody.

Components

1. Initial survey to asses magnitude of IDD
2. Supply of iodized salt (Fig. 3.7)

3. Resurveys for evaluation every 5 year.

No region in the country was found to be exempt; thus iodization of salt of entire country started in 1986; in 1992, in view of the broad spectrum of IDD, the name of the program changed.

Weaknesses

1. Lack of awareness
2. Lack of salt monitoring at all levels
3. Lack of intersectoral coordination
4. Salt manufacturing is still mostly outsourced and ran as a private business.

Vitamin A prophalaxis program (1970)

Objectives

Prevention of vitamin A associated blindness among under fives.

Beneficiaries

9 months–3 years old children.

Components

1. Supplementation (See Vitamin A Deficiency Prevention earlier)
2. Balanced diet with Vitamin A rich food
3. Measles vaccination
4. Additional attention to areas with night blindness/measles.

Nutritional anemia prophyaxis program (1970)

Objectives

Reduce iron deficiency anemia, thus ↓ MMR and low birth weight, and improve child nutrition.

Beneficiaries

1. Pregnant and lactating women
2. Under five children
3. Intrauterine contraceptive device users.

See section on 'nutritional anemia'; the program is now merged with RCH.

Integrated Child Development Sevices

See the chapter on maternal and child health.

Status of principal foods

Cereals[95]

Cereals, grains or cereal grains, are grasses (members of the monocot families Poaceae or gramineae). Cereal grains are grown in greater quantities and provide more food energy worldwide than any other type of crop. In their whole grain, they are a rich source all nutrients. However, when refined by the removal of the bran and germ, the remaining endocarp is mostly carbohydrate.

Most cereals are rich in B vitamins and minerals, but deficient in **lysine and threonine**. **Rice**, the staple of South Asia, has a calorific value of 3.45 kcal/g and 6.8g proteins per 100g, but is not a very good source of lipid soluble vitamins, and deficient altogether in calcium and iron.

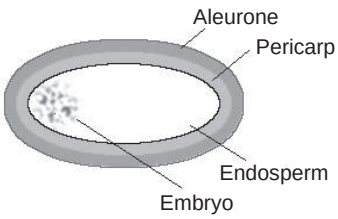


Figure 3.8 A rice grain

The endosperm is the food for the embryo and made entirely of starch. It is the aleurone, pericarp and embryo itself that contain the protein and vitamins (Fig. 3.8). *Milling* removes the outer coat and thus removes both proteins and vitamins from rice.

Parboiling

Parboiled ('partially boiled') rice is rice that has been boiled in the husk. Parboiling rice drives nutrients, especially thiamine, from the outer coats (bran) to the endosperm, so that parboiled white rice is 80% nutritionally similar to brown rice.

Method. Paddy is first hydrated (in water of 65–70°C for 3–4 hrs), → drain the water → steaming for 5–10 minutes. The pressure of steam pushes the nutrients inside.

Parboiled rice is more resistant to insects and suitable for storage. The starches in parboiled rice become gelatinized, making it harder and glassier than other rice. *Atta* is the main ingredient of most varieties of Indian bread. It is a whole wheat-flour made from hard wheat grown across the Indian subcontinent. Flat bread made from atta, such as chapati, roti, naan and puri, are an integral part of Indian cuisine. Hard wheats have a high protein content, so doughs made out of atta flour are strong and can be rolled out very thin. Flour (maida) is refined than atta and lost many nutrients.

Pulses

Like many leguminous crops, pulses play a key role in crop rotation due to their ability to fix atmospheric nitrogen into soil. They have the same calorific value as rice (3.45), but contain four times the protein (24 mg/100g). However, most pulses (except sesame) is deficient in **methionine and cysteine**. Germinating pulses contain a lot of vitamin C, which is lost overtime.

Lentils (masoor likely originated in the Near East,[96] and has been part of the human diet since neolithic times. In 100g of lentils there is a whooping 26g of proteins,[97] 31g of dietary fibers, 0.87 mg thiamine and 7.5 m of iron (one of the best sources of vegetable iron).

Soybean is mostly protein (40g/100g), but is lacking methionine, and stuffed with phytoestrogens, which is suspected to be responsible for early menarche in Indian girls.

Green leafy vegetables

They are a very good source of all micronutrients, specially iron, calcium, B vitamins and folate. The fiber content of GLV makes it a necessity for all heart patients and diabetics, and those with constipation. However, bioavailability of calcium and iron from GLV is greatly hampered by presence of organic acids.

Roots and tubers

Potato is a mass of complex carbohydrates, and carrot is the best source of vitamin A. But they are lacking proteins and B vitamins.

Nuts and oilseeds

Nuts have very high content of unsaturated lipids and good quality proteins, B vitamins, calcium, iron and phosphorus.

Fruits

Fruits are generally very safe sources of food as the plant usually saves it best for the fruits, and it is well protected from any kind of contamination.They are rich in

- Vitamin A (everything that is yellow or orange)
- Vitamin C (any fruit, especially oranges, lemons and *amla*)
- Sodium and potassium (all fruits)
- Iron (raisin, dates)
- Carbohydrates
- Dietary fibers.

Animal flesh

Animal flesh provides proteins of high biological value, vitamin B_{12} and iron in the heme form. In the respect of fats, however, fish score well over most animals. Fish oils provide much needed polyunsaturated fatty acids (which is deficient in animal flesh), their livers are very good sources of vitamin A and D, and seafish also contain iodine.

Eggs

Chicken eggs supply all essential amino acids for humans[98] and provide several vitamins and minerals, including vitamin A, riboflavin, folic acid, vitamin B_6, vitamin B_{12}, choline (essential for fetal brain development), iron, calcium, phosphorus and potassium. They are also an inexpensive single-food source of protein. The egg is one of the few foods that *naturally* contain vitamin D. A protein called avidine in raw eggs impairs absorption of biotin, so that eating raw eggs is not a good idea.

Composition. A large egg (60g) contains approximately 70 kcal (egg white = 15 kcal, yolk = 55 kcal), 6g protein, 6g fat . The yolk contains more than two-thirds of the recommended daily intake of 300 mg of cholesterol. People on a low-cholesterol diet may need to reduce egg consumption; however, only 27% of the fat in egg is saturated fat (Palmitic, Stearic and Myristic acids) that contains LDL cholesterol. And you can always take the egg white which has very little fat.

Milk

Human milk is produced from 12th gestational week. It is no coincidence that the composition of milk varies with each species, and even within the same species, depending on gestational age. Mothers who deliver preterm have less dense milk than those at term, because GI tracts of premature babies are yet immature.

	Human	Cow
Protein (g)	1.1	3.2
Fat (g)	3.4	4.1
Lactose (g)	7.4	4.4
Energy (kcal)	65	67
Ca^{++} (mg)	28	120
Vitamin C (mg)	3	2
Mineral (g)	0.1	0.8
Water (g)	88	87
Iron (mg)	-	0.2
Bacterial contamination	None	Likely

Proteins

Human milk has 3 times less proteins than cow's milk (cow's milk needs to be at least 3 times diluted to be fed to infants). It is especially rich in cysteine, taurine and his (which are essential for neural development of the newborn) and is completely digested and utilized.

Fats

Cow's milk has greater amount of fat, but the proportion of PUFA is abundant in human milk (necessary for myelination of developing nerve cells).

Carbohydrates

Human milk contains lactose in a huge amount. The lactise breaks up to form *galactose* which goes on to form the sphingolipids, the component of myelin sheath. The lactose also favors development of *Lactobacillus bifidus* in the intestine of the newborn which eliminates other pathogenic bacteria.

Vitamins and minerals

1. Human milk provides vitamin A, D and B vitamins, copper, Se, Co, Ca
2. It is poor in Na, P and iron (but lactoferrin, the compound that binds iron in milk, has a very high degree of bioavailability, so that more percentage of iron gets absorbed).
3. A water soluble form of vitamin K (K3) is present.

Other molecules

Human milk contains secretory immunoglobulins (IgA), many macrophages, the enzyme lysozyme, interferons and some lymphocytes which impart some passive immunity in the gut of the newborn.

Milk products

Skimmed milk. Milk minus the fat (and thus no vitamin ADEK). It is a good source of proteins and Ca^{++} ions.

Toned milk. 1 part water + 1 part milk + 1/8 part skimmed milk powder. It is cheaper than milk and composition nearly equals cow's milk.

Vegetable milk. Milk prepared from groundnut or soybean may be used as an alternative.

Milk powder. It is similar to cow's milk in many respect and lacks immunologic molecules.

4 Health and the Community

KEY FEATURES

- PEOPLE
  - The demographic cycle
  - Demographic trends in India
  - Fertility
  - Family planning
- SOCIETY
  - Social medicine
  - Sociology
  - The family
  - Cultural factors in health and disease
  - Socioeconomy
  - Social security
- WOMEN AND CHILDREN
  - The maternity cycle
  - Problems of women and children
  - Growth and development
  - The RCH package
  - RCH II (2005–2010)
  - Integrated child development services
  - School health
  - Postpartum program
- THE ELDERLY
  - Problems of the elderly
  - Ideal geriatric health services
- INFORMATION, EDUCATION AND COMMUNICATION
  - Common terms
  - Communication
  - Health education

PEOPLE

Because Community Medicine is all about taking health care to the people, it is time that we study the anatomy and physiology of the population, before we could move on to pathology and medicine. **Demography** is the study of population, of their size, composition and distribution. It is equivalent to anatomy and physiology in internal medicine.

There are five processes which control the size, composition and distribution of any population.

- Fertility—How many are being added to the population?
- Mortality—How many of them are dying?
- Marriage—Which will eventually (though not always) lead to fertility?
- Migration—How many of them are going elsewhere or coming in?
- Social mobility—The change of social status of an individual or family over-time (i.e. if a street child manages to get a decent job and a home for himself and his family, he is said to be 'upwardly mobile' or have 'climbed the social ladder').

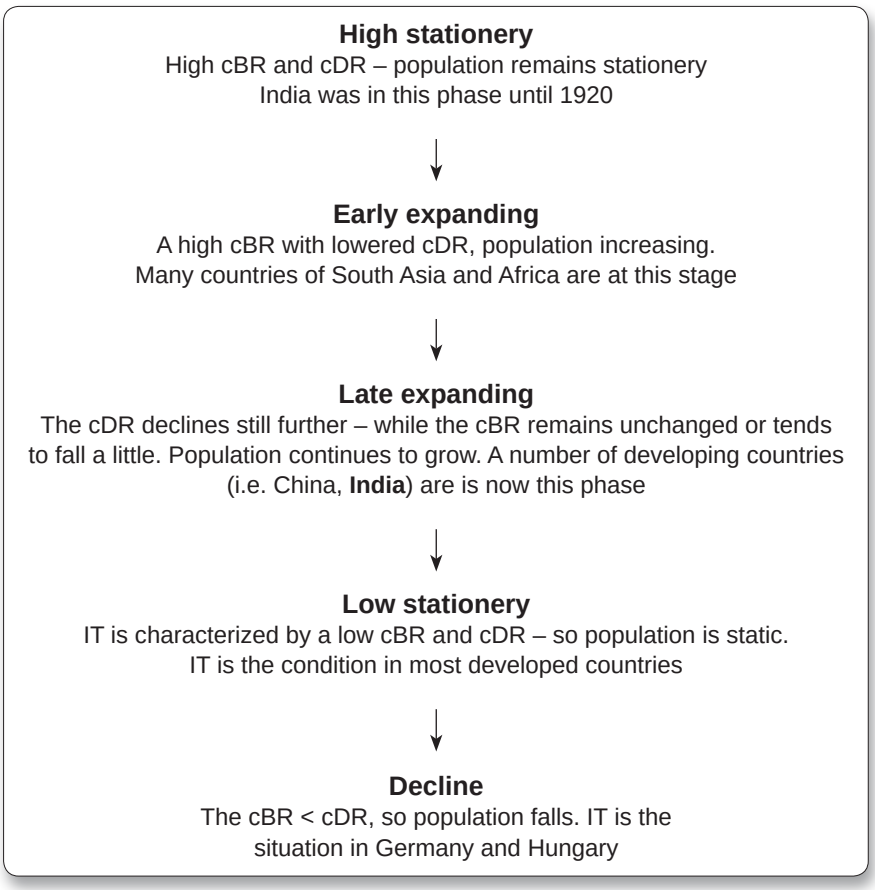
Demographic data can be collected from
1. Census.
2. National Sample Survey (an central government organization which conducts population surveys independent of census).
3. Registrations of birth, death and marriage.
4. Ad hoc demographic studies.

Two fundamental indicators

Crude Birth rate (cBR) is the number of live births per 1000 people in estimated mid year population, in a given year. It is given by the formula (number of live births during the year/estimated mid year population) × 1000. **Crude Death rate** is similarly defined for deaths in a year.

The demographic cycle

This is not truly a cycle, but a series of phase transitions that most of the nations go through.



World population trends

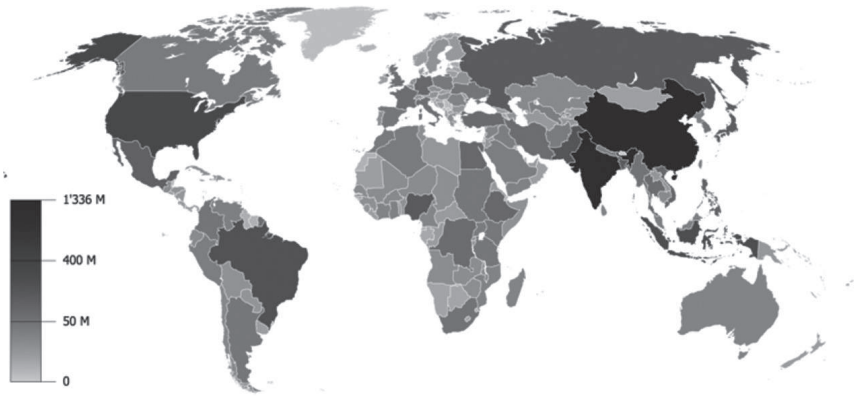


Figure 4.1. Distribution of world's population [Wikipedia user Emilfaro, under the Creative Commons Attribution Share Alike 3.0 License] based on the GeoHive estimates (www.geohive.com), obtained on the March 3, 2009

The world population touched 6 billion in 1999, and estimated to be 6.6 billion in 2007. It is no small wonder that the four countries of the South Eastern Asia Region (and five, if you include China), show up in the top 10 populous nations (Fig. 4.1) (India, Bangladesh, Pakistan, Indonesia). *Bangladesh*? You wonder. It is the size of a medium-sized state, and yet more populous than Canada or Egypt or France, all much larger in area. It seems India still fares better than its close neighbors.

The growth rate

The annual growth rate = (cBR – cDR + immigration rate - emigration rate) × 100. The **doubling time** of a population is approximately = 70/GR.

Table 4.1. State of nations according to growth rate

Slow growth	< 0.5
Moderate growth	0.5–1
Rapid growth	1–1.5
Very rapid growth	1.5–2

India is **very rapidly growing**, i.e. GR = **1.64** [2011 census provisional report].

World. In 2009 the estimated annual growth rate was 1.10%.[99] The growth rate is on the decline in recent times, although the population is still growing in actual numbers.

The demographic gap

IT is the factor cBR – cDR.

Demographic trends in India[100]

With a population of 1210 billion (2011 census) India houses 17.5% of world population) with 2.4% of its land.

Very rapid growth

The population is climbing since 1921, the year which is said to mark the 'big divide'. As per 2011 census, growth rate in India is 1.64 - very **rapid growth**. The population has increased by more than 181 million during the decade 2001–2011, however, 2001–2011 period is the first decade with exception of 1911–1921 which has actually added lesser population compared to the previous decade. Among the states and union territories, Uttar Pradesh is the most populous state and Lakshadweep the least.

Young age distribution

A **population pyramid** shows the age-sex composition of population in a single chart. India has a broad population base, i.e. huge number of dependent people below 15 years. Remember that every little toddler playing in the streets with a tire and a stick counts as one whole human being, and you will soon get the big picture. India is teeming with children, some of them have homes, parents, go to school and get two square meals a day, but most of them are lacking one or more of these.

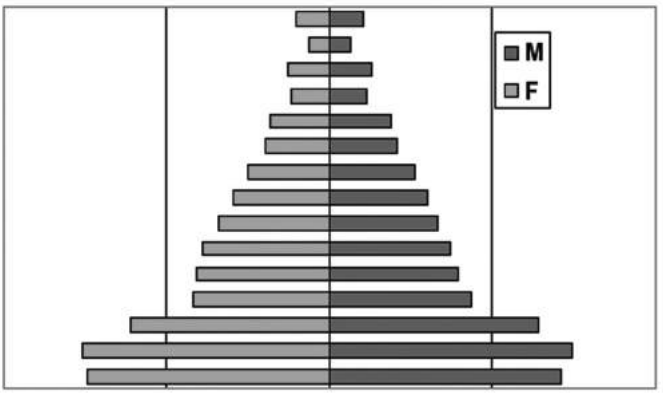


Figure 4.2. Population pyramid in India

In developed countries, this pyramid is thick at its waste, i.e. it is no pyramid at all but looks like a boem. Gradually, when each generation reproduces less than its forebearers, the pyramid will turn into an upside down. This upside down pyramid is the aim of the National Population Policy (an also its logo).

Figure 4.3. The logo of the National Family Welfare Program

Societal dependency ratio

It is the ratio of (people > 65 + people <15)/people of 15–65 years (working age). The large chunk of dependants in India are children (young age dependants). However, the dependancy ratio is an oversimplification, because everybody in the age group 15–44 is not employed, and many children under 15 have to support their families.

Sex ratio

It is the number of females per thousand males = 940 in India. Only two states are female predominant, Kerala and Pondicherry. However, the **child sex ratio** (0–6 years) is a dangerously low **914**, which is lowest since independance.

Why? It is a sordid truth that nobody in India wants a girl until she has turned sixteen. It is shocking to see the expression of profound gloom over the face of the minute old mother, when she is informed that she has delivered a girl. A boy is, supposedly, borne by God's infinite grace, and is presumed to be the returns of many prayers of the mother. If she comes home with a boy, her-in-laws are sure to hold her in high esteem than ever before, and the boy is reared with all the care the family can afford. A girl, in the other hand, is a scourge to majority of Indian families. Mothers are afraid even to mention that she has had a girl, and stories of the mother asphyxiating the newborn girl is not rare. If the girl child survives the neonatal period, she is treated with utmost indifference and disgust in many rural families, her health and education is usually ignored for the sake of her brother, if any, and she is pushed into housework 'to make a good wife'. And despite all this neglect, if she still manages to survive until her menarche, the parents get anxious to find a boy to hand this burden over. Girls in rural India are forced into marriage before they are through their teens, and their obstetric career is kick started by the husband, backed by his family. And soon, the girl child finds herself in the same position as her mother (if not, due to malnutrition or anemia, she dies of an obstetric accident). Add to this the problem of **trafficking** of women out of the country into the middle east for prostitution. Of course there are exceptions, and there are waves of change, but India still remains one of the worst places to be a girl.[101]

Density

The population density in India is **382/km^2**. The distribution of population is, however, very uneven and Bihar is the most population dense state. The effect of such a dense population is ↑ facilities and quality of life, and burgeoning pressure on natural resources.

Urbanization

Proportion of urban population in India is 27.8% in 2009.[102] There are two reasons

- The alarming tendancy of villagers to abort their native profession and gather in big cities for more profitable jobs, better health care, entertainment and chance of social mobility; however, most of them end up in slums, running small shops and adding to the already overcrowded city.
- Cities themselves are expanding at a drastic rate.

Literacy

A literate, by definition, is one who is > 7 years and can read and write any language **74.04%** of Indian population falls in this category.

Family size

It is the total number of children a woman has borne at a point of time. It depends on the duration of marriage, education of the couple, the number of live births, preference of male children (uneducated couples will go on reproducing until they have a boy), etc. The average family size in India is **2.8** (2006).

Fertility

In between 15–45 years, a woman can give birth to 15 children (a feat which is, however, seldom achieved). The number of actual children born to a woman is called **fertility**. (*Fecundity* is the capability to bear children). Causes of increased fertility in India are

1. **Universality of marriage**—Irrespective of financial safety, Indian families do not feel 'complete' until every child has been married.
2. **Low age of female marriage**—The Child Marriage Restraint Act (1978) still prohibits a marriage of girls before 18 and boys before 21. Whilst the mean age of female marriage in India has reached 20.5,[103] in rural areas 7.4% of all married women are between 15–19.[104] Girls who are forced to marry earlier produce a lot of kids, and are more prone to die of obstetric complications.
3. **Literacy**—Although about 74% in India are literate, only 65% of women are literate in contrast to 82% of men. The National Family Health Survey - 3 shows that duration of education is inversely proportional to fertility. People who have completed at least 10+2 have an average family size of only 1.80, whilst those with no education have an average 3.55 children.[105]
4. **Limited use of contraceptives**—*Spacing* the birth of each children by one year reduces fertilty. However, most spacing methods have not gained popularity in India.
5. **Tradition**—Ill placement of women in society, low value of girl children (couples usually want at least a boy, so if a girl is born – they usually go on reproducing), low standards of living, cast and religions are other contributing factors. Muslims seem to have a higher fertility rate than Hindus.
6. **Early childhood mortality**—A mother would not undergo tubectomy (or her husband would not allow) unless the doctor ensures her of safe births of at least two children.
7. **Economy**—It is seen that people with a low income bear more children[106] (when they are in no position to raise them). It may be with the anticipation that more children means more earning members for future. The NFHS-3 shows a 0.92 more TFR in rural people than urban ones.

Fall in death rate

This is because of absence of natural checks (famines are not so frequent nowadays), mass control of some diseases, medical advance, increased food supply

(specially after the green revolution), international aids and success of health programs.

Life expectancy

At a given age, the life expectancy is the years the person of that age may expect to live according to mortality pattern of that country. According to 2011 census, the life expectancy in India is 63.90 (men) and 66.90 (women). (The recent values, for male and female are, respectively, 67.4 and 72.61 years.[107]

Life expectancy is used in life insurance and studies of survival rate in malignancy, and also in the calculation of DALY and health adjusted life expectancy (HALE). *Expectancy at 1 year* is a better indicator than at expectancy at birth, because of the high infant mortality in India.

Table 4.2. India – core health indicators (WHO)

Indicator	Value (year)
Life expectancy at birth (years) males	61.0 (2004)
Life expectancy at birth (years) females	63.0 (2004)
Healthy life expectancy (HALE) males	53.3 (2002)
Healthy life expectancy (HALE) females	53.6 (2002)
Probability of dying (per 1000 population) between 15 and 60 years (adult mortality rate) males	275 (2004)
Probability of dying (per 1000 population) between 15 and 60 years (adult mortality rate) females	202 (2004)
Probability of dying (per 1000 population) under 5 years of age (under-5 mortality rate) males	81 (2004)
Probability of dying (per 1000 population) under 5 years of age (under-5 mortality rate) females	89 (2004)
Total expenditure on health as percentage of gross domestic product	4.8 (2003)
Per capita total expenditure on health at international dollar rate	82 (2003)
Population (in thousands) total	1,103,371 (2005)
Per capita GDP in international dollars	1,830 (2004)
Percentage of attended deliveries	42.5

Fertility

Indicators of fertility

Fertility is the actual bearing of children (c.f. fecundity which is the potential to bear children).

Crude birth rate

It is the number of live births during the year/estimated MYP × 1000. It is crude in the sense that the denominator includes (a) men (b) women other than reproductive age. It is 23.5/1000[108] in India in 2007.

Generalized fertility rate (GFR)

It is the (number of live births during the year/mid year female population aged 15–49) × 1000. The generalized *marital* fertility rate includes one more clause, *married*, in the denominator. These two indicators, one feels, have more meaning than cBR (only women who can give birth are included in denominator).

Age specific fertility rates

It is the usual 'age specific' variable which defines fertilities of women in different age groups between 15–45 (i.e. number of children borne by all women in an age group/total number of women in that age group).

Total fertility rate

It is the **average number of children a woman would have if she were to pass through her reproductive years bearing children at the same rates as the women now in each age group**, (i.e. this hypothetical woman symbolizes *all* child bearing women). It is actually Σ age specific fertility rates.

TFR is the accepted indicator for fertility, and indicates the average family size. According to National Population Policy, it should be 2.1. The TFR in India is 2.8 [Sample Registration System, report 4 of 2007] or 2.72 in 2009.[109]

Gross reproductive rate

It is the average number of *girl* children that would be born to a woman if she experiences current fertility pattern throughout her reproductive period, assuming no mortality of the children she has borne. It is TFR × (female births/total births). The GRR is 1.3 in India.

Net reproductive rate

It is the average number of girl children that would be born to a woman if she experiences current fertility as well as child mortality pattern throughout her reproductive period (i.e. if she gives birth to the number of children corresponding to each age group as she passes through those ages, and also some percentage of her children die, corresponding to child death rate in that age group). An **NRR=1** means one woman *replaced* by another of the next generation.

Child woman ratio

It is the number of children of 0–4 years/women of reproductive age × 1000. There are **439** children per 1000 women of reproductive age in India.[110]

Measures to check the Indian population

1. National Family Welfare Program.
2. National Population Policy.
3. Community participation—Community need assessment and cafeteria approach.
4. Delivery of family planning services at PHC and postpartum units.
5. Education on small family, spacing, timing, age of marriage and pregnancy.
6. Better primary health care and ensuring child survival.
7. Universal access to contraception.

8. Social engineering— ↑ age of marriage, female literacy and women empowerment, planned parenthood, antipoverty program, 20 point and minimum needs program.
9. Free and compulsory school education upto 14.
10. Coordination with NGOS.
11. Major changes in approach–No incentives, bottom up, ↑ quality of care, risk approach, life cycle approach.

Family planning

It is a way of

- Thinking and living that is
- Adopted *voluntarily*
- Upon the basis of knowledge, attitude and responsible decision
- By individuals and couples
- In order to promote the health and welfare of the family and group
- Thus contributing to the social development of the country.

Aims

Welfare states just cannot equate family planning with contraception. The aims of the 'family welfare services' are much broader.

1. Prevent unwanted pregnancy.
2. Bring about *wanted* pregnancy.
3. Regulate interval between pregnancies.
4. Time births between 20–30 years.
5. Set number of children.

Family planning from a swear word to a right

Because people will usually reproduce if given a chance, and abstinence is not very popular except among ascetics, family planning is not natural but must be artificially practised. There was once a time when 'Nirodh' used to be a swear word, unacceptable in social setting. However, it is a healthy sign that we have taken these matters more seriously, and began to speak more frankly. The UN has vested each nation with the responsibility to provide its citizens accessible means of family planning (i.e. no couple should have the excuse 'we could not afford a condom, and thus Chotu was born'; the state must provide both knowledge and means of family planning). In recent times, the right to be (or not to be) pregnant by choice is regarded a fundamental right of women.

Goal

SOCIAL DEVELOPMENT of the country

Scope[111]

1. Birth control
2. Management of sterility
3. Advice regarding parenthood
4. Education regarding STDs

5. Genetic counseling
6. Premarital and marital guidance
7. Services for unmarried mothers
8. Medical termination of pregnancy
9. Screening for reproductive tract disease.

Benefits

1. For the woman— ↓ morbidity and mortality due to pregnancy.
2. For the child— ↓ congenital anomaly, ↓ mortality, ↑ growth and development (more the number of children, more the competition for nutrition and survival).
3. For the society—DEVELOPMENT.

The Indian scenario

- Couple Protection Rate = 46.6%[112]
- Unmet need for contraception = 12.8%[113]
- One child is born every 1.25 seconds[114]
- 50–60% of births are of 3rd order or more
- With increasing teenage sex, there is rise in unplanned pregnancies and abortions.

Contraceptives

These are substances, devices to prevent pregnancy.

Table 4.3. Contraceptives

Terminal methods	Vasectomy, tubectomy
Spacing (i.e. to 'space' births)	Intrauterine contraceptives Barriers ('conventional contraceptives' which have to be used *during* intercourse) 1. Physical—Condom (m,f), diaphragm, polyurethane 2. Chemical—Spermicidal (nonoxynol) foam tablet/suppository/jelly 3. Combined—Vaginal sponge soaked in nonoxynol (TODAY) Hormonal 1. Oral—Combined, progesterone only pills 2. Injectable—DMPA, NET-EN, DMPA-subcutaneous 3. Implant—Norplant 4. Vaginal ring Postconceptional 1. Medical termination of pregnancy nonhormonal (Centchroman)
Traditional (Behavioral)	Abstinence (don't have sex) Coitus interruptus (withdraw before ejaculation) Rhythm (avoid sex between days 10–17 of menstrual cycle)
Natural	Rise in basal body temperature 'Ferning' of cervical mucus

A good contraceptive is:
1. 100% effective
2. Safe
3. Reversible
4. Of low cost
5. Convenient
6. Long acting
7. Acceptable.

Effectivity of contraceptives: Pearl index

Failure rate per 100 women-year of exposure = **number of accidental pregnancies (including abortion and stillbirths)/total woman years of exposure** × 100. At least 600 women-months should be studied to make it valid.

If a pregnancy occurs, 10 months need to be deduced from the denominator (4 months for an abortion).

What does a pearl index = 10 mean?

100 woman years will have 10 accidental pregnancies, so, 1 woman year will have 0.1 accidental pregnancies; reproductive period of a woman is usually 25 years, so she will have 0.1 × 25 = 2.5 accidental pregnancies through her reproductive period.

Eligible couples

An eligible couple is where

1. Female is in the reproductive age group (15–44)
2. Staying together

The number of eligible couples is maintained at each subcenter in the Eligible Couple and Child register. **Number of eligible couples in India = 168/1000 population**.[115]

Unmet needs

Women who feel they need contraception but are not actually using are said to have an 'unmet need' for contraception. This may be due to
1. Insufficient health services
2. Lack of information
3. Fear about contraception
4. Opposition from husband or relatives.

12.8% of married women in India have an unmet need for a contraceptive.

Recommended contraceptives (by National Family Welfare Program)

1. OCPs
2. Condoms
3. IUD
4. Sterilization.

Acceptance of contraceptives

The **Couple protection rate** = % of eligible couples using one of a recommended contraceptive. It is **46%** in India. The recommended target is 60% to achieve a **TFR 2.1** or **NRR 1**. This is because a CPR of 60% will cut out all births of order 3 or more (50–60% births belong to this class).

Condoms

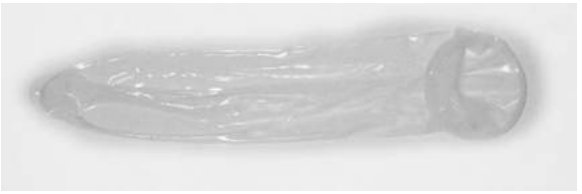


Figure 4.4. A condom

Condoms are sacs made of latex[116] with a teat infront to hold any excess sperm. Every latex condom is tested for holes with an electrical current. If the condom passes, it is rolled and packaged. In addition, a portion of each batch of condoms is subject to water leak and air burst testing.[117] Condoms are effective (pearl index = 2/100 woman year or 2% failure rate,[118] however may vary upto 14 due to incorrect use), and have some noncontraceptive benefits too (prevents most STDs like gonorrhea, chancroid, herpes, HIV, human papillomavirus infection and thereby, cervical cancer).

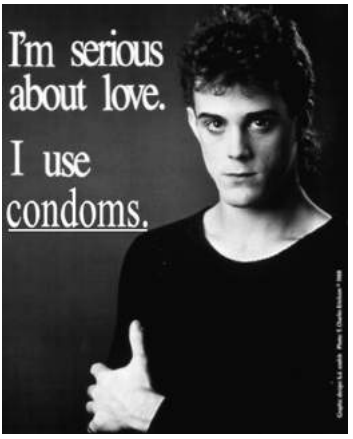


Figure 4.5. Are you serious about love?[119]

Condoms are also used for plastic repair of vaginal agenesis, and as a catheter.

Procedure

Wear a condom over erect penis without letting air in → use only water soluble lubricants, if required (oil based lubricants damage the latex, and are one of the causes of condom failure[120]) → after intercourse is over, remove it while the penis is still erect (or else it will slacken and semen may spill over in vagina) → the

condom should be disposed in the garbage bin (and not the drain, which it can block).[121] Use a new condom for each sexual act. The shelf life of a latex condom is 3 years.

Need

72 condoms/couple/year on average (6 unsafe days a month × 12 months).

Ideal candidates

1. AIDS endemic area.
2. Female with heart disease, thromboembolic disease or jaundice (can't be given OCP), pelvic inflammatory disease (contraindication IUD).
3. Lactating female.
4. When the female has missed OCP for 2 consecutive days.
5. First two months of starting OCPs.

Causes of failure

- Slippage
- Tear (more likely with new users than experienced users)
- Use of oil based lubricants
- Experimentation without a condom (let's have a go)
- Using two condoms at once (double bagging)
- Reusing condoms.

Female condom

Since 1988, the female condom is available. The female condom is a pouch made of polyurethane with two flexible rings at each end. The ring inside the pouch is inserted deep into the vagina, which blocks the external os. The other end of the pouch is open and stays outside the vagina.

Advantages. Female condoms can be reused, can be used for anal sex, and overall gives women a heightened sense of control over sexual intercourse.

Disadvantages. Female condoms cost more, less efficacious than male condoms, hard to put on and the open end hanging outside the vagina can be a turn off for many men.

Diaphragm

It is a soft latex or silicone cap with a spring molded into the rim. The spring creates a seal against the walls of the vagina, thus blocking the cervix. The failure rate of diaphragms vary between 6–39%[122,123] depending on proper usage.

Figure 4.6. A diaphragm

Procedure

Squeeze the rim into an oval shape for insertion → a *water-based* lubricant (usually spermicide) is applied to aid insertion. The diaphragm must be inserted sometime *before* intercourse, and remain in the vagina for 6 hrs after a man's last ejaculation.[124] For multiple intercourse, an additional 5ml of spermicide is inserted into the vagina before each act. Upon removal, a diaphragm should be cleaned with warm mild soapy water before storage. Without proper hygiene in inserion, diaphgram may introduce infections. After continuous use for more than 24 hrs, a staphylococcal infection (and even toxic shock syndrome) may develop.[125]

Spermicidal jelly/suppository

Nonoxynol-9 inhibits oxygenation of sperm, should always be used in combination with a barrier method. It is available as foam, cream, suppository and film form. **Side effects** include irritation, itching, or burning of the sex organs (either partner), and in women, urinary tract infections, yeast infection, and bacterial vaginosis.[126]

Condoms that are spermicidally lubricated by the manufacturer do not have enough spermicide to aid in preventing pregnancy, have a shorter shelf life, and may cause UTI. The World Health Organization says that spermicidally lubricated condoms should no longer be promoted. However, they recommend using a nonoxynol-9 lubricated condom over no condom at all.[127]

Intrauterine contraceptive devices

Although many earlier had tried inserting something in the uterus to prevent pregnancy, it was Dr. Ernst Gräfenberg of Germany published a report on an IUD made of silk suture (1929). He had found a 3% pregnancy rate among 1,100 women using his ring. In 1930, Dr. Gräfenberg reported a lower pregnancy rate of 1.6% among 600 women using an improved ring wrapped in silver wire. Unbeknownst to Dr. Gräfenberg, the silver wire was contaminated with 26% copper.

The first plastic IUD, the Margulies coil or Margulies spiral, was introduced in 1958. This device was somewhat large, causing discomfort to a large proportion of women users, and had a hard plastic tail, causing discomfort to their male partners (obviously). The **Lippes Loop**, a slightly smaller device with a monofilament tail, was introduced in 1962 and gained in popularity over the Margulies device.[128] The Lippes loop is usually regarded the *first generation* of IUDs.

Second generation copper—T IUDs were introduced in the 1970s, when it was found that copper gives definitive protection against pregnancy. They are more suitable for the nullipara, more tolerated than the Lippes loop, more effective and can be used for postcoital contraception. They has since been a smash hit.

Absolute contraindications

1. Pregnancy.
2. Puerperal sepsis.
3. Immediately after septic abortion.
4. Unexplained vaginal bleeding.

5. Malignant gestational trophoblastic disease.
6. Cervical cancer.
7. Endometrial cancer.
8. Distortions of the uterine cavity by uterine fibroids or anatomical abnormalities.
9. Current PID.
10. Current purulent cervicitis, chlamydial infection or gonorrhea.
11. Known pelvic tuberculosis.

Relative contraindications

1. Postpartum between 48 hrs and 4 weeks
2. Benign gestational trophoblastic disease
3. Ovarian cancer
4. Very high individual likelihood of exposure to gonorrhea or chlamydia
5. AIDS (unless clinically well on antiretroviral therapy).

Gen I: Lippe's loop

It is a nonmedicated radioopaque (barium coated) loop. It is not used much because of high expulsion rate.

Gen II: Copper T

It is a 'T' made of plastic coated with copper. It is sterilized by γ-ray irradiation. The set of a copper T comes with a *inserter* with a guard that moves over the inserter (to adjust for varying uterine size), and a P-shaped plunger.

- The Government supplies CuT-200B (i.e. 200 mm^2 of copper, *barium* coated) at hospitals, which has a failure rate of 3% and must be changed every 4 years
- The 380A copper T has more amount of copper, is coated with silver (Argentinuium), much lower failure rate (0.5–0.8%) and can be used for 10 years.

Figure 4.7. Lippes loop

Figure 4.8. The 380A copper T

Gen III: Hormonal IUD[129]

Hormonal uterine devices do not increase bleeding as earlier copper Ts. Rather, they *reduce* menstrual bleeding or prevent menstruation altogether, and can be used as a treatment for menorrhagia.

Progestasert, introduced in 1976, is a 'T' with 38 mg of progesterone, which is released at a rate of 65 μg per day. It must be replaced *every year*. Another such device, the MIRENA (from Bayer company) or LNG-20, releases 20 μg of levonorgestrel per day and may be used for *10 years at a stretch*. It has a failure rate of only 0.2%.

Mechanism

The presence of a device in the uterus recruits leukocytes and releases prostaglandins by the endometrium. These substances are hostile to both sperm and eggs; the presence of copper increases the spermicidal effect.[130] A few physicians have suggested they may have a secondary effect of interfering with the development of embryos (thus they are also effective as postcoital contraceptives).[131]

Ideal candidates

It is best for women with

1. Uncooperative husbands
2. Unipara
3. Normal periods
4. Willing to check the IUD tail
5. Can follow up regularly
6. Monogamous relation.

Insertion

The IUD is meant to be inserted in OPDs. Two things should be kept in mind

- Maintain strict asepsis
- To avoid rupture of uterus, use the withdrawal technique (see below) rather than forcibly pushing in the device

Full obstetric history
(exclude pregnancy, PID, DUB, uterine anomaly, cancer)

↓

Empty the bladder

↓

Set the woman in lithotomy position

↓

Antiseptic dressing

↓

Open cervix by Allis' forceps and speculum

↓

↓

Assess position (i.e. anteverted or retroverted) and depth of uterus by introducing an uterine sound (check your G and O department to know about that device)

↓

Fix the guard (a knob attached to the inserter) at the external os

↓

Push the T into the uterus with the plunger

↓

Withdraw plunger and insert T at the same time (withdrawal technique); 2–3 cm of thread should be hanging out

↓

Tell the woman to check the thread each time she goes to toilet, report immediately if its missing

Ideal time for insertion is

1. Within 10 days of LMP[132]
2. 6–8 weeks postpartum (the uterus is very soft immediately after delivery)
3. May be used within 120 hrs of intercourse to prevent implantation.

Follow-up

Ask the woman to come at next menstruation (to check whether the device has been expelled) → then at the 3rd menstrual period (to check for complications) → every 6 months/1 year (for motivation).

Advantages

1. Only a single act of motivation is required; unlike OCPs and condoms, which you must remember everyday, an IUD is 'fit and forget'.
2. Highest continuation rate.
3. No systemic side effects (c.f. OCPs).
4. Effect is reversible.
5. Inexpensive.

Adverse reactions

1. Pain—Mild pain, specially during menstruation, can be ignored; if the pain is severe, check whether the device has been placed properly, and always keep perforation in mind; if the device is found to be in proper place, try a smaller T or suggest a different contraceptive.
2. Menorrhagia and metrorrhagia—All IUD users are given free iron-folate prophylaxis from the Nutritional Anemia Control Program.
3. Expulsion during menstruation (which may be unnoticed and pregnancy may occur).
4. Perforation (specially Lippe's loop)—May present as acute intestinal obstruction/chemical peritonitis or asymptomatic (!); urgent laparotomy is required.

5. Ectopic pregnancy (specially with hormonal IUDs).
6. Pelvic inflammatory disease.
7. Infertility (rare).

What if the thread is missing?

X-ray pelvis with an uterine sound placed inside uterus (or an ultrasonography) > if the IUD is seen outside the uterus > laparotomy.

What if it fails (i.e. pregnancy occurs)?

Either

1. Let it be expelled during delivery (but this has a 25% chance of a safe delivery)
2. If the thread is seen, bring it out by hand
3. Go for abortion.

Hormonal contraceptives

Nothing symbolizes women's liberation more than oral contraceptives. The OCP has vastly contributed to women's modern economic role, in that it prolonged the age at which women first married allowing them to invest in education and other forms of human capital as well as generally become more career-oriented. Soon after the birth control pill was legalized, there was a sharp increase in college attendance and graduation rates for women,[133] and transition of women from 'housewife' to 'working woman'.[134]

Oral contraceptives

Two brands of combined pills (Norgestrel 0.3 mg + Ethinyl estradiol 30μg) are produced by the government. Mala-N is supplied free by the National Family Welfare Program. Mala-D is socially marketed in drug shops.

OCPs come as monthly packs with 21 OCP tablets and 7 placebo (iron-folate tablets). They are to be taken from **5th–25th day of cycle** (i.e. starting from 5th day of LMP) once daily at bedtime (they should be taken at the same time of the day each day). For the next 7 days, OCPs are not necessary, but iron-folate tablets are given anyway so that women make a habit of 'taking a pill' once daily. Once OCPs are started, there is no need for a pause, and a next pack should begin immediately after the first is over.

Are placebos necessary? It is possible to skip withdrawal bleeding altogether and still remain protected against conception by skipping the placebo pills and starting directly with the next packet.

Note that OCPs stop ovulation, so that the endometrium spends the whole 21 days in proliferative phase (no secretory phase), and thus it is underdeveloped. The bleeding which occurs in these 7 days is *withdrawal bleeding* and not true menstruation (i.e. we are forcing the uterine cycle to our artificial rhythm, instead the natural rhythm generated by the ovary). The blood loss in such bleeding is only half of that lost in normal menstruation. This bleeding is also confirmatory that a pregnancy has *not* occurred.

Quarterly pill. Starting in 2003, women have also been able to use a three month version of the pill. The SEASONALE pill is to be taken continuously

without intervening placebos. Thus it gives the benefit of less frequent periods, at the potential drawback of breakthrough bleeding.[135]

Biphasic and triphasic pills. Because of side effects of progesterone (nausea, hypercholesterolemia, etc.), phasic pills have been develop which begin with low dose of progesterone and gradually up titer. The aim is to lower the total intake of progesterone in a cycle.

Progesterone only pills. These pills act principally, via thickening the cervical mucus and variably inhibiting ovulation (and implantation of ovum). They are to be taken everyday (no placebo tablets). Lacking estrogen, they do not cause thromboembolic disease (and thus not contraindicated in sickle-cell disease). The progestin only pill is recommended over regular birth control pills for women who are *breastfeeding* because the mini-pill does not affect milk production (estrogen reduces the amount of breast milk). Like combined pills, the mini-pill decreases the likelihood of pelvic inflammatory disease.

Mechanism

1. Inhibition of ovulation (estrogen causes suppression of FSH, and progesterone suppresses LH).
2. Thickening of cervical mucus (progesterone).
3. Keeping endometrium 'out of sync' with ovary, so that implantation cannot occur.

Efficacy. If used perfectly, the failure rate is only 0.3%, however in typical use, failure rate varies between 2–8%.[136] The causes of failure may be.[137]

1. Delay in starting the first packet of pills (i.e. starting after 5 days of LMP).
2. Missing more than one pill.
3. Delay in starting the next packet of active pills (i.e. extending the placebo period beyond 7 days).
4. Intestinal malabsorption of pills due to vomiting or diarrhea.
5. Drug interactions (i.e. with rifampicin, phenytoin).

OCPs should not be used continuously for more than 5 years at a stretch.

Common problems

- Missed one pill—Take the next pill as soon as reminded, and continue the regular ones.
- Missed more than one pill—If active pills have been missed, take *one* when you remember + continue regular pills + use a barrier method for 7 days. If a placebo has been missed, however, do not take the rest and start a new pack.

Ideal candidates

1. Menorrhagic women (OCPs reduce bleeding)
2. Women in late lactation (after 6 months)—If used within first 6 months, otherwise OCPs may reduce duration and quality of milk (but progesterone only pills may be given during lactation)
3. Women aged under 40, nonsmokers (for CVS reasons).

Advantages

1. Oral.
2. No need of privacy (the woman can pop the pill anytime and pretend that its just an aspirin).

3. 98% effective even on typical use (no requirement for stringent use).
4. *Reversible* after 2 months of cessation.
5. Cheap.

Common adverse effects. Breast tenderness, weight gain (? fear of weight gain), headache, melasma, leukorrhea, hypertension.[138] The commonest side effect is bleeding in mid – cycle (**Breakthrough bleeding**), usually due to inadequate hormonal support to endometrium. A higher dose of hormones is required.

Serious side effects. Coagulation disorders (deep vein thrombosis, pulmonary embolism, stroke and myocardial infarction). The relationship of OCP with breast cancer is complex and much debated. The WHO maintains that "the health benefits of any method of contraception are far greater than any risks from the method".[139]

Noncontraceptive benefits[140]

1. Menstruation is regularized
2. Reduced incidence of **dysmenorrhea, menorrhagia, PMS, mittelschmerz syndrome** (ovulatory pain), **PID** (it thickens cervical mucus), **ectopic pregnancy**, benign breast disease, ovarian cyst, endometriosis, **endometrial and ovarian cancer**,[141] colorectal cancer (eh!)

In addition to being a contraceptive, the OCP can be used to treat polycystic ovary disease, endometriosis, adenomyosis, dysmenorrhea, menorrhagia and acne.

Contraindications to OCPs

1. **Absolute**—Pregnancy, thromboembolic disease, stroke, heart disease, breast lump, genital cancer, active liver disease, cholestatic jaundice, migraine, dyslipidemia.
2. **Relative**—Undiagnosed dysfunctional uterine bleeding, 1st 6 months of lactation; age > 40 (or Age > 35 + smoker), mild hypertension, gross obesity, epilepsy, depression, oligomenorrhea/amenorrhea, jaundice, sickle-cell disease.

Interactions. Rifampicin, anticonvulsants (phenytoin, carbamazepine) and antifungals may cause OCP failure by enzyme induction. Many broad spectrum antimicrobials impair the enterohepatic circulation of estrogens.

Follow-up. The Health Worker should make a home visit after 15 days of giving OCP and a second visit after 1 month, to check for headache/calf pain/scotoma/numbness, etc.

Injectable hormone analogs

1. Depot medroxyprogesterone acetate (DMPA) 150 mg IM injection every 3 months on perfect use (never late for more than 2 weeks for an injection), it has a failure rate of 0.3%.[142] It is most effective if given within first 5 days of the cycle.
2. Norethisterone enanthate (NET-EN) 200 mg IM injection every 2 months.
3. A subcutaneous preparation of DMPA is now available (DMPA-SC) 104 mg every 3 months. This injection contains 31 percent less hormone than the

IM injection. Because it has a lower dose of progestin, it may lead to fewer progestin-related side effects. The injection is also less painful.

Subdermal implants

NORPLANT ® consists of 6 silastic capsules containing 35 mg each of levonorgestrel, which, when implanted subcutaneously in upper arm, gives protection beginning from 24 hrs of insertion to 5 years.[143] It is one of the most effective (failure rate 0.05%,[144] although not the most available contraception. It is specially useful in the developing world, as it does not require daily administration or access to a hospital to be effective. In addition, no continual contraceptive supplies (pills, condoms, etc.) are necessary.

Disadvantages. Norplant is quite likely to cause irregular periods (but overtime, reduces menstrual bleeding); some women report weight gain, hair loss, migraine, depression. Also, fertility takes sometime to return after the implants have been removed. It has been discontinued in the UK and USA due to reports of side effects (and because it is interventional, OCPs have gained more popularity).

Like all progestogens, it is contraindicated in blood dyscrasias, liver diseases or breast cancer.

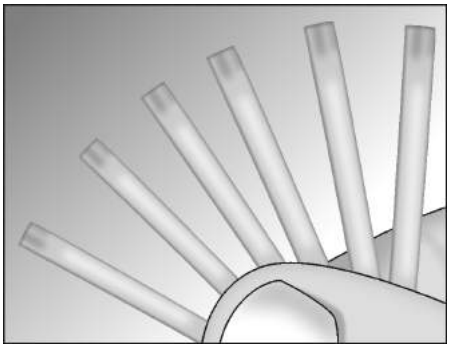


Figure 4.9. Norplant

Norplant II. Developed by the Population Council of USA, Norplant II or Jadelle® consists of *two* silicone rods each containing 75 mg of levonorgestrel. It was subsequently approved November 22, 2002 by the FDA as being effective for 5 years.

Vaginal ring

A vaginal ring containing levonorgestrel is effective contraception if worn for the first 3 weeks of each menstrual cycle. The NUVARING® is a ring which combines release of both estrogen and progesterones. Vaginal rings may cause all the effects of OCPs (except weight gain) and in addition, hypersensitive vaginitis. On perfect use, they have a failure rate of 1%.

Contraceptive patch

A contraceptive patch (i.e. the ORTHO-EVRA®) is a transdermal patch applied to the skin that releases synthetic estrogen and progestins. They have been shown

to be as effective as the OCP (failure rate 1%). It is applied on the day of LMP or within 5 days of LMP → changed every 7 days for 3 weeks → 7 day gap (like the placebo period of OCPs) → restart.

Natural methods

1. Lactation—To effectively shield pregnancy by lactation, it is useful to continue exclusive breastfeeding for 6 months and maintain interval between feeds of at most 6 hrs; it has 0.5% failure rate on perfect use, but rises to 2% if proper lactation is not done.
2. Abstinence—No failure on perfect use.
3. **Coitus interruptus**—A 4% failure rate even on 'perfect use'[145] and much more on 'typical' use.
4. **Fertility awareness methods**—Because the follicular phase of the menstrual cycle can show *extreme* variation, these methods show a high failure rate even on perfect use (9% with rhythm method).

Rhythm method (Knaus-Ogino method)[a]	Elicit menstrual history of 6 months. Let x = (longest cycle – 10) and y = (shortest cycle – 19). Then, unsafe period = yth – xth day of cycle
Standard days method (for women whose cycles are always between 26–32 days)	Days 8–19 are unsafe
Basal body temperature	Measure by BBT thermometer each morning before getting out of bed and touching the ground. A rise of 0.5–0.8°F is indicative of ovulation
Cervical mucus (Billing's method)	Observe appearance of cervical mucus in a napkin from 9th–20th day of cycle; it corresponds with ovulation

[a]Medical Eligibility Criteria for Contraceptive Use: Fertility awareness-based methods. Third edition. World Health Organization. 2004.

Sterilization

Criteria for sterilization

1. The husband should be between 25–50
2. The wife should be between 20–45
3. The couple should have at least 2 living children
4. If the couple has 3 or more children, age limits can be relaxed
5. Consent of both husband and wife is necessary.

Vasectomy

Vasectomy is a simple operation which may be done in an OPD. In the conventional form, it is simply severing and ligating the vas deferens in the scrotum.

- The **No-Scalpel**, in which a sharp hemostat, rather than a scalpel, is used to puncture the scrotum may reduce healing times as well as lowering the chance of infection. This method is promoted by NFWP and every doctor is to be trained with this technique. The plan is to make this available at all primary health facilities.

- An **open-ended** vasectomy seals only the distal end of the vas deferens, thus sperm do not build up inside the epididymis but accumulate in scrotum. Testicular pain (from "backup pressure") may also be reduced using this method.[146]
- The "Vas-Clip" method does not require cutting the vas deferens, but rather uses a clip to squeeze shut the flow of sperm. It is less successful than the conventional procedure.

Side effects. Sperm accumulates in the epididymis and will eventually spill into immune system, causing development of antisperm antibodies in most men. Sperm granulomas also form in the scrotum. Complains of reduced sexual desire after vasectomy have not been substantiated.

Efficacy. Very effective (failure rate 0.1–0.2%, equivalent to one in 2000 vasectomies). Until 2–3 months of vasectomy/30 ejaculations have passed, the man needs to use a barrier contraceptive, so that residual sperm dies away. *Failure* is rare if operation is properly done, the causes being

1. Recanalization
2. Something other than the vas has been cut.

How to draw men to vasectomy? Vasectomy is a much more simpler and more effective procedure than tubectomy, but is not yet prevalent in India due to irrational fear among men. The following may be advertised to attract more men for a vasectomy.[147]

1. Lower cost than tubectomy (expense in 5 vasectomy equals one tubectomy)
2. Simplicity of the surgical procedure (do not need to open the abdomen)
3. The lower mortality and morbidity of vasectomy (for example 0.1 per 100,000 vasectomies vs 4 per 100,000 tubal ligations in industrialized nations).[148]

Complications. Infections are uncommon after vasectomy; a bath should only be taken only after 24 hrs of the operation. **Postvasectomy testicular pain** may last from a few days to a lifetime. Some men experience depression or anger and go through a period of mourning over the loss of their reproductive ability, and may feel "less of a man". This emotion is similar to what some women experience after menopause.

Tubectomy

Method. In the **laparoscopic method**, the fallopian tubes are clipped. Laparoscopy not appropriate until 6 weeks postpartum, but may be done alongwith MTP. The woman needs to be followed up 7–10 days after operation and again after 12 months. In the minilaparotomy method, a 2.5–3 cm transverse incision in the middle gives enough access to both fallopian tubes so that a segment can be cut away/crushed. In the *Pomeroy technique*, small loop of the tube is tied by suture (at the amuplla isthmus junction) and the top segment of the loop is cut. This is a very simple operation, can be done under local anesthesia with one or two instruments, and is now carried out in BPHCs.

Efficacy. Failure rate is 0.5% only. Tubes may recanalize, and reversal from surgery is possible.

The Essure procedure (2002)

Microinserts are placed into the fallopian tubes by a catheter passed from the vagina through the cervix and uterus. Once in place, the device is designed to elicit

tissue growth (scarring) in and around the microinsert to form over a period of 3 months an occlusion or blockage in the fallopian tubes. It is permanent and has only 0.2% failure rate.[149] 3 months after insertion a hysterosalpingogram is done to confirm that the fallopian tubes are completely blocked. Until then, another contraceptive needs to be used. This procedure does not need anesthesia, and can be carried out in OPD. It has great potential to be used in developed countries.

Emergency contraception (the 'casualty' department of family planning)

Needs[150]

Unprotected sex. Where no contraceptive was used.
Inadequate protection.

- Condom breakage, slippage, or incorrect use.
- Three or more consecutive missed combined OCPs.
- If using progestogen-only pill, a more strict schedule is to be maintained; any such pill taken *three* hrs late amounts to inadequate protection.
- More than two weeks late for a progestogen-only contraceptive injection.
- More than seven days late for a combined estrogen-plus-progestogen monthly injection.
- Failed coitus interruptus (e.g. even ejaculation on external genitalia suffices as unsafe sex).
- Failure of a spermicide tablet or film to melt before intercourse.
- Dislodgment of a vaginal diaphragm.
- Miscalculation of 'safe period'.
- IUD expulsion.

Nonconsesual sex. Rape

Methods

The WHO-recommended regimen for emergency contraception is: 1.5 mg of levonorgestrel as a single dose.[151]

Table 4.4. Methods of emergency contraception

High dose estrogen within 72 hrs Diethylstilbestrol 50 mg × once × 5 days Ethinylestradiol 5 mg × once × 5 days	Not recommended due to side effects
Yuzpe method, within 72 hrs Ethinylestradiol 100 μg + Norgestrel 1 mg Repeat after 12 hrs	Equivalent to four Mala tablets
Progestrone only only, within 72–120 hrs LNG 0.75 mg repeat after 12 hrs LNG 1.5 mg single dose	Not effective once the process of implantation has begun, and will not cause abortion.
Antiprogestins Mifepristone, within 72–120 hrs Mifepristone 600 mg/50 mg/10 mg single dose	Recently recommended by National Family Welfare Program May cause ectopic pregnancy

Contd...

Contd...

IUD within 5–7 days	Only choice after 5 days have passed Continued contraception Risk of PID Unsuitable for nullipara

Efficacy

Failure rate is 0–2.4%, depends on type, time, method and stage of cycle. Levonorgestrel seems to be most effective.

Constraints

Premarital sex is still considered an offence tantamount to murder (or even worse) in our country, and brunt of the blame usually falls upon the girl, who also has to bear the misfortune of a teenage pregnancy. In such a social setting, problems are bound to plague emergency contraception services.

1. Misconceptions, lack of awareness, short period of time to access service.
2. Service provider related–Lack of awareness, judgmental attitude ('you two have committed a sin and now you must suffer'), lack of training and reluctance, lack of counseling.
3. Nonavailability of products.
4. Service related–Emergency contraception is largely ignored in NFWP (thus fostering the growth of many legal and illegal abortion clinics), there no distribution network for emergency contraceptives, and it still requires prescription by a physician.
5. Ethical–The family, kins and the service providers often view emergency contraception equivalent to abortion or even feticide; the girl is often wrongly blamed for promiscuity and 'being available', targeted at unmarried adolescents.

Table 4.5. Comparison between contraceptives

	Condoms	**OCP**	**IUD**	**Sterilization**
Reversible	+	+	+/–	–
Motivation needed	Less	Less	Moderate	High
Continuing motivation	Needed	Needed	Needed	Not needed
Perfect use failure rate	2%	0.3%	0.6%	0.1%
Noncontraceptive benefits	Yes	Yes	No	No
Side effects	Less	More	More	Less
Hospitalization	Not needed	Not needed	Needed	Needed

Suggesting a contraceptive

First, always give a 'cafetaria' (the exact word used by NFWP) choice to the couple. If you *have to* make the decision, consider culture, religion, socio-

economy, taboos and importantly whether the couple wants to delay children or stop altogether.

There is no single ideal contraceptive which meets all the social, cultural, esthetic and service needs of individual and community. Success will depend on

1. Efficacy (theoretical and practical)
2. Acceptability
3. Regular and proper use
4. Continuation.

Table 4.6. Suggested contraceptives

Everybody	Barrier methods
Newly married	OCP or condom, NOT an IUD (increases chance of PID in young women, increases menstrual bleeding and may cause mid cycle bleeding)
Post MTP	OCP, IUD
Postpartum	IUD after 6 weeks OCP after 6 months (it may reduce lactation) Lactation may itself be contraceptive in 1st 6 months if 1. EBF is done with one feeding at night 2. Amenorrhea continues
Women >35 years, smokers	IUD Advice to take permanent method

Termination of pregnancy

The successor to lack of sex education and lack of emergency contraception is, naturally, an abortion. The practice of abortion used to be a glamorous business in India because

1. Indians consider premarital pregnancy the greatest sin conceivable; even a serial killer gets more sympathy from the society ('he must be mentally ill') than a 16 year old unmarried mother ('she must have offered herself first').
2. Girls are more prone to go to an abortion clinic which promises 'secrecy' rather than a health center where nothing stays private.
3. In matters of abortion (and sexually transmitted diseases), rural Indians trust quacks more than doctors; they feel really 'shy' before a doctor, but not a charlatan.
4. Most Indian families (and the Indian Penal Code until 1971) consider abortion as murder, and prohibit it, even if at the cost of the mother.

All this resulted in a state where most abortions were done by nonmedical people by illegal means (i.e. a stick into the uterus), causing many young mothers to die in the process, and the general notion that 'mothers are expendable'. The **Medical Termination of Pregnancy act** was launched in 1971 with a viewpoint to change all this. The current legal clauses (1975) are as follows,

1. **Site**—Any Government hospital or institutions registered to Chief Medical Officer of Health (CMOH) of the district.
2. **Time**—Upto 20 weeks of gestation; if done after 12 weeks, consultation of at least 2 doctors are needed; MTP can be done after 20 weeks *only* on therapeutic ground.

3. **Surgeon**—A surgeon who is either DGO or MD in Gynecology and Obstetrics/has done 6 months of house staff ship in GandO/assisted at least 25 MTPs; they have to be certified by an CMOH of the district to carry out MTP. The surgeon must have one year of obstetrics practice under his belt (or 3 years if his registration is earlier than the MTP act of 1971)
4. **Consent**—Mother's consent is needed. For lunatic girls or those < 18, legal guardians must be asked for

Indications. MTP can be done with the following indications before 20 weeks of pregnancy on

1. Humanitarian ground—Where continuance of pregnancy constitutes grave risk to *mental health* of the mother (i.e. pregnancy of rape)
2. Therapeutic ground—Where continuance of pregnancy constitutes grave risk to the *physical health* (i.e. mother with a blood dyscrasia or severe heart disease)
3. Social ground—Where continuance of pregnancy constitutes grave risk to the health of the society (i.e. mother with an unwanted child of contraceptive failure; such a child is prone to be neglected)
4. Eugenic[152] ground—Where continuance of pregnancy will result in a child with developmental anomaly.

After 20 weeks, pregnancy can be terminated only on therapeutic ground. In such case, a single doctor may take the decision and perform MTP in an unrecognized center.

MTP register is maintained at directorate of health services in each state.

Amendment to MTP Rules, 2003

1. Medical Method of Abortion–In April 2002, Drug Controller of India approved marketing of Mifepristone for termination of early pregnancy. Currently its use in India is recommended upto 7 weeks (49 days of amenorrhea) in a facility with provision for safe abortion services and blood transfusion.
2. Manual Vacuum Aspiration (MVA) is recognized as an MTP procedure.

National Family Welfare Program, 1977

Milestones

1. 1952—India was first in the world to launch the family planning program
2. 1976—First National Population Policy
3. 1977—The Family Planning Program changed to National Family *Welfare* Program (Welfare concept = birth control + preventive, promotive, curative services for reproductive women)
4. 2000—New population policy.

Structure

NFWP is a 100% centrally funded project.

CENTER

MINISTRY OF HEALTH AND FAMILY WELFARE

Secretary to Government of India (economic matters)

Director general, health and FW (technical matters)

↓

↓

STATE

Health and FW directorate (State FW officer)

↓

DISTRICT

DISTRICT FW BUREAU

District FW officer (Deputy Chief Medical Officer III) - administrator

District mass education and media officer-in charge of family planning education

Statistics officer - evaluation

↓

URBAN

FW center/ postpartum unit

↓

PHC

Rural FW center (conducts MTP, IUD insertion)

↓

SUBCENTER

MPHW - asses and analyses situation of her area > makes action plan > sends to district > gets supply of contraceptives

↓

VILLAGE

Condoms, OCPs to be distributed by Village health guide

Strategies

Family planning is delivered through primary health care and postpartum units. The most stressed upon issues are spacing methods, timing of birth, enhancing child survival and increasing age of marriage.

1. Universal access to family planning as cafeteria approach and depot holder scheme.
2. Target free/Community Need Assessment (CNA) approach—No central targets has been set (i.e. no pressure to "distribute 10000 condoms within end of July"); every area has its own deficiencies which must be assessed and acted upon, peripherally.
3. Education on family planning.
4. Intersectoral coordination with NGOs, Departments of Rural Welfare, Panchayat raj.
5. Women empowerment, increasing age at marriage, increasing female literacy.

National population policy 2000

Objectives

1. Short-term—Meet unmet needs (contraceptives, manpower)
2. Intermediate term—Get TFR to replacement level (2.1) by 2010
3. Long-term—Stabilize population by 2045 (i.e. everyone gets enough food without pressurising the environment too much).

Sociodemographic goals

1. Meet unmet needs.
2. **Free and compulsory education upto 14 years** and reduce dropouts < 20%.
3. Universal immunization (100%).
4. **IMR < 30 and MMR < 100/100000** live births.
5. Age of marriage of all girls > 18 years.
6. **Institutional deliveries at least 80%**, and ALL deliveries by trained personnel even if at home.
7. Universal access to information and services of fertility control.
8. 100% registration of birth, death, marriage.
9. Integration of management of **reproductive tract infections**, STD and HIV; containment of infection.
10. Prevent and control communicable and noncommunicable diseases.
11. Integrate Indian medicine in reproductive health.
12. Converge related sectors.

Strategies

1. **Decentralization**[153] of planning, involvement of Panchayat.
2. Convergence of services at village level—All the family planning amentities should be available as a 'package' to the lowest level of health care.
3. Empowerment of women (literacy, income, political power, school enrolment for girls, access to safe water supply).[154]
4. Child health and survival—People won't stop producing children until you can guarantee survival of their present children.
5. Meeting unmet needs.
6. Provision of RCH services for **underserved population**—Slums, tribals, adolescents.
7. Involving *men* more into family planning.
8. Collaboration with private sectors, NGOs.
9. Mainstreaming **Indian medicine**.
10. Provision for health care of older population.
11. Legislation and public support, **freezing of Lok Sabha and Rajya Sabha seats based on 1971 census** extended upto 2026 (Lok Sabha seats are based upon population of the area; if distributed solely according to this criteria, the regions with more people will have a overtly greater say at the parliament).
12. Research on family planning.

Motivations for small families

1. Reward to Panchayat for excellent performance.
2. **Balika Samriddhi Yojana**—Cash of Rs. 500/- at birth of a girl of order 1 or 2.
3. **Maternity benefit scheme**—Rs. 500/- awarded to a mother who gives birth to 1st or 2nd child after the age of 19 years.
4. **Family Welfare linked Health Insurance scheme**—The couple undergoing sterilization, as well as the doctor doing it, are covered by insurance from any operative complication (including death).

5. **Incentives**—The Government provides many incentives for accepting family planning methods.

6. Janani Suraksha Yojana: See maternal and child health.

Any couple undergoing vasectomy/conventional tubectomy	Rs 800
Laparoscopic tubectomy	Rs 145
IUD	Rs 20
Government employees who sterilize after 2 or 3 children	Increment in pay
Death following sterilization or IUD insertion	Rs 20000 to spouse/heir

SOCIETY

It is imperative that we know a little something about the society we live in (the other name of Community Medicine is 'Preventive and *Social* Medicine'). People have grouped together in bands or flocks from prehistoric times, but it was only since the last five hundred years, that some serious insights were gained on the mechanisms of how such a group operates. The major impetus to development of the subject was the French Revolution, which had drawn many philosophers into the subject. The study of society, or the discipline of Sociology was firmly established on an academic basis by the philosopher Auguste Comte,[155] who is usually regarded as the 'Father of Sociology'.

Social medicine

In a very limited sense, social medicine = the 'social' parts of epidemiology. It is the study of man as a social being in his total environment. It emphasis those segments of health and disease which can be prevented or treated with the active effort of *many* people.

Social medicine

Study of man as a social being in his *total* environment, i.e. the health of an individual is not *personal* but social. This revolutionary idea was conceptualized by Salomon Neumann and Rudolf Virchow.[156] The term 'social medicine' was coined by **Jules Guerin**.

State medicine

Provision of free medical care by the government for all its people. The revenue from such a service is generated not by hospital bills but taxes that everybody pays.

Socialized medicine

Provision of free medical service and professional education by state but the program is operated by professional groups (like the National Health Service in UK)

Social-economic factors

The following social factors are responsible for incidence, course and outcome of a disease: **Poverty, malnutrition, sanitation, education** (or the lack of it), **housing and employment**.

Social sciences

There are five social sciences in the orthodox sense—Political science, social anthropology, economics, social psychology, and sociology.

1. **Sociology** is the study of society.
2. **Political science**—The study of the structural organization of society, its control mechanisms, its policies and legal framework; for instance, whether a society is democratic or a monarchy falls under the purview of political science.
3. **Social psychology**—Effect of social environment on attitude and motivation of an individual.
4. **Social anthropology**—Study of physical, social and cultural history of man which have led to the development of various kinds of societies of today.
5. **Economics**—Study of the production, distribution, and consumption of goods and services.

Sociology

It is the study of society, namely human behavior and pattern of relationships. It is time for us to again, define some 'common terms'. (This is a long list of definitions, but try to pick keywords from each instead of mugging them all).

Society

It is a group of individuals who have organized themselves to follow a *given way of life*. The principal role often society is CONTROL of the behavior of the individual by both law and customs. In this context, customs are often more effective than laws.

Medical sociology

It studies the *health*, *health behavior* (i.e. at what point of an illness does a person feel 'sick' and when does he consider a visit to the doctor) of individuals, as well as the *subsociety* of doctors, nurses, patients and health workers within a health facility, and the *relationship of the medical* profession to the rest of the society.

Community

It is a small part of society **delimited by geography** and **sharing common interests**. Its members know each other and interact between themselves freely. It functions within a particular *social structure*. It exhibits and creates norms, values and *social institutions*. A typical example of a community is a group of villagers, or the residents of an urban housing complex.
Two terms in the definition need further explanation.

Social structure

It is the *pattern of individual relationships* (i.e. who bosses over whom). It is a complex of major *institutions* (marriage, family, etc.), groups, *power structure* (i.e. parents usually have authority over children, police have power to dismiss a mob) and *status hierarchy* (a physician is usually well regarded in the community than a laborer).

Social institutions

An organized complex *pattern of behavior* in which a number of persons participate to promote interest of the group – i.e. family, school, hospital, clubs, marriage. Strictly speaking, people do not need to marry and have a family for satisfying their biological needs, they could go on living like animals if they chose too. But nevertheless, they do, for the sake of the society at large.

Socialization

It is the process by which the child gradually acquires *culture* (see later) and becomes *a member of a society*.

Social values

Those *standards of judgments* by which actions of an individual are *evaluated* to be good or bad. They are directive principles of human action.

Social norms

Social rules which define correct and acceptable behavior to which people are expected to conform.

Habit. It is a *personal affair* not entailing any obligation (i.e. somebody may have a habit of picking his teeth). When habits are shared by many people for necessity and sanctioned by society they are converted into *customs*.

Folkways. Customary ways of behavior enforced by informal social controls; they differ between various communities (i.e. prelacteal feeding is customary in some areas, while in other areas it is not).

Mores. Involve mental standards and sanctity but in an *informal* way (preventing incest, putting vermillion on married women). There is no iron-cast law that every married Hindu woman must bear a red mark over her forehead, but nevertheless, most women put it on. CUSTOMS = folkway + mores. They have a traditional, automatic and mass character.

Laws. The *formal* method of control of behavior, i.e. those rules set according to the constitution of the nation and 'must be' followed.

Social contract

First popularized by the great French philosopher Rousseau, a 'social contract' is an implicit bond of agreement between any two members of society over a certain action. When we ride a bus, we make an unstated bond with the driver to deliver us safely to the destination. Similarly, *marriage* is a social contract of sexual gratification, reproduction and conjugal life.

Culture

It is *socially inherited characteristic* of human groups. Culture is an experience which is *learnt*, *shared and transmitted*. Every kind of society rears a certain kind of culture, which is their own way of doing things. This is even true of people who are not bound by geographical limits but still form a coherent group (i.e. 'cyberculture'). *Acculturation* is diffusion of culture betwen two communities – a

two way process (like the Muslim reign in India, when culture of both Hindu and Muslim traditions were exchanged).

Learning

Any *permanent change in behavior* as a result of practice and experience. It includes not only acquiring knowledge, but also skills and formation of habits, development of perception. There are three kinds of learning.

Knowledge. Knowledge is *cognitive* learning, i.e. adding bits of information to the brain (not *mugging*, which is only temporary). When we speak of someone who is very 'knowledgeable' about classical music, what we are acually saying that he is not necessarily skilled in music but knows a lot of history and grammar of music.

Attitudes. Develop from *affective* learning, i.e. the child observes how others of his community behaves in a given situation and tries copying it. This imitation is later modified by the personal opinion and knowledge of the child, when he grows up, and develops into his/her attitude. When we say that a particular singer has a very 'flamboyant attitude' while singing, we are indicating the ease with which he or she sings. If the people of an area have a 'hostile attitude' to health services, they have *learnt* to be hostile for some reason, which must be investigated.

Skills. Skills are developed by *psychomotor* learning, a classical musician has to undergo years of rigorous training of his spinal reflexes, vocal cords and speech mechanisms before he becomes 'skilled' at singing/playing an instrument.

Social problems

Individual problems become social problems when they affect a large number of populations amounting to threat the welfare or safety of the whole group, i.e. poverty, crime, disease, population growth, and alcoholism.

Social pathology is said to be the relation between a social problem and development of a disease. It is the equivalent of clinical pathology in community medicine.

Social defense

Preventive, *therapeutic and rehabilitative* services for protection of society from antisocial acts, criminal or deviant conduct. Examples: Prevention of control of juvenile delinquency in children, Elimination of Prostitution and Suppression of immoral traffic of women and girls act (1956).

Social treatment

Measures for creating a healthy social environment.

Social security. The implicit sense of being safe, both physically and economically, afforded by the society. When a woman boards a train compartment full of men, she is assured that nobody will assault her because of the *implicit contract of security* which applies when being among many people. Similarly, the person who suddenly loses his job or liquifies all his bank assets can at least depend on his family, friends and society at large to 'buffer' him while he looks out for another job. Attemps have been made to formalise these informal contracts. Examples are

- Employee's State Insurance—The formal method through which employees of a certain organization contribute to support one of themselves when he is physically/financially distressed.

- Pension—A legislation of the gratitude of an organization to an employee who has served them for long.
- Life insurance—Many people accumulate money, little by little, into a central organization, which uses this money when anyone of these people are sick/distressed.

Fair distribution of wealth. Most criminals are not born but pushed into crime because of poverty.

Facility for exercise of leisure. It is important that children be given a chance for all round development, otherwise some of them will grow up to be sociopaths.

Educational facilities. Not only formal education, but value and moral education helps to foster the inculcation of social norms.

Propagation of healthy customs. Monogamy (specially in the time of HIV), freedom of expression, etc.

Framing and enforcement of laws. Protection of property, life and honor.

A burning social problem – Addiction

Repeated use of a psychoactive substance(s), to the extent that the user is

- Periodically or chronically intoxicated
- Shows a compulsion to take the preferred substance(s)
- Has great difficulty in voluntarily ceasing or modifying substance use
- Exhibits determination to obtain psychoactive substances by almost any means
- Typically, tolerance (tendancy to increase the dose) is prominent
- A withdrawal syndrome frequently occurs when substance use is interrupted.[157]

Addiction can be prevented by education of the target group and general public. The 'Narcotic drugs and psychotropic substances Act' of 1985 is the law in India against drug abuse.

Social organizations

Formal

These are groups of people rationally structured in a hierarchy and pursuing some specific goal like unions, clubs, hospitals, school and police.

Temporary social groups

Crowd. A group of people who has a common interest but no leader and disperses when interest is over (i.e. the crowd that gathers at the site of an accident).

Mob. A crowd with a leader and often a symbol/slogan. The leader leads the team into action (i.e. a protest march against inflation).

Herd. Crowd who have to blindly follow a leader (tourists following a guide).

Permanent social groups

Band. It is made of few families living together who have organized themselves to a pattern of life (gypsies).

Village. Collection of people permanently settled in a locality with their home and cultural equipments. Even after rapid urbanization. India remains a country

dominated by village scape. Caste, religion, rituals, kinship, marriage and economy are important aspects of Indian villages.

Town and city. A relatively large, dense and permanent settlement of socially heterogeneous individuals.

State/nation. Ecological or social group based on territory and common ideals – the definition is very heterogeneous in different parts of the world. Some nations, like the 'United States', is really collection of independently operating states. Others, like India, is a single nation divided into states for administrative purposes.

The family

It is a group of people

- Related by blood, marriage or adoption
- Following their individual roles and common culture
- Often constituting a single household
- Interacting and communicating with each other.

The family is the primary unit of all societies. An individual usually spends his/her life in two families.

1. Family of origin (in which the individual is born into)
2. Families of procreation (the one set up after marriage).

A group of people living under the same roof, but not related by blood/marriage/adoption, are said to be living in an **institutional household** (jail, hostels, and hotels).

Nuclear families	Couple + unmarried dependent children. Because one or sometimes both of the parents are working, the children have much more free time which they have to spend alone. Until one of the parents spares sometime, such families are not good for raising kids. A family <10 yrs is called a *new* family.
Joint families	Lateral extension of nuclear family where families of siblings live together. It generally has a headman and all the earning members bear the expenses together.
Extended families	Linear extension of nuclear family, made of three or more generation (grandpa → father → son).

A family is a

1. **Biological unit** as the members share a gene pool
2. **Social unit** as the members share a common physical and social environment
3. **Cultural unit** as the family reflects the culture of the wider society and determines behavior, attitude of the members.
4. **Epidemiological unit** because it is the unit for providing social services and comprehensive medical care.

Family cycle

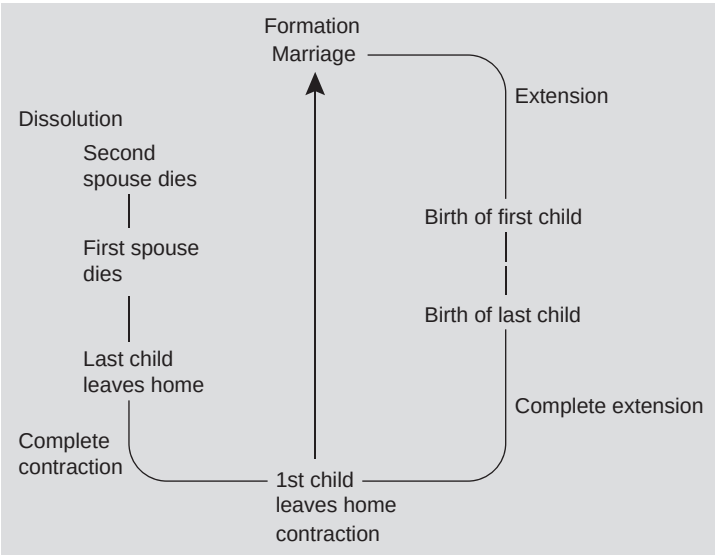


Figure 4.10. Family cycle

Dominance

Patriarchal family

It is prevalent in most cultures; the father is the principal legal guardian of the child and gives him his name. He is the head of household and takes major decisions.

Matriarchal family

It is seen in some hilly tribes and Kerala. It gives more social security to women and children.

Fundamental nature of families

1. Universal or permanent as an institution, but as an association it is only temporary (a particular family lasts for only a few decades, but the 'concept' of family has remained intact for thousands of years).
2. Provides emotional support for all its members (a joint family is kind of a shock absorber).
3. Economic unit both for production and consumption; everyone in the family works according to his capacity and receives his need (a kind of mini-socialism).
4. Active cooperation between family members is the best method for infant care, education, culture transmission, care of pregnant women, etc.
5. It has formative influence of children – It is the earliest social environment of life.

Functions of the family

1. Whilst many primitive (and some ultramodern) socities have experimented with casual sex, marriage (and optionally, having kids) have proved the only sure way to have sexual gratification without seriously disrupting the architecture of society.
2. Procreation—Marriage is not indispensable to have kids, but if those kids need to be properly incorporated into society, they will need a family (own or by adoption).
3. Legitimization of child birth—Children born outside marriage have difficulty establishing and legalizing their identity.
4. Viable production consumption unit—When measuring up a countries economy, it is most convenient if we count both production and consumption of goods in families (i.e. 'how many families live below poverty line?', etc.).
5. Social care—The family introduces each member to the society, sets up his/her status in society, shields the members from social problems, and regulates the political/social/religious activity of each member.

The family in health and disease

Child rearing

The family takes physical care of the dependent young – according to its resources, level of knowledge, state of technology and system of values. The family, other than food and clothing gives training on hygiene, discipline and ethics which are passed from generation to generation.

Socialization

The family teaches social values and transmits information, culture, belied, codes of conduct by example and precept; it is a kind of 'programming' which takes the child at par with the other members of society.

Personality formation

The family lays the foundation of physical, social and mental health of the child.

Care of dependent adults

The family cares for the sick/injured, pregnant and postpartum women, aged and handicapped. Often, the support of a sick person is more important in cure than medical treatment.

Familial disease

Genetic. Hemophilia, color blindness, diabetes, atherosclerosis, schizophrenia often run in the family.

Communicable. Diphtheria, measles, mumps, rubella, chickenpox, poliomyelitis, scabies and enteric fever spreads easily within the family.

Broken family

It is a family where either parents have separated/one or both parents have died. It plays havoc on the children and they show psychopathy, immature personality, growth retardation, speech and intellect retardation.

Problem families

While broken families are common in the west due to a large percentage of marriages ending in divorce, India seems to be teeming with **problem families**. These are the families which lag behind the community, i.e. their standard of life is much lower than the expected minimum. Parents are unable to meet the physical and emotional needs of their children. The underlying factors are often poverty, illness, mental instability (alcohol), lack of morality of parents, martial disharmony and drug/alcohol addiction in either of the parent. The children of such families, while mentally sound, usually become prostitutes/vagabond/criminals. These are also the families where 'selling' a girl child usually occurs.

Cultural factors in health and disease

Concept of etiology and cure

People still believe that disease have a mythical etiology. Precepts about bad water/air/blood are still very much prevalent, as well as taboos about 'catching a cold' because of eating rice at dinner (and many more you will encounter when you start clinical practice). Some of these beliefs have a rational basis, but most have none.

Environmental sanitation

It has long been the essence of Indian culture to concentrate upon the after life rather than the present one. Material well-being is not, supposedly, the Indian way of life. Thus Indian people chose to *consciously ignore* the problem of pollution and sanitation. Most of our population is at dark about the damaging effects of defecating here and there. They only *pretend* to know that mosquitoes breed in stagnant, waste water but still wash, drink and bath with their cattle in the same water.

Housing needs a special mention. The huge number of Indians living in slums are satisfied to live in damp and mossy kuchcha houses which forbid sunlight. They are used to sleep, eat, bathe (and often make love) in a single room. Even if offered a better deal, they refuse to abandon the slums.

Food habits

Hindu women are prone to fast in any occasion they find, sometimes without a drop of water (one of the causes of urinary tract infections); concept of 'hot' and 'cold' food is much prevalent in our country. Among the many myths regarding food, this one is the most hilarious: people in some regions still believe that pure milk, if dried – the cow which yielded the milk itself dries up (isn't that an excuse to add as much water as you like to the milk?).

Maternal and child health

This is critically important. Indian women breastfeed for longer periods, give regular oil message and sunbaths to the infants, so that infants are usually well to do for the period of lactation. The real problem starts after weaning from breast, and all kinds of useless food, sometimes inadequate in quantity, are offered to

the infant. Many families prohibit intake of egg/meat/fish protein in pregnancy (!). Some believe that colostrum is 'witch's milk' and do not breastfeed the baby for 1st three days. If all this is not bad enough, many pregnant women and their families distrust doctors and hospitals, and resort to a traditional (nontrained) *dai* during labor. About feeding the newborn, some find honey effective, and some go the extreme of giving opium/purgatives to the newborn.

Some customs are however, irrelevant – like pinching the baby (does nothing except irritate the child), applying oil over anterior fontanelle, etc.

Personal hygiene

Indian people bath regularly but are absolutely careless about oral hygiene. The general notion is anything will do to clean the mouth from broken herbs to charcoal; thus periodontosis and oral cancer is prevalent in India. Most barbers use the same razor over everyone's scalp and beard without any sterilization, and people are content with this practice. *Smoking* continues to be a major health problem after a quiescent period.

Marriage

Early marriage is usually associated with low birth weight, anemia, high maternal mortality. The Sharada act (1929), and later The definitive Child Marriage Prevention Act was passed in 1978 are examples of two social defenses to stop child marriage.

Socioeconomy

As wishful we are of an 'equal' society, where everybody gets according to need than according to his ability, until socialism prevails (and that day seems very far), there will be inequalities and maldistribution of resource in the society. The perception of 'social status' is, by this time, ingrained so deep into our subconsciousness that we are barely aware of it. But think of it: we judge people by their

- *Occupation*—We generally regard a doctor to be of higher status than a local businessman, even if the later earns more
- *Education*—Our tone changes drastically between speaking to a Professor and speaking to a bus conductor
- *Income*—The crudest measure of social status; when we are unsure of the above two, we use the income to simplify the process. Between two doctors of the same qualification, we generally regard the one with the larger fee to be more competent.

The *socioeconomic status* is the position of an individual/family with reference to prevailing available standards of cultural and material possessions, income and participation in group activity of the community.

Kuppuswamy scale for urban families

The scale is based on

- Education of head of family

- Occupation of head of family
- Per capita income of the family.

Modified Kuppuswamy scale for 2007

Education

Professional Degree, PG and Above	7
Graduate	6
Intermediate or Past High School Diploma	5
High School Certificate	4
Middle School Completion	3
Primary School or Literate	2
Illiterate	1

Occupation

Profession	10
Semiprofession	6
Clerk, Shop Owner, Farm Owner	5
Skilled Worker	4
Semiskilled Worker	3
Unskilled	2
Unemployed	1

Per capita income (Rs. per month)

19575 or above	12
9788–19474	10
7323–9787	6
4894–7322	4
2936–4893	3
980–2935	2
Below 980	1

Total score

Upper	26–29
Upper Middle	16–25
Lower Middle	11–15
Upper Lower	5–10
Lower	< 5

Pareek's SES for rural families

It measures the SES of a rural family with reference to caste (a major factor in rural India), occupation, education, social participation of head of the family, land holding, housing, farm power, material possessions, family (type and size and features of members other than head).

The combined score puts a family in either of 5 SES categories – Upper, upper middle, lower middle, upper lower, lower.

The caste system and reservation

After India gained independence, the Constitution of India listed some erstwhile groups as Scheduled Castes (SC) and Scheduled Tribes (ST). The framers of the Constitution believed that, due to the prevailing caste system in India , SCs and the STs were historically oppressed and denied respect and equal opportunity in Indian society and were thus underrepresented in nation-building activities. The Constitution laid down 15% and 7.5% of vacancies to government aided educational institutes and for jobs in the government/public sector, as reserved quota for the SC and ST candidates (totally 22.5%). These numbers were to be periodically updated, according to demographic data. The Supreme Court ruling that reservations cannot exceed 50% (which it judged would violate equal access guaranteed by the Constitution) has so forth set a limit reservations.

The percentage of SC/ST population in India is 24.4%.[158] However, there are another class of backward people not included in these schedules. The **Mandal commission** lead by Bindhyeshari Prasad Modal reported in 1980 that such suppressed people (other backward castes or OBCs) constituted 52% of population. However, this data was based on the 1931 census, and mostly derived from projections. But whatsoever, the recommendations of the commission were implemented and 49.5% reservations were allocated in Government Jobs. In 1999–2000 survey, the National Sample Survey organization reported the OBC population to be 36%. As political as reservations have been, in 2005 the 93rd Constitutional amendment brought an added 27% reservations for OBCs (adding to the already existing 22.5% for SC/ST) in all higher educational institutions, which spurred furious debates and protests from students all over India in 2006. The object of this book is to inform, not indulge into any *-ism*, so I refrain from debating whether reservations are good or bad.

An alternative to caste based reservations. **The Sachar Committee** which has studied the backwardness of Indian Muslims have recommended following scheme for identifying *really* backward and needy people.

- Marks based on merit: 60
- Marks based on household income (irrespective of caste): 13
- Marks based on district in which person studied (rural/urban and region): 13
- Marks based on family occupation and caste: 14
- Total marks: 100.

Common terms in economics

Gross domestic product

It is the income generated from within the country (the 'wealth' of a nation generated within it.

Gross national income

The Gross National Income is GDP + *net income received from abroad*. It is expressed 'at current prices' (which is useful for international comparison) or 'at constant prices'(according to prevailing prices at a fixed base period in the past). India's GNI/per capita in 2007 is $950.[159]

Net national product

It is the GNP – capital consumed in production.

Purchasing power parity

It is the no. of units of a country's currency required to buy the same amount of goods/services as a dollar would buy in the US. The GDP and GNI are usually expressed in purchasing power parity.

Poverty line

It is the income that allows an individual to eat 2400 cal/day in rural area and 2100 cal/day in urban area. In Indian currency, that amounts to 228.90/capita/month and 264.10/capita/month respectively (at 93–94 prices). The Planning Commission of India estimated that 27.5% of the population was living below the poverty line in 2004–2005, down from 51.3% in 1977–1978, and 36% in 1993–1994. The source for this data was the 61st round of the National Sample Survey (NSS) and the criterion used was monthly per capita consumption expenditure below Rs. 356.35 for rural areas and Rs. 538.60 for urban areas.

Accoding to the new international poverty line (income < US$ 1.25 purchasing power parity per day), 41.6% of Indian Population are below poverty line.[160]

Social security

It is the security that society furnishes through appropriate organizations against certain risks to which the members are exposed (sickness, invalidity, maternity, old age, death).

Assistance

Social assistance is a device to provide benefit for person of small means granted as *right* and in amount sufficient to meet a minimum standard of need and financed from taxation/general revenues.

1. Workmen's compensation (pension) act 1932
2. Central maternity benefit act 1961.

Insurance

Social insurance is a device

- To provide benefits for persons of small earnings
- Granted as of *right*
- In amounts which combines the contributive efforts of the insured with
- Subsidies from the employees and the state.

The chief difference between assistance and insurance is that, a part of the payment is taken from the person's own savings in the case of insurance. Examples of insurance are

1. ESI Act 1948
2. Family Pension Act 1971
3. Provident Fund
4. Community based Universal Health Insurance scheme (2003)—Insurance for Rs. 1 per day (poor) or Rs. 2 per day for a family of seven or more.

Employees State Insurance Act (1948)

It is the comprehensive act which covers all sectors of Government employment except mines, defence and rail. It is an insurance scheme financed by funding from employees, employers and Government.

Coverage

1. Nonpower using factories employing > 20
2. Power using factories employing > 10
3. Road transport
4. Newspaper
5. Shops, cinemas, theaters, hotels and restaurants.

Only employees earning < 7500/month are covered.

Structure

ESI Corporation, a board of
Chairman - Minister of Labor
5 members selected by Central Government
1 member submitted by each state and union territory
5 selected by employees all over the nation
5 selected by employers
2 doctors
3 selected by Member of Parliaments

↓

Vice Chairman

↓

Director General

↓

Four principal officers—
Medical commissioner
Insurance commissioner
Finance commissioner
Actuary

Benefits

The ESI offers two kinds of benefits

1. **Direct medical schemes**—In areas with 1000 or more employee families, **ESI clinics** are established, which provide preventive and curative services

to all the employees and their family. If the number of employees is between 750–1000, part-time clinics are run.

2. **Indirect medical schemes**—'Insurance Medical Practitioners', a special group of doctors selected by the ESI are appointed in the ratio one doctor per 750 employee families (maximum).
   ESI enjoys the highest doctor: Population ratio in India.

Benefits for employees

1. **Medical**: OPD, drugs, specialist, pathology and X-ray, domiciliary service, antenatal/natal/postnatal care, immunization, family planning, emergency service, ambulance, health education, hospital care, personal devices and appliances (prosthetics, etc.) for those incapacitated due to injury durng employment.
2. **Sickness benefit**: 50% wage for 91 days under treatment of ESI, 309 days for some chronic diseases; to reap this benefit, the person should remain under treatment of ESI and not any other organization, if not referred by an ESI doctor.
3. **Maternity benefit**: Full wage upto 135 days of pregnancy (6 weeks if a miscarriage occurs) and 30 days leave for puerperium.
4. **Disability benefit**: The act provides cash payment, besides free medical treatment, in the event of temporary and permanent disablement as a result of employment injury. Total permanent disability → life pension; partial permanent disability → life pension at partial rate; temporary disability → 85% wages.
5. **Dependency benefit**: In case of death from an employment injury, the spouse and children of the employee are paid 40% more than standard pension for until the children become 18 years of age or there is an unmarried daughter.
6. **Funeral expense**: Rs. 2500.
7. **Rehabilitation benefit**: Available at monthly Rs. 10 deposit.

Benefits for employers

1. Do not have to give social assistance (Workmen's compensation, Maternity benefit, etc.); everything is covered under ESI.
2. Do not have to pay medical allowance to employees.
3. Get an income tax rebate.
4. Lastly, get a healthy workforce.

WOMEN AND CHILDREN

Most National Health Programs have focused on women of reproductive age (15–44, constituting 22.2% of total population) and children (under 15, 35.3% of total population), because

1. By sheer numbers, they constitute 57.5% of population.
2. They are vulnerable to certain conditions not otherwise implied; while the developed world has grown out of it, pregnancy still continues to be a 'risk factor' for fatal illness in developing countries; again, a great amount of illness and death occurs within first five years of life in developing countries, a period of life which is supposed to be the foundation for future.

3. According to the WHO, reproductive and sexual ill-health accounts for 20% of the global burden of ill-health for women;[161] this underlines their pivotal role in women's health care - one cannot really set up any health care for women without reproductive health services.[162]
4. Most of the problem of women and children are preventable. Because during intrauterine life the mother and child come in one package, and the close association with the mother continues until late childhood, mother and child constitute a single 'unit' of Reproductive and Child Health care (RCH).

Social obstetrics is the study of social factors influencing the health of women and children, and the influences it has on health care delivery. It is the general framework of RCH.

The maternity cycle

Although number of ova are fixed by birth, they do not become mature and ovulation does not occur until puberty, when a rise of estrogen causes the now famous **LH surge**. The first one or two menstrual periods are irregular and an ovulatory, after which the girl settles into a rhythm. Pregnancy has occurred at 5 years of age,[163] but generally we consider 15–44 to be the reproductive age group.

The 'choice' of menstruation. In earlier times, when girls were married in their childhood, they often became impregnated with their first ovulation, and over the prolonged period of pregnancy and extend lactation (often 2–3 years), did not see another period, following which they were usually pregnant again. Menstruation was infrequent, occassional, an 'aberration' rather than the normal rhythm that it is today. The modern woman has *chosen* the minor hassle of menstruation over the risks of frequent pregnancy.

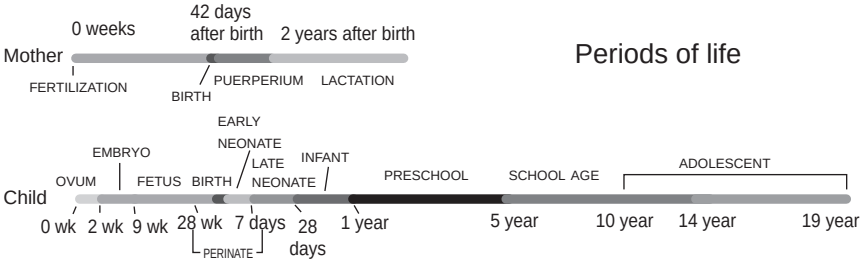


Figure 4.11. Periods of life

Fates of pregnancy

Delivery before viability (28 weeks IUL or 1000g weight of fetus)

- Spontaneous (abortion)
- Assisted - legal MTP (before 20 weeks), illegal MTP (after 20 weeks)
- Delivery after viability
- Spontaneous live delivery - term delivery (between 37–42 weeks), preterm delivery (earlier than 37 weeks)
- Dead delivery (stillbirth)
- Induced live delivery.

Problems of women and children

The problems of RCH revolve around this triad:

1. **Malnutrition**—Two phases of life are highly susceptible to develop malnutrition, intrauterine life and during weaning.
2. **Infections**—Maternal infections may lead to IUGR, congenital anomalies, abortion, LBW and puerperal sepsis. Infections in the child range from diarrhea and ARI to malaria and tuberculosis, and run a vicious cycle with malnutrition.
3. **Uncontrolled reproduction**—Most of MCH problems arise with pregnancies of too many and too frequent pregnancies.

Current RCH status of India

cBR = 23/1000 myp[164]
IMR = 54/1000 live births[165]
MMR = 301/100000 live births[166]
PMR = 37/1000 live births[167]
Under five mortality = 72/1000 live births[168]

Maternal mortality

Many people consider the day their child was born the happiest day in their life. In the world's wealthier countries, that is. In poorer countries, the day a child born is all too often the day its mother dies. The lifetime risk of dying in pregnancy and childbirth in Africa is 1 in 22, while it is 1 in 120 in Asia and 1 in 7,300 in developed countries. More than half a million women die in pregnancy and childbirth every year - that's one death every minute. Of these deaths, 99% are in developing countries. In addition, for every woman who dies in childbirth, around 20 more suffer injury, infection or disease – approximately 10 million women each year. One of the millennium development goals is to reduce MMR by 3/4th between 1990 and 2015.[169]

Maternal mortality = **Death during pregnancy** OR **intranatal period OR postnatal (6 weeks) period**, irrespective of site and duration of pregnancy, due to **causes related to or aggravated by pregnancy** or its management, and *not* accidental deaths.

Maternal Mortality Ratio[170]

$$MMR = (\text{maternal deaths in a year/live births}) \times 100000$$

National MMR = **301**/lakh live births (2003).

Causes

Antepartum/postpartum hemorrhage, sepsis, eclampsia, obstructed labor, ectopic pregnancy, embolism. *Anemia* contributes indirectly to all causes of maternal mortality.

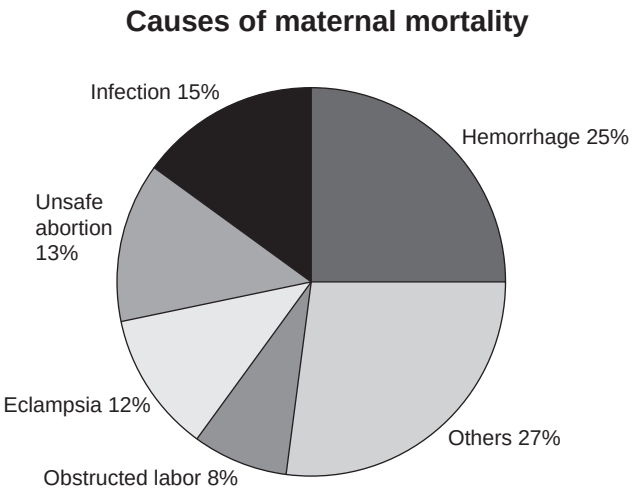


Figure 4.12. Causes of maternal mortality[171]

Postpartum hemorrhage

More than 500 ml bleeding from or into genital tract within 24 hrs of delivery. Primary PPH occurs in 2–3% of all deliveries.

1. Primary (with 6 hrs) – Due to separation of placenta
2. Secondary (after 6 hrs) – Due to infection.

Preeclampsia/eclampsia

Preeclampsia = Blood pressure > 140/90 developing after 20 weeks of pregnancy + albuminuria. It is manifested by

- Weight gain >3 kg/month
- Headache refractory to analgesics
- Epigastric pain and vomiting
- Blurring of vision.

ECLAMPSIA = preeclampsia + convulsion. *Treatment* of preeclampsia/or eclampsia is to stabilize blood pressure, sedate, maintain airway, rest the mother and counsel the mother and party about Caesarean section/MTP. When the mother is just a little stable, give 1st dose $MgSO_4$ at the PHC and refer to FRU. A person should accompany the mother to see if the tongue falls back.

Obstructed labor

Fetal descent < 1 cm/hr in primigravida or < 2 cm/hr in multigravida. The cause is usually something sinister (contracted pelvis/large baby, etc.) and must be investigated in an FRU.

Social factors

Social obstetrics is the study of social factors which affect reproductive health of women. It studies age at marriage, parity, socioeconomic status, environment and health services available to women.

The following social factors which adversely affect pregnancy have been identified.

1. **Age at pregnancy** <20 or > 35.
2. Birth spacing < 3 years; with frequent pregnancies, the body is not left with enough time to replenish all the nutrients, specially iron.
3. **High parity**—Each pregnancy weakens the smooth muscles of the uterus a little further, more so with a cesarean section.
4. **Low socioeconomic status**—A poor/uneducated woman is more likely to get married early, to get pregnant repeatedly and not to avail antenatal care.
5. Maternal undernutrition.
6. Insanitary environment which predisposes to puerperal sepsis.
7. Delivery at home/by untrained personnel which has been customary in India for a long time because mothers-in-law were horrified at the thought of a 'male gynecologist'; they wanted women doctors for their daughters-in-law, but paradoxically, were even more horrified at the thought of a girl going to school, let alone medical school to be a doctor.
8. Lack of referral facilities—Most maternal deaths are *intranatal and immediate postnatal*, so that referral to a FRU is essential in complicated pregnancies
9. Lack of empowerment of women.

Risk approach

All pregnancy is at risk, but a risk group has been identified.

1. Elderly primipara => 30 years of age
2. Short statured primi (≤140 cm)
3. Nullipara/multipara with 4 or more pregnancies
4. Teenage pregnancy
5. Bleeding in first trimester
6. Bleeding after 28 weeks (antepartum hemorrhage)
7. Malpresentation
8. Twins or hydramnios
9. Eclampsia/ preeclampsia
10. History stillbirth/IUFD/Cesarean section, difficult labor
11. Medical diseases (heart disease, diabetes, eclampsia, anemia, malaria, HIV)
12. Prolonged pregnancy (>14 days after EDD).

Prevention

See RCH package later.

Problems of Children

Developed countries

Prematurity, perinatal problems, developmental malformations, behavioral problems, accidents and injuries.

Developing countries

Low birth weight, malnutrition, infections, accidents, injuries, behavioral problems.

Major causes of mortality in children

1. Birth injury/asphyxia
2. Acute respiratory infections
3. Diarrhea
4. Tetanus.

Mortality is greatest in early neonates > late neonates > postneonatal infants > 1–4 years > 5–14 years. Similar to women, a group of at risk children has been identified.

Children at risk

1. Low birth weight
2. Artificial feeding
3. Birth order ≥ 5
4. Spacing < 24 months
5. Failure to gain weight in 3 successive months
6. Death of one/both parents
7. Severe acute infection
8. Moderate (<70%) malnutrition
9. History of death of sibling before age of 24 months.

Rights of the child

In recognition of the newfound awareness of health, the UN in 1959 declared a set of 'rights' of children, which includes free education, access to recreation, developing in an effectionate environment, social security, having a *name* and nationality, to be given priority in all relief activities, to be a useful member of society, to be given special care if handicapped. These rights are granted without bars of sex, race, color, religion, nationality or social status.
The **Children's Day** is November 14th.

Stillbirth

A **stillbirth** is the death (and subsequent delivery) of a fetus weighing 1000g (usually after 28 weeks of IUL) or more. The **stillbirth** rate is **number of stillbirths per 1000 (live + still) births** = 9 in India.[172] Stillbirths do not necessarily indicate bad obstetric care because causes of stillbirth are often

- Unknown
- Difficult to detect (chromosomal anomalies)
- Difficult to control[173] (maternal diabetes, Rh incompatibility)
- Impossible to control (fetal malformations, cord around the neck of fetus, multiple pregnancies).

Low birth weight (< 2.5 kg)

LBW babies may be classified into two groups

1. Preterm (delivered earlier than 37 weeks)—These babies survive with difficulty as their surfactants and respiratory centers are undeveloped, but once they live, their growth catches up with normal children in 2–3 years.
2. Full-term but growth restricted (small for date/Intrauterine Growth Restriction) – They weight < 10th percentile of the normal weight for any gestational

age. They are prone to death during neonatal period, infancy and also suffer malnutrition.

The prevalence of LBW is 28% of all live births in India, and more than half of these belong to the 'small for date' group. Because Indian babies are usually smaller (but no less developed) than western ones, the cut off should be 2 kg for Indian babies rather than 2.5 kg.

Table 4.7. Table of normal fetal weight at later pregnancy

28 weeks	1 kg
32 weeks	1.75 kg
36 weeks	2.75 kg
37 weeks	2.5 kg

LBWs are further classified as *very* low birth weight (< 1.5 kg) and *extremely* low birth weight (< 1 kg).

Causes

- **Preterm delivery**—Hard work, acute infections, multiple pregnancy, hypertension (all of which predisposes to early rupture of membrane and separation of placenta)
- **IUGR**—Low weight gain during pregnancy, short statured mother, teenage pregnancy, primipara, too many and too frequent pregnancies, anemia, preeclampsia, smoking, strenuous physical activity, illiteracy, low SES, intrauterine infections (specially TORCH infections), chromosomal abnormality.

Consequences

↑ morbidity and mortality (LBW has the most *direct* association with infant mortality); ↑ incidence of asphyxia, infection, hypothermia, malformations, mental retardation; high cost of neonatal care.

Prevention

Primary prevention

1. Identify at risk women according to etiology (see earlier), counsel them to avoid pregnancy and if they are already pregnant, provide extensive antenatal care and hospital delivery.
2. Increase food intake of pregnant women (the RCH proposes at least one extra meal per day); control maternal infections.

Secondary prevention

1. Early diagnosis of treatment of unrecognized infections (malaria, urinary tract infections, cytomegalovirus, toxoplasmosis, rubella, syphilis)
2. Early detection and treatment of anemia, hypertension, diabetes.

Treatment

Low birth weight babies (specially those below 2.5 kg) need to be treated in a Neonatal Intensive Care Unit, which is a very specialized disciplines; refer to any textbook of neonatology (or visit the NICU of your Medical College).

Infant mortality

Epidemiologist have long regarded mortality before 1 year of age as the most useful indicator of not only health status. but general standard of living of the people and effectiveness of interventions for improving maternal and child health in a country. This is because

1. It addresses the largest single age category of mortality
2. Deaths in this age group is due to a limited set of diseases, and not as diverse as the adult population
3. IMR is affected directly and quickly by health programs.

Infant mortality rate

IMR = (number of infant deaths (< 1 year of age) in a year/number of live births in the year) × 1000

National IMR = **54/1000 live births**;[174] (30.15 in 2009 estimates[175]

Regional variations. While IMR is low in Kerala, Tamil Nadu, Andhra Pradesh and Punjab, it is extremely high in Uttar Pradesh, Orissa, Bihar and Madhya Pradesh.[176]

Causes

Biological

1. Birth weight—Babies < 1 kg at birth will invariably succumb without intensive resuscitation measures.
2. Age of mother < 19 years and > 30 years.
3. Sequence—The first born is usually prone to die because of the inexperience of the mother in maintaining good pregnancy and taking care of the child; she learns from it, and the second born usually survives. However, from the 3rd born onwards death rates rise, partly because of maternal age, part neglect and partly due to too frequent pregnancies.
4. Birth spacing—Too frequent pregnancies (closer than 3 years) damage both ways: the mother cannot restore her stores, and she has to wean the first baby off the breast early so that the baby becomes prone to infection and malnutrition.
5. Multiple births often cause each baby to be of low birth weight.
6. A **large family** hinders maternal care to each child, so that they fall sick easily.

Socioeconomy. Needless to say that the average newborn in a slum has less chance of life than an average newborn in a middle class family.

Culture. It is a shrewd mystery why our people choose the worst of places (the cowshed), the worst of appliances (straw mats for a surface, husband's razor for a blade, cow dung for a cord stump, purgatives for feeding) and the worst trained of people (the indigenous dai) for the most welcome event of the family (the birth of a child).[177]

1. Malpractices regarding **breastfeeding**—Most Indian mothers will breastfeed for adequate periods if given a chance, but they are often interrupted by prelacteals and intermittent doses of top feeding (anything other than breast milk) by their mothers-in-law.

2. Customs and traditions regarding delivery.
3. Early marriage and pregnancy.
4. Neglect/murder of female child.
5. Quality of parenthood—Indian parents are generally very efficient at child rearing even in tough times, except when the one of them goes addicted/ antisocial (which is somewhat common in India) or they get divorced (much rarer in India).
6. Education of mother—It is ultimately the mothers who rear us into the world, see through our growing years and make an indelible impression on both our physique and psyche, it is only just that they should know what's right and wrong.
7. Unavailability of health care—India is yet to achieve the target of 100% deliveries by trained personnel.
8. Neglect of the *illegitimate child*.

Classification

Neonatal deaths (within first 28 days of life)	**Postneonatal deaths (from 29th day to 1 year)**
Due to causes carried over from intrauterine life (prematurity, birth injury, low birth weight, congenital anomalies, intrauterine infections) • Mortality in the first 7 days is **early neonatal mortality** is almost always caused by causes 'carrier over' or endogenous • Mortality in the rest 21 days, **late neonatal mortality** is often due to acquired infections	Due to newly acquired infections (ARI, diarrhea), malnutrition
Male children die more in this period	Female children die more in this period (they usually survive the neonatal period but fall victim to malnutrition and infections due to parental neglect)
55–60% of infant mortality occurs in neonatal period (and maximum within 1st 24 hrs after birth); the neonatal mortality in India is 37/1000 live births[a]	40% of IMR (23/1000 live births, as per SRS 2003)
[a] Sample Reporting System, Report 4 of 2007	

Prevention

This lists the essentials interventions alongwith the staff responsible for it.

Primary

1. Essential obstetric care, provided by nurses, anganwadi workers (AWW), MPHW and medical officer (MO); the focus should be on adequate nutrition, to which end nutritional supplementation should be provided to pregnant women by AWW.

2. Essential newborn care (prevent hypothermia, care of skin, cord, eye, breast-feeding, BCG and OPV vaccination prompt referral) by the nurse and MO.
3. Postneonatal care (immunization, growth monitoring, complementary feeding and exclusive breastfeeding) to be provided by nurse, MO, MPHW, AWW.
4. Immunization and vitamin A prophylaxis through public health nurses.
5. Growth monitoring by AWW.
6. Education on family planning, pregnancy and child care, by MPHW and AWW.
7. Planning and implementation—Establishment of first referral units, Baby Friendly Hospital Initiatives, allocation of funds, to be carried out by health officials.
8. Intersectoral coordination with departments of education, rural welfare, public works department (for environmental sanitation), etc.
9. Social measures—Education and empowerment of women, to ensure a basic level of income for every family so that they stop producing children as an 'insurance' for future.

Secondary

1. Early diagnosis and treatment (or referral to FRU) of complications of pregnancy—MO
2. Identification of neonatal distress and referral to NICU
3. Diagnosis and treatment of diarrhea and acute respiratory infections – MPHW.

Perinatal mortality

A *perinate* is a person from 28 weeks of gestation *OR* weighing > 1000g in IUL to 7 days after birth (early neonatal period). Perinatal deaths thus can be *late fetal or early neonatal*.

Perinatal mortality rate

PMR = (perinatal deaths in a year/live births in the year) × 1000
= number of late fetal and early neonatal deaths weighing > 1000g in a year/number of live births weighing > 1000g in the year
(WHO – for international comparisons)

PMR in India = **37/1000** live births[178]

Causes

Antenatal. Severe anemia (Hb < 5g/dl), hypertension, diabetes, syphilis, malaria, sepsis, hypoxia, pelvic diseases (contracted pelvis/pelvic neoplasms), uterine anomalies, Rh incompatibility, antepartum hemorrhage, congenital anomalies, cervical incompetence

Intranatal. Preeclampsia and eclampsia, birth injury, birth asphyxia, cord prolapse, dystocias, cephalopelvic disproportionation, amnionitis, premature rupture of membrane

Postnatal. Prematurity, congenital anomalies

Importance

1. Because *early* neonatal deaths are harder to prevent than later ones, PMR is a better indicator of RCH services than IMR.
2. Factors for early neonatal death and late fetal death are similar.

3. Many early neonatal deaths are, by mistake, recorded as stillbirths—Thus the IMR gets reduced while stillbirth rate shoots up; the PMR includes both kinds of deaths.

But there still exists lack of uniform definition (should we consider 1000g or 28 weeks as the landmark?), gross under reporting and difficulty in estimating gestational age of dead fetus (which often requires fetal measurements not easily carried out in a PHC).

Prevention

Primary prevention. Preconceptional care (counseling about age of pregnancy), family planning, genetic counseling, advice to avoid pregnancy when affected by major disease (heart diseases specially Eisenmenger syndrome, diabetes, etc.).

Secondary prevention. Prenatal diagnosis of birth defects, essential obstetric care, mandatory hospital delivery for high-risk mothers, careful monitoring of labor, maintenance of '5 cleans' in home delivery, essential neonatal care

Perinatal deaths should be classified as 'unnatural deaths' and an autopsy should be carried out, if possible.

1–4 year mortality

It is the age specific mortality of 1–4 year children. The **1–4 year mortality rate** = (number of deaths between 1–4 years of age/total number of children between 1–4 years) × 1000. It is,

1. More refined indicator of social situation than IMR, because it does *not* include those causes of deaths 'carried over' from uterus, and largely beyond social control (i.e. birth asphyxia, intrauterine infection, prematurity)
2. Usually causes by malnutrition, poor hygiene, infection (diarrhea and ARI), accidents.

The *2nd year* seems to be the most vulnerable.

Under five mortality

UNICEF uses under five mortality as the most significant indicator of social development, rather then HDI or GDP, because

- It measures an *end result* of efforts rather than the efforts themselves (i.e. percentage of school enrolment, which indicates how many children are admitted to school but does not tell how many are getting education)
- It is a sum of variety of efforts (treatment of infection, nutrition, environmental sanitation, antenatal care), etc.
- Unlike GDP or HDR, it is not biased towards a wealthy minority; a wealthy man might have an income 1000 times more than a poor man, and can alter the HDR significantly. But his child is not 1000 times less likely to die than that of the poor man while both live in the same country.

Under five (child) mortality rate

Under five mortality rate = (number of deaths < 5 years in a year/number of live births) × 1000; **72/1000** live births in India.[179] India ranks **49th** in under five mortality. The **Child survival index** is (1000 – U5 mortality)/10, **92.8** in India.

Handicapped children

The causes of handicap in children may be

1. **Physical**—Blindness, deafness, limb weakness (specially due to poliomyelitis), congenital anomalies (i.e. cleft lip), acquired defects (due to leprosy)
2. **Mental**—Retardation (IQ < 70) , which may be due to genetic causes (Down's syndrome), antenatal (folate deficient, CMV infection), perinatal (birth asphyxia) and postnatal (malnutrition, hypothyroidism, environmental exposure to lead and mercury, etc.)
3. **Social**—Orphans and street children who have the potential but not the opportunity to grow up as a responsible citizen.

Prevention

Primary

Genetic counseling to at risk (elderly) mothers, folate supplementation during pregnancy, avoidance of radiation and teratogens during pregnancy, polio vaccination of the newborn, good intranatal care and prevention of birth asphyxia. However, s*ocial handicap* is not amenable to medical measures and could only be corrected by socioeconomic development of the country as a whole.

Secondary

- **Early diagnosis of handicap/deformity**—Prenatal diagnosis of serious birth defects is a ground for MTP); even after birth, the parents should check whether the child attains normal developmental milestones in time (consult any textbook of pediatrics); ideally, each newborn should be screened for hypothyroidism
- **Treatment**—Some causes of handicap are amenable to treatment (hypothyroidism, leprosy), most other are not

Tertiary. The handicapped child must not be viewed as trash and treated like a normal child except allowances for his handicap. *Physical aids* such as muscle strengthening, crutches, automatic chairs, etc. solve only part of the problem. The more challenging problem is *mental* rehabilitation, that is digging the child out of his inferiority complex and make use of his faculties (albeit limited) to their full extent. The final step is *vocational* training which allows the child to pick up a skill of his or her choice, which provides some economic independence. Most Governments, including the Indian Government, provide aid and funds for rehabilitation of disabled individuals.

Child abuse and violence

Children are tired of being told they are the future. They want to see us fulfil our promises in the present, and enjoy their right to be protected from violence today.

—Paulo Sérgio Pinheiro, Independent Expert for the United Nations Secretary-General's Study on Violence against Children

I hate being a child

—Girl, 13, South Asia[180]

There are forms of child abuse and violence. *Extreme* forms of violence against children including sexual exploitation and trafficking, female genital mutilation/cutting, and the impact of armed conflict have provoked international outcry and achieved a consensus of condemnation (i.e. they are not 'acceptable' to the public at large). But many children are routinely exposed to physical, sexual and psychological violence in their homes, schools, care and justice institutions, the places they work and their communities, where violence against children remains legal, state-authorized, socially 'approved' (everybody knows proverbs such as 'spare the cane and spoil the child') and thus grossly under reported. It seems that most of adults believe that children are something subhuman, 'animals' whose reflexes are to be trained by violence, much like a ring master tames the tiger.

Types of abuse and violence

1. **Physical**—Corporal punishment (with canes, belts, etc.), forced into uncomfortable positions (like the 'chair'-a popular method in schools), female genital mutilation (prevalent in Africa).
2. **Mental**—Verbal abuse, locking in a room, prohibiting leisure activities, being 'bullied' by another child in school, seeing parents fight; **child labor** may itself be considered a form of abuse.
3. **Sexual**—India has had its share of pedophiliacs in recent times; most incidences of sexual abuse against children, however, do not make headlines. The 11-year-old girl in the joint family, due her own immaturity, could not possibly understand the significance of his 22-year-old cousin brother groping her, or even raping her, until it hurts. And when it comes out of the bag, the family will usually tell the *girl* to shut up rather than go to the police because of a certain abstract entity called 'family prestige'. Young boys are often sexually abused in their workplaces rather than homes, usually through anal sex (and thus may fall victim to HIV).

Acceptance of violence in children. The most frightening part of all this is that *children* may themselves accept some degree of physical, sexual and psychological violence as an *inevitable* part of childhood. They are often afraid to complain because they are taught to 'obey' adults even if the order is to put their pants down.

Effects

Exposure to violence in early childhood, whether as victims of or witnesses, can result in growth retardation, mental impairment, sociopathic behavior, substance abuse, early sexual activity and criminal tendency, anxiety and depressive disorders, impaired work performance.

Prevention

Early in the history of the UN, the international community recognized the need to protect the human rights of children. The Declaration on the **Rights of the Child**, proclaimed by the UN General Assembly in 1959, set out 10 principles aimed at providing special safeguards for children. The Convention on the Rights of the

Child establishes high standards for child protection both in the public sphere and the private sphere of the family.

- Laws (and enforcement of them) against child abuse, so that their are no legal premises left to justify violence against children in any form.
- To establish voluntary as well as governmental agencies to keep vigil on child labor, violence, prostitution and trafficking.
- Provision of recovery and social reintegration services for children.
- Create accessible and child friendly recovery systems, child guides and counsellors.
- Education to parents and would be parents.

Gender differences

Given a chance, boys and girls grow up, on average, to be equal citizens. But being the self destructive species that we are, it is no wonder that boys and girls are each burdened with their own set of problems since childhood, which are entirely preventable and unnecessary:

1. **Boys** are expected to live upto the 'male stereotype' of being macho and aggressive all the time. Through subtle methods, the parents, the family and society and general are always telling the boy 'you are the man, it's a big bad world out there, go fight it out'. Boys try to follow this model as closely as possible, and soon find that not only the society *accepts* some degree of violence from a boy, but also expects it. He finds fighting in school gangs, bullying weaker or younger children, spitting and smoking in public, swearing at each other, insulting passers-by and teasing/groping girls (or in absence of girls, groping each other) very 'manly'. The trouble is, every boy is not born equal. There will always be the misfit who will not fall for this cult, and he soon finds himself a victim or an outcast, depending on his intellectual position. In adult life, these 'manly' trends often transform into drug abuse/ hypersexuality/criminal behavior.
2. For **girls**, life is a struggle for survival. They are unwanted in the family, more likely to die due to parental neglect in infancy, more prone to malnutrition. Many parents in India still don't give a damn about sending their daughters to school. Like boys, a certain 'social sex' is imposed upon them, through dolls and kitchen sets in their childhood, and through sexual abuse in their adolescence. It is hard wired into their consciousness that they are objects to be subjugated, manipulated and disposed off at will of men. And if she escapes all this, there is still high possibility that she would be stolen/sold from her parents and trafficked into prostitution, or worse, be married in childhood with a dominating husband (through the incentive of a massive dowry), get pregnant in the teens and die in childbed. No country for women, eh!

One of the UN Millennium Development Goals is to eliminate gender disparity in primary and secondary education preferably by 2005, and at all levels by 2015.

Child labor

An estimated 20% of GNP of India is produced by child labor (!). India cannot find jobs for 60 million adults but can find space for 111 million (estimated by ILO, 2005) child laborers.[181] Working children are deprived of education, good nutrition, are forced into long hrs and often dangerous/toxic jobs (like cleaning garbage, making fireworks) with little wage. In short, they live the life of adults, without the pay and rights. In Northern India the exploitation of little children for labor is an *accepted*[182] practice and perceived by the local population as a *necessity* to alleviate poverty (Fig. 4.13). Most of the these workers are migrant children who cannot go home, sleep at their work place, and are 'trapped' because their family depends on their income. Rural families who are ailing with poverty perceive their children as an income generating resource to supplement the family income. Parents sacrifice their children's education to the growing needs of their younger siblings in such families and view them as wage earners for the entire clan (i.e. first they produce a litter and then dump the responsibilities to the eldest child).

Girls are often trapped in a far worse situation than this, known as a 'maid servant'. Although a major work force in India, the workplace of the maid servant is indoor, and never comes in public eye, unlike the boy who works in a tea stall. Apart from suffering all the disadvantages of child labor, they are also sexually exploited by the family members she works for, and she *has to* keep quiet for her job.[183]

Child trafficking. Children may be transfered out of their homeland by many means

- Being lured into a hypothetical job
- Forcibly abducted
- Sold by parents.

These 'effectively orphaned' children are then used for commercial sexual exploitation, prostitution or child pornography, forced labor or services, slavery, servitude, the removal of organs, illicit international adoption, early marriage, recruitment as child soldiers, for use in begging (usually after amputation of an organ) or as athletes (such as child camel jockeys or football players), and various other purposes. In total, children, specially **girls** are fast becoming a major export commodity of India.

Street children. Majority of street children have lost a parent, or both, or have left home because of abuse, or have been kidnapped. These children live the harsh life of adults, and become victim to sexual abuse, HIV, malnutrition, parasitic infections, drug addiction They are easy targets for drug dealers for production and distribution of illicit drugs. They are also often used as scape-goats by police and viewed as criminals or criminals-in-waiting.

Prevention

The Child Labor Prohibition and Regulation Act of 1986 allows employing children in 'family based work', and many employers take advantage of this clause and conjure up a distant family relation with his child employee. However, even if the law is strictly employed, the large amount of child labor and trafficking

Figure 4.13. Two roadside entertainers in New Delhi

that goes on unforeseen to public eye is likely to continue, because it is rooted in poverty, and with the current rate of inflation (and populism politics[184] that have become rampant), there seems no near end to poverty.

The **Immoral Traffic (Prevention) Act** (1956) provides for 7 years to life imprisonment for recruitment, transportation, transfer, harboring or receipt of a child (below 18 years) for the purpose of exploitation. Like child labor, the problem of trafficking is also socioeconomic than purely criminal/judicial.

Measures to circumvent the problems of MCH

This list will come helpful while answering any question regarding maternal and child health.

1. RCH package
2. Family planning
3. ICDS
4. Communicable disease control programs
5. Immunization
6. Social engineering
7. Better environmental sanitation
8. Improved socioeconomic conditions.

Growth and development

Growth

Growth is a net increase in the dry mass of tissue, due to hyperplasia and to a lesser extent, hypertrophy. It is the essential characteristic of a child which distinguishes it from an adult.

Assessment of growth: Anthropometry

Weight

Ideal birth weight is about 2.5–3 kgs. The birth weight reduces by 10% in the 1st few days of life, which is regained by the 10th day. Weight is recorded by a Detecto's scale (for < 10 kg babies), a standard scale (which you have to stand upon) or Salter scale (a spring balance, for community purposes).

Table 4.8. Expected weight

Birth weight	Attained at
× 2	5 months
× 3	1 year
× 4	2 years
× 5	3 years
× 6	5 years
× 7	7 years
× 10	10 years

Expected weight

Under 1 year, expected weight = (x + 9) / 2 kg (x = age in months)
Between 1–6 years, expected weight = (x + 4) × 2 kg (x = age in years)
Between 7–12 years, expected weight = (7x – 5) / 2 kg (x = age in years)

Weight is often used to classify malnutrition.

Length or height

Length is measured upto 2 years by an infantometer (a very atypical instrument which is to be used by two persons, one fixes the head and another the knee of the child). After 2 years, length converts to height (which is measured by a stadiometer, which is just those perpendicular scales you see in clinics). While measuring height make sure that the heels, buttock and shoulders touch the wall and make the child look straight so that Frankfurt plane (line joining external auditory meatus and inferior orbital margin) is horizontal.

Table 4.9. Expected lengths/heights

Birth	50 cm
1 year	75 cm
2 years	87.5 cm
3 years	95–98 cm
4 years	100–103 cm

Expected heights

Between 2–12 years, expected height = 6x + 77 cm (x = age in years).

Adolescence should add 8 cm/year (in girls) and 10 cm/year (in boys).

Unlike weight, the height seldom reduces once it is gained. A single measurement of weight gives information *only* about the present status of the child, whereas his height is a cumulative result of his nutritional history. A child of short height for age is suffering from chronic malnutrition or has some intrinsic disease. A child with normal height but low weight (low weight for height) is suffering from *acute* malnutrition recently; he has grown to normal stature due to good nutrition throughout his childhood, and it is only recently he isn't getting enough food, which caused his weight loss.

Head circumference

The maximum circumference of the head from the inion (occipital protuberence) to the supraorbital ridges is recorded (make sure the tape touches the mastoid process). The chest should grow to equal head circumference by 6–9 months if the child is properly nourished.

Table 4.10. Expected head circumferences

Birth	33–35 cm
3 months	40 cm
12 months	45–47 cm
2 years	48 cm
4 years	50 cm
12 years	52 cm

Mid arm circumference

It is the circumference of the arm midway between tip of acromion and tip of olecranon. It is an indicator of muscle mass *only* between 1–5 years.

Expected MAC

Between 1–5 years 16.5–17.5 cm. The **Shakir's tape** is used to measure MAC; it has colored markings for easy diagnosis of malnutrition

- 13.5 cm
- 12.5–13.5 cm
- < 12.5 cm

If a *bangle* of 4 cm could be passed across the entire forearm, the child is easily diagnosed to be malnourished.

Subcutaneous fat (skinfold) thickness

It is measured over triceps/subscapularis with *Harpenden calipers*. Expected thickness between 1–6 years = **10 mm**; if it is below 6, there exists moderate to severe malnutrition.

Growth chart

There are two 'standards' against which we judge the growth of a particular child.

1. The WHO-NCHS combined publication on weight for age in normal children (2006).
2. The ICMR standards of growth in Indian children.

For the purpose of easy diagnosis and follow up of malnutrition, **growth charts** have been devised from these data. The growth chart is used for:

1. **Growth monitoring** by health workers.
2. **Early diagnosis** of malnutrition; the visual impact of a falling growth curve is more appealing than tables of data.
3. **Planning and policy making**—Separate set of curves can be used in a growth chart for 'normal' growth and growth failure, which allows categorization of children according to growth.
4. **Education** of health workers and the mother.
5. **Evaluation of health services**—Whether children begin to show growth after some intervention.
6. **Taking action**—Depending on where in the growth chart a child is located, the health worker can deduce appropriate intervention.

WHO Growth Charts[185]

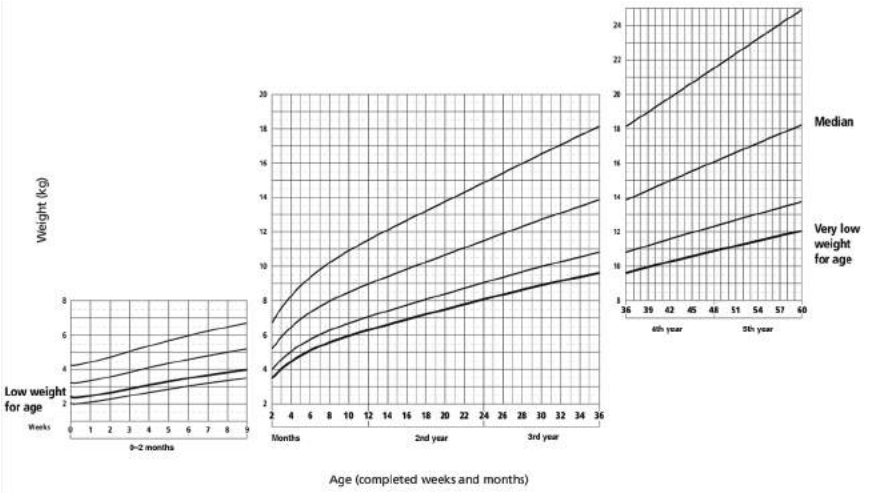


Figure 4.14. WHO growth chart for girls

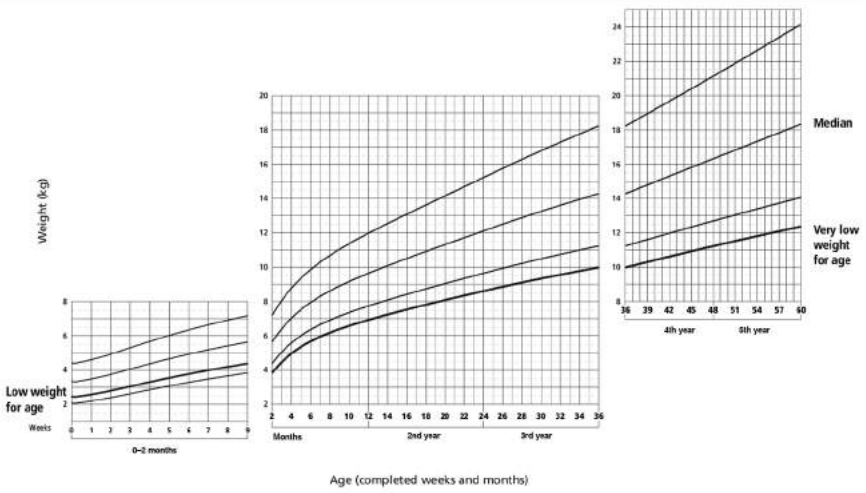


Figure 4.15. WHO growth chart for boys

ICMR growth chart

See 'protein energy malnutrition'.

The RCH package

Preventive, promotive, curative and rehabilitative services for mother and child. It is a need based, client centered, demand driven, integrated package with definite objectives, strategies and components.

Milestones

- 1952—National Family Planning Program
- 1974—National Family *Welfare* Program
- 1992–97—Child survival and safe motherhood program (CSSM)
- 1997–2002—RCH
- 2002 onwards—RCH II.

Aims

1. Enabling people to regulate fertility
2. Enabling women to go through safe pregnancy and childbirth
3. Ensure survival and welfare of mother and child
4. Enable couples to have sex free of fear of pregnancy or STDs.

Objectives

1. To reduce maternal and child mortality
2. Promotion of reproductive health
3. Promotion of physical and psychosocial development of child and adolescent.

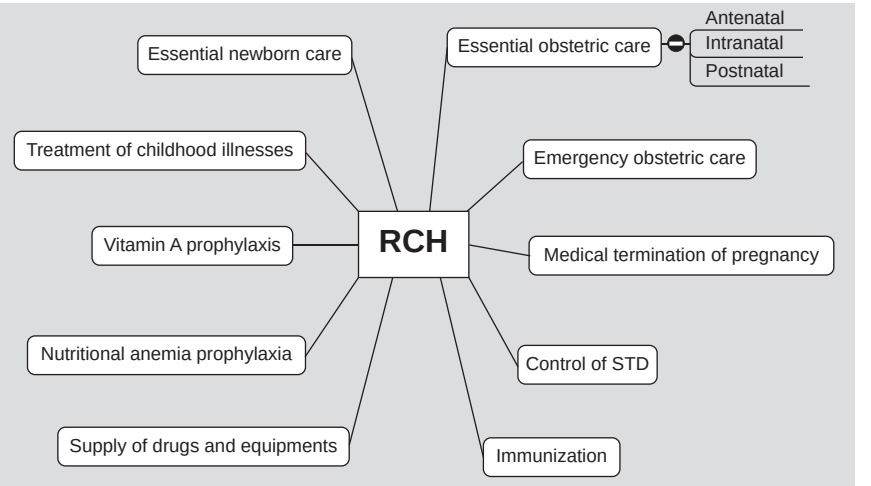


Figure 4.16. Activities of RCH

Changes in RCH from earlier programs

Community need assessment approach

The targets of RCH are set at the subcenter by the MPHW after house to house visit of his 5000 population and meeting the community leaders. The targets are sent to PHC to be reviewed by the MO. The MO makes demographic calculation to assess validity of the targets. Each year, expected rise in population is about 10%, so a 10% rise in targets is justified.

Differential approach

RCH has segregated districts according to cBR and female literacy, and provisions have been separated for each category. The weakest districts usually get the best support from RCH.

Life cycle approach

RCH targets at all stages of the development of a female – so that they live a healthy childhood and adolescence and give birth to a healthy child, and can take good care of the child.

Participatory planning

The community and village, subcenter level is the *planning center* of RCH (bottom up approach) rather than a central planning office.

Greater emphasis in quality

Strict quality control of services have been emphasized in RCH.

Preconceptional care (i.e. before fertilization takes place)

The RCH program extends well before pregnancy so that coming pregnancies may be made safer.

Adolescent (10–19) health care

Problems

Undernutrition, behavioral problems (teenagers are often reported to be 'difficult' by parents), unwanted pregnancies (due to lack of knowledge and unavailability of contraception), STDs.

Care for adolescents

1. Awareness and counseling regarding sex (through school curriculum, ICDS workers).
2. Extra food (specially iron and iodine); IFA tablets are distributed to adolescents of certain areas through schools and ICDS program.
3. **Rubella vaccination** at the beginning of reproductive age group; it is necessary for the vaccine to be given to exclude pregnancy first, and use some contraception for at least 8 weeks after vaccination; it is yet to be included in RCH program.
4. Screening for cervical cancer and HPV vaccination at the very beginning of sex life - not yet fully implemented.

Role of family and community

1. Safe and supportive environment
2. Encouraging interaction between parents
3. To provide correct information regarding sex.

Essential obstetric care

Antenatal care

'Ideal' births must be

1. Planned
2. Well-maintained during intrauterine life
3. Protected from congenital diseases.

Antenatal care is care during pregnancy to protect and promote health of mother and child.

Objectives

1. To promote, protect and maintain health of mother during pregnancy and to ensure a healthy baby at the end.
2. To detect high-risk mothers.
3. To monitor progress, foresee complications of pregnancy through risk assessment and its management.
4. To prepare the mother for delivery and child care.
5. To remove anxiety and fear of delivery.
6. To reduce maternal and infant morbidity and mortality.
7. To sensitize the mother to the need of family planning and MTP.
8. To teach the mother the basics of child care, nutrition, hygiene and sanitation.
9. To attend the children accompanying the mother.

Early registration

Register a pregnant mother *at first contact or within 12–16 weeks* (when the mother recognizes pregnancy).

Antenatal visits

Ideally, visits should be once monthly or in the later parts, once weekly. In the present situation of our country, we are not yet ready to provide the ideal. At least *three* checkups (**20 weeks**–fetus becomes palpable/audible, **32 weeks**–complete development of CVS, **36 weeks**) excluding registration is the bare minimum. At least *one home visit* should be made. In each visit

1. Assess duration of pregnancy (today – LMP).
2. Check whether the duration corresponds with uterine size.
3. Assess whether the mother is 'at risk'.
4. Examination.
   - Perception of fetal movements (appear in 18–20 weeks).
   - Height of mother (< 140 cm falls in risk group).
   - **Weight gain**—Normal pattern of weight gain is 0.5 kg/week in 2nd and 3rd trimester; weight gain outside 2–3 kg range/month is pathologic. **Excess weight gain** indicates polyhydramnios, preeclampsia or twin pregnancy. **Low weight gain** signifies IUGR.
   - Blood pressure—Measure a baseline for each individual. A BP > 140/90 after 20 weeks is indicative of preeclampsia, and a rise > 160/100 is dangerous.
   - Pallor (give IFA treatment).
   - Edema (to check eclampsia).
   - CVS and respiratory system.
   - Fundal height, fetal lie and presentation.
5. Any complaints? Ask for blurring of vision, pain abdomen, headache, edema which indicate eclampsia/preeclampsia.
6. Laboratory—Urine for physical and chemical examination, hemoglobin, blood group (including Rh) and blood sugar.

Table 4.11. The prenatal care chart

	First visit	15–20 wk	24–28 wk	29–41 wk
Complete history	+			
Updated history		+	+	+
Complete physical examination	+			
BP	+	+	+	+
Weight	+	+	+	+
PV	+[a]			
Fundal height	+	+	+	+

Contd...

Contd...

	First visit	15–20 wk	24–28 wk	29–41 wk
Fetal HR	+	+	+	+
Routine investigations				
Hb	+		+	
Blood group	+			
PP sugar	+			
VDRL	+		+	
HBsAg	+			
HIV	+			
Urinalysis	+			
Special investigations				
PAP smear	+			
Antibody screening	+			
Chromosomal anomalies/ birth defects	+	+		
[a] It should be done within 8 weeks of LMP to asses fetal size and importantly, ECTOPIC PREGNANCY				

Nutritional anemia prophylaxis and treatment

Iron-Folate (IFA) tablets

Adult—100 mg iron + 500 µg folate

Child—20 mg iron + 100 µg folate

Table 4.12. Nutritional anemia prophylaxis and therapy

Prophylaxis of anemia in pregnancy	IFA (100 + 500) × 1 × 100 days, after a meal, beginning from 2nd trimester; iron is usually avoided in first trimester because • Menstrual blood loss stops with pregnancy • Iron aggravates morning sickness
Therapy	For mothers: IFA (100 + 500) × 2 × 100 days, after lunch and dinner, beginning from 2nd trimester For child: IFA (20 + 100) × 1 × 100 days Add anthelmintics in endemic areas/worm passers

The IFA tablets are distributed by MPHWs with the advice that they are gastric irritants (take only after food). As a proof of consumption of tablets, the mother will usually pass black stools at the beginning. The MPHW will also advice the mother to eat more green leafy vegetables.

Tetanus immunization

The recommended schedule is two doses of tetanus toxoid: TT-1 at first contact and TT-2 after 4 weeks. If *documented evidence* of adequate immunization within

3 years is present, give *only* a booster dose. TT is effective in preventing neonatal tetanus *only* if given before 36 weeks. However, even after that period, it is still worth a try.

Advice

1. **Rest**—Sleep 8–10 hrs at night and 2 hrs during daytime; sleep over *left* side to avoid compression of inferior vena cava by uterus (the IVC lies on right side).
2. **Diet**—*One extra meal* in the afternoon; each day of pregnancy needs 300 extra kcals than normal, and weight gain in pregnancy should be 12 kgs in total.
3. **Hygiene**—Hand washing, food hygiene, water sanitation.
4. **Sex**—Avoid intercourse in the last 2 weeks of pregnancy as it may start off premature labor.
5. **Breastfeeding**—The antenatal clinic is an ideal opportunity to teach the importance of exclusive breastfeeding and when to start complementary feeding.
6. To avoid teratogenic drugs and radiation exposure (specially X-rays) during pregnancy.
7. Education on **family planning**.
8. The family members are advised arrange for transport facilities for referral to FRU, if necessary (the Government has set up schemes to arrange transport for at risk mothers).
9. Advice on newborn care (see later).
10. Teach how to identify **danger signs** of pregnancy (sudden vaginal bleeding, no fetal movements, inadequate weight gain, edema, blurring of vision, etc.).
11. Prepare for delivery—If delivering at home, get a trained dai with DDK or prepare such things beforehand; if delivering at an institution, select the institution and arrange for money, manpower and transport at the right time.

Specific diseases

The HIV +ve mother. HIV may pass onto the fetus from the mother both through placenta and breast milk. All HIV +ve mothers should be discretely counseled on what choice they make, i.e. MTP or proceed with delivery after prophylactic antiretroviral drugs.

Genetic counseling. See the chapter on genetics.

Rh iso-immunization. If an Rh –ve mother has an Rh +ve husband, there are chances of an Rh +ve baby too, in which case the first issue will be spared but the second child onwards have a grave risk of stillbirth (see any textbook of obstetrics). Ideally such mothers should be treated in secondary level health care.

- At 28 weeks, screen the mother's blood for Rh antibodies and if –ve, give prophylactic antiD Ig.
- At 35 weeks, screen again; if negative, keep her under observation and if positive, treat like a sensitized patient.
- At birth, collect cord blood and send for Hb, bilirubin, direct Coombs test, and blood grouping; administer 300 μg of antiD Ig to mother if the baby is Rh+ and there is no evidence of maternal antibodies.

Record keeping

1. Each mother is given an antenatal card, which is carried over to be the immunization card for the child after birth.
2. Each new mother encountered must be registered in the eligible couple and child register.

Intranatal care

Most maternal deaths are due to *intranatal* causes rather than antenatal. The things that usually go wrong are

- Any item used in delivery (the labor table, instruments, hands of the doctor) turn out to be contaminated, so that the mother develops puerperal sepsis.
- Some injury occurs to mother and/or the child.
- Uncontrolled postpartum hemorrhage.
- The labor gets prolonged/the mother develops eclampsia.
- Some obstetric complication like cord prolapse, malpresentation.
- A fault in neonatal care - either in maintaining temperature, asepsis, care of the eyes, etc.

Institutional deliveries

The national target is to get at least one trained personnel (doctor/ nurse/MPHW/ trained dai) beside 100% deliveries, and conduct at least 80% of deliveries in some health facility (institutional delivery). A book on community medicine is no place for a detailed discussion on how to conduct a delivery (go to the labor room of your medical college), I mention only a few things that greatly reduce maternal mortality.

1. Monitoring labor by a **partograph**; this chart has an *alert line* and an *action line* to indicate prolonged labor and fetal distress
2. Early identification of impending conditions like hypertension, preeclampsia, eclampsia, etc.
3. After delivery, examine the placenta for missing cotyledons, position of umbilical vessels, and any extra lobes; any missing cotyledon is *retained* within uterus and is a potential source of bleeding, search for it.

The mother should be discharged after 5 uneventful days of delivery.

Home delivery

Most rural women still prefer their homes for delivery rather than hospitals. The advantages of a home delivery are

1. Mothers feel *safe* and this emotional well-being greatly helps labor
2. No chance cross infections between patients, as frequently occurs in a hospital
3. No chance of mix up of babies
4. Easier to monitor the baby.

Home delivery is to be done by trained dais and if needed, female MPHW. Emphasize upon 5 cleans: **clean hands** (washing with regular soap will do), **clean surface** (a fresh polythene sheet), **clean cord tie** (ordinary cotton thread boiled for 20 minutes and dried in sunlight), **clean cord stump** (**nothing** to be applied over cord), **clean razor** (use a new blade for cutting the cord). Except the polythene,

all of these materials are supplied to antenatal mothers in a **Disposable Delivery Kit** (DDK).

Most homes in rural India are unsuitable for delivery, and the risk of complication is too great. Mothers are also forced to work too soon after delivery (being admitted in a hospital gives at least a week's rest from the chores of the house), and her nutrition is often ignored.

Indications for referral to hospital

1. No progress of labor (no pain) even after liquor has broken
2. Cord or hand prolapse
3. Meconium stained liquor
4. Fetal heart rate < 120 or > 160
5. Excessive bleeding during labor
6. Retained (not separated within half an hr of delivery) placenta
7. Postpartum hemorrhage
8. Core temperature > 38°C anytime during labor.

Newborn care

Half of infant deaths occur in neonatal period, and still half in the 1st 7 days. The prime causes, as already mentioned are asphyxia, hypothermia (axillary temperature < 36°C) and infections.

Levels of neonatal care in India

Level I: Primary health care	Staff: Pediatrician, obstetrician, anesthesist Aseptic delivery practice Essential resuscitation apparatus Promotion of breastfeeding
Level II: District and sub-divisional hospitals (FRU)	All complicated labors are to be referred here; facilities are • IV fluids and antimicrobials • Radiant warmer • Phototherapy (for jaundice)
Level III: Medical colleges	All intensive monitoring instruments facilities for total parenteral nutrition

Essential newborn care

1. Receive the baby in a clean surface (polythene sheet or sterile trays); if the baby does not cry, extend the neck and tickle the toes → still quiet? → ASSISTED RESPIRATION (ventilator/mouth to mouth).
2. Suck the mouth to clear oropharynx.
3. Ligate and cut the cord preserving at least 4 cm (on the babies side) *after it has stopped pulsating*; allow the stump degrade on its own in 5–8 days.
4. Examine the baby and take weight within ½ an hr (for home deliveries, weight may be taken within 48 hrs). The weight may be taken by a spring balance, flat balance, salter scale or a color coded spring balance.
5. Initiate breastfeeding within ½ an hrs of normal delivery, or 4 hrs of cesarean section.

Apgar[186] score

Sign	0	1	2
HR	Absent	<100	>100
Respiratory effort	Absent	Slow, irregular	Good, crying
Muscle tone	Flaccid	Some flexion	Active movements
Reflex response	None	Grimace	Cry
Color	Blue, pale	Body pink, limb blue	Pink
TOTAL	0–3 Severe depression	4–7 Mild depression	8–10 No depression

The Apgar score is taken after one and five minutes after birth. A score < 5 needs prompt action.

Resuscitation

Because birth asphyxia is a leading cause of death and future disability, it is worthwhile to read resuscitation thoroughly from a texbook of pediatrics.

Warmth

1. Just wipe the baby (very softly) and wrap in a *preheated* towel, do not rub off the vernix caseosa and allow it to dry; it is essential to cover the largest evaporating surface of the newborn, i.e. its *head*.
2. Wrap the baby in two layers of sterile cotton/clothing.
3. Put within 45 cm of a 200W bulb/place in an incubator/radiant heater, or better - get the baby to contact with mother (kangarooing) as soon as possible; this also facilitates breastfeeding.
4. If the temperature is still subnormal (< 36°C at axilla) refer to FRU.
5. If temperature is normal, the baby may be given a bath (although not practiced nowadays) in a few hrs and then again after 7 days.

Prevent infections

1. Tetanus immunization of mother prevents neonatal tetanus (*antenatal* pediatrics).
2. Maintain 5 cleans during delivery.
3. Clear the eyes with sterile saline from medial to lateral side; tetracycline 1% ointment may be given.
4. Breastfeeding within an hr of birth.
5. Restricted handling of baby (do not allow the kins to gather around the newborn).
6. Instill BCG and OPV-0.

Initial examination

The term infant is flexed, sleeps 80% of the time (specially in daytime) and when not sleeping, cries vigorously. First meconium is passed in 24 hrs, then a phase

of transitional diarrhea (green) goes on between 3–10 days until golden yellow formed stools come up. The first urine is passed in 48 hrs. Adequate urine output is 6–8 times a day.

Vitals. Color of the baby should be pink; there may be some peripheral cyanosis for 1–2 hrs. *Respiratory rate* should be between 40–60/min; **apnea** is defined as stoppage of respiration for > 30s, sometimes associated with bradycardia (a seizure, on the other hand, when manifested as apnea, will cause tachycardia).

Signs of respiratory distress in newborn

1. Tachypnea/bradypnea
2. Grunts - partial closure of glottis to maintain positive end expiratory pressure sign of surfactant deficiency
3. Stridor/wheeze
4. Use of accessory respiratory muscles
5. Chest and suprasternal in drawing
6. Cyanosis.

The *heart rate* (NOT pulse) should be between 120–140/min; count at least for 6 seconds. *Blood pressure* is in the range 60/40, which might not be measurable directly, so we have to resort to **capillary refill time** (indicative of blood pressure), which should be less than 3s. **Temperature** should be 36.5–37.5 (both in body and toes).

Maturity. The signs of a *term baby* are as follows,

1. Smooth thick black hair.
2. Elastic pinna (cartilage has developed).
3. Nipple > 5 mm in diameter.
4. Genitalia—Scrotum developed, rugous, pigmented and at least one testis is palpable (male); labia minora is wholly covered by labia majora (female).
5. Deep planter creases extend beyond anterior 1/3rd of sole.
6. Cannot cross elbow over midline, thus cannot touch opposite shoulder by hand (negative Scarf sign).
7. Can flex the wrist upto 90°; the premature has tense ligaments and cannot do so, because effects of maternal E and P have not taken full effect.
8. Can extend the ankle as to touch the leg.
9. Full Moro reflex develops after 35 weeks, so does sucking-swallowing coordination.

Anthropometry. The **birth weight** should be taken within 1 hr of birth. Normal newborn weighs > 2.5 kg and accumulates an additional 20–30 g/day). **Length** can be measured most accurately with an *infantometer* (ask the demonstrator of your CM department to know about it) within 3 days of birth; it is usually about 50 cm. Average **head circumference** of newborns is 35 cm and chest circumference 33 cm.

Secondary examination

The secondary examination should be carried out by a pediatrician with 24 hrs of birth, after the initial resuscitation is over and the baby is stable.

Head. Size, shape (may be altered by a caput or cephalhematoma), both fontanelle should be open just after birth; moulding may obliterate the sutures.

Check any trauma/forceps mark overhead and any anomalies - hydrocephalus, meningocele.

Back. Observe neural tube defects (specially a hairy nevus, meningocele or myelomeningocele).

Face. Many chromosomal anomalies (like Down's syndrome) are evident in face.

Eyes. Look for conjunctivitis, subconjunctival hemorrhage, microphthalmias, corneal dystrophies; massage the lacrimal duct if needed.

Nose. It is important to exclude choanal atresia by poking a catheter.

Mouth. Examine for cleft lip and palate, small tongue (mongolism), protruding tongue, oral thrush, presence of teeth (syphilis), epstein perals (epithelial inclusion cysts) on hard palate.

Skeleton. Look for common anomalies (*talipes equinovarus*, genu valgus/varus and developmental dysplasia of hip.

Sepsis. Look for sources of infection (conjunctivitis, thrush, umbilical discharge, more than 5 pustules in skin), diarrhea, convulsion, altered sensorium, respiratory distress.

Umbilical cord. The umbilical cord, if contains a single artery, is a marker of many congenital anomalies. An outpoking of intestine covered by skin is *umbilical hernia*; while one without skin is *exomphalos*. A foul discharge from cord indicates an *abscess*, and induration around cord denotes dangerous levels of *sepsis*.

Skin. Rashes, hypo/hyperpigmentation, jaundice, bleeding spots, hairy naevi must be searched for. *Milia* are harmless sebaceous distensions over nose. Mongolian spots and erythema toxicum are vascular phenomena.

Genitalia. Check for hypospadias, undescended testis, hydrocele, fused labia etc.

Danger signs in newborn

1. Poor feeding, lethargy, convulsion (subtle convulsions = staring look, smacking, apnea + tachycardia).
2. Respiratory distress (see above).
3. Tachy/bradycardia.
4. Cold/fever (normal temperature 36.5–37°C).
5. Central cyanosis, jaundice upto palm/sole or within the 1st 24 hrs.
6. Diarrhea, persistent vomiting, distension, no meconium within 24 hrs, hepatosplenomegaly.
7. Umbilical pus, pustules > 5 in number.
8. No passage of urine within 48 hrs, oliguria, kidney mass.
9. Bleeding from any site (some per vaginal withdrawal bleeding may occur in girls on 5–7th day; it is due to withdrawal of maternal sex hormones).
10. Poor weight gain.
11. Acidotic breathing.
12. Anemia, thrombocytopenia.
13. Raised ICP (papilledema, bulging fontanelle, disturbed infant).

Early diagnosis of congenital anomalies

1. History—Teratogenic/goitrogenic drugs, radiation, viral infections in 1st trimester, polyhydramnios (indicates an upper GI atresia), oligohydramnios (renal agenesis, obstructive uropathy).
2. Orifice counting
3. Finger and toe count.

Criteria for nursery admission

Immediate. Birth weight < 2 kg, born at 35 weeks or earlier

Late. Jaundice, sepsis, respiratory distress, cyanosis, convulsion, bleeding, hypoglycemia (loss of activity, jitteriness and convulsion), hypothermia.

Feeding of the newborn

The normal newborn loses 10% weight in 1st few days but regains the weight by 10th day. Thereafter, 20–30g gain/day is expected.

Requirements. This table addresses the fluid need of the newborn.

Table 4.13. Nutritional requirement of newborn

Birth weight	Amount	Method of feeding
> 1.8 kg or > 34 weeks	Day 1:60 ml/kg, increment 15 ml/kg/day	Breastfeeding on demand
1.2–1.8 kg or 32–34 weeks	Day 1:75 ml/kg, increment 15 ml/kg/day	Expressed milk gravitated by nasogastric tube at 1.5 hrs interval; each feed should be 10–20 ml
< 1.2 kg or < 32 weeks	Day 1:90 ml/kg, increment 15 ml/kg/day	IV 10% dextrose, add electrolytes after 48 hrs; when vitals are stable, and sucking-swallowing coordination has developed, breastfeeding may be started

Additional needs. Vitamin A, D (prevents osteopenia of prematurity), Fe and Calcium, specially in preterm. **Coconut oil** may be added in milk for calories (fortified human milk). In babies with cirrhosis/biliary atresia, lipid may be given as medium chain triglycerides (olive oil).

Postnatal care (upto 42 days after delivery)

Objectives

1. To restore mother's health
2. To prevent and detect complications of puerperium
3. Ensure successful breastfeeding
4. Educate parents about family planning.

Common problems

Puerperal sepsis. Fever, lower abdominal pain, delayed involution and foul smelling lochia within 3 weeks of delivery.

Deep vein thrombosis/thrombophlebitis. Pale legs and calf tenderness developing in 7–10 days after delivery.

Urinary tract infection. May result due to break of hygiene in hospital settings, inadequate water intake.

Secondary postpartum hemorrhage. Due to infection, retained bits of placenta.

Immediate postnatal care of normal labor

- Examine the patient throughly (specially the vitals, contraction of uterus, vaginal bleeding)
- Ensure a secure and private toilet for the patient
- Examine the perineum, clean the episiotomy wound with povidone iodine, and put a sterile dressing and a large absorbable sterile pad
- Give the baby to her immediately; initiate breastfeeding.

Early ambulation should be encouraged. The patient should stay in the hospital for 2–5 days, until the episiotomy wounds heal and the lochia dries up a little.

Bladder. Lack of privacy, insensitivity of bladder, the position on the bedpan and painful perineal sutures all contribute to the urinary retention after pregancy. Timely catheterization is recommended. At times it is better to drain the bladder continuously for a day or two.

Uterus. The uterus should reduce by half inches each day, and by 2 weeks it should disappear into pelvis.

Perineum. After every act of voiding and defecation, disinfect the perineum and cover it with antiseptic dressing

Care of breast. The nipples will usually be sore and can be infected while breastfeeding. Soothing ointments may be used.

Postnatal visits

At least 2 postnatal visit are required, the first within 10 days and the second after 6 weeks. Examine

1. Pulse, BP, temperature, pallor
2. Episiotomy wound
3. Discharge (lochia *rubra* in first, 2–3 days, then lochia alba follows)
4. Involution—After delivery, the uterus shrinks to a 20 week size. Then it reduces 1 cm/day in 1st 2 days and 2 cm/day in 3rd to 7th day)
5. Breasts—Check for sore nipples/mastitis.

Iron folate. Tablets should be provided if anemia persists. **Pelvic floor exercises** and **good nutrition** is a must for the mother if she wants get back in shape and replenish her stores. **Abstinence** must be practiced for at least 6 weeks (otherwise the vaginal wound may get disrupted). If the mother is breastfeeding, she will be amenorrheic but that should not be relied upon as effective contraception and **family planning** advice must be given.

Breastfeeding

Most Indian mothers breastfeed (about 98% in villages and 96% in towns), but it is the malpractices regarding breastfeeding that are troubling. But this was not the

scenario until 1940s, and until very recently, dialogues regarding breastfeeding was a cliché in Hindi films. After Independence, inadequate and wrong breastfeeding have risen gradually due to,

1. Lack of awareness in growing population
2. Promotion of infant milk substitutes by companies
3. Ignorance of doctors about breastfeeding.

Indian mothers secrete milk from 12th week of gestation, around 450 – 600 ml milk per day in lactation period (peaks around 6 months of lactation), of which the baby needs only 150 ml. This amount of milk is secreted *irrespective of maternal nutritional status*.

Table 4.14. Foremilk and hindmilk

Foremilk	Hindmilk
Secreted at start of feeding	Secreted at end
Thin and watery	Thicker
Rich in protein, lactose, vitamins, minerals and water	Rich in fat
Satisfies thirst	Satisfies hunger

Colostrum

It is the milk secreted in 1st 3 days after delivery. It is a thick yellow juice rich in protective molecules, high in vitamin ADEK, ensures maturation of intestinal mucosa. It is the first immunization (passive) of the baby.

Optimal feeding practices

Exclusive breastfeeding. The baby should be put to breast with ½ an hr (normal delivery) or 4 hrs (Caesarean section). For the next 6 months, the baby should be fed only breast milk for 6 months of life. No food, drugs or even water should be allowed unless medically indicated. Even no pacifiers should be used. If however, the mother feeds small amount of other drinks/food then the baby is *predominantly* but not completely breastfeed.

Position of baby

- Head slightly raised than body
- Head and neck in straight line
- Supported by left hand
- Facing the mother
- Close to the body of the mother.

Attachment (to areola)

- Wide open mouth
- Everted lower lip
- Chin touching the breast
- Areola, if visible, is seen more on the upper side.

Suckling. Must slow but deep with periodic gaps.
Frequency. Feeding on demand, at least 8 times daily.

Adequate complementary feeding (weaning) after 6 months with continuation of breastfeeding upto 2 years

Weaning is the period when infants are most vulnerable to malnutrition. Breastfeeding should not be stopped all of a sudden at 6 months but tapered off gradually. The infant is introduced to *semisolid* (NOT liquid) food like cereals with oil, pulse, smashed vegetables, egg, meat and fish. Start with small quantities (1 teaspoon between breastfeeds) and gradually increase amount and frequency. A *feeding bottle* should not be used in infant feeding. It is a potential source of bacterial infection and reduces sucking power of infants.

Food for weaning. The food should be highly energy dense, easily digestible, semisolid, low in bulk and viscosity, fresh and clean, affordable and available, acceptable, easy to prepare.

Amount of complementary feeding. Between 6–12 months, if breastfeeding is continued, then 1 katori thrice daily of complementary feeding is enough. If breastfeeding is discontinued, however, the infant will need feeding 5 times daily.

Malpractices regarding breastfeeding

1. **Prelacteals**—Feeding the newborn honey, sweet water, and artificial milk within 1st 3 days instead of colostrum.
2. **Artificial feeding (top feeding)**—No breast milk at all but only artificial feeding is given.
3. Delay in initiation of breastfeeding—It is customary in our country to separate mother and child for some period, even in hospitals. In cesarean sections, it gets delayed even upto 72 hrs.
4. Reduction of feeding during illness (illnesses actually *increase* nutritional demand of children).
5. **Bottle feeding**—A prominent source of infection.
6. **Token breastfeeding**—When the child is mostly fed artificial food and only occassionally breast milk.

Special situations

If a mother is working/separated from baby/baby doesn't attach to breast then breast milk is expressed and stored.

1. Storage—Breast milk can be stored in a clean, wide mouthed container for 10 hrs (in room temperature) or 24 hrs (refrigerator) or 3 months (in –20°C freezer).
2. Extraction—Manual or breast pump.
3. Method of feeding—Katori and spoon, tube feeding.

The mother may also take the baby to the working place and place it on a créche to feed intermittently.

Advantages of breastfeeding

For the child

1. Complete nutrition
2. Provides passive immunity

3. Easy digestibility and bioavailability
4. The very act of suckling helps develop teeth and gum
5. Causes less incidence of dental caries
6. Protects against allergy
7. Causes less incidence of ischemic heart disease in the future
8. Essential for proper development of brain.

For mother

1. Convenient and always available
2. Reduces PPH and helps postpartum uterine involution
3. Reduces risk of breast cancer
4. A natural contraception
5. Helps restore normal figure of the mother.

For the father—It is free!

For community—No pollution (c.f. with plastics containers and bottles), no dependance on large companies.

'REAL' contraindications to breastfeeding

REAL—Radiation, ergot, anticancer drugs, lithium; breastfeeding is contraindicated when the mother is exposed to any of these.

Efforts to promote and protect breastfeeding

Code on protection and promotion of breastfeeding (1992)

This was the first initiative to restore the lost art of breastfeeding.

Exclusive breastfeeding was included in RCH, in general and also in management of infections.

Baby-friendly hospital initiatives (UNICEF)

Every baby friendly hospital must

1. have a written breastfeeding policy
2. train staff to implement the policy
3. onform mothers about benefit and management of breastfeeding
4. help to initiate breastfeeding as soon after delivery
5. show mother how to maintain lactation even when separated
6. encourage exclusive breastfeeding
7. keep mother and baby in same room (rooming in)
8. encourage breastfeeding on demand
9. prohibit artificial teats or pacifiers
10. establishment breastfeeding support groups and refer the mother to such groups.

Kangarooing (skin-to-skin)

It helps maintenance of temperature of baby, facilitates and increases duration of breastfeeding. In addition, it helps from the close association between mother and child that goes a long way.

Infant milk substitutes, feeding bottles and infant foods act, 1992

1. No person shall advertise, promote, offer incentive for promotion of sale of IMS.
2. The label should contain no pictures of babies or mothers, only composition, and no words like "approved by medical professionals", "humanized food" or "complete food".
3. No display of posters and placards in public about benefit of infant milk substitute.
4. Health service providers cannot accept gifts incentives from companies for promotion of substitutes/feeding bottles.
5. Penalty: Fine/imprisonment for 3 years.

Family planning

Conducting a delivery present an opportunity to prevent the next one. The woman and her husband should be given a 'cafeteria' choice on contraception, and advised to space pregnancies for at least 3 years.

Early detection of complication

See intranatal care.

Emergency services for those who need it

See 'maternal mortality'.

Prevention and management of RTI (STDs)

See STDs in the chapter on communicable diseases

Child health and welfare

1. Exclusive breastfeeding
2. Immunization
3. Treatment of ARI, diarrhea, anemia.

Family welfare services

1. Provision of contraceptives
2. MTP.

RCH II (2005–2010)

The second phase of RCH program, i.e. RCH – II has been commenced from 1st April 2005. The general plan is to get as close as possible to the goals stated in Millennium Development Goals, the National Population Policy 2000 and the National Health Policy 2002.

Goals

1. Reduce IMR < 30 and neonatal mortality < 20 by 2010
2. Reduce MMR
3. Reducing total fertility rate.

Features

1. A **sector wise approach** which extends the program reach beyond RCH to the entire family welfare sector.
2. Building **state ownership** by involving states and UT's from the outset in development of the program.
3. **Decentralization** through development of District and State level need based plans.
4. **Flexible programming**, i.e. instead of a single plan throughout the nation, states have to develop need based work plans with freedom to decide upon program inputs.
5. Capacity building at the district, state and the central level to ensure improved program implementation.
6. Performance based funding to ensure adherence to program objectives, reward good performance and support weak performers.
7. **Convergence**, both intersectoral as well as intrasectoral to optimize utilization of resources.

Essential obstetric care

1. To promote institutional deliveries and 24 hrs delivery services in PHCs and CHCs; at least 50% PHCs and CHCs should be open for 24 hrs.
2. To provide a skilled attendant to every delivery.
3. Tertiary health workers like ANM have been given power to carry out emergency interventions to save a mother.

Skilled birth attendants

Apart from the trained dai who can conduct deliveries, the MPHW is to be empowered for emergency obstetric care, like—

1. Episiotomy repair
2. Repair of 1st and 2nd degree perineal tear
3. To control PPH by misoprostol/oxytocin
4. To give first dose of $MgSO_4$ in case of eclampsia and then refer to FRU
5. Active management of 3rd stage of labor, partography
6. To remove retained products of conception.

Emergency obstetric care

The secondary level of health care/first referral units (FRUs) are targeted as emergency obstetric service providers. A fully featured FRU should have:

1. **24 hrs delivery services**
2. **Obstetric surgery facilities**
3. **Newborn care**, a neonatal intensive care unit
4. Emergency care of sick children
5. **Anesthesists and obstetricians** all the time
6. Family planning (including laparoscopy) services
7. MTP
8. Treatment of STDs
9. **Blood bank**

10. Essential laboratory services
11. Referral service.

New initiatives in RCH

Training of MBBS doctors in Anesthetic Skills for Emergency Obstetric Care at FRUs.

To alleviate shortage of specialist manpower Government of India launched training of MBBS doctors for gaining Anesthetic Skills in Emergency Obstetric Care at FRUs.

Setting up of Blood Storage Centers at FRUs

Timely treatment for complications associated with pregnancy is sometimes hampered due to nonavailability of blood transfusion services at FRUs to facilitate establishment of blood storage.

Developing a cadre of Community Level Skilled Birth Attendant

A 'Community Level Skilled Birth Attendant' is a person who will be trained in midwifery to provide maternal care at the community level. She will be

1. Selected from the community where she will set up her practice after completion of her training of one year in midwifery
2. Left in the villages to practice the skills provided
3. Serve in the same community for a minimum period of three years
4. Not be given government services but stipend for the training period and hostel facility will be provided at ANM training centers.
   This scheme will be taken up during Phase II of RCH program.

Janani Suraksha Yojna

The scheme is a modification of National Maternity Benefit Scheme. It combines an incentive for institutional delivery with the institutional care.

Features

1. Incentives for delivering in institutions
2. Cash assistance for transport to institution for delivery
3. Cesarean section and obstetric operations subsidized by Government
4. Encouraging puerperal mothers to undergo tubectomy
5. Incentives to trained dai or ASHA for bringing mother to an institution.

Eligibility

This scheme divides states as low performing (LPS) where most deliveries occur at home, and high performing states (LPS), where most deliveries are institutional.

1. In low performing states, any woman aged 19 years of above and below poverty line will get the benefits upto first two live deliveries in an institution; the benefit may be extended to third child if the woman agrees to sterilization.
2. In high performing states, two children are the limit for benefits.

Accredited Social Health Activist (ASHA)

ASHA will act as a link among beneficiary at village level, Anganwadi Worker and ANM. The scheme is under consideration. She will help and guide women to

assess the health facilities for antenatal care, institutional delivery, postnatal care and counseling on nutrition and family planning services.

Vandematram Scheme

The scheme intends to provide free antenatal and postnatal check, counseling on nutrition, breastfeeding, spacing of birth, etc. through public private partnership. This is a voluntary scheme where in any OBG specialist, maternity home, nursing home can volunteer themselves in joining the scheme. Any lady doctor/MBBS doctor providing safe motherhood services can also volunteer to join this scheme. The enrolled 'Vandematram' doctors will display 'Vandematram' logo in their clinic, Iron and Folic Acid Tablets, oral pills, TT injections, etc. will be provided by the respective District Medical Officers to the 'Vandematram' doctors/clinics for free distributions to beneficiaries. The cases needing special care and treatment can be referred to the Government Hospitals and institutes, who have been advised to take due care to the patients coming with Vandematram cards.

Child health strategies

1. Continue existing interventions
2. Strengthening of skilled newborn care at birth
3. Integrated management of childhood illness (IMCI).

Integrated Management of Childhood Illnesses

Most sick children present with more than one single diagnosis. In reponse, WHO and UNICEF have developed the IMCI strategy.

- Improvement of case management skills of health staff
- Improvement of the health system as a whole to better manage childhood illnesses
- Improvement in family and community practicers.

Actions

1. Algorithmic approach to a sick child between 1–5 years, which can be followed by all categories of health workers.
2. Preventive and promotive interventions to be integrated with cure.
3. Use of affordable drugs.
4. Teaching parents how to care at home.
5. To be delivered by AWW, MPHW and ASHA.

Guidelines

Assess. Danger signs, nutrition and immunization status, other health problems.
Classify. Use a triage.

- *Urgent*, for referral
- Specific medical treatment and advice
- Advice and go home.

Identify. Specific disease, need for prereferral treatment (first dose of an emergency drug should be given at clinic, like atropine).

Treat. Train the caretaker on how to administer oral drugs, how to feed and give fluids; follow up on a specific date and identify danger signs in the meantime.

Counsel. To solve any feeding problems, the health of the mother herself.

Follow up. Reassess the child.

The Indian version of IMCI includes the 1st 7 days of life, and thus called IMNCI (N for 'neonatal'). Extracts from of the WHO IMCI guidelines for health workers are provided in the appendix. The entire document is available at http://whqlibdoc.who.int/publications/2008/9789241597289_eng.pdf.

Integrated Child Development Services

ICDS was initiated by the Ministry of Social Welfare, Rural development and Women and Children's welfare on 2nd October 1975, in pursuance of the national policy for children. It is not only 'just a program' but an exemplification of inter-sectoral coordination.

Objectives

1. To improve nutritional and health status of children in the age group 0–6 years
2. To lay the foundations for proper psychological, physical and social development of the child
3. To reduce mortality, morbidity, malnutrition and school drop-outs
4. To achieve an effective coordination or policy and implementation among various departments working for promotion of child development
5. To enhance the capability of the mother and nutritional needs of the child through proper nutrition and health education.

Beneficiaries

Pregnant women	Health check up Tetatnus immunization Supplementary nutrition Nutrition and health education
Nursing mothers	Health check up Supplementary nutrition Nutrition and healthy education
Women of 15–45 years	Nutrition and health education
Children < 3 years	Supplementary nutrition Immunization Health check up Referral services
Children 4–6 years	Supplementary nutrition Immunization Health check up Referral services Nonformal education
Adolescent girls	Nutritional supplements

Services

Supplementary nutrition

It is given to

1. Children < 6 years
2. Pregnant and lactating women.

Normal children, and those 1st or 2nd degree malnutrition	300 kcal + 8–10 g protein at least 300 days a year
Children with 3rd and 4th degree malnutrition	600 kcal + 16–20 g protein at least 300 days a year
Pregnant and lactating women All adolescent girls	500 kcal + 20–25 g protein at least 300 days a year

Nutrition and health education

All women of 15–45 years of age are educated, specially pregnant and lactating ones.

Immunization

The ICDS workers motivate mothers for immunization and bring them, along with their children, to immunization centers.

Health check up

1. Antenatal care: Immunization, IFA prophylaxis, protein supplements, 3 physical examinations, referral services.
2. Postnatal care.
3. Newborn care.
4. Care of children < 6–immunization, general check up every 3–6 months, growth monitoring every month, treatment of diarrheal diseases/ARI/intestinal worms, vitamin A prophylaxis, referral services.

Nonformal preschool education

Children of 3–6 years are imparted nonformal preschool education in an AWC in each village of about 1000 population. The objective is to provide opportunities to develop desirable attitude, values and behavior.

Structure

Ministry of Social Welfare

↓

COMMUNITY DEVELOPMENT BLOCK, one per 100000 population in rural areas and a TRIBAL DEVELOPMENT BLOCK, one per 35000 in tribal areas; the block is managed by the Child Development Project Officer (CDPO)

↓

Four *Mukhyasevikas* – a full-time government for training and supervision of Anganwadi Workers and record keeping, visits

↓

Each Mukhyasevika is mentor to 20–25 Anganwadi workers (AWW), who are allotted a 1000 population and selected from the community; they are honorary workers and not full-time

School health

School health services fill in the gap of RCH, namely, care of children of 5–14 years. These children are generally the healthiest among all age groups. Because this age group spends a large part of the day in the school, it is best to integrate their health services with education.

History

1909—1st school health check up, Baroda
1960—Renuka Roy committee on school health
1983–84—Review of school health problems by National Institute of Health and Family Welfare, and recommendation of:

1. Health check up in school.
2. Midday meals.
3. Health education to children.
4. Promote physical activity for children.
5. Improve school environment.

School health problems

Malnutrition, dental diseases (caries), eye (ametropia, vitamin A deficiency), ENT disorders (ASOM, rhinitis, tonsillitis), skin (scabies, ringworm), accidents and injuries.

Objectives

1. Awaken health consciousness in children.
2. To recognize child as a agent of change in the family (i.e. the child incalculates health education from school and spreads it into his home).
3. Prevention and treatment of disease in children.
4. Provision of healthful school environment.

Activities

Health appraisal

1. Periodic examination of students by teachers
2. Daily morning inspection of students by teachers, as a screening for illness
3. Medical check up by MO at school admission and every 4 years (physical examination, eye, ENT, speech, blood, urine).

Remedial measures and follow up

Children with any problems are referred to the nearest PHC with a student card for prompt treatment, or may be accompanied by the teacher. Special clinics may also be set up for every 5000 school children.

Prevention of communicable disease

1. Pursue the parents and the child for immunization
2. Education on personal hygiene
3. Improve school environment and sanitary facilities.

Emergency and first aid by teachers

Common emergencies like pain abdomen, fainting, injury and convulsions are to treated initially by teachers, for which they are to be given special training. A first aid post should be set up in every school.

Special care

1. Mental counseling—Regarding studies, aptitude, healthful behavior, avoiding drugs, sex education; the school age is no period for distinction between 'smart' and 'dull' students. Every student must be given equal opportunity.
2. Dental care—Emphasis on brushing and avoiding too much sugar in foods.
3. Eye examination—Special charts have been devised so teachers can assess visual acuity.
4. Education and support to handicapped children.

Nutrition

The midday meal program provides nutrition for school age children, thus integrating nutrition with education. In view of recent trend of obesity in school children, education on good feeding practices (see "Dietary Goals" in the chapter on Nutrition) is also necessary from a very early age.

School environment

1. The school building must be away from noise, at a higher level than surroundings (so that it is never clogged by water), away from cinema house, rail lines, bars, etc.
2. Provision of fire safety.
3. Playground (a must).
4. 10″ thick walls to cut off noise.
5. floor area 10 ft^2/student, no room should accommodate > 40 students
6. Desk with backrest.
7. Adequate natural lighting.
8. Potable water supply.
9. Latrines (1 per 100 students) and urinals (1 per 60 students)–m, f.
10. window + door area must equal at least 25% of floor area.

Postpartum program

All India Hospital Postpartum Program is an urban, hospital based program, with the aim to set up RCH in the urban areas and institute family planning immediately after delivery. It constitutes of postnatal care, immunization, iron and vitamin A supplementation to children, family welfare and in some medical colleges, PAP smear facilities for at risk women.

THE ELDERLY

About 7.2% population of India constitutes of people over 60, or geriatric population. The elderly:

1. consume most of health services
2. are a huge economic burden (pension, medical care, social services).

Problems of the elderly

Modern society is built by the young for the young, who do not give much consideration to the old or incapable. They are merely tolerated as so much liability or nuisance. The old, who were once young, helped to make the present society, and now they pay the price of their former neglect of the old. The cycle goes on—

- Poverty, isolation and neglect
- Hypertension, ischemic heart disease, heart failure
- Pneumonia, tuberculosis, asthma, COPD
- Peptic ulcers
- Diabetes, thyroid diseases
- Osteoporosis, osteomalacia, osteoarthritis, fractures
- Nodular hyperplasia of prostate, vaginal mucosal atrophy
- Dementia, Alzheimer disease
- Cancers
- Accidents.

Ideal geriatric health services

Goal

Improve *quality* of life rather than quantity (extending life is a punishment for most old people; let them enjoy whatever life they have left).

Objectives

Promote and maintain health.

Enable disability free independent life or minimize disabilities.

Primary prevention

Healthy lifestyle

1. Reduce diet—Calorie requirements reduce drastically with old age (m- 1800 kcal/d, f – 1400 kcal/d), eat more fibers, green leafy vegetables, fruits.
2. Exercise.
3. Avoid smoking/alcohol.

Immunization and chemoprophylaxis

Streptococcus pneumoniae and influenza have long been the old man's friend; there is scope of vaccination for these agents.

Hormone replacement therapy

For primary and secondary osteoporosis.

Avoid accidents

Every household/institution where old people reside should have good lighting, railed passages, a rough floor (so that they don't slip), window with grid and visual/auditory aids.

Secondary prevention

Screening for hypertension, diabetes, colorectal cancer.

Tertiary prevention

Reablement for independent life
Resettlement to own or other environment.

INFORMATION, EDUCATION AND COMMUNICATION

Common terms

Recall the fundamentals

1. **Information** are facts of some worth (look up the chapter on statistics and health information).
2. **Education** is the process of behavioral change from experience (one of several definitions of education; you need not agree to this).
3. **Communication**, at least from the perspective of community medicine, is an attempt to change behavior by information.
4. *Attitude* is the pattern of response to stimuli (recall the chapter of sociology).
5. *Behavior* are purposive acts arising out of decisions.

Communication

The principles of communication, at its basics, remain always the same, whether over the phone, over the internet, or gave to face.

Objectives

People communicate with each other with one or more of the following aims

1. To make somebody aware of a fact/event.
2. To develop proper perception in seeking solution.
3. To motivate, either positive or negative; the steps to motivation are awareness → interest → evaluation → decision.
4. To guide.

Communication process

Figure 4.17. Communication process

Sender

The sender must exactly know what he is trying at and who are his audience. He must also be aware of the channels of transmission he has.

Message

The message constitutes of three elements.

1. Form—The way it is presented (simple speech, video lecture, radio broadcast, interactive discussion, awareness posters ...).
2. Content—What it tries to say.
3. Treatment—How it is delivered to the audience (preparation, process, delivery).

Channels of transmission

1. **Direct (interpersonal)**—The most effective, because it provides the audience a scope to ask questions.
2. **Mass media**—Television and newspaper have a wider coverage, but offer no feedback facility (they are unidirectional); *radio* and *internet* are two mass media which can be made interactive.
3. Folk media—Presenting information through traditional art forms and festivals (i.e. a puppet show).

Receiver

An audience may be controlled (homogenous, i.e. everybody has certain common characteristic and background knowledge, as in a seminar of neurologists) or uncontrolled (heterogenous). The more homogenous an audience, the more effective communication becomes.

After listening to the message, the receiver will usually go through the stages of:

1. **Awareness** of the stated facts
2. **Interest** in the matter
3. **Evaluation** of whether the message was valid
4. **Decision** to adopt the recommendations of the message.

Feedback

Feedback may be elicited by interpersonal methods, opinion polls and KAP ('knowledge-attitude-practice') studies.

Barriers

1. **Physical**—Visual or auditory impaired audience; difficulty of expression in the sender
2. **Mental**—Emotional disturbances, lack of intelligence, foreign language
3. **Environmental**—Distance from speaker (as in a roadshow), noise, congestion
4. **Cultural**—Prevailing customs and belief (which often make people resistant to any kind of enlightenment), socioeconomic status (poor people are never in a mood to listen until they are fed), politics (political parties may deliberately misinform the people for their own purpose).

Principles of communication

1. Close perception between sender and receiver, they should not be too far apart in their viewpoints.
2. Message should be simple, accurate, adequate, specific, relevant, timely, appealing, practical, conforming to objectives.
3. Multimedia always works better than a simple lecture (unless that lecturer is somebody like Bertrand Russell).
4. Feedback is necessary.
5. Direct communication (one to one) is always more effective than indirect methods.
6. Sender skills—The sender must have a clear speech, must be a good listener to questions and comments, and should be able to discuss logically.

Purpose of health communication

1. To **inform** (most of the people don't know that they don't know) – about causation, prevention of disease and what the people can do.
2. To **educate** of healthful lifestyle and environment.
3. **Motivation**, which comprises the stages of awareness, interest, evaluation and decision.
4. **Persuasion**, as to influence people with your words.
5. **Counseling**, which is to help people understand and face their problems better.
6. Raising the **morale** of people.
7. Health organization, i.e. to achieve **community involvement** and intersectoral coordination.

Health education

Health education is

- 'A process that informs, motivates and helps people to adopt and maintain healthy practices and lifestyles, advocates environmental changes as needed to facilitate this goal and conducts professional training and research to the same end' [National conference on preventive medicine].
- 'A process aimed at encouraging people to want to be healthy, to know how to stay healthy, to do what they can individually and collectively to stay healthy, and to seek help when needed' [WHO (Alma ata, 1978)].

Objectives

1. Encourage people to adopt and sustain health promotion.
2. Promote use of health services.
3. Arouse interest, provide knowledge, improve skills of both the public and the health workers.
4. Enabling people to make rational decisions as to solve their own problems (community *involvement*).

Principles of health education

1. **Credibility**—The information must be updated and consistent.

2. **Interesting**—It often helps to picture yourself as one of your audience before you plan the session, *does it still seem interesting to you*? If yes, proceed.
3. Two way (provision for feedback).
4. **Motivation** (for or against something), and sometimes even **persuasion** if you know that your recommendation will do the audience good.
5. Comprehensible ('you know what I mean').
6. **Reinforcement**—As George Orwell famously remarked, even a *lie* becomes true if repeated enough.
7. Learning by doing *show* them rather than tell them.
8. Progress from *known to unknown* with graded doses of information (begin with what they are familiar with, and then scale up).
9. Good human relationship between sender and audience, and between the audience.
10. Use proper media of information—A warning for a thunderstrom should be broadcasted by radio or television so that it reaches a large audience, and because no feedback is necessary in such a case; but it is not a good method for education on family planning, because people will find it boring and turn off the television (or switch to a song and dance extravaganza).
11. **Evaluation**: KAP study.
12. Use community leaders (Panchayat pradhans, religious leaders, political leaders) as your *amplifier* or still better, *interpreters*.

Approaches to IEC

1. **Regulatory approach**—A primitive method of controlling people by laws and not by education (but unfortunately, laws never work until people begin to understand their gravity).
2. **Service approach**—As it happens, it turned health worker into door to door salesmen selling health services, but people felt no need of using these services - they were simply not aware of their needs.
3. **Education approach**—To make people understand and make a decision by themselves.
4. **Primary health care approach**—To integrate education along with health care.

Practice of health education

One of the jobs of the doctor is to teach (the reason that PhDs are often called 'doctors's). To some, teaching comes naturally; for other, here are a few tips. There are three levels of health education:

1. **Individual**—A part of the clinical appointment should be dedicated to education to the patient and his/her family.
2. **Group**—In teaching many people at once, there are two approaches. In the **formal** approach, there is dominance of the speaker and most others only listen (lectures, dialogues, symposium, colloquium). In the group approach, everybody does equal parts talking (buzz group, **group** discussion, workshop, role play, demonstrations). The later methods are better than the former because they involve active participation by all and direct feedback.

3. **Everybody**—For rapid and wide dissemination for some important piece of information, nothing suits better than newspaper, television, radio and the internet.

Check your communication skills

How frequently do you make eye contact? How good is your body language? Is your speech slow, loud, clear enough for everybody to understand? Do you repeat important lines? Do you answer the questions of people?

Educational aids

Not all occassions need a slide show; you could begin with a chalk and blackboard and build realms of knowledge. It is also very effective to inject local culture (such as a drama, dance or puppet show) in your session. Remember, the audience rules.

Planning for a session of health education

Suppose, as a medical officer, you are assigned to speak and educate about family planning in a rural community. How do you proceed?

Analyse

1. Major health problems (in this case, you need the demographic data of that area, the TFR, identify the barriers to family planning).
2. Existing program performance (what is the CPR in this area? why don't people accept contraception? Is there a cultural, social or religious barrier to contraception?).
3. Know your audience—Their demography, socioeconomic status, customs, segments (i.e. religions, economic classes).
4. Know local organizations—Their competency, commitment and coverage.
5. Check availability of media/audiovisual aids; in a rural area with no electricity, you just might have to depend on chalk and board.

Design

1. Assess needs of the community.
2. Set SMART objectives (specific, measurable, appropriate, realistic, time bound) of education; in this case you expect a change in attitude towards contraception (which is a *qualitative* target and could only be assayed with a KAP study) and a rise in CPR (which is a *quantitative target*).
3. Design a curriculum of your session; decide whether this education needs to be told on a personal basis (i.e. door to door visit), group basis or mass basis; in this case, a group talk would be most appropriate because door to door vendoring with condoms will not gain much success in rural India.
4. Select a suitable channel of transmission; for education on contraception, a group session with demonstration of contraceptives is most ideal.
5. Make a budget and a work schedule of who will do when and what; monitor performance of the workers.

Develop message and materials

1. The message should command attention, cater to hearts, clarify complex data, communicate its benefits, create trusts by retelling local success stories, conveys assistance and call for action (in short, the seven 'C's).
2. Pretest and retest the message with local groups and individuals; check whether the contents of the session are in anyway violating a religious/cultural belief of the community. These are very sensitive issues and to be dealt carefully; disburse information in very small, metered doses so that the community never gets a sudden shock.

Implementation

Distribute jobs among health workers; inform local NGOs and community leaders to take part in the session; advertise your session for maximum community involvement. The target is to reach the widest possible number of audience feasible through any possible media.

Evaluate the impact

Check the cumulative effect of the session by a KAP study in the community.

Counseling

It is an empathic ('put yourself in my shoes') process that helps people understand and deal better their own problems and communicate to their emotionally involved ones. It is specially important in

1. Genetics
2. Family planning
3. STD and HIV control.

Social marketing

Application of commercial marketing to advance a social cause, issue, behavior, product, service or even, idea. For example, the NFWP socially markets condoms and OCPs in chemist shops.

Method

1. *Analyse* health situation and set measurable objectives (which products need social marketing?).
2. Market research (what are the competing brands? what are their prices? how distinct will be your brand from them? who are the potential customers?); in case of contraceptives, OCPs and condoms are supplied free of cost in each health center; but still, some urban customers might like the convenience of just going to the drug store nearby, rather than visit a hospital or health center just for a condom.
3. Plan and develop product that correspond to the need.
4. Test and refine the product.
5. Advertise the product.
6. Market the product through a distributor; it is important to set a price that's not too high or too low (people assume price to be an indicator of quality).
7. Feedback.

5 Health and Environment

KEY FEATURES

- SANITATION
- WATER
  - How should ideal water be?
  - How much water do we need?
  - From where do we get this water?
  - Water pollution
  - What can unsafe water cause?
  - How can water be purified?
  - Principles of chlorination
  - Household water purification
  - Disinfection
  - Case study: How to clean a well?
  - Case study: Swimming pool sanitation
  - Water surveillance: How to investigate a complaint about water?
  - Water quality standards (WHO)
- AIR
  - Comfort
  - Air pollution
  - Prevention of air pollution
- Housing
  - The 'residential environment'
  - Overcrowding
  - Indicators of good housing
- WASTE DISPOSAL
  - Solid wastes
  - Liquid wastes
  - Hospital waste disposal
- DISPOSAL OF HUMAN EXCRETA
  - Excreta disposal in unsewered areas (on site' latrines)
  - Excreta disposal in sewered areas
  - Sewage treatment
- NOISE
  - Measurements of sound
  - Effects of noise
  - Control
- RADIATION
  - Unit of radiation
  - Types of radiation
  - Sources
  - Effects
- STERILIZATION AND DISINFECTION
  - Types of disinfection
- OCCUPATIONAL ENVIRONMENT
  - Aims of occupational health services
  - Psychosocial environment
  - Physical environment
  - Chemical environment
  - Biological environment
  - Sickness absenteeism
  - Prevention of occupational diseases
- DISASTERS
  - Classification
  - Disaster cycle
- (MEDICAL) ENTOMOLOGY
  - Vector dynamics
  - Control of (arthropod) vectors
  - Mosquitoes
  - Flies
  - Ticks
  - Mites
  - Lice
  - Fleas
  - Cyclops
  - Antivector chemical treatment-insecticides
  - List of vectors

Recall the fundamentals

1. Any set of objects can be defined to be as a *system*. Examples of systems include you, your house, a slide, a scalpel, the CM book, etc.
2. Everything that surrounds a system is called its *environment*.
3. So system + environment = The Universe.

SANITATION

The idea of environmental sanitation (*shoucha*–as it translates in Sanskrit) gained momentum with the demonstration by John Snow that spread of cholera was 'feco oral' (wonder what that term caused in public mind when used for the first time). Sanitation was, initially, centered around toilets and latrines, and how to keep flies away from them.

Gradually, the idea of sanitation has expanded to

> Environmental sanitation is the control of all those factors in man's physical environment which exercise or may exercise a deleterious effect on his physical development, health and survival
>
> —*WHO*

WATER

How should ideal water be?

Safe and wholesome water/potable water

1. should be free from pathogens and toxins
2. have a pleasant taste, color and smell (or else people won't drink it)
3. useful for domestic purposes (not hard water).
4. does not represent any significant risk to health over a lifetime of consumption.[187]

This 'ideal' kind of water is getting more scarce everyday. About a billion people around the world routinely drink unhealthy water. Most countries accepted the goal of halving by 2015 the number of people worldwide who do not have access to safe water and sanitation.[188] Even if this difficult goal is met, it will still leave more than an estimated half a billion people without access to safe drinking water and over a billion without access to adequate sanitation.

Poor water quality and bad sanitation are deadly; some five million deaths a year are caused by polluted drinking water. Almost "one tenth of the global disease burden could be prevented by improving water supply, sanitation, hygiene and management of water resources".[189]

Water, however, is not a finite resource, but rather recirculated in volumes many times higher than human consumption. We just need to know the right tricks to extract safe and wholesome water from this cycle.

How much water do we need?

An individual needs 2l/day to maintain homeostasis. An average person, however, for all domestic purposes, needs 150–200l/day. The target, in our country, is to deliver at least 40 liters of wholesome water to each individual in rural areas.

From where do we get this water? (Fig. 5.1)

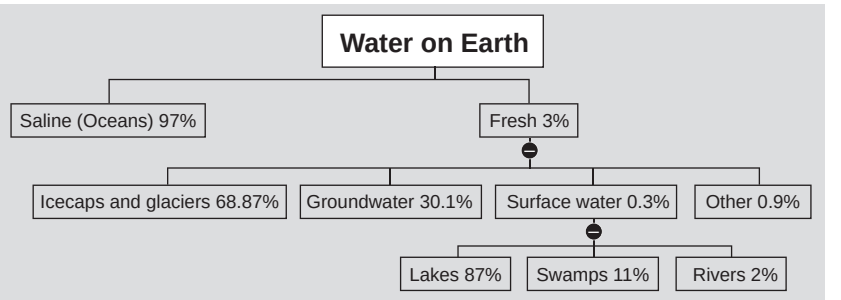


Figure 5.1. Distribution of water in this planet

Not all of these sources are usable. We have to extract water from such a point in the water cycle so that it gives a **safe yield** (adequate for 95% time of the year), i.e. you can't use a river that dries up in summer for a reliable source. So what do we do?

Why, Rain!

It is the purest of all natural water, and only contains traces of solids. Places like Gibraltar use it as the sole source of water. But rainwater tends to form acids with aerobic pollutants like SO_2.

Rivers and lakes

Surface water (like rivers, lake, ponds) is fed by rain (except the rivers that originate in Himalayan glaciers). Surface water is much susceptible to contamination and as such can't be used without treatment.

Wells

There are two kinds of wells (Fig. 5.2).

1. **Shallow wells** are only as deep as the top soil and does not reach the layer of rocks (impervious stratum) of the ground. They are more prone to be polluted by activity of surface.
2. **Deep wells** run below the impervious stratum, and are usually more protected from contamination.

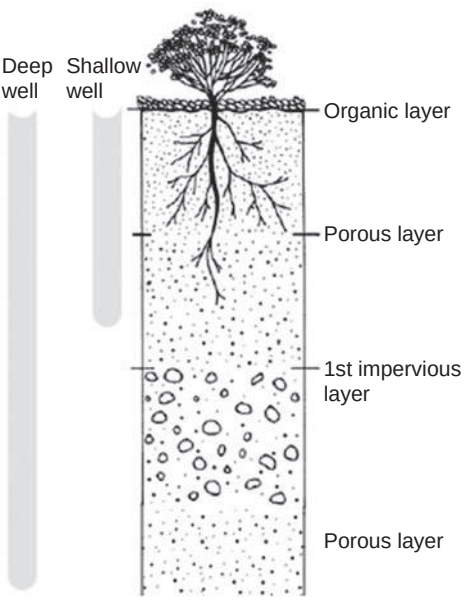


Figure 5.2. Shallow and deep wells

The wells and artesian wells are safe if built correctly. A **sanitary well** is one which is properly located, well constructed and protected against contamination with a view to yield a supply of safe water.

1. Location: => 15m/50 ft from any source of contamination (pond/ river/reservoir). It should not be >100m from users (Fig. 5.3).
2. It should be lined by bricks/stone, parapet covering and drainage of spilled water is needed.
3. There should be a sloping platform around and the well should at least have a handle.
4. The consumers should be responsible for maintaining the sanitation of the well.

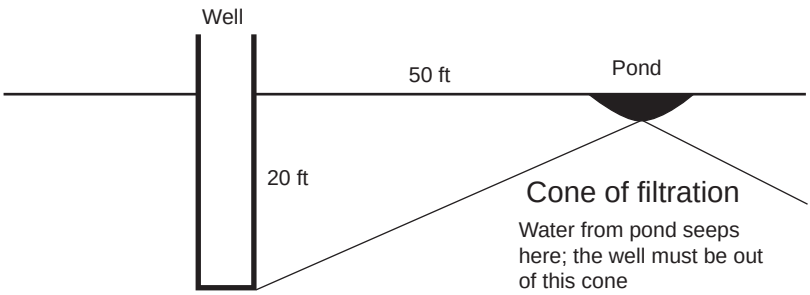


Figure 5.3. Sanitary well

Water pollution

Types of water pollution

Point source pollution. Point source pollution refers to contaminants that enter a waterway through a single discrete path, such as a pipe or ditch, or leakage from a ship.

Nonpoint source pollution. Nonpoint source pollution is accumulative effect of small amounts of contaminants gathered from a large area (i.e. leaching out of nitrogen compounds from agricultural land).

Causes of water pollution

Sewage. The source of most bacterial and viral pathogens in water; release of sewage in water occurs in any underdeveloped sewage plants or leaky sewage collection systems (pipes, pumps, valves). Some cities also have *combined* sewers, which may discharge untreated sewage during rain storms.

Organic substances. Detergents, disinfection by products (such as chloroform, which forms during chlorination of water), food processing waste (which can include oxygen-demanding substances like fats and grease, thus eating up oxygen content of water), insecticides and herbicides, petroleum (fuels and lubricants from urban run-off,[190] tree and bush debris (during storms), industrial solvents and cosmetic products (facewash, creams, shampoo, etc).

Inorganic substances. Acids (specially sulfur dioxide from power plants), ammonia, fertilizers containing nitrates and phosphates, heavy metals (from urban run-off and car washes), cement and gravel in run-off from construction sites, discarded trash, plastic and sullage.

Thermal pollution. Industries that use water as cooling agent release hot water into rivers and seas, endangering the marine life.

Radioactive material. With increasing dependance on nuclear power in developed countries, radiation pollution in water has also increased.

Indicators of water pollution

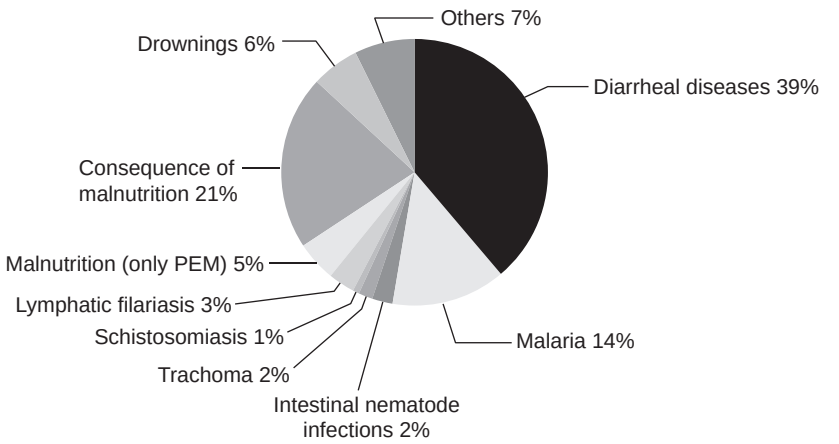
1. Total suspended solids
2. Biochemical oxygen demand at 20°C
3. Absence of dissolved oxygen
4. Chlorides
5. Nitrites and nitrates.

What can unsafe water cause? (Fig. 5.4)

Globally, 9.1% of disease burden (in DALYs) and 6.3% of all deaths are *directly* preventable by improved water safety.[191] Economically, investment to improve drinking water, sanitation, hygiene and water resource management systems makes strong economic sense. Every dollar invested leads upto eight dollars in benefits.[192]

Infectious diseases

Water residing agents. Poliovirus, Hepatitis A and E virus, Enterovirus, Rotavirus, *E. coli, S. typhi, V. cholerae, Giardia, Entamoeba, Ascaris, Chlamydia* (that which causes trachoma) and hookworms. The burden of water borne diarrhea equals **1.4 million preventable child deaths/year** and over long-term, diarrhea/intestinal



PEM; protein-energy malnutrition

Figure 5.4. Diseases contributing to water sanitation and hygiene related burden[194]

nematodes stunts the growth of the child and is one of the causative factors of protein energy malnutrition.[193]

Hosts residing in water. Cyclops (hosts for guineaworm), mosquitoe, snails (hosts for Schistosoma).

Others

Fluorosis (caused by excess fluorines), cyanosis (caused by nitrates), arsenic and lead poisoning, build up of insecticides inside body; regular intake of extremely soft water may cause cardiovascular diseases.

How can water be purified?

Nature does its part

Rivers dilute the polluted water, the evaporation condensation cycle robs water of its pollutants, aeration and oxidation by sunlight kills many bacteria, storage and sedimentation in ponds removes the particles, UV ray from sunlight is bactericidal.

Artificial

The fundamental methods applicable to all water purification systems are distillation and condensation, sedimentation (gravity dependent separation of insoluble particles), coagulation (separation of soluble particles by turning them insoluble by a chemical agent), aeration, adsorption (into the surface of a porous material), filtration (through a sieve) and disinfection (killing pathogens).

Table 5.1. General principles of water purification (Fig. 5.5)

To remove	Do
Turbidity	Storage/sedimentation/aeration/rapid sand filtration
Color	Coagulation/aeration/rapid sand filtration
Odor and taste	Aeration/adsorption (activated charcoal/algicides)
Hardness	Boiling/lime/lime and soda ash/zeolite
Iron	Lime/aeration/rapid sand filtration
Lead	Lime/filtration
Bacteria	Sedimentation/slow sand filtration/boiling/chlorine
Algae	Activated charcoal/$CuSO_4$

Large scale purification

The aim is to provide safe and wholesome water – thus ground water (specially) from deep wells, need no treatment. But surface water needs some cleansing.

1. **Storage**: Storage sediments most particles, allows dissolved oxygen is used by aerobic bacteria to destroy most organic matter, and also the bacterial count itself drops by 90% in one week. But long-term storage may cause algal growth.

2. **Filtration**: This means passing water through small slits, literally. Two kinds of filters are in use.

Table 5.2. Slow and rapid sand filters (Fig. 5.5)

Slow sand filter	Rapid sand filter
Supernatant water (1–1.5 m) ↓ VITAL LAYER: Layer of algae (zoogleal) which forms over the sand in several days. It destroys bacteria and removes organic matter ↓ SAND BED (1m) – Effective diameter is 0.2–0.3 mm so that water passes at a rate of 0.1–0.4 m^3/hr/m^2 of sand bed ↓ Gravel and stones to support the sand ↓ UNDER DRAINAGE SSF requires a large surface area. It is kept open (for sunlight to enter). The overhead pressure (level of water) maintains rate of filtration, so it is always necessary to check supernatant water level with a *venturimeter*. It is of less cost but also of low filtration rate. Removes 99% bacteria. Pretreatment of water by sedimentation is required before filtering in a slow sand filter	The raw water has to be first treated with alum ↓ Then the alum is violently mixed in a chamber. The water is now gently stirred in a flocculation chamber to precipitate the $Al(OH)_3$ ↓ It is now stored for sedimentation and then filtered Sand of 0.4–0.7 mm diameter is used, depth being 1 m. The other components are same as SSS. Alum does the work of the vital layer ↓ The RSF requires a small area, and gives ↑ rate of filtration (40–50 times than SSF), and gives increased physical quantity of water. But it is of higher cost, and of removes 98–99% of bacteria

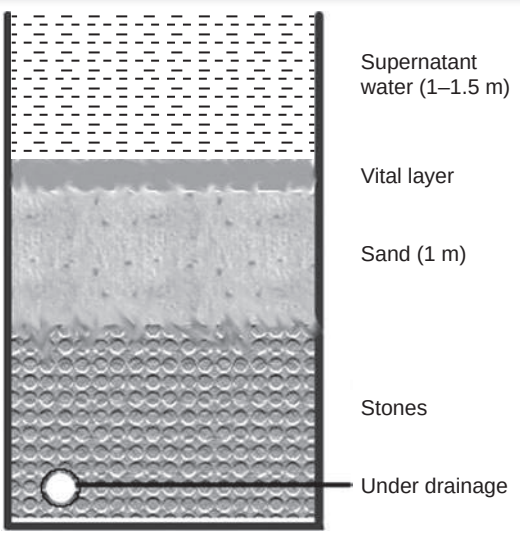


Figure 5.5. Elements common to sand filters

3. **Disinfection**: An *ideal disinfectant* for water is that which is
   - Capable of destroying the pathogens in a short contact
   - Removes taste/odor/color producing agents
   - Is easily detectable
   - Leaves some residue
   - Nontoxic.

In water works, disinfection is synonymous with *chlorination* (*Ozone* can be used as it causes no odor and smell, but to its disadvantage, has no residual amount). Chlorine removes bacteria (but not spores) and many viruses (except poliovirus, hepatitis virus), parasites (except their ova); oxidizes iron, Mn and H_2S; destroys some taste and odor producing compounds, destroys algae and slime.

Principles of chlorination

- Water should be free of turbidity
- **Chlorine demand** = Amount of chlorine added – residual chlorine after specific contact period (standard is one hr) at a given temperature and pH. It is needed to destroy all bacteria and organic matter. When the chlorine demand is met, free chlorine begins to appear, and this is known as *break point*.
- **Residual chlorine** = Chlorine left after all bacteria and organic matter is destroyed. There should be 0.5 mg/l (0.7 mg/l during disasters) residual chlorine.
- Either chlorine gas, chloramines (NH_2Cl) or prochloron (NaOCl, HOCl) is used to chlorinate water.
- **Orthotolidine** test detects free and combined chlorine in sequential colors; free chlorine shows up as yellow in 10s. *Orthotolidine arsenite* test checks free and combined chlorine separately
- Wait ½ an hr before drinking chlorinated water.

Household water purification

Purification of water in household can reduce bacterial population by 35%. The following five point water purification system[195] can be used in most households.

Protect your water source

- Keep a protected and covered well; use a clean rope and bucket
- Build a platform under the pump or tapstand
- Protect your spring by building a catchment box
- Collect and store rainwater in covered tanks
- Keep animals away from water sources by using fences
- Maintain a separate area for animals to drink protect the source of water by planting trees along creeks and rivers
- Locate latrines away from water sources and points of use
- Build a soak pit for wastewater, so that wastewater and drinking water can never mix.

Sedimentation

Sedimentation alone removes much of the bacteria, but not the turbidity or color of water. In the **three pot method**, water is consecutively passed through sedimentation in three different clean and *sealed* containers for 24 hrs each before drinking.

Sedimentation is accelarated by **coagulation**. Three common coagulants used are aluminium sulfate, polyaluminium chloride (also known as PAC or liquid alum), and ferric sulfate. Natural adsorbents/ coagulating agents like cactus leaves and moringa seeds may also be used.

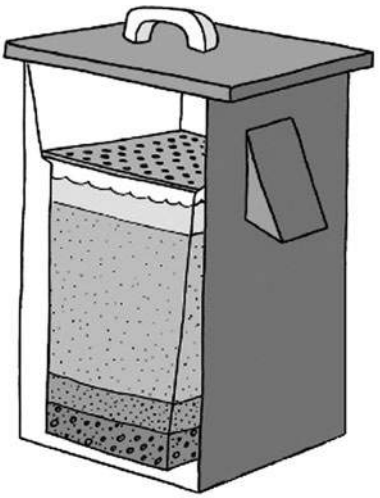


Figure 5.6. A biosand filter[196]

Filtration

Straining. A clean, cloth fabric can be used to strain particles out of water. Typically in South Asia, a sari is folded 7–8 times and used as a filter. Water is poured through the folded sari cloth and collected in a bucket underneath. Sari cloth filters are known to reduce the risk of cholera by filtering out particles and plankton which harbor the cholera bacteria.

Biosand filter. A biosand filter is a small slow sand filter, which can be assembled locally, removes 90–99% of pathogens and filters 60–80l of water per day (Fig. 5.6).

Arsenic filters. The Kanchan™ arsenic filter was developed at the Massachusetts Institute of Technology (MIT) in collaboration with the Environment and Public Health Organization of Nepal (ENPHO). The filter can remove both pathogens and arsenic contamination. The design is similar to the biosand filter, but 5 kg (11 lb) of nongalvanized iron nails and a layer of brick chips are added.

Ceramic silver impregnated filters. These filters are locally baked clay pots, where the inside is coated with colloidal silver fluid (which is bacteriostatic).

Candle filter. A candle filter consists of two containers and one or more ceramic filter elements, shaped like a thick candle, screwed into the base of the upper container. Water is poured into the upper container and then allowed to filter through the ceramic filter element into the lower collection vessel. Candle filters can have very low flow rates, so it is common to find filters with two or more candle filter elements. The best known manu-

Figure 5.7. A candle filter[197]

facturer of candle filters is the Swiss company called Katadyn® who produces the Katadyn® Drip Filter (Fig. 5.7).

Disinfection

Chemical disinfection

Chlorination is the most widely used method for disinfecting drinking water. Disinfecting water with chlorine will kill bacteria and viruses, but it does not deactivate parasites like giardia, cryptosporidium and worm eggs. Chlorine must be added in sufficient quantities to destroy all pathogens, but not so much that taste is adversely affected. Chlorine is available as the following:

- Bleaching powder (Ca(OCl)Cl) provides 33% available chlorine, but very unstable if left open (disintegrates and clorine evaporates quickly); has to be stabilized with excess of lime.
- Chlorine tablets (sodium dichloro isocyanurate) provides 50–60% available chlorine; very long shelf life (5 years).
- High test hypochlorite/prochloron—Stabler than bleaching powder, 60–70% available chlorine.

Solar water disinfection

This method is ideal to treat small quantities of water. Water is filled into *transparent* plastic bottles and exposed to full sunlight for a minimum of six hrs. During the exposure, the sun's UVA radiation and increased water temperature destroys the pathogens.

Boiling

Boiling water at 100°C will kill most pathogens and many are killed at 70°C. The recommended boiling time is one minute at sea level, adding one minute for every additional 1000 m in altitude. Boiling kills bacteria, spores, cyst, ova, and also removes temporary hardness (that due to bicarbonates). It does not give residual protection, and needs a lot of fuel to sterilize even a small amount of water.

Case study: How to clean a well?

Often after a flood, the wells are full to the brim with dirty water. Here is a rescue plan.

1. Find the volume of well (usually $\pi r^2 h$, where r is radius and h, the depth)
2. Find the volume of bleaching powder needed by **Horrock's apparatus**. Make a 200 ml solution of 2g bleaching powder. Now, fill 6 cups with water to be tested, each of 200 ml. Add one drop of bleaching solution to first cup, two drops to second cup and so on. Wait half an hr for chlorine to act. Now, add 3 drops of starch iodide indicator to each cup. Note the first cup where free chlorine appears (a distinct blue color develops). Suppose distinct color appears at 4rd cup, so bleaching powder required = $4 \times 2 = 8$ g per 455 l of water. (the number 455 is a constant, nothing to do with our calculations).
3. **Prepare chlorine solution**: Dissolve bleaching powder in a bucket full of water, and pick the supernatant in another bucket. Do not dip the residual lime in water (it will only increase hardness).

4. Dip the bucket in water several times.
5. Allow one hr contact period.
6. Test for residual chlorine (0.5 mg/l) by orthotolidine arsenite test.

During epidemics, to ensure a constant supply of chlorine, the **double pot** may be used. A smaller pot with a hole is put inside another large one. A hole is made in the larger pot which is in the opposite side of the hole in the smaller pot. A mixture of coarse sand and bleaching powder is loaded in the inner (smaller) pot and then submerged in water. The sand causes slow release of chlorine (much like a sustained release drug capsule).

Case study: Swimming pool sanitation[198]

Diseases like athletes foot (fungal), planter warts (virus), ENT infections (bacterial), UTI and intestinal infections may spread through insanitary swimming practices and *accidents* are always waiting to happen wherever there is water.

1. Regular circulation of water by a pump (to avoid stagnation).
2. Filtration of water while being pumped (sand filter/diatomaceous earth filter).
3. Use of **skimmers** (holes in the walls of the pool with a valve to prevent backflow of the debris into the pool) to collect debris/floating leaves from the pool.
4. An **overflow gutter** to collect spilt water.
5. A saline chlorination unit (for disinfection), electronic oxidation unit (to generate free radicles that kill bacteria) and heating units (to maintain a comfortable water temperature).
6. Recommended area 2.2 m^2/swimmer.
7. Surveillance: Do not allow person with communicable disease or very young children (without sphincter control), bath with soap water before dipping in the pool; stop spilling, spouting and spitting.
8. Regular monitoring of bacteriological quality.

Water surveillance: How to investigate a complaint about water?

In a town it is desirable to have piped water supply in adequate amount. This kind of water supply system has 3 components: Source, water works and distribution networks.

Sanitary survey

1. On the spot inspection an evaluation for detection and correction of faults
2. Identification of suspected source of water by fluorescent dye/chromobacteria.

Sampling

Samples are collected from source of water, reservoirs of water works, standpost on street and consumer tap by trained professionals.

1. For physical/chemical examination, collect in a 2 l Winchester Quart bottle.
2. For microbiological examination, collect in a 200 ml sterilized bottle.

Methods

When collecting from a tap, open it fully, let the initial water flow (for 2 minutes), and heat the tap > cool and collect. When collecting for a river, collect from not too near the bank but not too far from the point where people draw. Collect against the tide maintaining sterilization.

Many contamination events are sharply restricted in time, thus one time "grab" samples are often inadequate for analysis. Scientists gathering this type of data often employ autosampler devices that pump in water at fixed time intervals.

Transport and storage

If bacteriological examination is to be done, store in ice unit not more than 48 hrs.

Examination

Physical

Temperature of water should be measured at the point of collection without sampling. Turbidity should < 5 nephelometric units and color < 15 true color unit.

Bacteriological

The common feature of all bacterial screening procedures is that the primary analysis is for indicator organisms rather than the pathogens that might cause disease. Indicator organisms are bacteria such as nonspecific coliforms, *Escherichia coli* and *Pseudomonas aeruginosa* that are very commonly found in the human or animal gut and which, if detected, may suggest the presence of sewage. Indicator organisms are used because even when a person is infected with a more pathogenic bacteria, they will still be excreting many millions times more indicator organisms than pathogens. It is, therefore reasonable to surmise that if indicator organism levels are low, then pathogen levels will be very much lower or absent.

Presumptive coliform test

Multiple tube method. Estimate most probable number (MPN) of coliform/100 ml by inoculating increasing volumes of water (0.1, 1, 10, 50 ml) in McConkey's bile salt broth. All organisms producing acid and gas at 37°C are *presumed* to be coliforms. From the number of tubes showing acid and gas, the MPN is calculated. A confirmatory test is done for coliforms if the water sample was chlorinated. *E. coli* is Indole +ve, Methyl Red +ve, Vogues Proskawuer –ve, Urease –ve and Citrate –ve.

ATP testing. An ATP test is the process of rapidly measuring active microorganisms in water through detection of Adenosine Triphosphate. ATP is quantified by measuring the light produced through its reaction with the naturally-occurring firefly enzyme Luciferase using a Luminometer. The amount of light produced is directly proportional to the amount of biological energy present in the sample.

Membrane filtration, inoculation and colony count. Serial dilutions of the sample are vacuum filtered through membrane filters and these filters are themselves laid on nutrient agar within sealed plates. Membranes have a printed millimetre grid can reliably count number of colonies that have grown (20 hrs are allowed for growth of colonies) under a binocular microscope. The number of colonies = number of bacteria in original sample.

Fecal streptococci

Presence of fecal streptococci indicate recent fecal contamination.

Clostridium perfringens

Spores of *C. perfringens* (*welchii*) (without any coliforms) indicate old fecal contamination.

Colony count

Simple colony counts are done in nutrient agar at 37°C and 22°C in frequent intervals. A sudden rise in colony count gives earliest indication of contamination.

When samples show elevated levels of indicator bacteria, further analysis is often undertaken to look for specific pathogenic bacteria (*Salmonella typhi, Salmonella typhimurium, Cryptosporidium,Vibrio cholerae*, etc).

Chemical and physical surveillance

Basic tests. pH, temperature, color, turbidity, levels of chlorides, ammonia, chlorine demand and residual chlorine.

Complete analysis. Polynuclear aromatic hydrocarbons, radioactivity, pesticides, heavy metals.

Water quality standards (WHO)

Acceptability

Because end users have no direct method to judge water quality, they depend on their senses. They won't touch the water which looks dirty, smells bad, or has even a hint of color, or if it is impractically hard water. Rather they will find a souce of *unsafe* water that looks good.

Table 5.3. Acceptibility of water

Turbidity	No guideline value; turbidity > 5 nephelometric turbidity units is detected by people. *Chlorination* of water requires turbidity to be < 0.1 NTU
Color	≤ 15 true color units
Taste and odor	Minimal (no guideline value)
Temperature	Moderately cool (no guideline value)
Total dissolved solids	No guideline value; water becomes impalatable if TDS exceeds 1000 mg/l, and tastes good at 600 mg/l
Chemical	
pH	No guideline value; pH should be kept between 6.5–8 in distribution networks, otherwise pipes are easily corroded
Sodium	No guideline value; however, saline taste is detected in excess of 200 mg/l
Chlorides	No guideline value has been proposed; people can usually detect a level > 250 mg/l from taste
Free chlorine	5 mg/l; however, in concentrations 0.6–1 mg/l, most consumers will detect the taste of chlorine
Hardness	Ca^{++} 100–300 mg/l; however, people in some parts of the world tolerate a calcium level over 500
Iron	No guideline value; however, the taste of iron is detected, and laundry gets stained at a concentration of 0.3 mg/l

Microbiology

Coliforms (gram –ve enterobacteria fermenting lactose) are chosen as indicators of fecal pollution because

1. They survive natural disinfection better
2. They are constantly present in excreta
3. They live longer than other pathogens
4. They are easily detected.

Fecal streptococci indicate recent fecal contamination. *Clostridium perfringens* spores (without any coliforms) indicate old fecal contamination.

Guidelines values for microbial quality of water.[199] In all samples of water intended for drinking, and all samples of treated water entering the distribution system or *within* the distribution system, no 100 ml sample should have *E coli* or thermotolerant coliforms. Any presence of *E coli* warrants immediate investigation.

Most viruses are cleared by 0.5 mg/l of free chlorine. Parasites are removed by sand filtration.

Chemical

Guideline values

For most kinds of toxicity, it is believed that there is a dose below which no adverse effect will occur. For chemicals that give rise to such toxic effects, a tolerable daily intake (TDI) should be derived as follows, using the most sensitive end-point in the most relevant study, preferably involving administration in drinking-water.

1. The TDI is an estimate of the amount of a substance in food and drinking-water, expressed on a body weight basis (mg/kg or mg/kg of body weight), that can be ingested over a lifetime without appreciable health risk.
2. The maximum concentration of a substance that causes no harm in the consumers is called 'No observed adverse effect level' (NOAEL). It is usually detected from animal studies/retrospective studies on humans.
3. If NOAEL is not available (i.e. everyone has developed toxicity because of the substance), the minimum level of that substance that *causes* harm in study is called 'Lowest observed adverse effect level' (LOAEL).

TDI = NOAEL or LOAEL/Uncertainty factor

Sources of uncertainity in NOAEL determination

- Interspecies variation (animals to humans): 1–10
- Intraspecies variation (individual variations within humans): 1–10
- Adequacy of studies or database: 1–10
- Nature and severity of effect: 1–10

Guideline value = TDI × body weight × fraction of TDI attributable to water (i.e. what percentage of that toxic substance comes in through water)/daily drinking water consumption (1–2 l).

Table 5.4. Guideline values for selected chemicals

Arsenic	0.1 mg/l; use *surface water* to avoid arsenic (but surface water can not be used without treatment
Fluoride	1.5 mg/l
Mercury	0.006 mg/l
Lead	10 μg/l
Nitrate	50 mg/l; indicate pollution by fertilizer long ago
Nitrite	3 mg/l; indicates recent fertilizer pollution
Polynuclear aromatic hydrocarbons	Benzene 10 mg/l, Toluene 700 mg/l, Xylenes 500 mg/l
Pesticides	DDT 1 μg/l, Lindane 2 μg/l

Radiation

In safe water, gross α activity should be ≤ 0.5 Bq/l and β activity ≤ 1 Bq/l.

Problem village

A village where,

1. No source of safe water is available within 1.6 km (100 m in hills)
2. Water is available at a depth > 15 m
3. Where water has excess saline, iron, fluoride
4. Where water is exposed to risk of cholera.

Water supply programs in India

National Water supply and Sanitation program (1954). The effort started out with the objective of supplying safe water and adequate drainage for *everybody*.

Accelerated rural water supply program (1972).[200] Set some realistic targets and defined problem villages.

International water supply and sanitation decade (1981). Further specified the targets as 100% potable water supply, 80% sanitation coverage in urban area and 25% coverage in rural, to supply 40 l of water to everybody, and install one handpump/250 people.

Swajaldhara (2002). Shifting from a supply driven (i.e. the government decides how much water people need), Swajaldhara introduces a demand driven approach (people decide how much water they need) by decentralizing service delivery responsibility to rural local governments and user groups and building awareness about sanitation, water use and conservation (including rainwater harvesting).

AIR

Comfort

Comfort is "the state of mind that expresses satisfaction with the surrounding environment", which is a very subjective feeling.

Determinants of comfort

Air temperature. The earth is the only known planet which provides a habitable temperature. Extremes of hot and cold equally contribute to discomfort.

Air movement. Thermal comfort is maintained when the heat generated by human metabolism is allowed to dissipate, and flowing air helps the dissipation.

Metabolism. Each person has a different metabolism rate; so much that even people in the same room can feel significant temperature differences.[201]

Clothing. The amount of clothing is measured against a standard amount that is roughly equivalent to a typical business suit, shirt, and undergarments. Activity level is compared to being seated quietly, such as in a classroom. This standard amount of insulation required to keep a resting person warm in a windless room at 70°F (21.1°C) is equal to one *clo*, which is the unit of insulation by clothing. Higher clo values (due to either more layers of clothing *or* increased physical activity) causes greater thermal discomfort.[202]

Relative humidity. The recommended level of indoor humidity is in the range of 30–60%.[203] A way to measure the amount of relative humidity in the air is to use a system of dry-bulb and **wet-bulb thermometers.** A **dry-bulb thermometer** (which you usually see in your home or laboratories) measures the temperature not relative to moisture. This is generally the temperature reading that is used in weather reports. In contrast, a **wet-bulb thermometer** has a small wet cloth wrapped around the bulb at its base, so the reading on that thermometer takes into account water evaporation in the air. The wet-bulb reading will thus always be at least slightly lower than the dry-bulb reading. The difference between these two temperatures can be used to calculate the relative humidity. The larger the temperature difference between the two thermometers, the lower the level of relative humidity.

Indices of thermal comfort

Attempts at quantifying comfort ("I am 3.79% more comfortable than yesterday") have generated the following indices.

1. Air temperature—Not very effective; people in Rajasthan are more comfortable at the same temperature at which people of West Bengal feel suffocated (because Rajasthan has a lot less humidity).
2. Temperature + humidity—Still ineffective, because *stagnant* air, however cool or dry, will become hot and moist after you have exhaled a few times.
3. **Cooling power** = temperature + humidity + movement, measured by a Kata thermometer.
4. **Effective temperature**—an ET= 30°C in an area means that the comfort in that area at that particular time is comparable in comfort level to an area where temperature is 30°C + relative humidity is 100% + no air movement.
5. **Corrected effective temperature** = ET + correction for radiant heat (using globe thermometer). The comfort range of CET is 25–27, and intolerably hot over 30. Effective temperature is a weighted average between air temperature and radiation temperature and is closer related to the human perception.
6. **McArdle's maximum allowable sweat rate**— 4.5 l/4 hrs.

Air pollution

It is the

- Presence of any substance in air generated by activities of man
- In a concentration that interferes with human health or comfort
- Injurious to animal/plant kingdom directly/indirectly.

Air pollutants

Pollutants can be classified as either primary or secondary. Usually, *primary* pollutants are substances directly emitted from a process, such as ash from a volcanic eruption, the carbon monoxide gas from a motor vehicle exhaust or sulfur dioxide released from factories. *Secondary* pollutants are not emitted directly. Rather, they form in the air when primary pollutants react or interact. An important example of a secondary pollutant is ground level ozone — one of the many secondary pollutants that make up photochemical smog.

Primary pollutants

- **Sulfur oxides (SOx)**—Especially sulfur dioxide, is produced by volcanoes and in various industrial processes. Coal and petroleum often contain sulfur compounds, their combustion generates sulfur dioxide. Further oxidation of SO_2, usually in the presence of a catalyst such as NO_2, forms H_2SO_4, and thus acid rain.[204]
- **Nitrogen oxides (NOx)**—Especially nitrogen dioxide can be seen as the brown haze above industrial cities, which has also a sharp odor.
- **Carbon monoxide** is a colorless, odorless, nonirritating but very poisonous gas. It is a product by incomplete combustion of fuel such as natural gas, coal or wood (vehicular exhaust is a major source of carbon monoxide).
- **Carbon dioxide** is a greenhouse gas emitted from combustion but is also a gas vital to living organisms.
- **Methane** is an extremely efficient greenhouse gas.
- **Other volatile compounds** such as the aromatic compounds benzene, toluene and xylene are suspected carcinogens and may lead to leukemia through prolonged exposure.
- **Particulate matter** convert air into *Aerosol*, so that air actually becomes 'visible'. Dust storms, forest and grassland fires, burning of fossil fuels in vehicles and power plants generates significant amounts of aerosols. Particulates take a heavy toll over the lungs. **Cairo** has the maximum particulate matter among the world's cities, followed by Delhi and Kolkata in second and third place.
- Toxic metals, such as lead (generated from combustion of leaded petrol), cadmium and copper-generated from plastic, paint and metal industries.
- **Chlorofluorocarbons** (CFCs)—Harmful to the ozone layer. It was once used in compressors of refrigerators, now banned from use.
- **Ammonia** is emitted from agricultural processes. It is very caustic to the skin, very pungent to the nose and very irritating for the eyes.

- Odors such as from garbage, sewage, and industrial processes
- **Radioactive pollutants**—Produced by nuclear explosions, war explosives, and natural processes such as the radioactive decay of radon.

Secondary pollutants

- Particulate matter formed from gaseous primary pollutants and compounds in photochemical smog. Smog is a portmanteau of smoke and fog. *Classic* (those encountered in Victorian London) smog results from large amounts of coal burning in an area caused by a mixture of smoke and sulfur dioxide. Modern smog does not usually come from coal but from vehicular and industrial emissions that are acted on in the atmosphere by sunlight to form secondary pollutants that also combine with the primary emissions to form **photochemical smog** (Fig. 5.8).

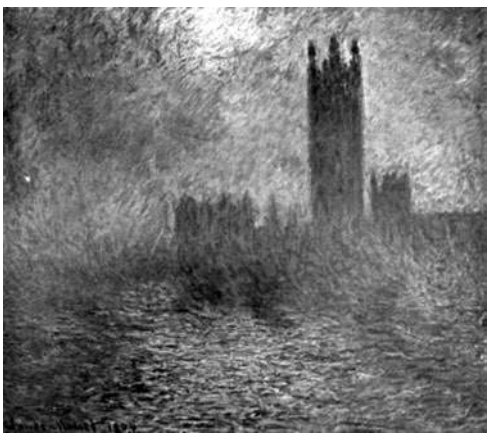


Figure 5.8. Claude Monet's impression of the London Smog[205]

- **Ground level ozone** formed from nitrogen oxides and volatile organic compounds. Ground level ozone is a component of smog.
- **Peroxyacetyl nitrate (PAN)**—Similarly formed from nitrogen oxides and volatile organic compounds.

Sources of air pollution[206]

Anthropogenic sources (human activity) mostly related to burning different kinds of fuel

- **Stationary sources**—Power plants, factories and waste incinerators, furnaces
- **Mobile sources**—Vehicles, marine vessels, aircraft
- Chemicals, dust and controlled burn practices in agriculture and forestry management
- Fumes from paint, hair spray, varnish, aerosol sprays and other solvents
- Waste deposition in landfills, which generate **methane**. Methane is explosive and also an asphyxiate. It may displace oxygen in an enclosed space
- Military, such as nuclear weapons, toxic gases, germ warfare and rocketry.

Natural sources

- **Dust** from natural sources, usually large areas of land with little or no vegetation, dust storms, landslides
- **Methane**, emitted by the digestion of food by animals, for example cattle; also from decomposed vegetation in marshlands
- **Radon gas** from radioactive decay within the Earth's crust. It is the second most frequent cause of lung cancer, after cigarette smoking.
- Smoke and carbon monoxide from wildfires
- Volcanic activity, which produce sulfur, chlorine, and ash particulates.

Effects of air pollution

The World Health Organization states that 2.4 million people die each year from causes directly attributable to air pollution, with 1.5 million of these deaths attributable to indoor air pollution.[207] The health effects caused by air pollutants may range from subtle biochemical and physiological changes to difficulty in breathing, wheezing, coughing and aggravation of existing respiratory and cardiac conditions. These effects can result in increased medication use, increased doctor or emergency room visits, more hospital admissions and premature death.

Table 5.5. Effects of pollutants

Substance	Effect
Nitrogen oxides	Form ground level ozone which is irritant and component of smog
CO_2	A greenhouse gas
H_2S, SO_2	Acid rain
Lead	Anemia, encephalopathy (particularly in children)
Polynuclear aromatic hydrocarbons/ volatile organic compounds	Lung cancer
Particulate matter <10μm (which enter alveoli)	↓ lung capacity, hypersecretion of mucus, asthma and cystic fibrosis, reduced visibility in cities (sometimes leasing to accidents)
Radon gas	Lung cancer

Global warming

Nowadays everybody is concerned about 'climate change' and 'global warming'. It is embarrassing, as physicians, to not have even a faint understanding of these terms, although it has little to do with day-to-day clinical decision-making.

Global warming is the increase in the *average temperature of the Earth's near-surface air* and oceans since the mid-20th century and its *projected continuation*. Mind the term 'global'. That this year was colder in your city than last year does

not rule out global warming. It has been shows, although only by statistical[208] methods, that average global surface temperature increased 0.74 ± 0.18°C between the start and the end of the 20th century.[209] Most of this 'warming' is attributable to deforestation, burning of fossil fuels and emission of greenhouse gases (Fig. 5.9).

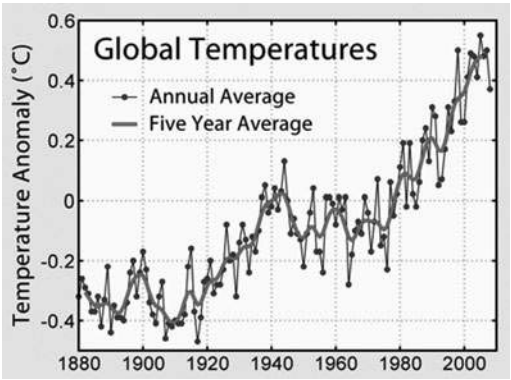


Figure 5.9. Instrumental record of global average temperatures as compiled by the NASA's Goddard Institute for Space Studies[210]

The greenhouse effect

The greenhouse effect is *essential* for life on Earth and is one of Earth's natural processes. It is the result of heat absorption by certain gases in the atmosphere (called greenhouse gases because they effectively 'trap' heat in the lower atmosphere) and reradiation downward of some of that heat. **Water vapor** is the most abundant greenhouse gas, followed by **carbon dioxide** and other trace gases (volatile organic compounds). Without a natural greenhouse effect, the temperature of the Earth would be about –18°C) instead of its present 14°C.

All this was very well until the industrial revolution in Europe, and we began burning our fossil fuels at a tremendous rate. Over 250 years, human activity has been increasing the concentration of greenhouse gases in the atmosphere (mostly carbon dioxide from combustion of coal, oil, and gas; plus a few other trace gases). There is no scientific debate on this point. Preindustrial levels of carbon dioxide (prior to the start of the Industrial Revolution) were about 280 parts per million by volume (ppmv), and current levels are greater than 380 ppmv and increasing at a rate of 1.9 ppm yr-1 since 2000.[211]

Effects of global warming

It is impossible to attribute to global warming any single meteriological event. Instead, long-term clustering of events may be caused by global warming. A 2001 report by Intergovernmental Panel on Climate Change suggests the following

- Retreat of large glaciers
- Disruption of large ice shelves
- Rise of sea levels—Global mean sea level has been rising at an average rate of 1.7 mm/year (plus or minus 0.5mm) over the past 100 years[212]
- Shrinkage of artic and antartic regions
- Change in rainfall patterns and alteration of seasons—As it is already being observed.

Additional effects as predicted by various authorities include—increasingly intense (but less frequent) hurricanes and extreme weather events,[213] reductions

in the ozone layer, changes in agriculture yields (which may throw millions into starving), changes in the range of climate-dependent disease vectors,[214] which have been linked to increases in the prevalence of malaria and dengue fever[215] and ocean oxygen depletion.[216] Increased atmospheric CO_2 increases the amount of CO_2 dissolved in the oceans, which alters its pH, so that many organisms may become extinct.[217]

Mitigation and preparedness

Because global warming is 'global', it is imperative that concerned nations sit down and sort it out. Herein lies the dilemma. Emission of greenhouse gases is linked to the industry and GDP of a country, an nobody's ready to submit themselves to any treaty that obliges them to reduce emissions, which is another way of saying 'stop your factories and do not ride cars'. The most industrialized nations have had their share of the pie of economic development, and now none is ready to listen to the others. Because cleaner air is nobody's property, and each country will benefit as much as any other, if not less, if anyone chose to reduce emissions, it is common sense that everybody should come to an agreement. But our species has been lacking common sense since Hiroshima. So nobody is ready to reduce emissions and give cleaner air to anybody else. The result is that things go on as usual, and nobody loses except our species.

The world's primary international agreement on reducing greenhouse gas emissions is the **Kyoto Protocol** (1977). The Protocol now covers more than 160 countries and over 55% of global greenhouse gas emissions. As of June 2009, only the United States, historically the world's largest emitter of greenhouse gases, has refused to ratify the treaty. The treaty expires in 2012. International talks began in May 2007 on a future treaty to succeed the current one. The 2009 United Nations Climate Change Conference met in Copenhagen in December 2009 to agree on a framework for climate change mitigation - however, it was a disappointment and no binding agreement was made.

What we can do at community level. We can certainly burn less fossil fuels, use more solar and wind power, conserve plantations, learn to conserve water, and use a dark color scheme in our computers (light colors emit more radiant heat).

Adaptation to global warming. People sense that global warming is inevitable, and they have to live with it. A wide variety of measures have been suggested for adaptation to global warming. These measures range from the trivial, such as the installation of air-conditioning equipment, to major infrastructure projects, such as abandoning settlements threatened by sea level rise, water conservation, water rationing, adaptive agricultural practices, construction of flood defences, changes to medical care, interventions to protect threatened species, and even colonization of Mars (sic) have all been suggested.

Indoor air pollution

As important as 'global warming' is, lets not forget to peek into our rooms, where we live most of the time, and check out the air inside. Because more people are affected by *indoor* rather than outdoor pollution.

Table 5.6. Indoor air pollutants

Agent	Effect
Radon is a colorless radioactive inert gas generated from decay of radium	2nd most common cause of lung cancer (after smoking)[a]
Molds and other allergens (house dust mites); mold is always associated with moisture, and its growth can be inhibited by keeping humidity levels below 50%[b]; regular vaccuming, especially in creaks and crevices, cleans mites	Allergy and asthma
Carbon monoxide (from fireplaces and combustion of kerosene etc.)	Asphyxia, unconsciousness and death (usually occurs in winter lamps, when many people sleep tightly in a closed room with a burning lamp)
Volatile organic compounds (from paints, tar, varnish, wax, cosmetics, cleansing agents, ink from printers and copiers, glues, adhesives, carbon paper)	Carcinogenic
Legionella pneumophila (resides in cooling towers and air conditioners)	Legionnaires' disease
Asbestos fibers (from building materials, insulation and fireproofing appliances)	Interstitial lung disease, lung cancer
Particulate matter (from tobacco smoke, inks, aerosol sprays, mosquitoe coils)	Reduced lung function
Carbon dioxide (increases with increasing number of people in a room)	Drowsiness, poor performance
Ozone (effect of electric arcs/UV ray sources over room oxygen)	Skin irritation, reduced lung function

[a] # US EPA Indoor Environment Division

[b] WHO guidelines for indoor air quality: Dampness and mould. Report on a working group meeting, 17–18 October 2007

Measures to control indoor air pollution. Continuous air exchange with outside air, use of air filters, indoor plantation, control of moisture.

Indicators of air pollution

1. Dust and grit in air (in tonnes/ km^2 of area/ month)
2. Concentration of SO_2 and NO
3. Suspended particles ($\mu g/m^3$)
4. Smoke/soiling indicator ($\mu g/m^3$)—The staining of a filter paper by a specified volume of air
5. Haze coefficient (for assesing aerosols in air)
6. CO, NO_2, Pb in air.

Table 5.7. Guideline values of some substances in air[218]

Substance	Guideline value
Carbon monoxide	Average 100 mg/m^3 over 15 minutes of measurement
Sulfur dioxide	350 $\mu g/m^3$ over 1 hr of measurement
Lead	0.5–1 $\mu g/m^3$ over 1 year of measurement
Particulate matter	100 $\mu g/m^3$

Air pollution index

SO_2 + smoke/soiling indicator + haze coefficient. However, countries may differ on their derivation of the index.

Prevention of air pollution

1. **Containment** of pollutants at source—Many devices arrest a pollutant at its origins-wet scrubbers and electrostatic precipitators (remove particulate matter), selective catalytic reduction and low NOx burners (remove nitrogen oxides), adsorption systems, vapor recovery and thermal oxidation systems (for volatile organic compounds), wet/dry scrubbers and flue gas desulfurization (for sulfur dioxide).

Scrubbers

Scrubber systems are a diverse group of air pollution control devices that can be used to remove some particulates and/or gases from industrial exhaust streams.

A wet scrubber is used to clean air, flue gas or other gases of various pollutants and dust particles. Wet scrubbing works, via the contact of target compounds or particulate matter with the scrubbing solution. Solutions may simply be water (for dust) or solutions of reagents that specifically target certain compounds.

Dry scrubbing systems are used to remove acid gases (such as SO_2 and HCl) primarily from combustion sources. The media used is typically, an activated alumina compound impregnated with materials to handle specific gases such as hydrogen sulfide. Media is used, can be mixed together to offer a wide range of removal for other odorous compounds such as methyl mercaptans, aldehydes, volatile organic compounds, dimethyl sulfide, and dimethyl disulfide.

2. **Replacement** of bad processes with good ones, such as Red phosphorus replacing white phosphorus, disuse of CFC, disuse of leaded petrol, use of electricity rather than coal for heating
3. Find alternate, "greener" energy source such as use of biofuel, solar, water, geothermal and wind energy.

Biofuels

Alcohols (commonly ethanol, also propanol and butanol) derived from fermentation of sugar is being already used in gasoline engines. Transesterification of

plant oils (derived from soy, rapeseed, jatropha, mahua, mustard, sunflower, palm oil) produces **biodiesel** which can be used in any diesel engine when mixed with mineral diesel. A number of other biofuels are available, including the raw vegetable oil.

4. **Traffic management**: Enforcement of emission rules and good driving practices, to increase total area covered by roads (30% in urban areas) and to increase offset of housing from roads.

Indian emission standards. Since the year 2000, India started adopting European emission and fuel regulations for four-wheeled light duty and heavy duty vehicles. Indian own emission regulations still apply to two and three-wheeled vehicles. Current requirement is that all transport vehicles carry a fitness certificate that is renewed each year after the first two years of new vehicle registration. Currently, Bharat stage III guidelines are in effect since 2005, and Bharat stage IV guidelines are to be introduced in 2010.

5. Establishing green belts (plantations) around industrial areas
6. Judicial use of insecticides, cosmetics, volatile compounds
7. Awareness campaigns
8. Legislation—"The Air Act" in India was set up in 1981.

HOUSING

Admit it. We like our homes. We *love* our sweet homes. Be it a cottage or a mansion, everybody shares equal affection for their respective homes. And in no place you will realise this better than in a slum. You will literally be frightened by their unstable little huts, where they live two families per room. However, no amount of incentives will move them from their homes, no promise of better housing, income or education will make them abandon the place they belong. Because 'housing' is not only the brick and mortar, but also includes the neighbors, the environment and the culture acquired over a prolonged period of stay in a certain place. Removing someone from his house equivalents to uprooting him. No community intervention should have 'change of housing', for any reason.

The 'residential environment'

It is the physical structure that man uses and the environment of the structure – includes all necessary services, facilities, equipment and devices needed for physical, mental and social well-being. A "healthful house" is that which provides

1. **Physical environment**—Personal space, ventilation, dry soil/floor, subsoil water (at a depth of > 3m), protection from communicable diseases, sanitary disposal methods for garbage and excreta
2. **Mental environment**—Privacy, safety, noise and odor control, good interpersonal relationship
3. **Social environment**—Social amenities (such as nearby parks and community recreation centers), good neighbors, a safe and cultured neighborhood.

Rural housing

Rural houses are often impracticable – damp, squirmy, without a kitchen, sometimes lacking a latrine and often adhered to the cattle shed. A 'model village' is where the houses are laid geometrically, the soil is a mixture of clay and sand, the area is dry and not flooded every year, every house has at least two rooms with separate kitchen and latrine, and cattle sheds are 8–10 m away from home.

Urban housing

Environmental housing committee (1947) recommends the following for urban houses.

1. A **site** which is not flooded regularly, does not rear mosquitoe breeding places, located over dry soil, connected by at least one and away from noise pollution
2. Open space in 4 sides (**setback**)
3. The **floor** should be impermeable (so that insects do not creep in) and easily washable, with no cracks, free of rats, and at least 3 ft high from road level
4. The **walls** should be solid and impermeable
5. The **ceiling** should be least 3m above floor (unless air-conditioned), should **not** transmit much heat
6. 100 ft^2 floor per room (at least 50 ft^2/person)
7. At least **two windows per room** (total window area = 1/5th of floor area) and area of windows + doors > 2/5th of floor area
8. **Daylight factor** (illumination inside the room/illumination outside the room) > 1% (ideally 8%) over half of floor; smallest newsprint should be legible after closing the doors during daytime
9. A separate **kitchen**
10. One or more **sanitary latrine(s)** with manageable privacy
11. Potable **water supply**
12. Separate facility for bathing and washing
13. Good provision for **garbage and refuse disposal**.

Overcrowding

It is the situation in which more people are living in a single dwelling than there is space – so that movement is restricted, privacy secluded, hygiene impossible and rest difficult.

Effects of overcrowding

Infectious diseases

Among the physical parameters of housing, the per capital floor area available or overcrowding has been shown to be a very important factor in increasing the frequency of all diseases transmitted through direct interhuman contact, viz measles, meningitis, influenza, upper respiratory infections, scabies and tuberculosis.[219]

Personal space violation and its aftermath

The idea of personal space. Every person has a rim of space surrounding his body that he considers his own 'personal space'; he is very reluctant to let anyone

in this personal space, except very intimate ones. Personal space does not have a fixed measurement, but varies depending on situation (i.e. can be reduced to few millimeters in a crowded train, or extend to a few meter in a leper colony).

Overcrowding drastically violates the personal space. It is in human nature to protest this onslaught against privacy, and overcrowding frequently leads to irritability, loss of concentration and domestic violence. Because personal space is a lot wider in women, they are especially vulnerable to the effects of overcrowding.

Criteria of overcrowding

Person per room. Should be always < 2 (children <1 years of age equal '0' person, and children 1–2 years of age consists only half a person),

Floor space. At east 50 ft^2 for each person; ideal range is 90–110 ft^2 per person

Sex separation. Two (or more) people of the opposite sex > 9 years of age forced to living in the same room is overcrowding.

Indicators of good housing

1. Physical—Floor space, volume, height of rooms, persons per room, rooms per house
2. Economic—The cost of building the house, the rent/mortgage and recurring expenditures of maintenance of house
3. Social[220]
   a. Related to disease prevention—Diseases due to inadequate sewage or garbage management *or* contaminated water *or* vector borne diseases or overcrowding; frequency of accidents, degree of proximity to animals, and access to medical facilities
   b. Related to comfort—Thermal comfort (not too warm or cold inside the house), visual comfort (the fences of the house should not be ridden with posters or graffiti), acoustic comfort (not too near a railway, highway or factory) and spatial comfort
   c. Related to mental health and social well-being—Prevalence of neglected/unemployed youth in the neighborhood, frequency of suicides and drug/alcohol abuse in the surroundings.

WASTE DISPOSAL

This is where the 'disgusting' part of community medicine really comes in. But think of your own home; do you clean your own room regularly? Where do you dispose the ends of your nails after a nail removal? Where do you dispose the strew of graphite after you have sharpened your pencil? Where have all the books from your nursery and kindergarten classes gone? What happens to the onion peels and vegetable residue after your mom is done cooking? The problem of waste management doesn't strike as readily, because it takes sometime for a heap of garbage to build up in your room, but it is just as sure if you do not have the habit of cleaning up once in while. Without your intervention, things in your room will naturally, overtime, fall into disarray. It is called *entropy*.[221] Fighting this tendency of material objects is what this chapter is all about (Fig. 5.10).

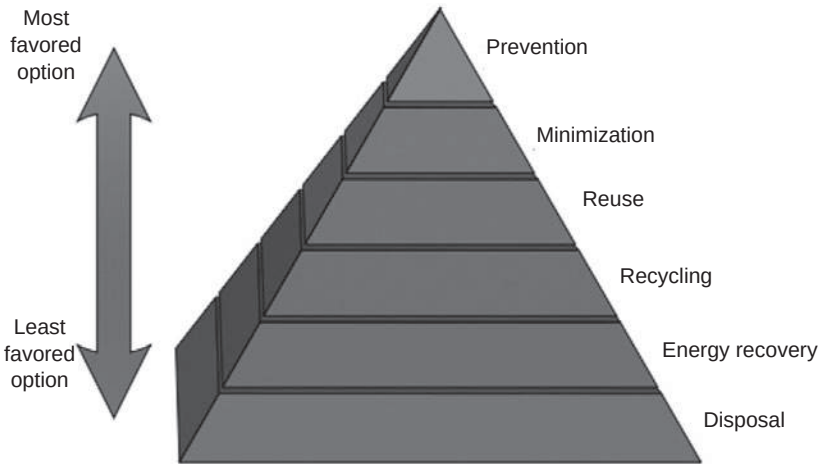


Figure 5.10. The waste disposal hierarchy[222]

Solid wastes

Solid wastes consist of street refuse (everything that you see lying over streets), market refuse (vegetable and animal remnants), stable litter (animal feeds and feces), industrial waste (may range from inert substances like chalk, or dangerous ones like batteries), office waste (mostly paper, carbon paper, plastic) and domestic refuse (plastic, ash, garbage and rubbish). *Garbage* is organic solid waste (i.e. vegetable peel) and *rubbish* is inorganic solid waste paper, etc.).

Waste minimization

The more waste we generate, the more we have to dispose. Instead of discarding old clothes and getting a new PC every holidays, wouldn't it be better if we try to get more out of every product? You can always use the leftover pages of your old diary instead of starting a new one this year (remember, paper is tree). We could do shopping in old-fashioned big shoppers made of jute rathern than the plastic carry bags. And we could altogether stop using any 'use and throw' items (what a derogatory phrase that is!). When you think of it, there is a lot we can do.

Sorting and recycling (Fig. 5.11)

Sorting and recycling allows the recovery of raw materials, which can be later used to manufacture new products. We can sort our waste in our homes and place the recyclable ones in a different bin. Those which can be recycled are glass, plastic, aluminium, cardboard, steel, paper, card, wood, metal, plastic, building site waste, cans. Developed countries have also devised automatic sorting machines in treatment plants.

Figure 5.11. The symbol for recycling

Storage

The best place to store solid waste, until it is picked up, is a *closed* dustbin or disposable paper bag. Whatever the design, it must be ensured that *flies* do not have access to it.

Collection

We Indians (especially those in cities) have a habit of packing everything in a plastic bag and throwing it out to the open street. It is only recently that door to door collection of waste has been initiated in major cities of India.

Methods of disposal

Dumping

This most unreasonable practice is still in Kolkata.

Composting

Composting is a process that has always existed in its natural state. Today, with modern technologies, this process can be accelerated and monitored efficiently. It is an excellent way of regenerating soils impoverished by intensive farming. Four types of waste are accepted in the composting facilities:

- Waste from the wood industry (bark, shavings), the paper industry and the food industry
- Farming waste: Manure, mineral fertilizers, etc.
- Household waste (food, garden)
- Sludge from the sewage plants.

Composting is a process based on the decomposition of organic matter by microorganisms in the presence of oxygen. The result is a stable organic product, which is both hygienic and rich in humus. This process, which normally takes several months, can be speeded up and controlled using various techniques. Once the composting process is finished, a product called compost is obtained, which is mainly used for agriculture manure.

Landfill

Waste that can neither be reused nor receive special processing is stored in landfills. A landfill requires high technical standards in order to protect the environment and local communities. It is very important taking into account

- Location should not be too close to housing or a water source
- To ensure that waste does not leak into groundwater
- To block flies and rodents from access to the waste
- The construction of a cover, in order to minimise rainwater penetration.

Overtime, waste, which decomposes produces **biogas**, a mixture of carbon dioxide and methane. Biogas is collected and then used in energy production, (at the same time the emission to the atmosphere of methane is avoided). Apart from biogas, the decomposition of landfilled waste produces a liquid known as "leachate". Leachates contain heavy metals, salts, nitrogenous compounds and various types of organic matter. Due to its high polluting potential, it is needed that this leachate is collected and treated to avoid environment contamination.

Energy from waste

Nowadays, energy recovery goes hand-by-hand with waste elimination. There are different ways of getting energy from waste: Incineration, gasification, anerobic digestion.

Incineration allows obtaining energy at the same time that the volume of waste is reduced, as well as the use of landfill. Three different kind of waste are accepted in an incineration facility:

- Household waste
- Nonhazardous industrial waste
- Rejects from sorting centers.

The waste is then burnt at temperatures reaching 1000°C, producing steam that turns turbines, which in turn produce electricity. The fumes produced in the combustion are treated by a dry or wet method. Ashes can be reused in civil engineering.

In the **anerobic digestion** process, the organic portion of the waste is separated and then placed in a sealed reactor. In the reactor, the conditions necessary for biological degradation are created which allows the production of biogas, that can be used as a fuel to produce electricity.

Gasification and advanced plasma arc gasification are used to convert organic materials directly into a synthetic gas (syngas) composed of carbon monoxide and hydrogen. The gas is then burnt to produce electricity and steam.

Liquid wastes

1. *Industrial liquids* should ideally be treated and detoxified before any contact with groundwater or any water source (i.e. rivers).
2. *Sullage* is the water from bathroom and kitchen, and is usually free of major pathogens, and thus can be allowed to drain into groundwater.
3. *Sewage* is the water from latrine which is teeming with pathogens and should NEVER come in contact with any natural source of drinking water; treatment of sewage is a major investment for long- term well-being.

Hospital waste disposal

Disinfection → disposal → drainage

Health care waste is generated by health care establishment, research facilities from various activities. They may be

1. General wastes.
2. Hazardous wastes—Infectious material, genotoxic material (mutagen/teratogen/carcinogen) and sharp wastes (which may puncture skin); all bags/bins/trolleys and vats containing hazardous waste should be marked with the biohazard symbol (Fig. 5.12).

Figure 5.12. The biohazard symbol

Table 5.8. Categories of health care waste

1.	Human anatomical waste (i.e placenta)
2.	Animal waste (i.e. guinea pig intestine)
3.	Microbiology and biotech waste
4.	Waste sharp
5.	Discarded medicine, cytotoxic drugs
6.	Soiled waste
7.	Solid (casts, drip tubes, catheter)
8.	Liquid waste (disinfectants)
9.	Incineration ash
10.	Chemical waste

Assuming there is 3–3.5 kg of biomedical waste being produced by a hospital bed,[223] one could imagine how much of waste is accumulated by the thousands of beds in the country. Of all the health care wastes, 66–80% are recyclable.

Impact of health care waste

Health hazards

1. Infections (especially hepatitis B and C, HIV)
2. Toxic waste (drugs, disinfectants) may get inhaled or come in contact with skin if not properly disposed
3. Radioactive waste (from radiotherapy departments) may get into environments.

Environmental hazard

Hospital waste, apart from looking foul and unsightly, clogs drains (because of its high polymer content), suffices as a vector breeding place, contaminates ground water and volatile compounds (many drugs and disinfectants) get into the air.

Population at risk

1. Health care staff (doctors, nurses, auxiliaries)
2. Hospital maintenance personnel
3. Patients and visitors
4. Hospital support service
5. Waste disposal workers
6. General public in vicinity.

Principles of management

1. Minimize amount of waste; try reusing old items (with repeated sterilization) instead of demanding new ones
2. Segregate wastes *at time of generation* into anatomical, infectious nonsharp and sharp waste
3. Disinfect infectious waste before disposal.

Segregation and collection (Fig. 5.13)

Table 5.9. Which class of waste is to be put in which colors of bags and bins?

	Bag	Bin
• Discarded medicine, cytotoxic drugs • *Solid* chemicals used in disinfection/laboratories (such as oxalic acid)	Black	Black
Anatomic wastes (biodegradable)[a]	Yellow	Yellow
• Infected material from microbiology laboratory (all samples/cultures, vaccines, dishes and devices) • Soiled items (items stained with anybody fluid such as cotton, dressing, plaster casts) • Solid, possibly infected waste generated from ward (catheters, saline infusion sets, etc.) - to be cut in pieces before disposal (to prevent reuse before sterilization)	Red	Red
Sharp items (to be broken and disinfected in bleaching powder before dropping in bin)	Blue	Red
[a] Placenta should be stored, for extraction of hormones, wherever facilities are available.		

Segregated internal transport

The waste is to be carried in dedicated trolleys (black – uncovered, red – covered) to a double chambered vent, located near or inside hospital campus.

On site treatment

1. Needles/nozzles to be cut by needle cutter, disinfected by 1% bleach for 1 hr (but sharps generated in laboratories must stand 10% bleaching overnight)
2. Wet thermal treatment (autoclaving) of *reusable* biomedical waste.

On site segregated disposal

1. Rural/small hospital → landfill (see waste management)
2. Large hospitals → store in double chambered vat near exit
3. Liquid wastes → Neutralize in acid/alkali and drop in septic tank.

Off site segregated transport

Daily final disposal in dedicated, covered vehicles.

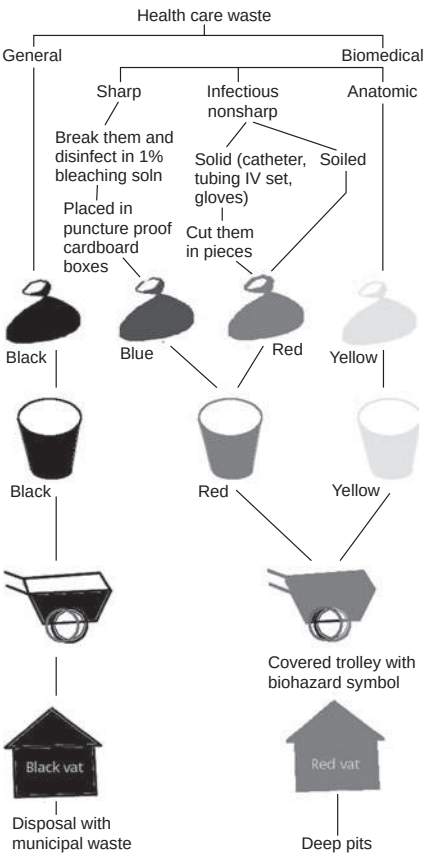


Figure 5.13. Bags and bins simplified

Off site disposal

1. **Incineration**—Not suitable for plastics, silver salts, reactive chemical wastes, ampoules and waste with heavy metals (batteries, thermometers, lead).
2. **Chemical disinfection**—Suitable for microbial culture media and sharp wastes.
3. Wet thermal treatment (autoclaving)—For all reusable waste except sharp ones.
4. Dry thermal treatment—Dehydrates most biological waste and thus makes them shrink; not suitable for cytotoxic or radioactive waste.
5. Microwave irradiation—For disinfection of reusable waste.
6. Landfill.
7. Inertisation (mixing with cement).

DISPOSAL OF HUMAN EXCRETA

> If you consider the contribution of plumbing to human life, the other sciences fade into insignificance.
>
> —*James Gorman*

Very few scholars have documented the history and state of toilets of the world. The Nobel Prize winner for Medicine (1913) Charles Richet attributes this silence to the disgust that arises from noxiousness and the lack of usefulness of human wastes. But the toilet is part of the human history, and a critical link between order and disorder and between a good and a bad environment. The subject of the toilet is as important, if not more, than other social challenges like literacy, poverty, education, and employment. In fact, the subject of the toilet is more important because lack of excremental hygiene is a national health hazard while in other problems the implications are relatively closer to only those who suffer from unemployment, illiteracy, and poverty.

> We have had women's liberation, sex revolution, workers' revolution, we can talk about everything now—the toilet is the last taboo which must be broken.
>
> —*Jack Sim, founder of World Toilet Association*

Effect of insanitary toilets. Human excreta, in addition to polluting water, soil, air and acting as breeding grounds of vehicles, also is the largest vehicle for transmission of bacteria (the 'feco-oral' route) and most unsightly. It is important to create a **sanitation barrier** between feces and the five routes of transmission (water, fingers, flies, food and soil), to control enteral infections.

The issue of dirty toilets. Not only do dirty toilets contribute directly to vector borne disease, but they affect the social psyche. A large number of people avoid going to public toilets just because they are unclean. Particularly women and school children avoid toilets for a long period of time when they are out of home. This causes various health problems such as dehydration, urine infection, bladder disease, and anal fissure.[224]

Excreta disposal in unsewered areas ('on site' latrines)

India is still mostly 'unsewered' (without sewage treatment and disposal systems), and many people (38%) are still regularly visiting the 'outfields' for relief.[225] Even more shameful is the existence of 'service types latrines' from where excreta is collected by human laborers.

Sanitary latrines

These are the best solution for unsewered areas. A sanitary latrine is one which

1. is not unsightly or malodorous
2. does not give access to flies and mosquitoes, rats
3. does not contaminate air, water, soil
4. provides water for ablution
5. located at least 15 m away from a water source.

The types of sanitary latrines in use are discussed as follows (Figs 5.14–5.20):

1. **Bore hole latrine**: It is basically a pit (20 ft × 16″ diameter) lined by cement, refusing access to flies and prohibiting contact with water or soil. It is however, hard to construct and needs a device called 'auger'. It is for family use and life period is 1 year. To construct such a latrine, subsoil water must be more than 10 ft and soil should not be loose.
2. **Dug well latrine**: It is easier to construct and longer life. The pit is wider (10–12 ft × 30" diameter).

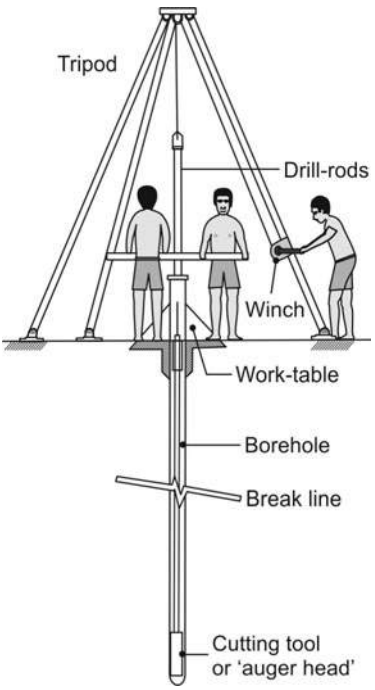


Figure 5.14. Construction of a borehole latrine using an auger

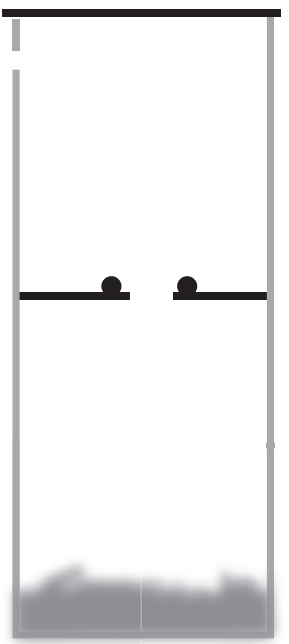


Figure 5.15. A bore hole latrine[226]

Raised latrines. When the groundwater is high, the area is flooded regularly or the ground is too rocky to excavate by hand there is a case for using a *raised* latrine.

3. **Ventilated pit latrine**—During the 1980s the VIP latrine was developed in Zimbabwe. The main drivers for design were to eliminate two unpleasant aspects of using on site latrines, namely flies and smell. Furthermore, the reduction of flies can also reduce the transmission of disease. Put simply, the technology facilitates the flow of air through the system. One important aspect is that the inside of the toilet should remain dark as means of attracting flies up a vent pipe where they will eventually die and fall back into the latrine.

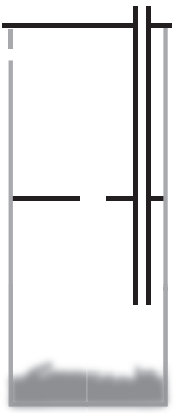


Figure 5.16. A 'VIP' latrine; note the vent nozzle[227]

4. Water seal latrine/RCA latrine: It is basically a pan connected (underground) to a dug well latrine directly or by a connecting pipe; the specialty of this kind of latrine is the **water seal** or trap, a U- shaped pipe just below the pan which maintains a constant level of water, as to prohibit entry of flies into the dug well.
   a. Located at least **15 m** away from any water source.
   b. The squatting plate should be accomodating for one person (3 ft × 3 ft), dry, periodically exposed to sun; a gentle **slope** should run towards the pan.
   c. The **pan** should have uniform slope and smooth surface.
   d. The water seal/**trap** should be at least 2 cm deep.
   e. Should have a superstructure (for privacy).
   f. The dug well should be lined by earthenware rings to withstand pressure of loose soil and groundwater (where groundwater levels are high).

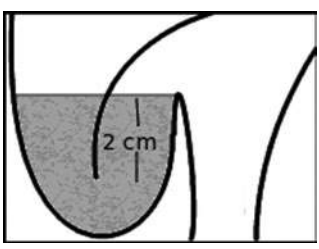


Figure 5.17. A plumbers trap

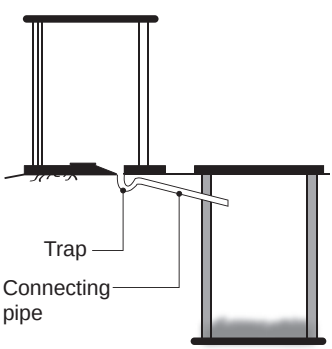


Figure 5.18. Water seal latrine[228]

Septic tank

It is suitable for

1. families
2. small groups of houses
3. institutions without access to sewerage system.

Criteria

1. Volume = 20–30 gallon (2.5–5 ft^3/ person)
2. Length = 2 × breadth

3. Depth = 5–7 ft; of this depth, only 4 ft should be occupied by liquid and the rest by the sludge below.
4. Provision of inlet and outlet; the sludge should slope towards outlet.
5. A cover.
6. At least 24 hr retention period for anaerobic oxidation.

There are two stages in the working of a septic tank: the *anaerobic digestion* occurs inside the tank. The layers of fat and grease (scum), liquid and sludge separate out within the tank. The effluent liquid passes into soil. The bacteria and cysts in the effluent are now aerobically oxidized in soil.

Because it is necessary to have some anaerobic bacteria within the tank, sludge from another tank is placed within a new tank ('ripening' of the tank).

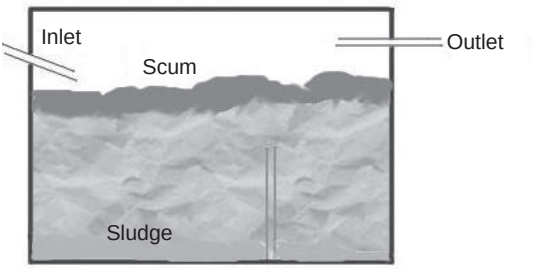


Figure 5.19. A septic tank[229]

Aqua privy

An aqua privy functions in a similar manner to a septic tank whilst avoiding the need for a consistent water supply to operate a flush toilet. The water will drain off the top and the sludge needs to be emptied on a regular basis. An advantage of the aqua privy is that it reduces odors. However, regular emptying could become an onerous requirement.

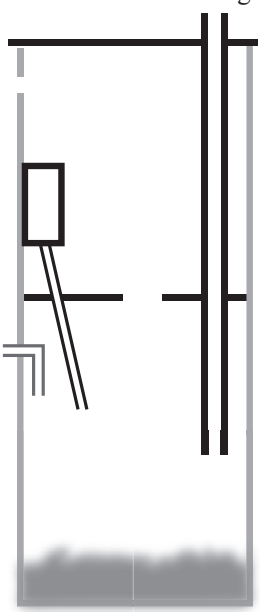


Figure 5.20. An aqua privy[230]

Trench latrines

A trench latrine is a rectangular hole in the ground. The hole should be dug as deep as possible — about 2m and may be lined with timber where there is danger of collapse. It may be of any convenient length, usually between 5 and 10 m, and between 1 and 1.5 m wide. By setting up partitions, a whole camp can use one trench latrine. Each week the contents of the trench are covered by a 100–150 mm deep layer of soil. This will reduce the smell and prevent flies from breeding in the trench. When the bottom of the trench has risen to within 300 mm of the surface, the trench is filled in and the latrine is closed.

A trench latrine system is very labor intensive and requires constant supervision. Not only must the contents of each latrine be covered each day, but new latrines must be prepared, old ones filled in, and regularly used latrines cleaned. It is suitable in emergency relief camps.

Excreta disposal in sewered areas

> The underground galleries which are the organs of the big city (sewage pipes) will work in the same way as organs of the body, without being revealed
>
> —*Haussmann (1858)*

Most older cities use a *combined* sewer system, where sewer and rainwater drains through the same pipes. During dry weather (and small storms), all flows are handled by the publicly owned treatment works (POTW). During large storms, the relief structure allows some of the combined stormwater and sewage to be discharged untreated to an adjacent water body (Fig. 5.21).

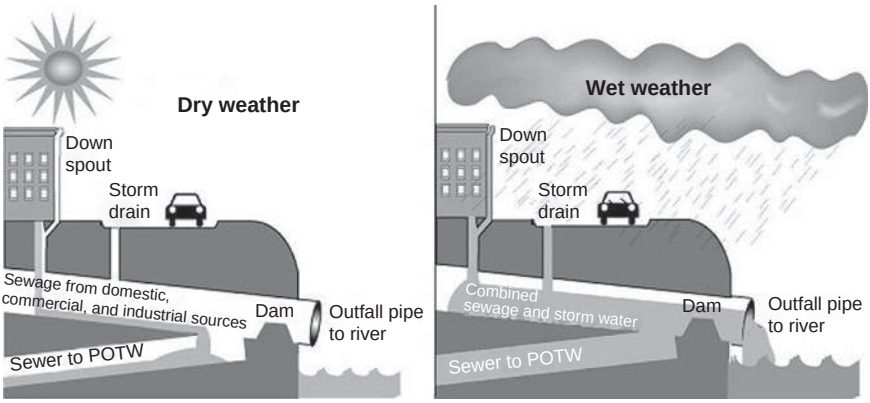


Figure 5.21. Combined sewer overflow

Thus new cities are now built with a *separate* sewer network. Advantages of a *separate* water carriage system for human excreta is that

1. Sewer pipes need to be less wider
2. Amount of sewage is less
3. Estimation of total bulk of sewage is possible
4. Lesser fluctuations in bulk of sewage.

The water carriage system in a sewered area consists of the following.

Household fittings. The water closet (the toilet which you are used to see), water seal and the cistern.

House sewers. An underground pipe of 4″ diameter usually drains into the street sewer; an antisiphon pipe may also be used; the drain should have a slope towards the street sewer (for a velocity of 1–2 ft/sec) , smooth inner surface, should be flushed properly, and should join in 'Y' fashion with street (and not in 'T' fashion); a ventilator nozzle should be connected to this drain for expulsion of gases. A trap should also be placed where the house drain joins the street sewer.

Public sewers.[231] Public sewers should have 9″ diameter (often they are much larger), placed 10 ft below ground, with such a slope towards treatment plant as to allow a 'self cleansing' velocity of sewage. *Manholes* should be placed where—

1. Sewers change direction
2. Two or more sewers meet
3. Each 100 m in linear course.

Traps are placed in the house latrine, where house drain joins public sewer and wherever surface water may gain entry into sewer.

Sewage treatment

Sewage consists of water (99.9%) and sludge (0.1%), which remain as solution, suspension or settle down. Organic substances constitute 40–70% of sludge.

Dry weather flow is the average amount of sewage flow in 24 hrs. It varies according to whether the sewage system is combined or separate, the time is day or night and the season is summer or winter. It is the indicator for amount of sewage.

Objectives of treatment of sewage

1. Protecting soil, water and marine life from sewage
2. To convert effluent of sewage to 'graywater' (nontoxic water free of pathogens) which can be disposed in a river or sea or reused for washing purposes.

Power of sewage

1. **BOD**: The biochemical oxygen demand is the amount of oxygen absorbed by a sample of sewage (at 20°C) in 5 days, indicator of organic matter and bacteria. If BOD > 300 mg/l, the sewage is said to be 'strong'.
2. **COD**: Chemical oxygen demand is the amount of oxygen absorbed after using an oxidizer (it is used where industrial pollution may have killed all the bacteria).
3. *E. coli* count.
4. Total suspended solids (signifiant if > 500 mg/l).

Overview of sewage treatment. Conventional sewage treatment may involve three stages, called primary, secondary and tertiary treatment.

- *Primary* treatment consists of temporarily holding the sewage in a quiescent basin where heavy solids can settle to the bottom while oil, grease and lighter solids float to the surface. The settled and floating materials are removed and the remaining liquid may be discharged or subjected to secondary treatment.
- **Secondary** treatment removes dissolved and suspended biological matter. Secondary treatment is typically performed by indigenous, water borne microorganisms in a managed habitat. Secondary treatment may require a separation process to remove the microorganisms from the treated water prior to discharge or tertiary treatment.
- **Tertiary** treatment is sometimes defined as anything more than primary and secondary treatment. Treated water is sometimes disinfected chemically or physically (for example by lagoons and microfiltration) prior to discharge into a stream, river, bay, lagoon or wetland, or it can be used for the irrigation of a

golf course, green way or park. If it is sufficiently clean, it can also be used for groundwater recharge or agricultural purposes.

Municipal method

Primary treatment

Sewage is screened for large objects (i.e. dead animals); these are treated as general waste and placed in a landfill/incinerated

↓

GRIT CHAMBER

A long passage where sewage is passed slowly, so that all the gravel and insoluble matter sediments

↓

PRIMARY SEDIMENTATION TANK

In the primary sedimentation stage, sewage flows through large tanks (at a velocity of 1–2 ft/min). The tanks are large enough that sludge can settle and floating material such as grease and oils can rise to the surface and be skimmed off. Sewage is separated into a homogenous liquid (effluent) and a 'sludge' that can be separately treated

↓

The sludge is processed by SLUDGE DIGESTER where it is 'self digested' in 2–3 weeks to form a dry, tarry manure and methane gas

Secondary treatment

Treatment of the effluent

There are several ways to deal with the effluent.

Trickling filter. Effluent is spread onto the surface of a deep bed made up of coke (carbonized coal) or limestone chips so that it 'trickles' through. Very soon, biological films of bacteria, protozoa and fungi form on the media's surface and reduce the organic content.

Activated sludge process. Effluent is throughly mixed with sludge from the final setting (to import bacteria), and *aerated* to help the bacteria grow and digest all the organic matter until it forms a *second* batch of sludge which is sent again to sludge digester. This process is much more effective in removing pathogens like Salmonella and Vibrios, and can convert ammonia to aerial nitrogen (through the denytrifying bacteria), but cannot deal with a sudden increase in sewage volume.

Biological aerated filters. Biological Aerated (or Anoxic) Filter combine the best of both processes. BAF usually includes a reactor (similar to activated sludge process) filled with a filter media. The dual purpose of this media is to support highly active bacteria that is attached to it and to filter suspended solids.

Rotating biological contactors. Rotating biological contactors (RBCs) are mechanical secondary treatment systems, which are robust and capable of withstanding surges in organic load. The rotating disks support the growth of bacteria and microorganisms present in the sewage, which breakdown and stabilize organic pollutants. To be successful, microorganisms need both oxygen to live and food to grow. Oxygen is obtained from the atmosphere as the disks rotate. As the

microorganisms grow, they build up on the media until they are sloughed off due to shear forces provided by the rotating discs in the sewage.

Secondary sedimentation

The final step in the secondary treatment stage is to settle out the filtered effluent in another tank for 2–3 hrs, where a 'secondary sludge' is formed. This sludge is rich in bacteria and some part of it is used in activated sludge process.

Tertiary treatment (effluent polishing)

This stage improves the quality of effluent before it is released into environment.

Filtration. Sand filtration removes much of the residual suspended matter. Filtration over activated carbon removes residual toxins.

Lagooning. Lagoons are highly aerobic man-made ponds and where, in the presence of suitable bacteria, effluent is digested overtime. Small filter feeding invertebrates such as Daphnia and species of Rotifera greatly assist in treatment by removing fine particulates.

Constructed wetlands. Constructed wetlands provide a high degree of aerobic biological improvement and can often be used instead of secondary treatment for small communities.

Nutrient removal. Waste water may contain high levels of the nutrients nitrogen and phosphorus. Excessive release to the environment can lead to a build up of nutrients, called **eutrophication** → overgrowth of weeds, algae, and cyanobacteria (blue-green algae) → decomposition of the algae by bacteria uses up so much of oxygen in the water that most or all of the animals die → which creates more organic matter for the bacteria to decompose. The **activated sludge process** removes most nitrogen very well (and liberates nitrogen in air). Phosphorus may be removed by specific bacteria (polyphosphate accumulating organisms) or by precipitation with salts (ferric chloride, alum, lime).

Other methods to deal with raw sewage

1. Outfall in multiple points of deep sea—Not recommended
2. River outfall—An age old practice, but contraindicated
3. Oxidation pond—The sewage from a small community may be treated in a pond consisting of anaerobic + aerobic bacteria + algae with adequate sunlight and air. The aerobic bacteria oxidize the organic matter into CO_2, which is used by the algae to grow; again, the algae will release oxygen during photosynthesis, the same oxygen which is used by bacteria
4. To be released in farmland for 'sewage farming' (which may cause diseases like worm infestation or neurocysticercosis).

NOISE

"Wrong sound at wrong place or time". The word 'noise' derives from latin *nausea*, a sickness.

Measurements of sound

You already know of frequency (η), wavelength (λ) and amplitude (A) of sound. There is two other aspects of sound pertinent to sound perception. One is the

intensity (I) - better called **sound pressure level**. Intensity is rate of flow of sound energy per unit area. Thus its unit is Watt/m^2. Our ear can detect a sound as little 10^{-12} Watt/m^2 (0.00024 dyne/cm^2/s) intensity at 1000 Hz, which is called the **threshold of hearing**. Again, sounds greater than 1 Watt/m^2 are painful to the ear. In view of this large range of audibility, a logarithmic scale is most appropriate to measure intensity.

Bel. The difference in intensity level of two sound of intensities I and I_0 is defined as Log_{10} (I/I_0) Bel. Because bel is an impractically large unit, a decibel has been devised which is 0.1 bel. Thus difference in intensity level of two sound I and I_0 is 10 Log_{10} (I/I_0) decibel.

Obviously, a tenfold increase in intensity will create a difference of 10 dB, and 100 times increase comprises 20 dB and so on. By International standards, I_0 is chosen to be the threshold of hearing = 10^{-12} W/m^2 at a frequency of 1000 Hz. Then, one bel is the intensity of sound which is ten times the threshold of hearing. It is obvious that the intensity of threshold of hearing is 10 Log (10^{-12}/ 10^{-12}) = 0 dB and intensity of threshold of pain is 10 Log ($1/10^{-12}$) = 120 dB.

Table 5.10. Levels of sound encountered in daily life

Sound	Decibels	Sound	Decibels
Threshold of hearing	0	Traffic	70
Whisper	20	**Recommended maximum**	85
Conversation	65	Pain	120

However, our tolerance of sound depends not only on its intensity, but the context and setting as well. We tolerate sounds of such intensity while in a train that feel unbearable in our bedroom. It seems that we 'expect' trains, markets, hospital wards and public places to be noisy.

Effects of noise

Auditory

1. *Fatigue* appears with noise of 90 dB and greatest at 4000 hz. There is also associated whistling and tinnitus in ears.
2. Deafness: Temporary deafness (by a large blast of sound) lasts < 24 hrs. Chronic exposure to noise (> 100 dB) may cause noise-induced hearing loss.[232] A comparison of Maaban tribesmen, who were insignificantly exposed to transportation or industrial noise, to a typical US population showed that chronic exposure to moderately high levels of environmental noise contributes to hearing loss.[233] Sound greater than 160 dB ruptures the Tympanic membrane.[234]

Nonauditory

1. Annoyance—A 2005 study by Spanish researchers found that in urban areas households are willing to pay approximately four Euros per decibel per year for noise reduction.[235]
2. Interference with speech—Noise also makes animals (that includes humans) communicate louder, which is called Lombard vocal response.[236]

3. Loss of efficiency and sleep, ↑ BP and ↑ HR; high noise levels can contribute to cardiovascular effects and exposure to moderately high levels during a single 8 hrs period causes a statistical rise in blood pressure of and vasoconstriction leading to the increased blood pressure noted above as well as to increased incidence of coronary artery disease.[237]

Control

City planning

1. Separate industries, railways, roads from residential zones
2. Green belts between roads and residence (15 m)
3. Widen streets.

Vehicle control

Use of noise barriers, limitation of vehicle speeds, alteration of roadway surface texture, limitation of heavy vehicles, use of traffic controls that smooth vehicle flow to reduce braking and acceleration, setting up no horn zones and tire design.

Architecture

The best way to protect citizens from noise is to set up residential zones far apart from transport zones. If this is not plausible, sound proofing will help.

Personal

Rotating duty of workers in a noise dense industry, use of ear plug and muff near high decibel devices, regular audiometric check up of workers.

Legislation

Noise laws. In many developing countries, noise is regarded as a 'nuisance' rather than something that 'pollutes' the environment. Speaking of developing countries, Indian law limits a certain maximum of noise (85 dB) but does not take into account the effects of political meetings and FM radio in public transport, which manage to be annoying while still maintaining a level lower than 85 dB.

Worker's compensation. Must include sound induced damages.

RADIATION

Unit of radiation

Table 5.11. Units of radiation

Quantity	Conventional unit	SI unit
Amount of radiation (number of nuclei breaking per second)	Curie	Becquerel (disintegration /second)
Exposure (number of ions produces in the air near radioactive substance)	Roentgen	Coulomb/kg of the medium around radioactive substance

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Quantity	Conventional unit	SI unit
Absorption	Rad	1 Gray = the amount of ionizing radiation that rises the energy in 1 kg of the medium by 1 joule
Dose equivalent (absorption × modifying factors)	Rem (for X-rays and γ-rays, Rem and Rad are equal)	Sievert

Types of radiation

1. Ionizing—γ-ray (an electromagnetic wave, has deep penetration into living tissue), β particles (electrons) and α particles (the helium nucleus, penetrates very little but causes maximal damage), X-rays (another electromagnetic wave).
2. Nonionizing—UV ray, visible light, infrared waves, microwaves and radiowaves (all part of electromagnetic wave spectrum).

Sources

1. Cosmic rays—Usually of very little concern to people living over the surface of earth, as the atmosphere protects us well; however, jet pilots have to be careful.
2. Terrestrial radiation—Radioactivity is present everywhere (and has been since the formation of the earth). According to the IAEA, one kilogram of soil typically contains the following amounts of the following four natural radioisotopes 40K, 226Ra, 238U and 232Th, totaling 445 Bq on average.[238] Radioactive atoms from soil may reach human body through three main roots—crops, animal flesh and milk.
3. Air (radon gas, which is a natural disintegration product of radium).
4. Internal—Radioactive elements in body (specially 40K and 14C are commonly found in living tissue).
5. X-rays (0.02–3 rads/film)—Harms the *personnel* doing the X-ray more than the patient (the patient is exposed only once at a time, but for the operator, its his job).
6. Nuclear fallout, nuclear power plants, radiotherapy departments.

Effects

Acute radiation sickness

Radiation sickness is generally associated with acute (a single large) exposure. Nausea and vomiting are usually the main symptoms. The onset of symptoms is an indicator of the absorbed dose.[239]

- Nausea and vomiting generally occur within 24–48 hrs after exposure to mild (1–2 Gy) doses of radiation.
- Moderate (2–3.5 Gy of radiation) exposure is associated with nausea and vomiting beginning within 12–24 hrs after exposure, with fever, hair loss, infections, bloody vomit and stools, and poor wound healing.

- Nausea and vomiting occur in less than 1 hr after exposure to severe (3.5–5.5 Gy) doses of radiation, followed by diarrhea and high fever in addition to the symptoms of lower levels of exposure.
- Very severe (5.5–8 Gy of radiation) exposure is followed by the onset of nausea and vomiting in less than 30 minutes followed by the appearance of dizziness, disorientation, and low blood pressure.

Delayed effects

Leukemia, cancers, loss of lifespan, sterility, familial diseases, teratogenicity.

Maximum limits of tolerable radiation

- Outer space (background) radiation < 0.1 rad/year
- Man made source < 5 rad/year
- Genetic dose (radiation to reproductive organs) < 5 rem over a period of 30 years
- Gross α activity in water > 0.1 Bq/l
- Gross β activity in water > 1 Bq/l.

Protection

1. Proper X-ray machines, dose reduction (i.e. reducing unnecessary X-ray examinations).
2. 0.5 mm lead coat for workers.
3. A film badge/dosimeter shows the accumulated radiation since the machine was last charged.
4. Periodic check ups of workers, division of working hrs.
5. Stop nuclear tests and avoid a nuclear war (its time somebody listens to this advice).

STERILIZATION AND DISINFECTION

Disinfectants are agents that destroy/inhibit growth of *pathogens* and thus break the chain of transmission. *Antiseptics* are disinfectants in low concentration used in vitro (i.e. over human body).

Types of disinfection

1. **Concurrent**—Running through the course of disease (disinfection of vomitus, feces, gloves, etc.).
2. **Terminal**—Carried out after competition of infective period (room fumigation, incineration of soiled bedding).
3. **Prophylactic**—To stop development of infection (wound care, disinfection of drinking water, etc.).

The *contact period* is the minimum time needed for a disinfectant to be effective. You have known most of the procedures of disinfection from your microbiology classes. The following table lists common disinfectants used in day-to-day hospital and primary health care setting.

Tests for efficiency of a disinfectant

Phenol coefficient. It is the (*minimum dilution* of a disinfectant to sterilize a fixed volume of substance)/(minimum volume of same concentration of phenol to do the same). If this is greater than 1, the disinfectant is effective.

Riddle walker ratio. It is the (minimum dilution of 1 ml of a disinfectant to sterilize a media)/(the same to do with phenol). **Chick Martin ratio** is same as Riddle walker ratio, only the media is 1% stool.

Table 5.12. Common disinfectants

To disinfect	Use
Water (see water treatment earlier)	Chlorine (for 1 hr) Ozone
Rooms	Formaldehyde (with or without $KMnO_4$)
Skin	Tincture iodine/ Betadine (with or without alcohol)/Chlorhexidine/Ethanol
Blood/serum spill over table or floor	NaOCl (which is *virocidal*)
Wounds	Chlorhexidine + cetrimide (SAVLON)
Serum/vaccines	Vaccine bath/Seitz filter
Plasma	H_2O_2
Floor of room	Lysol/cresol (PHENYL)
Test tubes/corks/glass slides	Flaming
Inoculating loop/forceps/spatula/ needle	Red heat
Glass syringe/pipette, powder, petroleum jelly, vaseline, paraffin,cotton	Hot air oven 160°C for 1 hr
Rubber tubes and gloves/ Ryle's tube	Boiling and steaming
Latex/Linen	Washing and autoclaving
Sutures	Glutaraldehyde and Lysol
Instruments with a lens	Glutaraldehyde (2%)
Disposable syringe/catheter/mask/ petri dishes	γ -ray/ethylene oxide
Prosthetics	Ethylene oxide
Milk	Pasteurization
Water	Chlorine/ozone
Viral vaccine preparation	Propriolactone, Detergents (subunit vaccine), Methanal

Contd...

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To disinfect	Use
Sharp instruments	Chlorhexidine
Instruments (nonsharp)	Boiling for 20 minutes
Feces/urine	Equal volumes of one among 5% bleaching powder/10% phenol /5% cresol/1:4 lime for 1–2 hrs
Bedpans/urinals	2.5% cresol for an hr; bedpans could also be boiled
Sputum	5% cresol for 1 hr Collecting in paper hankeys and incineration
Catgut/plastic disposables	γ-ray

Example: Sterilizing a room

In a town. All windows are closed, then a bowl containing 500 ml 40% formalin per 1000 ft^3 of the room is placed over a heater (which automatically shuts off after 15 minutes) → Room closed → Opened after 24 hrs → NH_4OH (ammonium hydroxide) soaked bandage is thrown into absorb residual formalin → Another 24 hrs wait → Open room.

In a village with no electricity. All windows are closed, then 500 ml 40% formalin/1000 ft^3 + 375 g $KMnO_4$ is placed in a bowl → Formate (HCOOH) released in air → Opened after 24 hrs → NH_4OH soaked bandages thrown → 24 hr wait → Enter room.

$$KMnO_4 \rightarrow K_2O + Mn_2O_3 + 5[O]$$
$$HCHO + [O] \rightarrow HCOOH$$

OCCUPATIONAL ENVIRONMENT

The occupational environment is the "sum of external environmental influences which prevail at work which have a bearing on health of the worker". Because, in modern life, we spend as much time in our jobs as in our homes, the 'occupational environment' is as important as domestic environment.

Since 1950, the International Labor Organization and WHO have agreed on a common definition of **occupational health.**

The promotion and maintenance of

- The *highest degree of physical, mental and social* well-being of workers
- The prevention of illnesses caused by their working conditions
- The protection of workers in their employment from risks resulting from factors adverse to health
- The placing and maintenance of the worker in an occupational environment adapted to his physiological and psychological capabilities

and, to summarize, the *adaptation of work to man and of each man to his job.*

—WHO/ILO joint committee on occupation health, 1995

Ergonomics is, however, *fitting the job to the worker* (i.e. an 'ergonomic keyboard' rather than 'ergonomic environment').

Aims of occupational health services

1. Promote the highest level of health in workers.
2. Prevent occupational diseases (primary, secondary and tertiary prevention).
3. ↑ *productivity* of the individual; a helpful environment is always a boost to performance.

Many governments have realized that the money saved from not building good occupational safety standards will ultimately be drained by employee's illness and social security insurance. In addition, a sick worker equals a nonproductive worker, which adds to the burden of economic loss. Lastly, exposure of bad working environment and immoral labor practices in mass media is such negative publicity which no corporation can afford.

Psychosocial environment

Maladaptation to an alien psychosocial environment (i.e. hostile colleagues/grumpy boss/unsatisfactory job) may result in

1. Behavioral diseases—Anxiety, depression, drug abuse, absenteeism, alcoholism.
2. Psychosomatic disorders—Fatigue, peptic ulcer, hypertension, heart disease.

Physical environment

Table 5.13. Physical hazards of occupation

Agent	Ideal	Adverse effects
Heat	69–80°F	Syncope, cramp, 'stroke' (not the neurological kind of stroke), fatigue, prickly heat, burns
Cold		Trench foot, frostbite, chilblains
Light	Uniform, adequate, not flickering, no sharp contrasts, no glare	Low light: Eye strain, pain, headache, accidents, Miner's nystagmus Excess light: Discomfort, fatigue, glare accidents
Noise		Deafness, fatigue, BP, arrhythmia
UV light		'Welder's flash' keratoconjunctivitis
Radiation	Maximum limit 5 rem/year for whole body	Acute burns, dermatitis, skin cancer, cataract, teratogenicity, leukemia
Vibration		↑ vascular sensitivity and easy bleeding (happened in those who work with a drill)
Long working hrs in fixed posture		Back and shoulder pain (this is a relatively new hazard brought into India by IT companies and call centers)

Chemical environment

Table 5.14. Chemical hazards of occupation

Agent	Disease	Industries
Coal dust	Anthracosis	Coal mine
Silica	Silicosis	Gold, silver, mica, pottery, steel, slate
Asbestos	Asbestosis Lung carcinoma	Tiles, talc, cement, fireproof garments
Iron	Siderosis	Mines, steel, welding
Cane (bagasse)	Bagassosis	Sugarcane, paper, board
Cotton dust	Byssinosis	Textiles
CO_2, CO, HCN, Methyl isocyanide	Asphyxia, fatigue, emphysema	Coal workers, traffic police, fertilizer factory workers
Metal solvents	Cancer, dermatitis	Mines, chemical industries
Pesticides	Organophosphate poisoning	Farmers

Occupational dermatitis

Causes

Irritants – like acids and sensitising agents which are particular to a person (i.e. some particular lotion or shampoo may cause dermatitis in a person). Those working at beauty parlors, catering, motor repair and printing are especially susceptible to dermatitis.

Prevention

1. Pretesting with the suspected antigens
2. Protection by using gloves
3. Hand washing
4. Periodic examination.

Lead poisoning

Lead is used in paints, oil, battery, lead pipe, food cans, fruit ripener, vermillion, etc. The usual compounds that cause toxicity are lead carbonate, lead oxide and lead arsenate. Tolerable blood level of lead is 25 μg/dl; symptoms appear at 70 μg/dl. Lead inhibits synthesis of hemoglobin so that Hb precursors, aminolevolinic acid and porphyrins accumulate in blood and spill into stool and urine. Lead poisoning is a notifiable disease and subject to compensation.

Routes

Inhalation from fumes; ingestion (from lead pipes carrying water), contact.

Clinical course

Early signs

1. Blue lining of gum (Bartoneal line) which is a deposition of PbS
2. Basophilic stippling of RBC (Fig. 5.22)

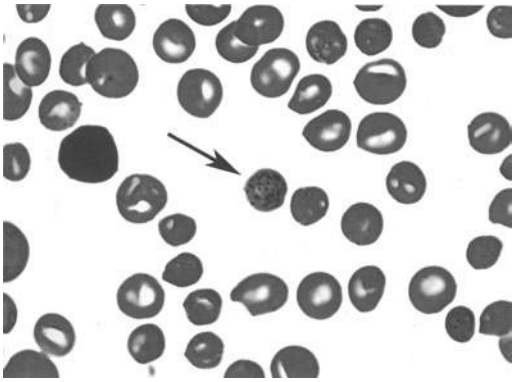

Figure 5.22. Compare the dotted (stippled) RBC with a normal one

3. Retinal stippling.

Late signs

- Abortions (repeated)
- Loss of bone density
- Abdominal colic, constipation
- Wrist and foot drop
- Lead encephalopathy
- Fatigue (due to anemia)
- G failure and mental retardation in children
- Hypertension
- Impotence, irregular menstruation.

In addition, *organic* lead compounds cause sleepiness, dizziness, fatigue, insomnia, psychosis.

Diagnostics

- Urine coproporphyrin > 150 μg/dl
- δ-ALA in urine > 5 mg/l
- Pb in urine > 0.8 mg/l
- Blood lead > 70 μg/dl
- Basophilic stippling.

Prevention

Primary.

1. Moistening devices to prevent inhalation
2. Lead masks (to prevent inhalation of lead from fumes)
3. Good housekeeping—Keeping every surface free of dust all the time
4. Regular shift change of workers
5. Abolition of lead pipes and containers
6. Avoid eating in direct vicinity of workplace
7. Vigil over children (so that they don't suck on painted toys)
8. Substitution of lead compounds with nontoxic ones.

Lead limit in air is $2mg/10m^3$

Secondary

Frequent health check up of workers.

Dust pneumonitis (Pneumoconiosis)

Dust is composed of coarse particles + *aerosols* which are suspended in air for a prolonged time. Of the aerosols, the ones < 100 μm are inhalable; of these, those < 25 μm reach lower RT (*thoracic fraction* of aerosols). Now, only those particles < 10 μm get in alveoli (*respirable fraction*) and deposit, but particles less than <0.5 μm do not deposit as they are expired out.

Table 5.15. Pneumoconioses

	Silicosis	**Anthracosis**	**Asbestosis**	**Byssinosis**	**Bagassosis**
Agent	SiO_2	Coal	Fibrous silicates (crocidolite)	Cotton dust, hemp, etc.	*Thermo-actinomy-cetes sacchariae* in sugarcane dust
Environ-mental assess-ment	Size selective dust sampling in breathing zone (personal sampler)		Personal sampler Static sampler X-ray, lung function tests, sputum for asbestos body		
Limit	0.5–5 mg/m^3			2–6 mg/m^3	
Radio-logy	Coales-cing nodules in upper lung fields (‘*snow storm*’)	Small round opacities → progressive massive fibrosis	Interstitial fibrosis + basal pleural thickening (‘*ground glass*’)		Mottling in lungs
Clinical	Chest tightness, incapacity, respiratory failure, cor pul-monale		Fine basal rales and creps; dyspnea, cyanosis, clubbing, broncho-carcinoma, mesothe-lioma	Chest tightness on 1st working day in the week → permanent air flow obstruction	Breath-lessness, cough and fever

Contd...

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	Silicosis	Anthracosis	Asbestosis	Byssinosis	Bagassosis
Check ups	Preplacement examination: Respirartory system, history of tuberculosis, X-ray and LFT 1 yearly medical examination				
Control	Dust control Personal protection	Wetting, Ventilation Personal protection Air-conditio-ning for excavation operators	Use safer asbestos (chrysolite, amosite) Substitution of other insulants (glass, etc.) dust control	Enclose dusty operations Ventilation Personal protection	Keep moisture content > 20% (so that the organisms doesn't fly around in air) and propionic acid spray exhaust ventilation and wet processes for dust control

Occupational cancers

Occupational cancers arise after prolonged exposure, often earlier than cancers in general population, may develop even after cessation of exposure and is remarkably localized in anyone population.

Table 5.16. Occupational cancers

Agent	Industry	Organ affected
Mineral oil, tar, pitch, dyes, X-ray	Coke oven, gas worker, tar distillers, oil refiners, dye markers, road makers	Skin
Ni, Cr, asbestos, tar, radiation	Gas, asbestos, Ni, Cr, Radioactive mining	Lung
Auramine, β-naphthylamine, benzidine	Dye, rubber, gas, electric cables	Bladder
Benzol, radiation	Radioactive mining	Leukemia

Biological environment

Table 5.17. Biological hazards of occupation

Occupation	Hazard
Agriculture, animal husbandry, forestry	Arbovirus, hookworms, leishmaniasis, malaria, schistosomiasis, Farmer's lung
Construction, sewers mining	Coccidioidomycosis, hookworm, histoplasma, leptospira, tetanus, wound sepsis
Meat and fish	Bovine tuberculosis, brucella, fungal infections, Q fever

Contd...

Contd...

Occupation	Hazard
Poultry	Newcastle disease, avian flu
Hair, hide wool	Anthrax, Q fever
Veterinarians	Tuberculosis, brucellosis, leptospira, ornithosis, Q fever, rabies
Doctors, nurses laboratory workers	Viral hepatitis, tuberculosis, HIV

Farmer's lung

Although it is not acquired in factories, Farmer's lung is the largest occupational disease of India. It is caused by Micropolyspora faeni growing in > 30% moist hay, hemp, siscal. Repeated attacks cause inevitable pulmonary fibrosis and cor pulmonale.

Sickness absenteeism

As production technology improves, absenteeism tends to counter the benefits. The major causes of sickness absenteeism, is surprisingly, not medical at all.

1. **Social**: To attend the marriage of cousin's elder sister's younger nephew
2. **Economic**: To get sickness benefits from the company
3. **Medical**: Well, sometimes they really fall ill
4. **Others**: Addiction, etc.

Prevention

1. Good management and HR department
2. Preplacement examinations (whether the worker really has a chronic disease)
3. Ergonomics, so that workers don't feel *too* fatigued after a days work.

Prevention of occupational diseases

Health promotion

Premedical examination

It is done at the time of employment. The purpose is to fit the job to the worker, or if it can't be done, reject the worker. It includes

1. Medical, family, occupational and social history
2. Physical examination
3. Risk factors (COPD, anemia, hypertension)
4. Special tests (chest X-ray, ECG, vision, urine and blood).

The premedical examination also gives baseline data for future comparisons of the same worker, and it also serves as a legal document for the worker.

Periodical examination

Depending on the industry, the frequency of periodical examinations will vary. In factories dealing with chromium, even *daily* examinations are required.

Health education

It is necessary to train every worker on how to do his job well as well as how to save himself. An occupational disease is a chronic burden on the worker, the

employer and the state in general. An *accident* usually costs much more to the employer than the worker. Thus, educating workers are economically beneficial.

Environment

1. Sufficient supply of safe and wholesome water
2. Sanitary food vendors
3. Sanitary latrines, separate for men and women
4. Safe disposal of garbage and sewage
5. General cleanliness: The walls, ceilings and passages should be painted with water washable paint, washed at least once in six months and painted each three years; dust should be cleared immediately
6. Space: 500 ft^3/worker under 14 feet
7. Light: Minimum 6–12 foot candles lighting should prevail; 50–75 foot candles illumination is necessary for skilled work
8. Ventilation and control of temperature
9. If the organization provides housing, the housing complex should be separate from the working complex.
10. Regular environmental monitoring.

Mental health

The industry should encourage an atmosphere of support, innovation and excellence. Performance based incentives are a great way to keep up the morales of workers.

Welfare[240] facilities

Insurance for disability, maternal health and family welfare service, crèche (a room for infants to play while their mothers are at work) service if > 30 women are working, first aid facilities, recreation facilities, a good human resource department.

Nutrition

The Indian Factories Act asks for a canteen if number of workers exceed 250. The aim is to provide balanced diet at low cost in sanitary control. If the worker carries his lunch, provision should be made to store it in some sanitary place.

Stop population growth

It is rare to find a worker who does't know that the gas in his factory is killing him. It is only his obligation that he has to work in that very place, because if he doesn't, there will always be one to replace him. In most western societies, employees are the assets of employers because it has hard to find a skilled worker. In our country, however, because there's always somebody to replace you, employers have an unending choice of hire and fire, and people work as if there's no tomorrow. Labor is dirt cheap in India, and this is what multinational companies exploit. Their message is simple—"If you don't like inhaling lead, get out of here, we will find someone who will do it for less pay than you". Thus Indian workers have to persist clinging to their jobs, and MNCs get richer as usual.

Specific protection

Immunization

Immunization against HBV, rabies, tetanus, anthrax and other occupational diseases may be appropriate.

Technical measures

1. Substitution of harmful agents (i.e. white P by phosphorus sequisulfide, which eliminated 'fossy jaw'[241]
2. Automation (as to avoid direct contact with harmful substances and make robots do the dirty work)
3. Moisture and wet drilling methds to combat dust
4. Enclosure of harmful machines and/or isolation of workers
5. Local exhaust ventilation
6. Personal protective equipments (lead suits, masks, ear plugs, helmets, shoes, aprons, gloves, etc.)

Early diagnosis and prompt treatment

Medical care

The Employees State Insurance provides medical care not for the worker only but his family. The periodical medical examination may serve as a tool for diagnosis.

Supervision of working environment

The physician must visit the factory frequently and have a hands on experience.

Disease notification

The Factories act and the Mines act require notification of occupational diseases (i.e. pneumoconioses).

Laws

The Indian factories act, 1948.

Scope

A *factory* is a establishment with

> 10 employees in a factory using electrical power

> 20 employees in a factory using no electrical power

It applies to whole of India except J and K.

Health, safety and welfare

The following provisions are to be made in any factory.

1. **Space**- 500 ft^3 per worker under the height of 14 ft
2. Water supply
3. Waste treatment
4. **Ventilation**
5. Control of temperature
6. Maximum bearable **weights** for men, women and children to be set by state Government
7. Hand and personal equipment washing

8. First aid
9. Canteen if > 250 employees are working
10. **Creche** where > 30 women are working
11. **Safety officer** where there are > 1000 employees, **welfare officer** if > 500 employees.

Employment age

People < 14 are barred from working. Adolescents (14–18) can work only after surgeon's clearance, and within 6 am to 7 pm. They cannot work in hazardous processes.

Hours

1976 amendment prescribed a *maximum 60 hr week*, with maximum 12 hrs per day including rest. *Rest* of ½ an hr is permissible after 5 hrs of work (or 4.5 hrs for adolescents).

Leave with wages

A leave is due every 20 days of work, and may be accumulated upto 40 days.

Occupational diseases

On report of any occupational disease, the factory management must carry out.

1. Notification
2. Health surveys to identify disease source
3. Control measures.

Hazardous processes

A Site Appraisal Committee is entitled to check factories and identify hazardous factories.

The Indian Working mother

1. Maternity leave for 135 days (or 6 weeks for abortion)—ESI act
2. Gets a crèche if > 30 women are working (Factories act)
3. Gets lesser weight to bear (Factories act)
4. Gets lesser working hrs (Factories act)
5. Does not have to go into mines (Mines act)

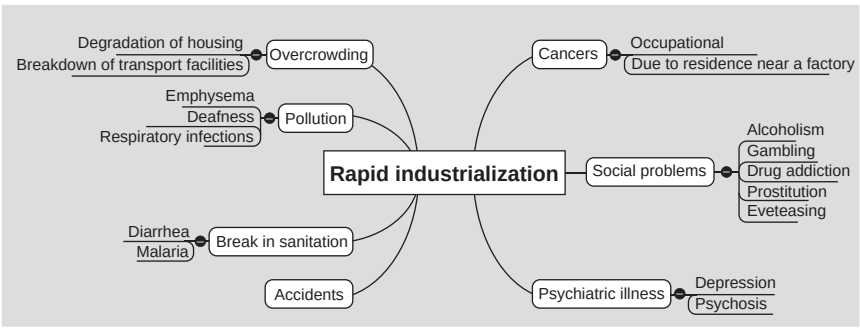

Figure 5.23. Problems of rapid industrialization (the situation India is going through)

6. Does not have to work in radioactive factories (Factories act)
7. Doesn't have to work in 7 pm to 6 am (Factories act)
8. Gets antenatal and postnatal services from ESI.

DISASTERS

The first reported disaster is when God flooded the earth and Noah set out to survive in his ark. Because the troposphere (the lowest layer of atmosphere) is very restless, and the core of this planet is still liquid, the surface of earth is disaster prone. We note the large disasters, but to each large disaster, 20 smaller ones go underreported.

And if it soothes you in anyway: Global warming is a disaster in making.

A disaster is a

- Damage
- Ecological disruption
- Casualty
- Deterioration of health services.

that warrants an extra ordinary response and requires help from *outside* the community.

—WHO strategy and approcahes to humanitarian action

Classification

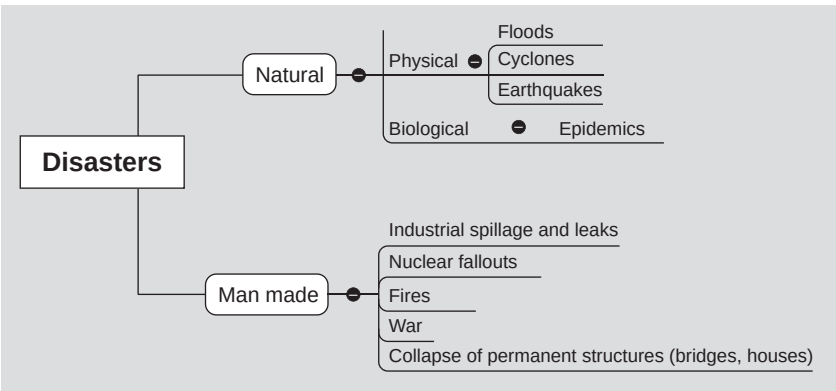

Figure 5.24. Types of disasters

Disaster cycle

The situation is usually like this: A certain area is hit by a storm ↓ everyone rushes into the area and starts relief and restoration of facilities. This is the **response phase**. It would have only been better if this enthusiasm stayed on and people thought to put up a contingency plan for the next disaster, which is the **mitigation and preparedness** phase.

Figure 5.25. Disaster cycle

Impact

Table 5.18. Effects of disaster

Effect	Earthquake	Winds	Tidal waves/ flash floods	Slow floods	Landslide	Volcanoes
Death	+++	+	+++	+	+++	++
Injuries	+++	++	+	+	++	+
Communicable diseases	+	+	+	+++	+	+
Damage to health facilities	+++	+++	+++ Localized	++ (only equipment)	+++	+++
Damage to water systems	+++	+	+++	++	+++	+++
Food shortage	+	+	+++	+++	+	+
Major population movement	+	+	+++	+++	+++	+++

Nonliving

In addition to destruction of physical structures and immediate loss of human life and cattle, a disaster also destroys the sanitary facilities, medical facilities of the region and people have to often take shelter in relief camps. The lack of personal space, inadequate food, breakdown of hygiene and contamination of water sources (i.e. in a flood) often cause insurgence of epidemics (diarrhea, respiratory infections, vector borne diseases, zoonoses), malnutrition and posttraumatic stress disorder.

Objectives of management

1. Prevent deaths of starvation
2. Prevent epidemic breakout
3. Restoration and rehabilitation of the affected
4. Prepare for the next disaster.

Situation analysis

It is very useful to carry out a quick spot survey to assess the damage to humans and habitat.

Immediate response

1. Search and rescue—The first few hrs should focus on rescuing the maximal number of people rather than full medical attention to everybody.
2. Field care—First aid only (try only splints, no plasters or reduction immediately).
3. Assess task on hand (the final number affected will usually be *twice* the number encountered in first hr).
4. Coordinate local resources (i.e. find a school where the people may be shifted, or an owner of a truck/ambulance).
5. Inform the authority, bring in health workers from nearby sites, organize mobile camps and inform NGOs.

Triage

"Management based on likelihood of survival in the face of scarce resources." Triage attempts to focus medical attention who need it from those where it can be delayed.

Table 5.19. Color coding of patients according to chances of survival

Red	Significant benefit on immediate hospitalization
Yellow	Less urgent need
Green	On the spot management
Black	Dead/moribund/questionable benefit on hospitalization

Mortuary

Promptly remove the dead after identification (if possible), and either leave it to the family or arrange a rudimentary cremation. Dead bodies are not a great health hazard, except their appearance.

Relief

Acquire items → store → distribute equitably (women and children 1st).

Short-term response

Renovation of structures

This is essential to begin the relief service. A few rooms and some tables and chairs will suffice. In a few days, you may also have to start immunization, antenatal/postnatal care.

Episodic vaccination against typhoid, cholera, tetanus is often a political move to woo people than for medical reasons; these vaccines are multidose and not very effective.

Nutrition

Begin with *survival* ration (1200 kcal + 30 g protein/day) for 3–4 days until everybody is rescued. Provide *cooked* food in 1 packet per person

↓

↓

After everyone is rescued, make community tents and start maintenance ration (1600 kcal, 45 g protein) for 1 week

↓

Ensure that everyone is rescued and you have enough supplies of food. Now start the regular ration (2100 kcal, 55 g protein).

1. Cooked food/mixtures of food are preferred than raw ones (otherwise people tend to sell the food); you can also distribute dried skimmed milk (which is too impalatable to be sold)
2. Promote breastfeeding even in the worst of times
3. Special arrangement for women, children and elderly
4. Snacks for children (so that they at least agree to your impalatable diet for the incentive of a tasty snack).

Once you start regular ration, you simply can't go back to earlier rations.

Food fortification during emergency. Fortifying foods such as peanut butter sachets and Spirulina have revolutionized emergency feeding because they

- can be eaten directly from the packet
- do not require refrigeration or mixing with water (water is scarce in disaster situations)
- can be stored for years
- can be absorbed by extremely ill children.

Water

1. Find a potable water source
2. Chlorination: Use double pot method (see earlier) for 0.7 mg/l residual chlorine in water (2.5 g bleaching powder/1000 l); it is important to disinfect ALL water sources within 2 km of tent
3. Protect these water sources (especially from sewage that accumulates in the relief camp)
4. In dire conditions you may need to *ration* water.

Environment

1. Sanitation—Make a trench latrine (see earlier) which can be used by many
2. Intensify vector control.

Epidemiological surveillance

Keep an eye on overcrowding, sewage failure, abundant vectors, population displacement, loss of food hygiene, disruption of routine health programs.

Reestablish health care

Start, in slow progression, the services of immunization, vitamin A prophylaxis, growth monitoring; reinstitute vector control measures, recruit fresh village health guides and try to make up for lost time.

Rehabilitate

Create jobs for these people, and the most rewarding job for them is to build their own village back. Start with food for work scheme (which is more immediately gratifying), and gradually change to *money* for work.

Coordinate other sectors

1. Pool resources from any source available
2. Train local people for rescue and community care.

Mitigation and preparedness

A better prepared community suffers less from a disaster than a nonprepared one. IN most nonprepard communities the picture is this (Fig. 5.26):

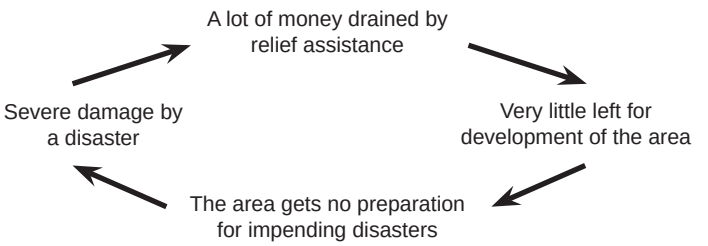

Figure 5.26. Funds tend to flow after disaster than before it

This reliance on relief, rather than preparedness, is a vicious cycle, and to break this cycle is successful mitigation.

Vulnerability assesment

Just as the epidemiologic triangle describes host vulnerabilities to disease, communities too vary on their vulnerability to disasters. Identify hazardous processes, possible incident scenarios, vulnerable population early and estimate the impact of a possible disaster.

Policy development

To prepare a laid down document for future course of accountable action, bottom up strategies, community involvement and intersectoral coordination.

Prevention of occurrence (Contingency plan)

1. Disaster management cell
2. Proper location of rescue shelters
3. Proper constructions (i.e. houses in earthquake prone zones should be built of very light material)
4. Prior identification of potable water sources
5. Public awareness programs
6. Teaching schoolchildren about disaster management
7. Coordinating information system on weather, earth movements, sea levels, human movements in that location
8. Timely forecast and warning systems for disasters.

Emergency preparedness

The goal is to be prepared for any calamity that might strike and rehearse everything to be done.

- Resource allocation—To identify and make inventory of available resources beforehand.

- Training the community on what to do when disaster strikes.
- Ensure one emergency kit in every house.
- Mock drills (rehearsals) of disaster.

(MEDICAL) ENTOMOLOGY

Entomology is the study of arthropods. Wonder what they have got do with public health?

- They act as vectors to spread diseases
- They may themselves cause disease (lice, scabies mite)
- They destroy crops and reduce food production.

Vector dynamics

A 'vector' is an organism which transmits disease

1. By inoculation into or through the skin/mucous membranes by biting
2. By depositing the agents on skin/food fomites.

Types of transmission

1. Direct contact—Scabies, pediculosis (lice).
2. Indirect contact—Scabies and lice may spread through fomites.
3. Mechanical transmission—Houseflies transport many pathogenic agents over their body.
4. Biologic transmission—The pathogenic agent spends some part of its lifecycle inside the vectors; most agents are carried by only a single generation of the vector. But **tick typhus** infects the progeny of ticks from their mother, which is known as transovarian transmission. Again, scrub typhus may affect ticks from one developmental stage to another, known as trans-stadial transmission.

Extrinsic incubation period

The time required for the infective form to develop within the vector (i.e. sporozoites of Plasmodium develop inside mosquito in about 10 days).

Vector 'infestation'

This is distinct from a parasitic infestation. A vector is infested when it lodges and multiplies in a surface, be it body or clothing, etc.

Control of (arthropod) vectors

Integrated pest management

Integrated pest management (IPM) is "a pest control strategy that uses a variety of complementary strategies including: Mechanical devices, physical devices, genetic, biological, cultural management, and chemical management. These methods are done in three stages: Prevention, observation, and intervention. It is an ecological approach with a main goal of significantly reducing or eliminating the use of pesticides while at the same time *managing pest populations at an acceptable level*".[242]

For their leadership in developing and spreading IPM worldwide, Dr. Perry Adkisson and Dr. Ray F. Smith received the 1997 World Food Prize.

Environmental control

This offers best and permanent results if properly applied. It includes source reduction (destroying habitats and breeding grounds of vectors), proper drainage of water, intermittent irrigation, proper waste disposal (so that flies do not have access to waste), piped water supply, intensive health education, etc.

Chemical control

They are efficient and cost-effective, but prone to resistance[243] and not very eco-friendly. More biodegradable alternatives (i.e. methoxychlor) have to be found.

Biological control

1. Larvivorous fish (Gambusia affinis, Lebister reticulatus) (Fig. 5.27)

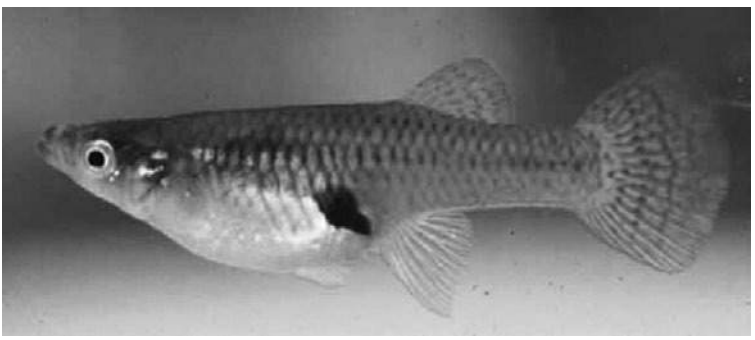

Figure 5.27. The larvivorous fish, Gambusia

2. Bacteria (Microsporidia, Coelomomyces, Bacillus thuringiensis
3. Exotic natural enemies of insects (hydra, flatworms).

Genetic control

Sterile male technique, cytoplasmic incompatibility–are still within laboratories.

Newer methods

Many of the newer pesticide groups are derived from plants or naturally occurring substances (e.g. nicotine, pyrethrum and insect juvenile hormone analogues), and further 'biology-based' or 'ecological' techniques are under evaluation-pheromones (gossiplure), chemosterilants (alphalite, TEPA), chitin[244] inhibitor (sentricon), ecdysoids (ecdysetrone-forces 'ecdysis' or deaquamation of epithelial layer of insets).

Integrated approach for vector control

It is the utilization of *all appropriate technologies and management techniques* (that are listed above) to bring about *an effective degree of vector suppression* (NOT total eradication of all insects, which will prove disastrous) in a cost-effective manners.

Need. (1) Insect resistance to chemical agents (2) Very high price of newer chemicals (3) Risk of pollution with chemical agents (4) Offers operational flex-

ibility for local and focal programs (i.e. where one method cannot be used, another will suffice.

IVC consists of chemical, biological and environmental control; along with personal protection measures (mosquitoe nets, repellants) and health education. IVC has enjoyed some success in guineaworm eradication (1984), Filariasis control by Vector Control Research Center Pondicherry (1980–85) and Integrated Control of Malaria – Kheda, Gujarat (1985–90).

Mosquitoes

Van Nostrand's Scientific Encyclopedia describes mosquitoes quite simply: "A small two-winged fly with slender body, long legs, and narrow wings bearing scales along the veins."

Sounds innocuous, right? Not a word about the mosquito's mouth, which is designed to furtively slip through your flesh. Nothing about the itching you feel as your body tries to decompose and eliminate chemicals the mosquito injected during the bite. Not a word about the grave diseases you could catch from mosquito: West Nile, encephalitis, yellow fever, dengue, malaria, etc. Not a word about the clever anticoagulants the whiner makes so your blood will flow freely into its gut. Not a mention of the need that females (of many mosquito species) have to suck blood so they can reproduce.

And for the whine weary-mosquitoes don't sing, they just beat their wings.

Genus differentiation (Fig. 5.28)

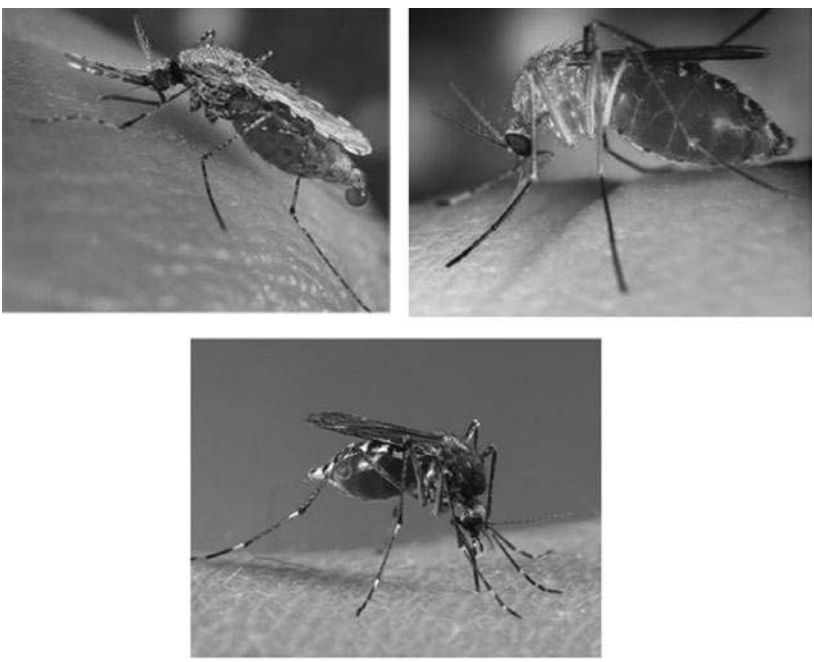

Figure 5.28. Adult mosquitoes—Anopheles and culex [US CDC Public Health Image Library] and Aedes [US Department of Agriculture]

A few general outlines will help. Anopheles sits in about 45°, has equal lengths of proboscis and palpi (the two antennae on both sides of the proboscis). Culex has a round abdominal tip and sits horizontally. Aedes has white stripes all over (Tiger mosquitoes).

Habits

Feeding. Males live on tree juices. Females are hematophagus. However, only some anthropophilic strains prefer humans to animals, Except Aedes, most bite at

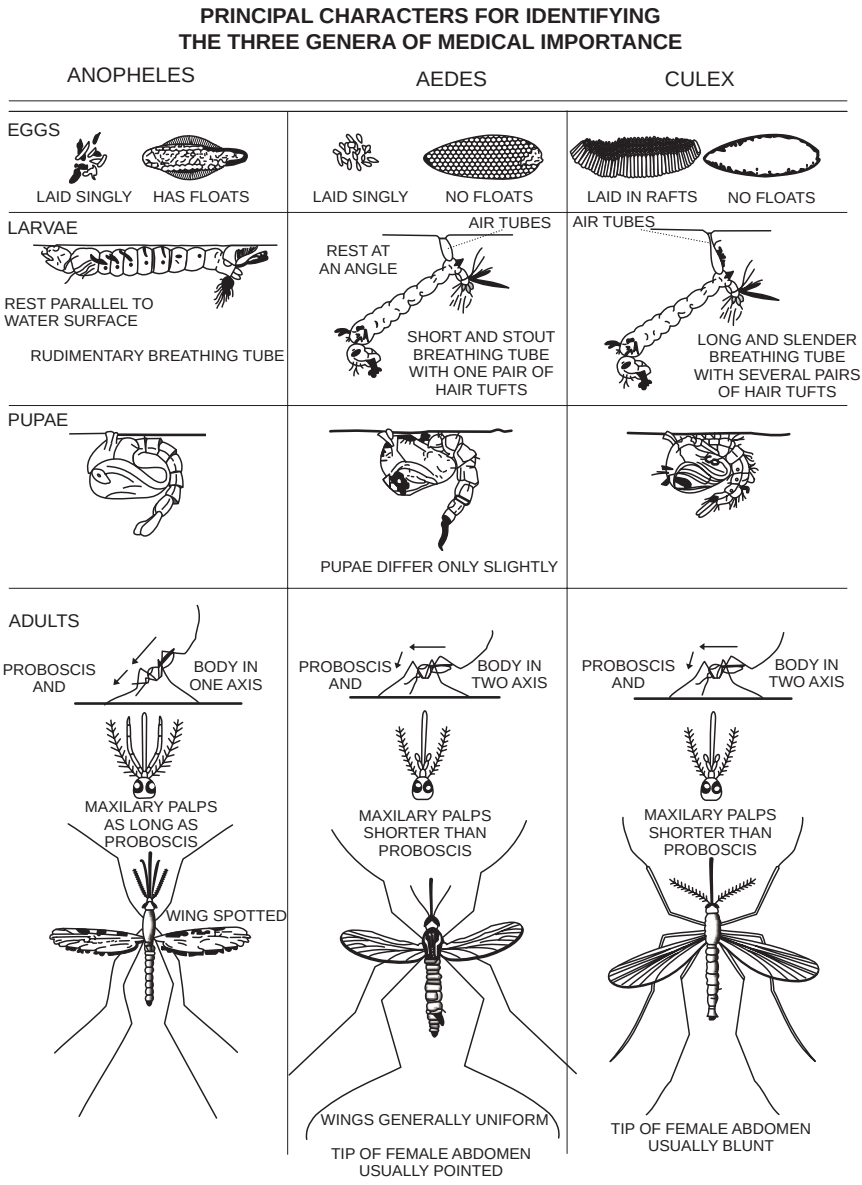

Figure 5.29. How to differentiate mosquitoes

evening and night. They may rest inside house in walls (at least 2 ft from floor), crevasses, beyond pictures, etc. (endophilia) or outside (exophilia).

Breeding. Anopheles prefers clean, oxygenated water (the reason the larva doesn't need a siphon) and Culex prefers dirty water. Aedes breeds in artificial, small collection of water, and Mansonia deposits below leaves of aquatic plants.

Dispersal. Only Culex is a strong flier of 11 km. Aedes and Anopheles are limited to 100–200 m. Aircrafts, storm and wind help their dispersion.

Anthropophilicity. Culex has maximum preference for humans and called 'nuisance mosquitoes'. Rapid unplanned urbanization has given rise to the dominance of these mosquitoes.

Life cycle (Fig. 5.30)

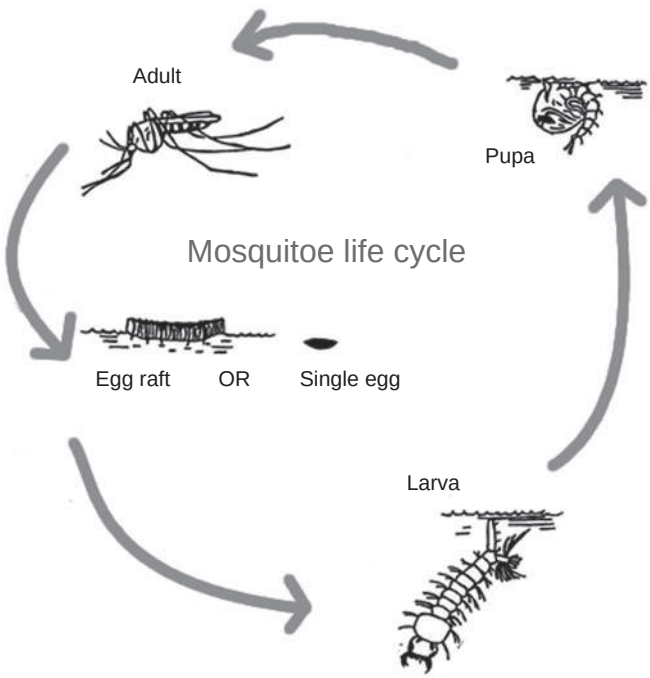

Figure 5.30. Life cycle of mosquitoes

Control

Mosquito-control operations are targeted against three different problems

- *Nuisance* mosquitoes bother people around homes or in parks and recreational areas
- *Economically important* mosquitoes reduce real estate values, adversely affect tourism and related business interests, or negatively impact livestock or poultry production
- *Public health* is the focus when mosquitoes are vectors, or transmitters, of infectious disease.

Monitoring mosquito populations. Adult mosquito populations may be monitored, via

1. Landing rate counts—The number of adult female mosquitoes that land on a part of the body, such as an arm or both legs, within a given time interval.
2. Mechanical traps use a fan to blow adult mosquitoes into a collection bag that is taken back to the laboratory for analysis of catch. The mechanical traps use visual cues (light, black/white contrasts) or chemical attractants that are normally given off by mosquito hosts (e.g. carbon dioxide, ammonia, lactic acid, octenol) to attract adult female mosquitoes.

Monitoring larval mosquito populations involves collecting larvae from standing water with a dipper or a turkey baster. The habitat, approximate total number of larvae and pupae, and species are noted for each collection.

Source reduction. Source reduction is as simple as regular recycling of all water containers. For larger wetlands, the following methods may be taken[245]

- **Open water marsh management (OWMM)**—Shallow ditches are dug to create a network of water flow within marshylands and to connect the marsh to a pond or canal. This gives predators from the pond (fishes) access to the mosquito larvae and can result in long-term mosquito control.
- **Rotational impoundment management (RIM)**—It involves the use of large pumps and culverts with gates to control the water level within an impounded marsh. Water is pumped into the marsh in the late spring and summer to prevent the female mosquito from laying her eggs on the soil. The marsh is allowed to drain in the autumn, winter, and early spring. Gates in the culverts are used to permit fish, crustaceans, and other marsh organisms to enter and exit the marsh.

For the larvae.

- **Mineral oils**: It forms a layer of oil over water which blocks entry of oxygen into the air tube. They are ineffective for Anopheles, as Anopheles requires no aerobic oxygen
- Chemicals: Paris green has been found very useful, especially for Anopheles. Insectides like abate,malathion are effective.
- **Biological**: Effective biocontrol agents include predatory fish that feed on mosquito larvae such as mosquitofish (*Gambusia affinis*) and some cyprinids (carps and minnows) and killifish. Other predators include dragonflies, the predator mosquito Toxorhynchites and predator crustaceans *Mesocyclops*, nematodes, and fungi. Also useful are the bacteria *Bacillus thuringiensis*, especially *Bt israelensis* (BTI). BTI is used to interfere in the digestion systems of larvae. It can be dispersed by hand or dropped by helicopter in large areas. BTI is no longer effective after the larvae turn into pupae, because they stop eating.

For adults. Control of adult mosquitoes are the most familiar aspect of mosquito control to most of the public. DDT was formerly used throughout the world for large area mosquito control, but it is now banned in most developed countries. Controversially, DDT remains in common use in many developing countries, which claim that the public-health cost of switching to other control methods would exceed the harm caused by using DDT.

- Residual spray—DDT is applied on walls and resting surfaces 1–3 times a year (1–2 g/m^2).
- Space sprays—Pyrethrin (the extract of Pyrethrum flowers) is very effective for spraying in closed rooms. Malathion and fenitrothion have been used for Ultra Low Volume (ULV) spraying.
- Some newer mosquito traps or known mosquito attractants emit a plume of carbon dioxide together with other mosquito attractants such as sugary scents, lactic acid, octenol, warmth, water vapor and sounds. By mimicking a mammal's scent and outputs, the trap draws female mosquitoes toward it, where they are typically sucked into a net or holder by an electric fan where they are collected.[246]

Personal protection

- Mosquito nets—To give effective protection, the size of holes must be <0.0475 inch in any diameter.
- Screening windows.
- Repellants: Diethyltoluamide (DEET).

Flies

Flies are true insects of the class diptera (two wings).

Sandflies

Sandflies are *very* small (about one fourth of a mosquito) brown insects, easily distinguished from mosquitoes from their (1) hopping movement, (2) second longitudinal vein of wing branches *twice*, (3) their wings, unlike mosquitoes, are upright (4) their legs are longer than their bodies, (4) they are much more hairy than any other flies. These are ecologically sensitive insects, fragile and cannot withstand desiccation.

There is only one sandfly vector of kala-azar in India, Phlebotomus are gentipes.

Life cycle. Consists of egg, four instars of larvae, pupa and adult. The whole cycle takes more than a month.

Habits. Sandflies are troublesome nocturnal pests, with painful bites but scarcely ever themselves being noticed. The females alone bite every third or fourth day. They can't fly long distance and generally hop about. Their movement is restricted to 50 yards of breeding place.

Control. Can be achieved by

1. Sanitation
2. Insecticides—DDT 1–2 g/m^2 or Lindane 0.25 g/m^2.

Houseflies

This is the one which *everybody* knows. It breeds anywhere there is decaying organic matter (that includes open wounds), and that makes it the filthiest of creatures (from a human viewpoint, of course). It has been suggested that this fly came originally from Africa but nowadays it has followed us to all parts of the earth. In Northern Europe it probably didn't become established until man started

keeping domestic animals indoors during the winter, a practice that didn't start until about the beginning of the Iron Age, 400 BC.[247]

Figure 5.31. The menacing compound eyes[248]

More than 100 pathogens associated with the housefly may cause disease in humans and animals, including typhoid, cholera, bacillary dysentery, tuberculosis, anthrax ophthalmia and infantile diarrhea, as well as parasitic worms. Pathogenic organisms are picked up by flies from garbage, sewage and other sources of filth, and then transferred on their mouthparts and other body parts (Fig. 5.31), through their vomitus, feces and contaminated external body parts to human and animal food. In addition they can be intensely irritating when they occur in great swarms, settling on man and beast alike.

Monitoring. People show variable degree of fly tolerance depending on the setting. In general, at homes the threshold is very low and control actions are taken with a very few flies. However, we tend to ignore the flies in sweetshops, dairy, meat vendors which has no reason at all. The complaint threshold density of the housefly at waste management sites may be 150 flies per flypaper per 30 minutes. Houseflies are monitored with baited traps, sticky ribbons, or spot cards on livestock facilities.

Control

1. **Good sanitation**—Ensures that organic matter, food debris, etc. are always inaccessible to flies, or at least cleaned twice weekly (the fly breeding completes in just one week). Spilled feed should not be allowed to accumulate but should be cleaned up two times a week.[249]
2. Traps—Houseflies are attracted to white surfaces and to baits that give off odors. Indoors, ultraviolet light traps collect the flies inside an inverted cone or kill them with an electrocuting grid.
3. Insecticide
   a. Residual DDT, methoxychlor for adult flies
   b. Larvicides (Diazinon, permethrin and dichlorvos)—Residual wall sprays can be applied where the flies congregate. Resistance to permethrin develops more rapidly in fly populations from farms on a continuous permethrin regime than in farms in which permethrin and dichlorvos have been alternated.
   c. Space spray of pyrethrium and DDT
4. Personal protection by screening
5. Health education.

Ticks

Ticks are small arachnoids (i.e. belong to the same group as spiders). **Hard ticks** (family Ixodidae) are frequent eaters, have their head in the dorsal aspect of the

body, and a single nymph stage. A shield of chitin, called the 'scutum', gives hard ticks their name. *Soft ticks* (family *Argasidae*) are larger, have *five* nymph[250] stages, require less frequent meals and the head is *ventral*.

Common to all ticks and mites is the lifecycle: All of them go through the stages of egg → larva (which usually has 6 legs) → nymph (eight legs, but no reproductive organs) → adults.

Ticks are ectoparasites, living on the blood of mammals, birds, and occasionally reptiles and amphibians. Ticks are vectors of a number of diseases, including

- Hard ticks—Lyme disease, Colorado tick fever, tularemia, babesiosis, ehrlichiosis and tick-borne meningoencephalitis.
- Soft ticks—Q fever, tick-borne relapsing fever, Kyasanur forest disease.

Interestingly, ticks show the phenomenon of transovarian (mother to daughter) and transstadial (i.e. the immature stages are also capable of infection) transmission of pathogenic agents.

Mites

Mites are the arachnids, like ticks. For its size, mites present considerable menace to humans (Figs 5.32–5.34).

Figure 5.32. Ixodus heaxagonus, a hard tick (note the 8 legs, like that of a spider)[251]

Figure 5.33. A trombiculid mite[252]

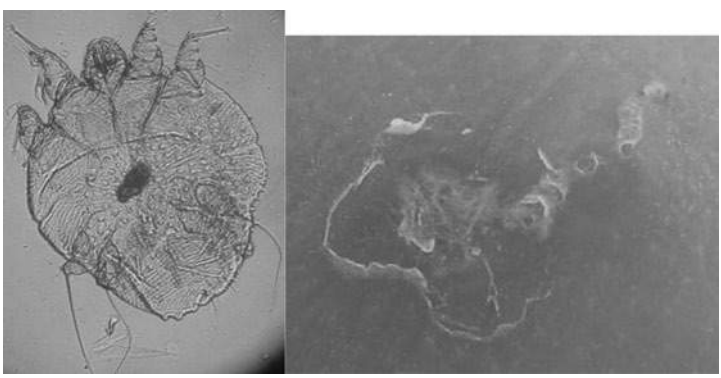

Figure 5.34. An itch mite—The first organism proved to be a human pathogen (1687)[253] and a scabetic lesion (right)

Mites, likes ticks, do not fly or move about very much. They are spread by close contact and fomites, and cling onto the skin without any symptoms for a few hrs until the itch begins. There are three kinds of medically important mites.

- Trombiculid mites (*Leptotrombidium deliense* and *Leptotrombidium akamushi*) are vectors for scrub typhus.
- Larvae of *Eutrombicula alfreddugesi*, called Chiggers, attacks pores of human skin and causes itchy rash in campers and hitchhikers.
- Of special importance in India are itch mites (female members of *Sarcoptes scabiei*) who caused scabies. The disease classically affects the hands, wrist, extensor aspect of elbows, axilla, buttocks, lower abdomen. The patient presents with follicular lesions that itch, especially at nights. Often the whole family is affected. Diagnosis is confirmed by finding of a *burrow* of mites in the skin.

Control of scabies

Scabies is common in India and usually the first thing in the morning you have to face in the outdoor of a public health center. Remember that ALL the members of the household must be treated.

1. Benzoyl benzoate (25%) twice weekly (at 12 hrs intervals) to be applied over whole body below chin (in babies, the head must also be covered). After 12 hrs of 2nd application, ask the patient to take a scrubbing soap bath and change all garments.
2. Topical Lindane lotion (0.5%) to be applied twice in 2–3 days interval.
3. Sulfur ointment (2.5–10%) is the cheapest option.

Lice

Lice are small, wingless ectoparasites of mammals and birds. School age children are the commonest victims.

Adaptations for a parasitic life. Lice are highly adapted animals to host conditions; the adaptations include thier size (0.5 to 8 millimeters), their stout legs, and their claws which allow them to cling tightly to hair, fur and feathers; other adaptations include being wingless and dorsoventrally flattened. They have only small compound eyes; many species have no eyes at all. Most lice have relatively simply chewing mouthparts, but in some they are highly adapted for piercing and sucking.

Spread. Lice cannot jump or fly. They are spread very easily from head to head through fingers and fomites.

Biology. Most lice are scavengers, feeding on skin and other debris found on the host's body, but some species feed on sebaceous secretions and blood. They produce eggs (nits) which are attached to cement of hair by their saliva; eggs hatch in 8–9 days (only if temperature > 22°C); the larva (nymph) looks much like the adult except that it is small, and takes three moulds to become an adult. The adult has antennae, pointed head, 6 legs with claws and the last segment is bilobed in females (Figs 5.35–5.36).

Types. The human harbors three types of lice: Head lice (*Pediculus capitis*, body lice (*Pediculus pubis*) and pubic lice (*Phthirus pubis*). The DNA differences

between head lice and body lice provide corroborating evidence that humans started wearing clothes about 72,000 years ago, give or take 42,000 years.[254]

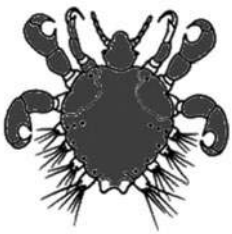

Figure 5.35. The small, extremely inert pubic (crab) louse clings tightly to a hair with its powerful claws

Figure 5.36. A head louse under dissecting microscope[255]

Vectors. In addition to being an itch, lice transmit epidemic typhus, relapsing fever and trench fever (Fig. 5.37).

Control

1. Combing—The most effective method; combing for lice is a tiresome, but effective practice of ridding a child of an infestation.
2. Malathion and Carbaryl shampoo—A lotion of 0.5% malathion given 12–24 hrs contact is effective; there are already reports on resistance to malathion.
3. Malathion powder for "mass delousing" (one shot louse removal from many children at once) of body louse.
4. Regular bathing and change of clothes.

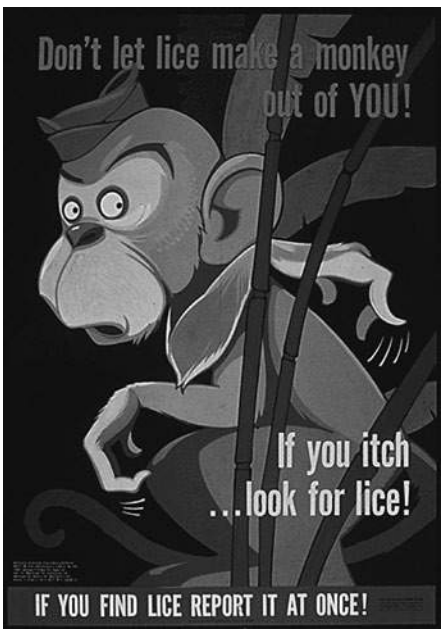

Figure 5.37. War poster : Don't let lice make a monkey out of you[256]

Fleas

Fleas are small, wingless, chitinous, blood sucking insects, usually host specific (a different species infests dogs, cats, rats and humans). They have 10 abdominal segments, bilaterally compressed; males can be detected from females by a penis and spermatheca. Fleas are found in hosts and in their nests. Both the sexes can suck blood. They can't fly but jump 3–4 inches vertically/drift sideways. They are transmitted by their hosts, vehicles, luggage, and food grain transport (Fig. 5.38). Important flea species include

- Human flea (*Pulex irritans*)
- Northern rat flea (*Nosopsyllus fasciatus*)
- Oriental rat flea (*Xenopsylla cheopis*)

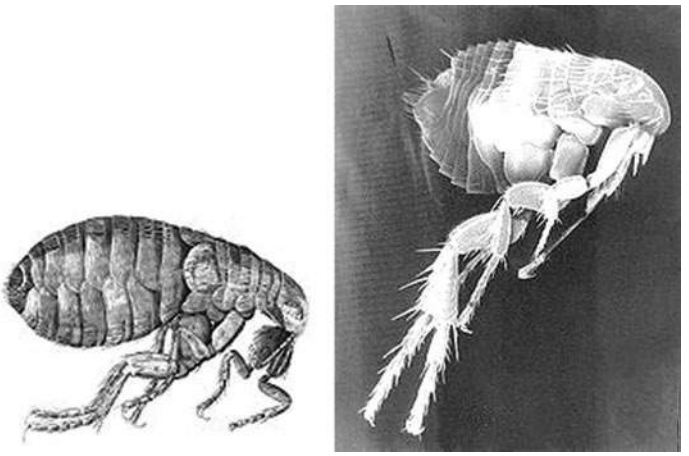

Figure 5.38. The flea was one of the first organisms to be studied under a microscope by Robert Hooke;[257] centuries later, the flea is seen in a scanning electron microscope [CDC Public Health Image Library]

Habits

The flea is adapted to feeding on the blood of their hosts. Their bodies are laterally compressed, permitting easy movement through the hairs or feathers on the host's body (or in the case of humans, under clothes). **They are very good jumpers** and jump upto 200 times their body length.[258] They usually hop away sideways. Their tough body is able to withstand great pressure (like scratching or a slap which will normally kill a mosquito).

Vectors

Besides the biting problems posed by the creature itself, fleas can also act as a vector for bubonic plague (between rodents and humans), murine typhus (endemic typhus) , and in some cases Hymenolepiasis (tapeworm). They are transmitted by their hosts, vehicles, luggage, and food grain transport.

Modes of disease transmission

1. Biting: A blocked rat flea transmits plague, i.e. whose mouths have been blocked by bacilli. A partially blocked flea can have some blood and live, so it is even more dangerous
2. Mechanical transmission
3. Feces (or excess blood split out by the flea in the fur of animals).

Monitoring

1. **General flea index**: Number of fleas/rodents in a locality.
2. **Specific flea index**: Flea/rodent according to each species of flea. It Xenopsylla index>1, it indicates potential explosiveness of plague.
3. **Percentage flea index**: Proportion of each species of flea found in each rodent.
4. **Burrow index**: Free living flea (species specific)/number of rodent burrows in a locality.

Control

Treat the animal to kill adult fleas. In case of domestic animals, a veterinary consultation is required. In epidemics of plague, DDT dust (or BHC, Carbaryl, Malathion) is to be sprayed each 2–4 months, upto 1 ft height in the walls (the height where rats will pick up the insecticide). Rat burrows should also be stuffed with 30 g powder. 5 miles around an infected area should be sprayed within 24 hrs of report. Flea control should be followed by rodent control.

Personal protection. Diethyltoluamide

Control the larval stage. In case of domestic pets, wash pet bedding in hot water/ steam it to kill flea larvae. If animals sleep with family members, all bedding must be washed. Next, find those outdoor areas where the pets spend most time and spray inset growth regulators (IGR). In epidemic situations, it is difficult to control larva of rat fleas, however.

Cyclops

They are fresh water crustaceans (the same group as crabs), just visible to the naked eye, with a noticably forked tail and jerky swim. Cyclops are the vectors for guineaworm and the fish tapeworm *Diphyllobothrium latum*.

National guineaworm eradication program (from archives)

1. Case detection
2. Epidemiological investigation
3. Case management
4. Vector control.
   - Because the infection is acquired by drinking, straining (filtering) by nylon mesh or boiling water before drinking is an effective solution
   - Treatment of drinking water with chlorination (5 ppm) or lime 4 g/gallon; or abate 1mg/l
   - Biological control with *Gambusia* fish
   - Eradication of step wells and provision of piped water/sanitary well.

Antivector chemical treatment—Insecticides[259]

Inorganic insecticides

Silica (SiO_2). This acts as a dessicant and strips off the waxy coating off the cuticle of the insect thus causing suffocation. This is sometimes referred to as diatomaceous earth or "kieselguhr" and is made up of the frustules of diatoms (Bacillariophyceae).[260]

Boric acid (H_3BO_3). Also known as boracic acid and is used for incorporating into baits for ant control.

Organic insecticides

Organochlorines

Diethyl dichlorophenyl trichloroethane (DDT)

DDT was originally prepared in 1873, but it was not until 1939 that Paul Muller of Geigy Pharmaceutical in Switzerland discovered the effectiveness of DDT as an

insecticide (he was awarded the Nobel Prize in medicine and physiology in 1948 for this discovery). DDT is a CNS poison which kills insects by paralysis and convulsion. The action of DDT persists for about 18 months (residual effect). As a spray, 1–2 g/ m^2 is applied on walls. It may also be used as a powder to be applied in creeks and burrows of fleas and ticks. Lastly, it can be used as aersol in space sprays. The use of DDT increased enormously on a worldwide basis after World War II, primarily because of its effectiveness against the mosquito that spreads malaria and lice that carry typhus.

The chemical stability of DDT and its fat solubility came to be known later, and thus its disadvantages. DDT is not metabolized very rapidly by animals; instead, it is deposited and stored in the fatty tissues. The biological half-life of DDT is about eight years; if ingestion continues at a steady rate, DDT builds up within the animal overtime. This has lead to the ban of DDT from many developed countries.

Other organochlorines

Benzene hexachloride (BHC/HCH) was prepared by none other than Michael Faraday. The γ isomer of BHC is called Lindane. Aldrin and methoxychlor are newer organochlorines.

Organophosphates

Organophosphates are cholinesterase inhibitors which paralyse insect by overstimulation of neuromuscular junctions. However, by the same mechanism, they are also human toxins. Examples include chlorpyrifos, chlorpyrifos-methyl, diazinon, dichlorvos, pirimiphos-methyl, fenitrothion and malathion.

Carbamates

Functionally, these share the same mechanism as organophosphates. These are effective against a wide range of pests. Moderately residual and effective at higher temperatures, but broken down if alkalinity is too high. There are loads of carbamates, but propoxur and carabaryl are most used. Propoxur is the choice for DDT resistant Anopheles.

Pyrethroids

Pyrethrum is extracted from Chrysanthemum flowers. It is given as space spray with ½ an hr contact period. WHO recommends a combination of pyrethrum + DDT as space spray for synergistic action. The downside to using natural pyrethrum is that it is very expensive (artificial pyrethroids are now being manufactured, of which the best known is permethrin).

Mineral oils

Kerosene and malarial form a layer of oil over water and suffocate mosquito larvae (except Anopheles); however, they are very harmful to aquatic life.

Stomach poisons

Paris green (Cu Aceto arsenite) was given as powder over water to control mosquito larva before the advent of DDT. NaF is a good replacement.

Insect growth regulators(IGR)

These are hormones which interfere with the insects growth cycle inhibiting full development (methoprene, hydramethylnon, pyriproxyfen, flufenoxuron).

Fumigants

These are volatile gases and only to be used by qualified personnel (methyl bromide, aluminium phosphide, magnesium phosphide, calcium cyanide, hydrogen cyanide).

Toxicity of insecticides

A common measure of the acute toxicity is the lethal dose (LD_{50}) or that causes death (resulting from a single or limited exposure) in 50% of the treated animals. LD_{50} is generally expressed as the dose, in milligrams (mg) of chemical per kilogram (kg) of body weight. Chemicals are considered highly toxic when the LD_{50} is small and practically nontoxic when the figure is large. However, the LD_{50} does not reflect any effects from long-term exposure (i.e. cancer, birth defects or reproductive toxicity) that may occur at levels below those which cause death, these are covered by things such as OEL (Occupational Exposure Limit).

List of vectors

Table 5.20. List of vectors

Class	Group	Name	Agent	Disease
Insecta	Mosquitoes	*Anopheles fluviatilis* *Anopheles stephensi* *Anopheles sundaicus* *Anopheles minimus*	Plasmodium sp.	Malaria
		Culex quinquefasciatus *Culex vishnui* *Culex tritaeniorhynchus*	*Wuchereria bancrofti* Arbovirus JE virus	Filariasis Viral fever JE
		Aedes aegypti	Arbovirus	Viral fever (dengue, JE, West Nile) Viral hemorrhagic fever (yellow fever, chickunganya fever, Â dengue) Rift valley fever

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Class	Group	Name	Agent	Disease
		Mansonia annulifera *Mansonia uniformis* *Mansonia longipalpis*	*Brugia malayi* Arbovirus	Malayan filariasis Chickunganya fever
	Flies	*Musca domestica* *Musca nebulo* *Musca vicina* *Musca sorbens* (housefly)	Many	Diarrhea, dysentery, typhoid, paratyphoid, cholera, amebiasis, helminthic infestations, poliomyelitis, conjunctivitis, trachoma, anthrax, yellow fever
		Phlebotomus argentipes *Phlebotomus papatasi* *Phlebotomus sergenti* *Sergentomyia punjabensis* (sandflies)	*Leishmania donovani* *Leishmania tropica*	Kala-azar Oriental sores Oriental sores Sandfly fever Oroya fever
		Glossina palpalis (tsetse fly)	*Trypanosoma brucei*	Sleeping sickness
		Simulium (black fly)	*Onchocerca volvulus*	Onchocerciasis
	Lice	*Pediculus capitis* (head) *Pediculus corporis* (body)	*Rickettsia prowazekii* *Borrelia recurrentis* *Bartonella quintana*	Epidemic typhus Relapsing fever Trench fever
	Flea	*Xenopsylla cheopis* *Xenopsylla astia* *Nasopsyllus fasciatus* (rat fleas)	*Yersinia pestis* *Rickettsia typhi* *Hymenolepis diminuta*	Plague Endemic typhus Hymenolepiasis Chiggerosis

Contd...

Contd...

Class	Group	Name	Agent	Disease
		Pulex irritans (human fleas) *Cynocephalus canis* (dog flea) *Ctenocephalides felis* (cat flea)		
	Bugs	Reduviid bugs	*Trypanosoma cruzi*	Chagas disease
Arachnida	Ticks	*Dermacentor andersoni* *Haemaphysalis spinigera* (Hard ticks)	*Rickettsia rickettsii* *Babesia microti* *Francisella tularensis* Arbovirus	Rocky mountain spotted fever Babesiosis Tularemia Viral fevers (Colorado tick fever) Viral hemorrhagic fever (Kyasanur forest fever) Tick paralysis
		Ornithodorus moubata Soft ticks	*Coxiella burnetii* *Borrelia recurrentis* Arbovirus	Q fever Relapsing fever KFD
	Mites	*Leptotrombidium* (trombiculid mites)	*Orientia tsutsugamushi* *Rickettsia akari*	Scrub typhus Rickettsial pox
		Sarcoptes scabiei (itch mite)	Female Sarcoptes scabiei var hominis	Scabies
Crustacea	Cyclops	*Mesocyclops*	*Dracunculus medinensis* *Diphyllobothrium latum*	Dracunculiasis Fish tapeworm disease

6

Health and the State

KEY FEATURES

- HEALTH CARE DELIVERY
  - How must a good health care service be?
  - The Indian situation analysis
  - Changing terminologies
  - The public health sector
  - National rural health mission (2005–12)
  - National health policy 2002
  - The Indian Public Health Standards
  - Private health sectors
  - Voluntary health agencies/NGO
  - Drugs
- HEALTH INFORMATION
  - Sources of information
- PLANNING

HEALTH CARE DELIVERY

So how does a nation plan to keep its people healthy? It is no doubt a mammoth task, as it must include four distinct services to the people,

- prevention of diseases
- promotion of health
- therapy
- rehabilitation.

Health care services are those provided to individuals/communities by agents of health services or professionals for the purpose of promoting, maintain, restoring or monitoring health. Mind that the focus in *health care* is upon health, not disease. *Medical care*, however, is a *subset* of the health care service, which ranges from care in home to care in hospitals and refers to those personal services that are provided by a physician to his patients. The focus of medical care is on *disease*, not health. The duty of the state is to provide the entire gamut of health care, not *just* medical care.

How must a good health care service be?

Comprehensive

It must provide ALL the four services mentioned above.

Accessible

1. Physical—Health centers should, preferably, be located where normal people and those with disabilities can reach; it should not be located atop a mountain cliff or deep into a forest.

2. Social—People at health service must be supportive and sympathetic, or at least caring for the visitors.[261]
3. Economic—The basic health services (the eight components of primary health care listed below) should be socialized, i.e. they should be free and reimbursement drawn from taxes of the people only.

Acceptable

This does not have fixed parameters. Two examples will suffice.

1. Distilled and disinfected water, although safe, is impalatable; people expect water to have a certain taste, but no more than that. If you supply tasteless but safe water in a locality, people will drink from their common pond instead and get themselves ill.
2. Immunization schedules must be carried out at a time of the day when mothers are free from housework (i.e. 11 am–4 pm).

Relevant

No matter how rich your country is, their is a limit to every fortune. FUNDS ARE ALWAYS LIMITED. It is a matter of delicate planning on how to put them into best use. Thus, health services must not be placed out of context. We need no control program for achondroplasia (because it is too rare), or eating disorders (not so rare, but we have other pressing problems in hand) or Alzheimer's disease (there's very little tools to control it) in India, but we do need a control program for tuberculosis. Similarly, we do not need to build an ICCU in a village of a thousand people. We could spend the some money on improving the roadways between the village and the nearest district hospital.

Community activity

There are three levels for the community to take part

Support (i.e. monetary support or lending some men into health services)

↓

Participation (by taking some active part in the implementation of health services, i.e. village health guides)

↓

Involvement (carrying out both the planning and implementation by themselves, i.e. people's health in people's hands)

The Indian situation analysis

Demography[262,263]

India is in the **late expanding stage** of population. The total population of India is **1121 million**, with a **cBR 23 and cDR 7.5** (2007). Annual growth rate is **1.64%**. Of this population, a majority (64%) are dependent population. Adult literacy remains at **74%,** and sex ratio is **940.** Average family size is **2.8**, and average age of female marriage is 20.1. 41.6% of population live below new international poverty line (i.e. < 1.25 US$/day purchasing power parity).[264] **16.2%** of people

belong to scheduled castes (2001 census) and 8.2% to scheduled Tribes (2001 census). The population is characterized by:

1. Very rapid growth (GR=1.64%) – part of which may be attributed to immigration.
2. Large population base (children are the predominant group in population), pyramidal population pyramid.
3. High fertility.
4. Declining mortality.
5. Rapid urbanization.
6. 35.35% of population are young (below 15) accoding to 2001 census.
7. Literacy is still inadequate, especially among women.
8. Dependency ratio is 62/100, that is almost 1 dependent per earning member.

Table 6.1. CIA data updated for 2009 [CIA world factbook]

Total population	1,166,079,217 (July 2009); rural population 72.2%
Age structure	0–14 years 30.8% 15–64 years 64.3% 65+ years 4.9% The median age of Indians is 25.1 years[a]
Population growth rate	1.548%
Crude birth rate	22.22
Crude death rate	6.4
Unemployment rate among working age group	7.8%
Net migration rate	–0.05 migrant(s)/1,000 population (2007 est.); it is *negative* because more people are coming into India (often illegally) than going out
Sex ratio	1.064 male(s)/female (2006 est)
Infant mortality rate	30.15 deaths/1,000 live births (2009 est)
Life expectancy at birth (2007 est)	Total population: 69.89 years Male: 67.46 years Female: 72.61 years
Total fertility rate	2.72 children born/woman
[a] Ideally, if the life expectancy is 66, the median should be about 33; but the large fraction of children in India draw the median down	

Mortality

The cDR is 7.5 (2006, significantly reducing over the years). The life expectancy has gone upto 66 years in 2007. However the IMR in India is a drudging 54/1000, and the MMR **407/105**. There is wide variations in death rates among urban and rural areas.

The age group at the maximum risk of death in India is, however, **0–4 years**; that is, being a child in India is very risky.

Public health problems

Communicable diseases

1. Malaria, specially increase in falciparum malaria, chloroquine resistance and insecticide resistance of mosquitoes.
2. Tuberculosis—As high as 30% Indian population is infected with the bacillus, and 1.5% have a radiological disease; India accounts for **one fifth** of the global burden of tuberculosis.
3. Diarrheal diseases—It causes 1.07 million cases per year, mostly among under five children.
4. Acute respiratory infections—It is the 2nd most frequent cause of infant and child death (often claiming first rank, however).
5. Leprosy—With a prevalence of 0.74/10000, India has eliminated leprosy on a national level; however, there is high degree of regional variation of leprosy prevalence.
6. Filariasis—The population at risk (i.e. those who live in the vicinity of filaria cases and not adequately protected from mosquitoes) has risen to 500 million.
7. AIDS—It was estimated at the end of 2007, there are 2.4 million HIV +ve cases in India; it is interesting because at the end of 2003, number of HIV positive cases were estimated to be 5.1 million; this means, not only have many of those 5.1 million have died, but not many new HIV +ve cases have arrived.
8. Others—Kala-azar, JE, dengue, chikungunya, enteric fever, helminthic infestations (specially neurocysticercosis).

Noncommunicable diseases

With changing lifestyle among the well to do, India is the diabetic capital of the world, and in no better state for IHD, cancer, blindness. By 2005, these diseases have overcome communicable diseases in proportional mortality (53%) and almost caught up in total number of DALYs (44% of DALYs were caused by non-communicable diseases).

Nutrition

The Indian society is clearly watermarked between extremes of nutrition and total lack of it. The more poor get poorer, the more shopping malls seem to grow.

1. PEM—46% of children in India are underweight.[265]
2. Anemia—About half of women are children are suffering from IDA, the percentage even greater in pregnancy.
3. LBW—28% babies in India are born with LBW.
4. Xerophthalmia—It is the leading cause of childhood blindness in India, usually affecting between 1–3 years.
5. IDD—No part of India is secure from the effects of IDD.
6. Obesity—It is slowly rising to epidemic proportions.
7. Others—Food adulteration, use of saturated fatty acids, fluorosis, lathyrism.

Environment

1. Safe water is available to most 96% of urban population and 86% of rural population.
2. Sanitation is available in only 18% of rural population; it seems 'the great sanitary awakening' is yet to happen here.
3. Pollution has got out of control (people simply don't seem to understand) – more factories are born, more water bodies filled, more highrisers constructed, more homes converted into apartments, more grounds converted to dumping yards, more fuel is burnt, more bikes sold, more garbage and toxic waste dumped in the rivers – and nobody cares until floods bring the filth from outside gushing into their homes.

Health care

With no national health service, the existing hospital based service has proved spectacularly ineffective to control public health problems. Most medical services, including private ones, are centered around urban areas.

The following summarizes, concisely, the state of public health in India:[266]

- Public health expenditure in India has declined from 1.3% of GDP in 1990 to 0.9% of GDP in 1999, and currently rising to about 3%. However, Central Government contributes 15% of this expenditure, and States contribute 85%.
- Vertical Health and Family Welfare Programs (i.e. referral through primary → secondary → tertiary levels) fail to coordinate.
- Because public health is not socialized (not owned by community) it loses efficiency and accountability.
- Lack of integration of sanitation, hygiene, nutrition and drinking water issues.
- There are striking regional inequalities.
- Population stabilization is still a challenge.
- Curative services favor the nonpoor: for every Re.1 is spent on the poorest 20% population, Rs.3 is spent on the richest quintiles.
- Only 10% Indians have some form of health insurance, mostly inadequate.
- Hospitalized Indians spend on an average 58% of their total annual expenditure in hospital bills.
- Over 40% of hospitalized Indians **borrow heavily or sell assets** to cover hospital bills.
- Over 25% of hospitalized Indians **fall below poverty line** because of hospital expenses.

Resources

Men and women

As of 2009, there is one doctor for every 1588 Indians (that's 62 doctors per 100000 population), well above the target of 1/3500.[267] However, about three quarters of them chose to stay in cities, so the ratio is thwarted in rural areas.

In recent years, most of the focus has been given to MPHWs, village health guides, ICDS workers and ASHAs instead of doctors.

Money and material

WHO recommends a minimum 5% of GNP be spent on health, while India spends 2%.[268] It is heart-wrenching to see the Government spending millions on 'computerising' every sector,[269] building IT parks, flyovers, amusement parks and all things glittering, while ignoring the basics such as health and education.

Time

Most of the time of health workers is consumed by transport (and the occassional strikes), file browsing and paperwork in government offices, and very little time in actual work.

What solutions may we have

This section is a summary of whole of this book. You are advised to go through this list at the end of your course and revise each topic. It serves as a to do list.

Population control

1. National Family Welfare Program
2. National Population Policy 2000 (NPP)
3. Family welfare linked incentives, disincentives and insurance
4. Stop illegal immigration across borders.

National Population Stabilization Fund

The National Population Stabilization Fund (Janasankhya Sthirata Kosh, www.jsk.gov.in) is a combination of many government agencies and civil society working together. Its goal is to:

- To achieve population stabilization, by 2045.
- Meeting the unmet needs for contraception.
- Introduce innovative ideas in the in population control.
- To develop a vigorous people's movement in favor of population stabilization.
- To provide a window for contributions from individuals, trade organizations. and others within the country and outside, for cause of population stabilization.
- Reach out to needy population groups through innovative strategies.

The JSK has introduced many innocative strategies

1. Setting up **call centers** for questions on family planning.
2. *Prerna* award for couples practicing family planning and Santushti award for gynecologists performing a required number of sterilization operations.
3. A virtual resource center on family planning for public education.
4. Observing World Population Day on 11th July.

Control of communicable diseases

1. **Control** programs for tuberculosis, ARI, diarrheal diseases, malaria,[270] AIDS, STDs.

2. **Elimination** programs for leprosy, filariasis, kala-azar, tetanus
3. **Eradication** program for poliomyelitis
4. Universal immunization program
5. Quarantine measures at seaports and airports
6. Containment of epidemics
7. Integrated vector control measures
8. Improve sanitary conditions
9. Education on diseases and their prevention
10. Improve general quality of life through education and better income.

Control of noncommunicable disease

1. Education on traditional ways of living
2. Genetic counseling (to prevent genetic diseases)
3. National programs for control of blindness
4. National cancer control program
5. National mental health program.

For women and children

1. RCH program
2. Janani Suraksha Yojana
3. Nutritional anemia prophylaxis program
4. Integrated Management of Neonatal and Childhood Illnesses
5. ICDS
6. Occupational benefits for women and children
7. School health services
8. Postpartum program.

Environment

1. Water, air and soil pollution control acts
2. National water supply and sanitation program

Nutrition

1. National programs—Vitamin A prophylaxis, IDA control, ICDS, midday meal, iodine deficiency disorders control program.
2. Food fortification.
3. Public distribution system.

Health services

The **National Rural Health Mission** has been framed incorporating all the above mentioned components.

Current targets

Demography

- Stabilize population by 2045 (NPP)
- Get TFR to replacement level (2.1) by 2010 (NPP and NRHM)

- Birth rate 21 by 2007
- Death rate 9 by 2007.

Demography

- IMR < 30 by 2010 (NPP and NRHM)
- MMR < 100 by 2010 (NPP and NRHM).

Maternal and child health

- Neonatal mortality < 20 (RCH II)

Morbidity

- Eradicate poliomyelitis and yaws by 2005 (NHP 2002).
- 100% immunization.
- Reduce malarial mortality by 50% till 2010, another 10% by 2012 (NRHM)
- Reduce dengue mortality 50% by 2010 (NRHM).
- Reduce Japanese encephalitis mortality 50% by 2010 (NRHM).
- Eliminate leprosy < 1/10000 (the NHP targeted 2005 fr elimination, and at a country level, we *have* achieved elimination).
- Control tuberculosis by maintaining 85% cure rate.
- Eliminate neonatal tetanus < 1/10000 live births.
- To control diarrheal disease, vector borne diseases and ARI.
- Eliminate LF by 2015 (NRHM).
- Eliminate kala-azar by 2010 (NRHM).
- Achieve xero level growth of HIV by 2007 (NHP).
- ↑ cataract operations to 46 lakhs/ year in 2012 (NRHM).

Health service

- Upgrading all community health centers to Indian Public Health Standards.
- Increase utilization of FRUs to 75% (i.e. 75% cases from PHCs should go to FRUs, not tertiary hospitals) (NRHM).
- Engaging 250000 ASHAs in 10 states (NRHM).
- Increasing utilization of public health services to > 75% (NHP).
- Establish nationwide integrated health surveillance and information system by 2005 (NHP).

Changing terminologies

Comprehensive health care

It was coined by Bhore committee, 1946 as –'provision of integrated preventive, promotive and curative services from womb to tomb, to every individual in a defined geographic area'. It was with this idea that the Government of India built a chain of public health centers (PHC).

Basic health services

WHO/UNICEF coined the next term as–'a group of functions essential to the health of the people'.

↓

But the problems remained same as ever with health services, namely:

1. City and hospital oriented
2. Biased towards cure, not prevention
3. Inadequate community participation
4. Accessible to a very small population
5. Inadequate staff, drugs and material
6. Health was dissociated form socioeconomic context.

Thus there was growing dissatisfaction with these services, which demanded a new approach.

Primary health care/health for all

International conference on primary health care, Alma ata, USSR (WHO/UNICEF), 1978

1. This conference defined the concept and dimensions of health and primary health care.
2. Set the aim 'Health for all' by 2000: *Attainment of a level of health by all which permits socially and economically productive life*. The basic principle of this idea is close to socialism, i.e. health is a form of 'social justice', a responsibility of state towards its citizens, rathern than the responsibility of the individual.
   However, it does NOT mean that
   - health is a single quantifiable entity
   - nobody will fall ill
   - there will be provision for *medical* care for *everybody* for *all* diseases by the state
3. Interested member nations were asked to formulate strategies, national policy and plan of action.

The millennium development goals

More recently in September 2000, the UN declared the Millenium Development Goals. Three of the 8 goals are health related, namely

1. Reduce child mortality (under five mortality) to 2/3rds between 1990 and 2015.
2. Reduced MMR by 3/4th between 1990 to 2015.
3. To halt the progress and begin to reverse the progress of HIV/AIDS by 2015.

The public health sector

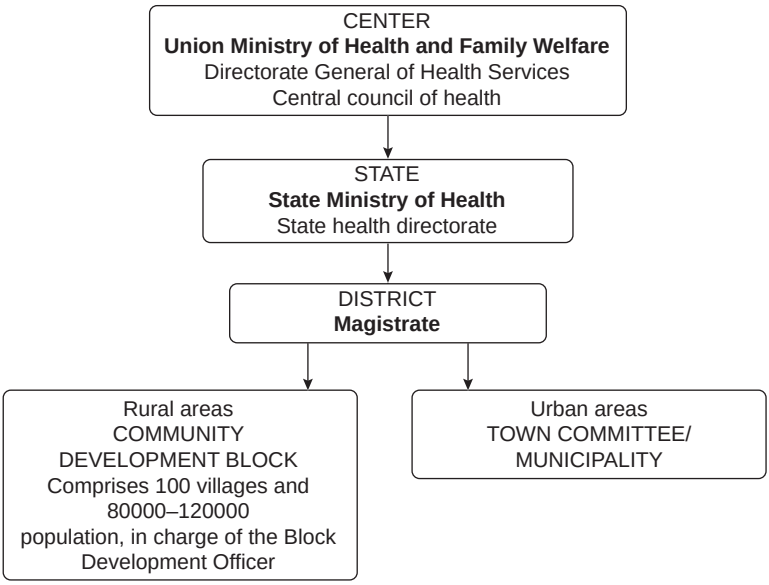

Figure 6.1. Administrative levels of public health sector

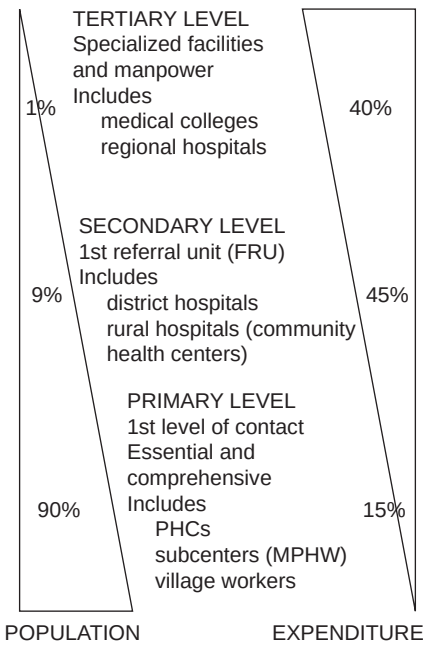

Figure 6.2. Three levels of health care in India; note the population and health care discrepancy

Primary care	Specialized (Sec. and Tert. care)
Low cost	Expensive
Difficult to introduce (because it is COMPREHENSIVE)	Easier (it is THERAPEUTIC)
Appropriate for meeting national health problems	Inappropriate (deals with 1% of population)

Primary health care

It is the

- *Essential* health care which is
- Practical and scientific
- Made *accessible* and *acceptable* universally (individual, family and society) by
- *Full participation* of the community
- At a cost which the community and country can afford
- Spirited by self reliance and determination of the people.

Essential components

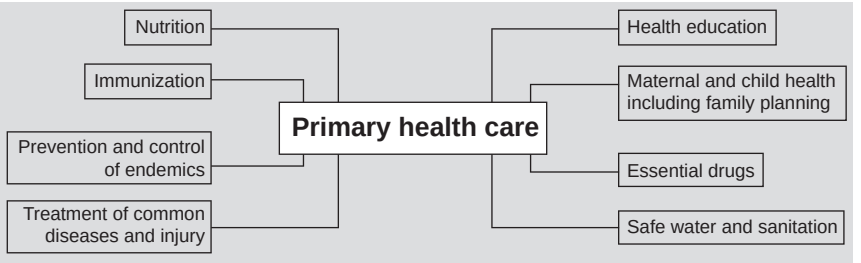

Figure 6.3. Essential components of primary health care

Principles of primary health care

1. **Equitable distribution**, not equal distribution; resources should be distributed according to need, not numbers. For example, PHCs are denser in hilly areas than in planes, although population in planes is much denser. This is because of the difficulty in transport in hilly areas. Similarly, classification of districts in malaria and tetanus programs helps to identify districts which are most affected and send maximum resources there.
2. **Community participation**—No health service is complete until the target population takes some part in its implementation. For example, village health guides, trained dais, ASHAs and Anganwadi workers are chosen from within the village.
3. **Appropriate technology**, which is cheap to implement but effective (i.e. ORS, immunization, vaccine vial monitors, disposable delivery kits).
4. **Intersectoral coordination**—No health service is a matter of health department alone, but requires collabration from many other sectors. The water supply and sanitation in an area has to be maintained by public health engineers,

not doctors. The midday meal scheme is run by education department, which plays an important role in child nutrition in India. The ICDS program is a collaboration between departments of Women and Children's Welfare, Rural development and Social welfare.

Primary health care in India

After the Alma ata conference, India brought out its own National Health Policy in 1983, which described the plan of action for reorienting and shaping existing facilities with specific goals. This is the basis of health care delivery in India.

Primary health care in rural India has been divided in three tiers:

1. Primary health centers/Block primary health centers or Community health centers.
2. Subcenters.
3. Village.

Village

1. Health promotion: MCH, family planning, IEC (information, education, communication).
2. Curative: Minor ailments, first aid.
3. Supportive: To help multipurpose health workers from subcenters.
4. Referral: To refer to subcenters.
5. Holding stock of ORS, condoms, etc.
6. Information: Vital events, enumerate beneficiaries.

Village health guides

One VHG is chosen per 1000 population in village directly from the people, by a scheme launched on 2nd October, 1977. The qualifications are,

1. Should devote at least 2–3 hrs a day to service (part time)
2. Should be local, preferably women
3. Able to read and write (minimum class VI)
4. Acceptable to all sections of the community.

The VHGs are trained locally with a stipend of Rs. 200/month, for a period of 200 hours over 3 months. They receive a honorarium of Rs. 50/ month + drugs amounting to Rs 600 per year.

What they cannot do

1. Injections
2. Prescribe outside their manual
3. Treat > 3 days if there is no improvement.

VHGs are the *face* of primary health care to the people of rural India.

Trained dais

Because people will usually resort to local dais for a delivery, the Government thought why not train these dais, rather than mothers in labor into hospitals that are far away? The trained dais are usually traditional midwives, trained to increase knowledge and skill. They are trained with stipend of Rs. 300 for 2 days at PHC/SC and 4 days field training with MPHW. They must conduct at least two deliveries during training.

Emphasis is laid on

1. Family planning education to the dai, so that she can spread it to the community
2. Asepsis during delivery (she is provided with disposable delivery kits)
3. Postnatal care and guidelines on infant feeding.

They receive Rs. 10 for each *registered* delivery.

Anganwadi workers

They are selected under ICDS scheme, 1/1000 population. They are trained for 4 months with knowledge of health, nutrition and MCH. They receive a honorarium of Rs. 1400/ month and their helper, Rs. 900/ month.

Services that they provide (are the same as services of ICDS)

1. health checkup
2. collecting candidates for immunization and bringing them to MPHW
3. growth monitoring
4. supplementary nutrition
5. health and nutrition education
6. nonformal preschool teaching
7. care of pregnancy and lactation.

Their beneficiaries are

1. children of 0–6 years
2. pregnant/lactating women
3. women of 15–45 years (they provide health education).

Subcenters

These are the peripheral outposts of health, distributed 1/5000 population in planes and 1/3000 in hilly areas (because hilly areas have widely distributed population with sparse connections).

Multipurpose health workers (MPHW) – Male, female

The subcenter is manned by two MPHW, one male and one female. The Indian Public Health Standards enlist the following 'assured' services at subcenters

1. Preventive service: Immunization, nutritional prophylaxis programs.
2. Antenatal care (minimum **3 check ups**, iron folate prophylaxis, routine blood tests, identification of risk factors.
3. Intranatal care (aseptic delivery, initiation of breastfeeding).
4. Postnatal care (at least **2 postnatal home vistis**, education on child nutrition and family planning).
5. Treatment of minor ailments including diarrhea, acute respiratory infections and injuries.
6. Integrated diseases **surveillance** (HIV, vector borne diseases, mosquito control, tuberculosis, etc.) weekly reporting to PHCs.
7. Family planning service and referral for *safe* abortion to higher level.
8. Adolescent health care services.

9. Training and guidance of lower level workers.
10. Refer patients to higher level.
11. Collect and report vital events.
12. Monitoring local environment, especially **water quality**.
13. Assist schools in implementing School Health Services.
14. Carrying out National Health Programs at lower levels.

Primary health centers

PHC is the core institution for proving integrated preventive, promotive and curative services to the people of a geographic area. PHCs are distributed in 1:6 ratio with subcenters, i.e. 1/30000 in planes and 1/20000 in hills.

PHCs should be[271] staffed by

1. Medical officer—3 (at least one female)
2. AYUSH practitioner—1
3. Pharmacist—2
4. Health assistant (male, female)
5. Health worker (female)—1
6. Clerks
7. Compounder—1
8. Auxilliary nurse midwives (ANM)—5
9. Laboratory technicians
10. Health educator—1
11. Drivers and class IV staff.

Functions of a PHC [Alma Ata Declaration]

1. Medical (curative) care
2. Maternal and child health care including family planning
3. Prevention and control of local endemic diseases
4. Vital event reporting
5. Safe water supply and basic sanitation
6. Health education
7. Refer patients to higher level if needed
8. Training of lower level workers
9. Carrying out National Health Programs
10. Basic laboratory services
11. Coordinate activity of subcenters (meeting on each Saturday).

The **Indian Public Health Standards** list the follwoing assured services in a PHC

1. **Outdoor**—4 hrs in the morning and 2 hrs in the evening.
2. 24 hr **emergency** service, first aid and splinting of injuries.
3. Proposal of at least **6 beds** in a PHC for management of emergency patients.

4. All elements antenatal care, intranatal, postnatal and newborn care (except difficult cases that must be referred); provision of facilities under Janani Suraksha Yojana.
5. **IMNCI**, promotion of breastfeeding, immunization, nutritional prophylaxis.
6. Family planning, MTP using **manual vacuum aspiration**.
7. Health education for prevention of STDs, treatment of STDs by syndromic approach.
8. **Growth monitoring** and coordination with ICDS centers.
9. Testing local **water quality**, promoting sanitation and waste disposal through health education.
10. Assisting in school health services.
11. Adolescent health care.
12. Carrying out **national health programs**.
13. Recording and reporting of **vital events**.
14. Training of lower level workers.
15. Basic laboratory tests (routine blood, sputum testing, **pregnancy** test, RPR test for syphilis, typhidot tests, **malaria parasite and antigen tests**, rapid test kit for **fecal contamination** of water, orthotolidine test for **chlorination of water**).
16. Selected minor **surgical procedures** (sterilization, MTP, cataract, hydrocele, etc.).
17. Using Indian systems of medicine (AYUSH) in mainstream.

PHC	Hospital
Preventive, promotive, curative	Curative
Defined area	Undefined
Optimum mix of medical and paramedical workers	Curative staff

Block PHC

It serves the same function as PHC, only that it is better equipped (it is a **bedded hospital** and distributed 1 BPHC per 1–1.2 lakh population. It has all the staff and facilities of a PHC, plus

1. Block medical officer of health (BMOH)
2. Medical officers – 2–3
3. Dental, ayurved, homeopathy officers
4. **Beds**: 10–60
5. **Operation theater** for family planning operations laboratory.

3 PHCs are tagged with one medical college for medical students to have a hands–on experience of village life (the 'Reorientation of Medical Education' program).

What you have to do as an MO

So in this extensive network of health care delivery, you find yourself as a medical officer, i.e. the team leader of your army, the king of your health center and its downline. Or, for the present purpose, you find yourself facing a question like 'Your block is experiencing 342 cases of cholera; how do you investigate and

control?' in your exams. In both situations, follow this routine. This routine applies to *anything* that is to be done by leadership and motivating people (i.e. controlling cholera, organizing a college fest, etc.) (Fig. 6.4).

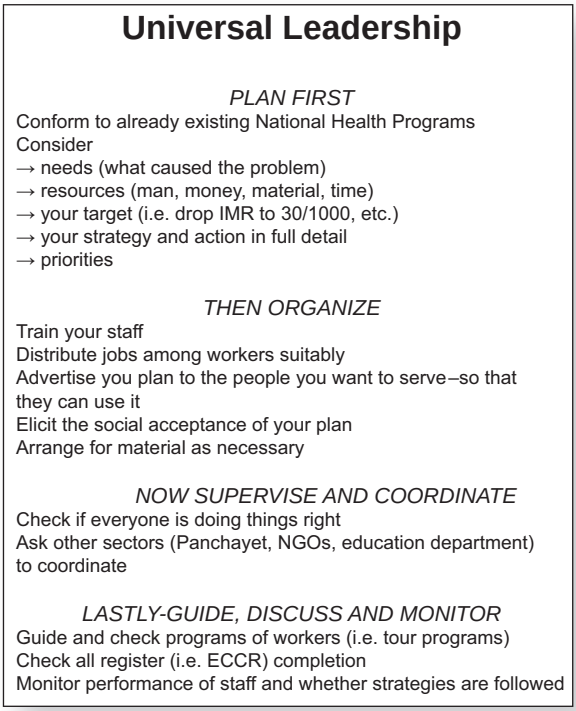

Figure 6.4. Universal leadership

Primary health care in urban India—Krishnan Committee (1982)—'Urban revamping scheme'

Primary health care is still very disorganized in towns. In the urban revamping scheme, towns with 40% slum population were initial targets. The idea was that basic health services should be within 15 min walking distance from every residence, so that poor people in urban areas do not have to line up in large hospitals. For this purpose, towns have been classified in the following manner.

- Category A (< 5000 population), B (5000–10000), C (10000–25000)—Attached to hospital for referral and supportive service. Only Lady Health Worker/Public Health Nurse needed, no doctors are necessary in primary health care.
- Category D (> 25000 population)—Attached to a hospital for referral, MTP and sterilization, doctor is required in PHC.

Staff

Nurse-midwives were proposed as 'backbones' of urban primary health care. They will provide:

1. preventive services
2. family planning
3. curative services
4. outreach with services (door to door visits)
5. report of vital events.

Secondary health care

A **community health center**/ 'Rural hospital' is an upgraded BPHC, which serves same population but has two extra facilities:

- **specialist** doctors in medicine, gyne-obs, surgery, anesthesia and pediatrics
- X-ray facility.

Nowadays, 1 of four BPHC is upgraded to CHC. Gradually, all BPHC will become CHC. The CHC enters the secondary level of health care.

Indian public health standards – assured services for CHC

- Emergency care of all medical and surgical patients
- 24 hrs facilities for delivery, both normal and assisted deliveries
- Essential and emergency obstetric care (including cesareans)
- Family planning services including laparoscopic sterilization
- Safe abortion (MTP)
- Newborn care
- Blood bank
- Essential laboratory services
- Referral and transport to higher levels
- Presence of Rogi kalyan samiti.

A **first referral unit** (FRU) is a district hospital/CHC with following facilities

1. Blood transfusion
2. Obstetric surgeon
3. Anesthetist and anesthetic equipments.

This is the place where to send in referred mothers (specially those in labor) from PHC. The FRU is a pivotal step in preventing maternal mortality because, mothers die because of bad *intranatal* care, not antenatal care.

National Rural Health Mission (2005–12) (Fig. 6.5)

The National Rural Health Mission (2005–12) seeks to provide effective health-care to rural population throughout the country with special focus on 18 backward states, which have weak public health indicators and/or weak infrastructure. This mission is time bound, unlike previous health programs, and it seeks to alter the architecture of health service by introducing some radically new measures such as ASHAs, Indian systems of medicine, etc. It also aims to increase health expenditure upto 3% of GNP.

Figure 6.5. The NRHM logo

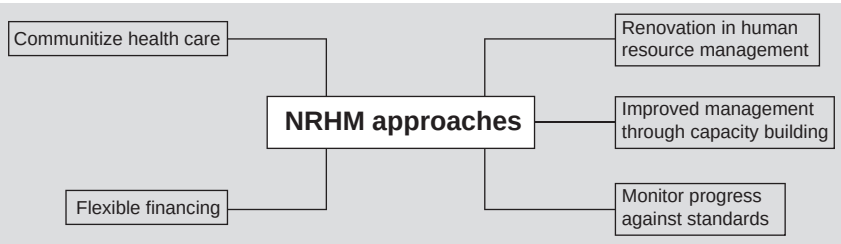

Figure 6.6. The 5 key approaches of NRHM[272]

Goals

1. Reduce IMR in MMR (especially in 18 selected states).
2. Create universal access to public health services such as women's health, children's health, water sanitation, hygiene, immunization and nutrition.
3. Prevent and control both communicable and noncommunicable diseases.
4. Access to integrated primary health care.
5. Stabilization of population, gender and demographic balance.
6. Revitalize local health traditions; mainstream 'AYUSH' (Ayurveda, Yoga, Unani, Siddha, Homeopathy).
7. Promote healthy lifestyles.

Targets

National

1. Infant mortality rate **30/1000** live births.
2. Maternal mortality ratio reduced to **100/100,000**.
3. Total fertility rate reduced to **2.1**.
4. Malaria mortality reduction rate: 50% upto 2010, additional 10% by 2012.
5. Kala-azar mortality reduction rate: 100% (**elimination**) 2010 and sustaining elimination until 2012.
6. Filaria/Microfilaria reduction rate: 70% by 2010, 80% by 2012 and **elimination by 2015**.
7. Dengue mortality reduction rate: 50% by 2010 and sustaining at that level until 2012.
8. Japanese encephalitiss mortality reduction rate: 50% by 2010 and sustaining at that level until 2012.
9. Cataract operation: Increasing to 46 lakhs per year until 2012.
10. Leprosy prevalence rate: Reduce from 1.8/10,000 in 2005 to **less than 1/10,000 (elimination)**.
11. Tuberculosis DOTS services: Maintain **85% cure rate** through entire mission period.
12. Upgrading Community Health Centers to Indian Public Health Standards.
13. Increase utilization of **First Referral Units** from less than 20% to 75%.
14. Engaging **250,000** female Accredited Social Health Activists (ASHAs) in 10 states.

Community Level

1. Availability of trained community level worker at village level, with a drug kit for generic ailments.
2. Observing **Health Day** in ICDS centers on a fixed day every month for immunization, ante/post natal check ups, child nutrition and family planning.
3. Availability of **generic drugs** (not branded drugs) for common ailments at Subcenter and hospital level.
4. **Good hospital care** through availability doctors, drugs and quality services at PHC/CHC.
5. Improved access to immunization through induction of Auto Disabled Syringes, alternate vaccine delivery and improved mobilization services.
6. Improved **institutional delivery** through provision of referral, transport, escort and improved hospital care subsidized under the **Janani Suraksha Yojana (JSY)** for the 'Below Poverty Line' families.
7. Availability of health care for free/very little expense by Community Health Insurance schemes.
8. Provision of household **toilets**.
9. Improved outreach services through **mobile medical unit** at district level.

Funds

It is a welcome move that all National Health Programs have now moved under the 'umbrella' of NRHM and one single budget finances them all. This allows states to allot funds to that particular program which is failing in that state (**flexible finance**). Earlier, separate budgets for each caused waste of time and paper, and often duplication of efforts.

Strategies

Core strategies

1. **Decentralize**: Train and enhance Panchayat Raj to own, control and manage public health services (rather than the health minister plan it all himself, it is better if the target population themselves do the planning).
2. Accredited Social Health Activist (ASHA): Female health worker to promote access to improved health care at *household* level.
3. Strengthen **existing health system**
    - Involve subcenters in local health planning and broaden duties of MPHW; each subcenter is provided Rs 10000 per annum and essential drugs, both modern and AYUSH, and additional MPHWs).
    - Strengthen PHCs and offering better medical care (24 hrs emergency service is to be available in at least 50% of PHCs), essential drugs (including syringes for immunization) so that people grow some trust on PHCs.
    - Strengthen CHCs by providing **30–50 beds per lakh population**, and 24 hr referral service as FRUs, presence of at least one anesthetist all the time, and involving Stakeholder committees (Rogi Kalyan Samiti) in CHC management.
4. Intersectoral planning of health programs at district level: To be carried out by District Health Mission, which will include doctors as well as adminis-

trator, managers (MBAs) and computer operator; the DHM will become the **unit** of health care and will plan all aspects of health (including water supply and sanitation) for the district.

5. Converging sanitation and hygiene: **Total Sanitation Campaign** (TSC) is presently implemented in 350 districts, and is proposed to cover *all* districts in 10th five year plan. Components of TSC include:
   - IEC activities
   - rural sanitary 'marts' (like shopping marts)
   - individual household toilets
   - sanitary complex for and maintained by women
   - school sanitation.

The TSC is also implemented through Panchayati Raj. ASHA would be paid incentives for promoting household toilets.

6. Strengthen national disease control prgrams and the Integrated Disease Surveillanc Project through supply of generic drugs (both modern and AYUSH) and setting up **mobile medical units** at district level.
7. Integrate family welfare programs at national, state and block level.
8. Technical support to National, State and Distric health missions.
9. Strengthen capacities for data collection, analysis and review for evidence based planning.
10. Formulate a transparent plan for career as a health worker (so as to woo young people into health service).
11. Assimilation of NGOs within health service.
12. Promote healthy lifestyles.

Supplementary strategies

1. Regulate private sectors, especially the 'informal' rural practitioners, so that they provide quality service at a reasonable cost.
2. Promote public private partnership—Since almost 75% of health services are being currently provided by the private sector (and people will usually visit private practitioners than health centers), there is a need to refine regulations. The public sector has to play the lead role in defining the framework and sustaining the partnership. A good example is a CT scan center run by a company on subsidized prices for patients referred from a PHC.
3. Mainstreaming of AYUSH—Indian systems of medicine, by providing AYUSH practitioners at every PHC and upper levels.
4. Reorientation of medical education, so that medical students have some experience of rural health facilities during their student life.
5. Health insurance for poor, so that they can avail good hospital service if necessary.

Accredited social health activists (FEMALE)

Each village will have an ASHA chosen by and accountable to panchayat to act as a bridge between auxiliary nurse – midwives and village, and to create awareness, mobilize community and increase utilization. She is an honorary volunteer and will receive performance based compensation for—

1. promoting immunization
2. referral
3. escort services for RCH
4. promotion of household toilets, etc.

ASHAs are selected from the community, with due proportion from the disadvantaged groups. They undergo induction training of 23 days per month × 12 months. They will carry a drug kit (allopathic/ ayurvedic) for common ailments.

Eligibility

1. A resident woman between 25–45
2. Educated at least upto class VII
3. Married/divorced or widowed.

Monitoring

Monitoring is to be done peripherally, i.e.

- Subcenters to report on performance to Panchayats
- Hospitals to Rogi Kalyan Samitis
- District Health Mission to Zila Parishad.

Annual District Reports on People's Health is prepared by DHM, which are compiled to form State and National Reports on People's Health to be tabled in Parliament. In accordance to Right to Information Act, the health reports should be available at all CHCs and over the internet for the public to scrutiny.

National health policy 2002

The ministry of Health and Family welfare evolved a NHP in 1983 keeping in view the national commitment to achieve 'Health for all'. Since then there has been significant changes in the determining factors relating to health sector necessitating revision of policy. A new NHP-2002 was thus evolved, whose aim is to achieve an acceptable standard of good health amongst general population.

Strategies

1. Increases access to decentralized public health system by establishing new infrastructure in existing institutions.
2. Equitable access to health services.
3. Priority to preventive and first line curative initiatives at primary health level.
4. Importance to tuberculosis, malaria, blindness, HIV.
5. Rational use of drugs.

Goals to be achieved by 2015

Eradicate polio and yaws	2005
Eliminate leprosy	2005
Eliminate kala-azar	2010
Eliminate lymphatic filariasis	2015
Achieve zero level growth of HIV/AIDS	2007

Contd...

Contd...

Reduce mortality by 50% from tuberculosis, malaria and other vector borne and water borne disease	2010
Reduce prevalence of blindness to 0.5%	2010
Reduce IMR < 30 and MMR < 100	2010
Increase utilization to 75%	2010
Establish National Health Accounts system	2005
Increase health expenditure to 2% of GDP	2010

The Indian Public Health Standards

The Indian Public Health Standards is the defining document for services available at all public health sectors. It lists assured services to be availed at every level of health care. The current recommendations (2007) have been listed earlier for subcenters, PHCs and CHCs.

Private health sectors

This consists of nursing homes, private practitioners of Indian systems of medicine. The NRHM aims to accommodate these people in health services, either officially or through public private partnership.

Voluntary health agencies/NGO

These are autonomous, privately funded organizations who conduct programs directly to furthering public health (health service/health education/research/health legistation, etc.)

Functions

1. Supplementing government agencies.
2. Guiding government agencies (especially in disputed territories where 'Government' is unknown).
3. Health education—Because NGOs are often closer to the public than Government bodies, they are more successful as an educational medium.
4. Research on health.
5. Demonstration of health related information.
6. Health legislation—These organizations often help the progress of social medicine through Public Interest Litigations (PILs).

Drugs

Essential medicines

> Those drugs that satisfy the health care needs of the majority of the population; they should therefore be available at all times in adequate amounts and in appropriate dosage forms, at a price the community can afford."
>
> *--WHO: Health topics—Essential Medicines*[273]

The WHO has published a model list of essential medicines. Each country is encouraged to prepare their own lists taking into consideration local priorities.

The *core* list from WHO presents a list of *minimum* medicine needs for a basic health care system. Time tested drugs such as morphine, allopurinol, atropine, diazepam, antidotes for poisons, penicillins, macrolide antimicrobials and antiretroviral drugs are few specimens from this list. The *complementary* list includes drugs for diseases which require specialized diagnostic or monitoring facilities are needed. The WHO list is important because it forms the basis of national drugs policy in many countries.

The list of WHO essential drugs can be obtained from http://www.who.int/medicines/publications/essentialmedicines/en/index.html.

The disarray of the medicine market

Most developing and many developed countries are yet to implement a 'public drug repository' (i.e. state funded production of drugs and identification by generic name rather than trade names). Meanwhile, we have to depend on pharmaceutical companies to produce drugs. The pharmaceutical field is one of the more strictly controlled businesses, but it is still possible for the price of a single drug to vary over an wide range (for example, the price of one atorvastatin 10 mg tablet varies from Rs 2.25 to Rs 12).[274] Because of our evergrowing population, the demand for drugs will only increase, and people have a natural tendency to go for the cheapest alternative. This has caused the meteoric rise of **counterfeit medicine** as a cottage industry.

Counterfeit medicine

Medicines which are produced with the intention to cheat

- have no active drug at all
- a wrong drug
- a right drug but in an insufficient quantity
- lie about their expiry date.

Substandard medicines are those which do not meet the quality specification described on the label.

Efforts at quality control

Drugs marketed in India are regulated by Drugs and Cosmetics act (1940 and 1945). The Central Drug Standard Control Organization looks after the safety, efficacy and quality control of drugs.

Good manufacturing practices (GMP)

Good manufacturing practice" or "GMP" refers to the quality control of manufacturing for foods, pharmaceutical products, and medical devices. Although there are a number of them, all guidelines follow a few basic principles.

1. Manufacturing processes are clearly defined and controlled.
2. The processes are controlled and any changes to the process that have an impact on the quality of the drug are evaluated.
3. Instructions and procedures are written in unambiguous language.
4. Operators are trained to carry out and document procedures.
5. Records are made, manually or by instruments, during manufacture that demonstrate that all the steps required by the defined procedures and instructions were in fact taken and that the quantity and quality of the drug was as expected.

6. Records of manufacture are maintained that complete history of a batch of drugs can be traced.
7. The distribution of the drugs minimizes any risk to their quality.
8. A system is available for recalling any batch of drug from sale or supply.
9. Complaints about marketed drugs are examined.

The World Health Organization (WHO) version of GMP is used by pharmaceutical regulators and the pharmaceutical industry in over one hundred countries worldwide, primarily in the developing world. The European Union's GMP (EU-GMP) enforces more compliance requirements than the WHO GMP, as does the Food and Drug Administration's version in the US.

HEALTH INFORMATION

A mechanism for *collection*, *processing*, *analysis* and *transmission* of information required for organizing and operating health services, and also for research and training.

Right to information act

Right to Information act is a law enacted in 2005 which applies everywhere except J and K.

- **Any citizen may request information from a "public authority" (a body of Government or "instrumentality of State") which is required to reply expeditiously or within thirty days**.
- The Act also requires every public authority to computerize their records for wide dissemination and to proactively publish certain categories of information.

The Act specifies that citizens have a right to

1. request any information
2. take copies of documents
3. inspect documents, works and records
4. take certified samples of materials of work
5. obtain information in form of printouts or in any electronic media.

Each Government office must employ a **Public Information Officer** (PIO) who will overlook requests for information. For Central Departments as of 2006, there is a fee of Rs. 10 for filing the request, Rs. 2 per page of information and Rs. 5 for each hr of inspection after the first hr. If the applicant is a Below Poverty Card holder, then no fee shall apply.

Objectives of a health information system

1. To give reliable, relevant, upto date, adequate, timely, reasonably complete information for health managers at all levels.
2. For educational purposes.
3. Monitoring health services.

   Data collection > transmission > processing > INFORMATION for use in planning and management.

Data

Raw data are the discrete observation of attributes, variables and events. Regarding health statistics, any data obtained directly from the mouths of a person is *primary*, and all other sources (i.e. hospital registers) are *secondary*. The statement "there have been 125000 new small cars bought in India since last year" is an example of a *datum* (singular for data).

Information

Reduced, summarized data adjusted for variations (e.g. age and sex) so that comparison over time and place is possible. Whilst data is presented in large tables, information is presented in a more palatable form like charts, pictograms or plain text.

Intelligence

Intelligence is the *transformation* of information, through integration with experience and perception, based on the social status of the person and his political[275] standing. Unlike data and information, which is open for everyone to see, **intelligence** is every man's own perception over the same information. It is entirely cerebral and very difficult to express to others without the proper linguistic skills.

Criteria for health information systems

1. Based on population (raw data should come directly from the target population)
2. Avoids unnecessary agglomeration of data.
3. Problem oriented.
4. Employs functional and operational items (thing which can be readily measured, like episodes of illness and laboratory tests).
5. Information should expressed briefly and imaginatively (more with pictures than tables).
6. Provision for feedback data (what people think about health services).

What kind of information do we need?

1. Demography, vital events
2. Environmental conditions
3. Health status (morbidity, mortality, disability, quality of life)
4. Health resources
5. Utilization of health services
6. Outcome of health services
7. Financial statistics.

Uses of health information

1. Know health status of people
2. Quantify health problems
3. Know health care needs
4. Compare health status between localities, states and nations

5. Planning, administration and effective management
6. Assess attitude and satisfaction of beneficiaries
7. As a research and education tool.

Sources of information

Census

A **census** is a generic term for collecting data about *every* member of a population (a census of lions in Africa). The **demographic census** is the total process of

- collecting, compiling and publishing
- demographic, economic and social data
- pertaining at a specified time
- to *all* persons in a country/area.

The census in India is usually carried out between January-March in the 1st year of each decade. The 1st census was carried out in 1881, and each 10 years since.

Information gathered in census

1. *Demography*: Population size, composition, distribution, fertility (no. of surviving children, no. of live borns, martial status, age at marriage), migration.
2. *Socioeconomy*: Education, income, occupation, language, religion, condition of housing, work and workplace, disability. The 2011 census will also include cast.

Methods

1. *De facto*: Count actual population present on day of counting.
2. *De jure*: Count only permanent residents of an area irrespective of their presence on that day.

Phases of census

1. House listing—Completed an year before the census.
2. Enumeration—Done at 4th week of February; the result of census is referred to as '1st March population'.

Uses of census

1. Vital tool for planning and policies
2. Provides denominator for health indices (i.e. mid year population, population in a certain area exposed to some risk, etc.).
3. Gives overall idea of population composition, size and growth rate.

Methods to determine intercensal population

Natural increase method. If registration of births and deaths is reliable enough, then naturally, population in 2006 = population in 2001 census + (total births + immigrations into India) – (total death + emigration from India).

Arithmetic progression method. Assuming a constant growth rate of population, population on 1st July, 2006 (the mid year population) = population on 1st March, 2001 + (5 × annual growth rate) + (4 × monthly growth rate).

Census, 2011

Census 2011 is the 15th Census of India since 1872, held in two phases: House listing and Housing census (April to September 2010) and Population Enumeration (9th to 28th February 2011). It was the first census to take cast into account.

Figures at a glance[276]

- Population: 120193422
- Growth from 2001: 17.64%
- Density: 382/km^2
- Sex ratio: 940
- Literacy: 74.04% (m – 82.14%, f – 65.46%).

Registration of vital events

In a welfare state, the state acknowledges each citizen the right to life, education and health. To ensure these rights, however, the state must *know* whether a certain person exists or not. The process of registration of vital events is the—

- Legal recognition, statistical recording and reporting.
- Of the occurrence and collection, compilation, presentation, analysis and distribution.
- Of statistics pertaining to vital events, i.e
  - Live births
  - Deaths
  - Fetal deaths
  - Marriages
  - Divorces
  - Adoptions
  - Legitimations
  - Recognition
  - Annulments and legal separations.

Central birth and death registration act, 1969

1. **Time limit for registration**—Birth → 14 days; death → 7 days (both 21 days in West Bengal).
2. **Responsibility**—Parents, spouse, head of hospital/nursing homes/jails where the event has occurred.
3. **Defaulters**—Fine of Rs. 50.

Organization

CENTER Registrar General of India
↑
STATE
Chief registrar of birth and death
Deputy Chief registrar of birth and death
Additional Chief registrar of birth and death
(the SBHI or ADHS)

↑

DISTRICT
District registrar of birth and death
Additional District registrar of birth and death
(the District Magistrate/ CMOH I/ CMOH II)

↑

BLOCK
Registrar of birth and deaths
(the Block Sanitary Inspector)

↑

VILLAGE/TOWN
Subregistrar of birth and death
(Panchayat pradhan/Municipal officer)

Lay reporting

One of the functions of the MPHW is to register vital events. This is necessary because the 'official' system is grossly under reported.

Sample registration system

Since civil registration system is grossly under reported, an SRS was initiated in 1960s to provide estimates of birth and deaths. It is a dual recording system.

Components

1. A sample is selected from target population and its vital events recorded continuously. School teachers are used as enumerators (informants).
2. Every 6 months an independent survey of births and deaths is done over the same sample.

The data from the two surveys should match. If they do not, a field reverification of unmatched/ partially matched events is carried out. Estimation of missing events can be calculated with Chandrasekhar-Deming formula.

Disease notification

Notification is necessary for initiating early control of diseases. Failure to notify highly communicable diseases may result in epidemics.

Diseases notifiable to WHO

1. Under International Health regulation: Cholera, plague, yellow fever.
2. International surveillance: Louse typhus, relapsing fever, poliomyelitis, influenza, malaria, rabies, salmonellosis.

Disadvantages

1. under reporting
2. subclinical/atypical cases remain hidden
3. depends upon untrained workers who often make wrong diagnosis.

Hospital records

Advantages

1. The geographic **area** of the patient can be known.
2. Age sex composition easily available.
3. Associated **diseases** of patients are found.
4. **Risk factors** that preceded the development of disease can be recorded (from the history sheet).
5. **Expenditure** per patient may be measured.
6. Only hospital data gives the **bed occupancy rate**, **turnover rate and duration of stay**, which are useful in planning for health services.

Disadvantages

1. Only clinical cases (tip of the iceberg) come to hospital.
2. Admission policy may vary from hospital to hospital, and even between attending emergency physicians; the same patient with chest pain can be regarded a case of IHD in one hospital and GERD on the other.
3. Because hospitals have no catchment area hospital records only give numerators (i.e. number of people affected with diseases), not denominators from which rates can be calculated.

Disease register

It is a permanent record where cases of a certain disease are followed up, basic statistic tabulation is done, and patients are subjected to special studies. This register usually holds detailed information on duration of illness, case fatality/survival, frequency of disease, natural causes of disease and prognosis. But only a few diseases have a permanent register in India (stroke, AMI, cancer, blindness, congenital defects, tuberculosis, leprosy).

Record linkage

To maintain all the health information of a person (name, birth, sex, blood group, marriage status, history of previous illnesses, current illnesses, any genetic disease, any psychiatric illness, number of hospital admissions, etc.) in a single register. This is still to be implemented in India (but some countries have done it and given everyone a 'Social security number'; this number could be punched in a central server to access all information regarding that person). Recently (2009) the central government has planned to issue ID cards and numbers to all Indians.

Surveillance

Surveillance programs for malaria, tuberculosis, leprosy, filariasis are important source of data.

Other health service records

A lot of information is also found in special clinics in hospitals, polyclinics, private practitioners, abortion centers, MCH centers, school health records, ICDS centers, etc. However, these data are not regularly monitored and are inherently segmental (cannot be integrated easily into national data, because they work in isolation within a very limited zone).

Environmental data

These schemes run only in a piecemeal manner in India: one NGO measures radiation in Benaras, another measures dust in Kolkata air and so on. But these remain to be integrated by the government into an unified environmental monitoring scheme.

Health manpower statistics

The MCI maintains an excellent database of doctors in India. The Department of Health and Family Welfare keeps a similar record of all health care staff.

Economic data

The consumption of goods, employment rate, per capita income, etc. are indicators of health, as discussed in first chapter. The Ministry of Finance usually maintains all economic data pertaining to the country.

Social insurance schemes

Insurance schemes are very tightly regulated (every episodes of illness of every insured person is rigidly and unambigously recorded by agencies, because there are monetary compensations involved), and thus make it possible to study the occurrence and prognosis of illness in *insured* population, as well as data on sickness absenteeism.

Surveys

Surveys can inquire into three aspects

1. health problems – distribution in time, place, person
2. analysis of the problems
3. availability and use of health services.

The survey is conducted in 5000–10000 households – A size considered adequate to provide national estimates on health status, anthropometry, food consumption, income, expenditure, housing, literacy, etc. Follow ups give more accurate results.

The **National Sample Survey Organization** conducts country wide interview based surveys on morbidity, family planning and vital events. To nullify the effects of an interviewer, they usually *mail* the questionnaire over post. This is effectively double blinding, because there is no interviewer involved. The disadvantage of this process is that some level of education in the part of the target population is necessary for a response, and rate of nonresponse is very high.

The cheapest survey is of course a Health Record Survey (scanning the pages of the register in a PHC/ hospital). But this kind of survery is not *population* based, data is *unreliable* and lack of uniformity/standardization (each hospital may record different parameters of the patient in a different tabulation form) makes difficult to integrate large amounts of data.

PLANNING

Planning is one of the managerial tasks a doctor needs to perform. It is the *organized, conscious and continual attempt to select the best available method to achieve a goal*.

Steps

Make a plan → Execute it →; See whether it works.

Needs of the community

There are two kinds of need

1. Felt need – what the people *feel* they need
2. Scientific need – what will actually do them good

The two often mismatch. For example, you think that a school in a village will do the people good. The people want a cinema hall instead.

Resources to fulfil this need

There are four kinds of resources (and this applies to every sector, not only health)

1. **Men (or women)** who will do the work
2. **Money**, which is the universal incentive
3. **Material**, ranging from drugs, vaccines, good road, vehicles, laboratory facilities
4. **Time**.

If we had all the best men, all the money and all the material for all the time, we would not have needed planning. But as I have iterated time and again, RESOURCES ARE ALWAYS LIMITED. The **purpose** of *Planning* is the method to invest these resources in the most effective manner.

What you want to achieve by planning

1. A GOAL is a large, final, almost vague aim (which is seldom achieved, or it is difficult to tell when it is achieved) like 'Health for all'.
2. An OBJECTIVE is a defined end of certain kind of activities, when couple protection rate > 60% you can stop worrying about family planning (supposedly).
3. A TARGET is an end in itself, usually in small quanta of activities, often limited in time (distribute 1097 condoms in a month or examine 100 slides a day).

How do you do that?

A PLAN is blue print for action. It consists of

1. A POLICY is a *guiding principle* to abide ('National Population Policy' or 'non violence policy') – but not inviolable commandments (a 'doctrine' or 'manifesto', on the other hand, is something to be rigidly adhered to, like the 'communist manifesto').
2. A PROGRAM is the actual implementation of a policy to accomplish the objective.
3. A SCHEDULE is the time-routine of the program.
4. A BUDGET is the money in hand.

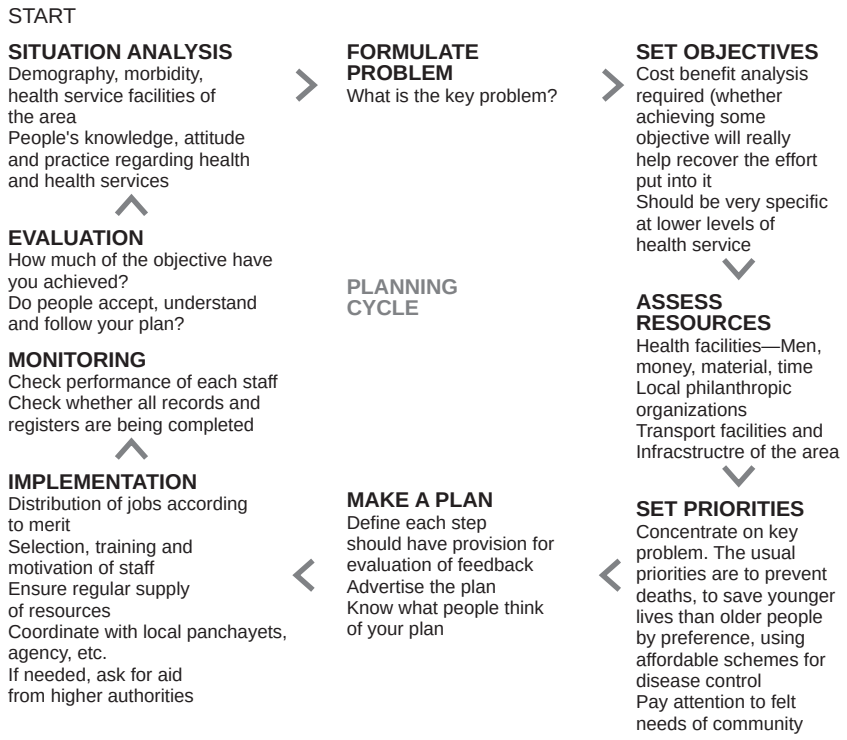

Figure 6.7. Planning cycle

The planning for the whole country, in general, is carrried out by the **Planning Commission of India**, which, since Independence, has published 5 years plans. The latest of such is the 11th five year plan (2007–2012).

Landmarks in health planning

Table 6.2. Landmarks in health planning

	Aims	Recommendations
Bhore Committee 1946	Survey health condition and organization; recommend for future developments	Integration of preventive and curative services at all administrative levels **Setting up 'Comprehensive health service'** with primary and secondary levels First suggested a **primary health center** to cater a population of 10000–2000 with 6 medical officers 3 months training in preventive and social medicine in MBBS course to make *social* physicians (i.e. that *you* have to read this book is because of Bhore committee

Contd...

Contd...

	Aims	Recommendations
Mudaliar committee (Health survey and planning committee) 1962	Assess performance of health sector Identify health needs and resources	Strengthen PHCs Strengthen subdivisional and district hospitals with specialists 'All India Health Service', similar to administrative and policy services Improve medical education, research and health organization
Kartar Singh Committee (Committee on MPHW and FP) 1973	Study the framework for integration of service and feasibility of having MPHWs	Various categories of peripheral workers to be organized into single cadre of MPHWs The auxilliary nurse midwives to be renamed MPHW female The posts of basic health worker, vaccinator, malaria surveillance, health education assistance were merged into MPHW male

7

Health and the Planet

KEY FEATURES

- HEALTH INTERNATIONALE
  - Beginnings
  - WHO's WHO
  - United Nations International Children's (Emergency) Fund
  - International Committee of the Red Cross
- OTHER INTERMEDIATE HEALTH ORGANIZATION

HEALTH INTERNATIONALE

Beginnings

As people came to realize that nothing is more 'international' than a pandemic, they enforced a 40 day quarantine on international travelers, specially sailors and tradesmen, who were recognized modes of transmission. Because of the trade inconvenience and subsequent political friction it had generated, the European countries organized the **first International Sanitary Conference** (1851) without much effect.

The next milestone happened to be in the Americas. The **Pan American Sanitary Bureau** (1902) was the **1st health agency** of the world, who devised the pan American sanitary code (which, by the way, is still in effect in the USA). Later, the same went on to be the Pan American Health Organization, the WHO wing in America.

WHO's WHO

After World War I, the League of Nations was formed and its Health Organization came to exist in 1923. Although the league was failure, its health organization did significant work in the public health sector. After the second war, the league was dissolved but the Health Organization continued as WHO.

The constitution of WHO was passed on **7th April, 1948** – renowned as the **World Health Day**. All members of UN + Switzerland ascribed to WHO.

The WHO is divides in 7 regions, of which New Delhi is the headquarter of South East Asia Region.

Objectives

'Health for all'—To attain a level of health by everyone as to live a socially productive life; the 1978 Alma-ata conference shifted the WHO approach to primary care.

Functions of WHO

The WHO acts as coordinator for health functions of all the member nations.

Prevention and control of diseases

Almost all communicable diseases have been subject to WHO focus sometime or the other. The eradication of smallpox is a magnificent example. With the same energy and vigor who is now battling against AIDS, and up against measles and poliomyelitis.

The WHO regularly carries out epidemiological surveillance of communicable diseases, chiefly on diseases subject to **international health notification** (cholera, plague, yellow fever) and other diseases under **international surveillance** (malaria, poliomyelitis, influenza, louse borne typhus, rabies, salmonellosis) This prevents the spread of diseases across boundaries by early notification. Member nations can also make use of WHO' emergency scheme for epidemics, in case diseases gets out of hand.

The WHO has published guidelines for the control of major NCD, and its range extends to vector control, immunization, quality control of drugs, health laboratory set ups and drug evaluation. The Expanded Program on Immunization is now a priority concern of WHO.

To build up comprehensive health services

The WHO helps member nations to build their own comprehensive health service by training manpower and providing resources, setting up laboratory, distributing drugs, etc. The leprosy eradication program is typical of a WHO guided program.

Family health care

The health of the mother and child is of greatest concern to WHO, as well as the improvement of quality of life.

Environmental guarding

WHO advises governments on providing basic sanitary facilities, protection of air, water and soil, occupational health, radiation hazards. A number of programs has been devised such as the Environmental Health Criteria Program.

Health information

The WHO has the following publication lines

1. weekly epidemiological record.
2. world health statistics quarterly.
3. world health statistic annual.
4. the International Classification of Diseases, published every 10 years.
5. the WHO website.
6. the Medical Literature Retrieval and Analysis System (MEDLARS)–at National Library of medicine (www.nlm.nih.gov).

Research

The WHO does not actually do research, but coordinates and funds research all over the world. Six tropical diseases—Malaria, leishmaniasis, schistosomiasis,

trypanosomiasis, filariasis, leprosy are the targets of WHO special program for Research and Training for Tropical Diseases.

Coordination

The WHO collaborates with UNICEF and FAO, etc.

WHO activities in SEAR (SOUTH EAST ASIA REGION)

- Malaria eradication
- Tuberculosis and leprosy control
- LF elimination
- Health laboratories service
- Vaccine production
- Drug distribution
- Health statistics
- Public health administration
- MCH, nutrition, immunization
- Health and medical education

International health regulations

The International Health Regulations (IHR) are an international legal instrument that is binding on 194 countries across the globe. Their aim is to respond to acute public health risks that can cross borders and threaten the entire humankind.

Need

In the globalized world, diseases like SARS and influenza can spread far and wide, via international travel and trade. Noninfectious emergencies such as chemical spills, leaks and dumping, or nuclear melt-downs can also affect a large part of the globe. The IHR aims to limit interference with international traffic and trade while ensuring public health through.

Strategies

The IHR, which entered into force on 15 June 2007, require countries to

- **Report certain disease outbreaks** to WHO.
- Strengthen their existing capacities for public health surveillance and response.

Notifiable diseases

1. Should always be notified: Smallpox, wild type poliomyelitis, human influenza caused by a new subtype, severe acute respiratory syndrome (SARS).
2. Should be notified depending on magnitude of problem: Cholera, pneumonic plague, yellow fever, viral hemorrhagic fevers (Ebola, Lassa, Marburg), West Nile fever, other diseases that are of special national or regional concern, e.g. dengue fever, or any event with unknown case that could cause public health problem.[277]

United Nations International Children's (Emergency) Fund

The UNICEF was found in 1946 to provide emergency care to war stricken children. But subsequently, it came to reside on a permanent basis and the 'emergency' was erased. The headquarters of UNICEF are at the UN building, New York. Delhi is the headquarters from south central Asia region. Earlier, UNICEF worked together with WHO on urgent problems like Malaria, Tuberculosis and STDs. The recent trend, however, is to focus on campaigns providing direct benefit to mother and child. The concept of 'whole child' has come up, in which the focus is not only the health and nutrition of the child, but his personal development and the development of the country at large. This is known as country health programming.

Functions

1. **Child health care**: In India, UNICEF has made a BCG + DPT plant, iodized salt plant and drinking water plant. It has also emphasized safe drinking water, immunization, sanitation and family planning.
2. **Child nutrition**: In the 1950s UNICEF began supplementing low cost protein rich diets, and helped nation, including India, in the 'Applied Nutrition Program'. UNICEF also made dairy plants in India, and provided specific prophylactic measures like Vitamin A prophylaxis schedule, iodized salt, IFA supplementation and food fortification.
3. **Family and child welfare**: UNICEF encourages improved child care both in and out of home through parent education, day care centers, child welfare agencies and women's clubs.
4. **Education** (formal/nonformal) to children and mothers.
5. **Urban basic project**: To upgrade basic health care services for women and children of urban areas, emphasizing women to income and improve economic standards.

Principal activities

- Growth monitoring
- Oral rehydration therapy
- Breastfeeding
- Immunization.

International Committee of the Red Cross

The International Red Cross was founded by Henry Dunant in 1864. The 1st meeting was held in Geneva in 1864, and a treaty was signed for a nongovernmental, neutral organization with the red cross emblem to aid wounded soldiers of war. The Red Cross has since been the symbol of neutrality and brotherhood, can be used only by military personnel and red cross members. Soon, Red Cross extended its services to disasters, war veterans, first aid and nursing refugee health services.

Indian Red Cross

Indian Red Cross was established in 1920 with objectives of health promotion, disease prevention and disaster mitigation. In peacetime, the society serves army

hospitals with newspapers, periodicals, music and other comfort goods and also services for exservice men.

In times of disasters, the society provides milk, medicine, vitamins, codliver oils and a hundred other ancillaries. It is involved in MCH and FP clinics in several cities and some branches have started blood banks of their own. The **Saint Johns Ambulance Association** is a 1st aid branch of the Indian Red Cross.

The **Junior Red Cross** offers boys and girls to have a hands on experience of village uplift, first aid and epidemic management.

OTHER INTERMEDIATE HEALTH ORGANIZATIONS

The **International labor organization (ILO)** is concerned with the working class (from miners to blue chips). FAO is based in Rome and looks over our alimentary needs; it works closely with United Nations Development Program (UNDP).

Cooperative for assistance and relief everywhere (CARE) was founded in North America in 1945, and has grown to be a large body of nongovernmental, nonprofit relief and development organization. In India, CARE works hand in hand with ICDS and also many child health and nutrition programs.

8 Noncommunicable Diseases

KEY FEATURES

- PROBLEM
  - Epidemiologic transition
- CARDIOVASCULAR SYSTEM
  - Problem
  - Summary of cardiovascular risk factors
  - Ischemic heart diseases
  - Hypertension
  - Cerebrovascular accidents (stroke)
  - Rheumatic fever/rheumatic heart disease
- CANCER
  - Problem
  - Agent
  - Host
  - Prevention
  - Epidemiology of selected cancers
- ENDOCRINE
  - Obesity
  - Diabetes mellitus
- EYES
  - Blindness
- ACCIDENTS
  - Problem
  - Agent
  - Host
  - Environment
  - Road traffic accidents
  - Preventing accidents

NCDs differ from communicable disease in few important points,

1. They are noncommunicable.
2. They **do not have a defined incubation period**—Chickenpox has an incubation period between 7–21 days, but we can never tell when a person affected with insulin resistance will begin to show features of Type II diabetes.
3. Agents are vague (**multifactorial causation**)—We can safely say that a certain herpes virus (varicella zoster virus) causes chickenpox, but we are not exactly sure what, if anyone agent, causes diabetes.
4. **Natural history if often uncertain/unpredictable**—Most infectious diseases follow a certain pattern of clinical presentation (i.e. chickenpox shows up as polymorphic vesicles spreading centrifugally, along with fever; but there are a myriad of ways in which diabetes can present.
5. The **boundary between diseased and nondiseased is very blurry**—One either has or does not have chickenpox, but regarding diabetes, one can have anyone of obesity, metabolic syndrome, impaired fasting glucose, impaired glucose tolerance, insulin resistance or frank diabetes; it's hard to say at exactly what point of time someone acquires diabetes.
6. **Slowly progressive**—Barring some exceptions (i.e. leprosy), most infectious diseases have rapid progress which results in either death or complete cure; but diabetes won't kill you quickly, it will slowly gnaw at your eyes, nerves and blood vessels.

7. **Usually nonreversible and permanent pathology**—Infectious diseases are the only 'curable' diseases in the trust sense of the word, most noncommunicable diseases are not.
8. Leave some **residual disability** and rehabilitation is necessary—Most communicable diseases (except some like leprosy) can be completely cleared from the body, but not diabetes, which will usually produce some disability (blindness, stroke and residual weakness) over long term.
9. Definite **lag period** between behavioral change and disease progression—There is a certain gap between change of food habits (eating refined carbohydrates and fats) and development of Type II diabetes.

PROBLEM

Erstwhile a problem of developed nations, NCDs are rising in developing countries due to

1. Demographic transition (greater life expectancy).
2. Medical transition (most people now survive a single myocardial infarction, to suffer from a second).
3. Risk transition (we are acquiring all the bad habits of the west).

Epidemiologic transition

The epidemiologic transition theory was proposed in a 1971 paper by AR Omran.[278] The "epidemiologic transition" refers to relatively constant patterns of changes in patterns of disease as societies develop. With a somewhat Biblical turn of phrase, Omran named these the "age of pestilence and famine," "the "age of receding pandemics," and the "age of degenerative and man-made diseases."

Age of pestilence and famines	Age of receding pandemics	Age of degenerative diseases
Frequent epidemics and famines Endemic infections and parasitic diseases Chronic malnutrition Maternal and child health problems Serious environmental health problems: Unsafe water, fecal waste contamination, insects and rodents, poor housing.	Epidemics and famines somewhat reduced in frequency People live long enough for heart disease and cancer to occur Infection and parasitism somewhat reduced Occupational health problems rise Sanitation begins to improve Urban health problems become acute Accidents at home and in industry.	Morbidity overshadows mortality as the main issue Chronic disease, mental illness, drug addiction, pollution rise in importance Infectious disease mainly in certain pockets of population Geriatric problems take over from MCH Electrical or chemical hazards become main occupational health dangers Rising cost of medical care.

CARDIOVASCULAR SYSTEM

As in other forms of machinery, it is the moving parts that go astray first. The circulatory system – the pulsating heart and arteries – is man's Achilles' heel in the long run. His progress in conquering death has raised disorders of this system to the rank of the number one killer. Circulatory diseases are responsible for just over half the deaths in the US, and out of these diseases, as single one, atherosclerosis, accounts for one death out of four."

--Asimov, Isaac: Asimov's Guide to Science

... I come not, friends, to steal away – your hearts...

--Shakespeare, William: 'Julius Caesar', Act III, scene ii

In today's world, most of the deaths are due to noncommunicable diseases (32 million, and just over half of these are a result of cardiovascular diseases. Even in some developing countries cardiovascular diseases have become first or second causes of death.

Problem

Some figures[279]

1. CVDs are the number one cause of death globally: more people die annually from CVDs than from any other cause.
2. An estimated 17.1 million people died from CVDs in 2004, representing 29% of all global deaths. Of these deaths, an estimated 7.2 million were due to coronary heart disease and 5.7 million were due to stroke.
3. Low- and middle-income countries are disproportionally affected: 82% of CVD deaths take place in low- and middle-income countries and occur almost equally in men and women.
4. At macroeconomic level, CVDs place a heavy burden on the economics of low- and middle-income countries. Heart disease, stroke and diabetes are estimated to reduce GDP between 1 and 5% in low- and middle-income countries experiencing rapid economic growth, as many people die prematurely.

Beyond the figures

I do not need to reiterate the problem of heart diseases. Most of you students come from moderately affluent families living in middle to upper socioeconomic strata. How frequently have you heard of somebody dying from cholera or malaria or meningococcemia? And how frequently have you seen deaths due to a 'heart attack' or 'stroke'? A century earlier, when it came to diseases, people talked about cholera and plague and malaria. Today, at least to the educated middle and upper class, they are mere fragments of history (ok, malaria still lives, but not up to its earlier proportions). Today people talk about their hearts, their serum cholesterol, their waistline, lipid lowering drugs, angiographies and bypass surgeries. The statement of mere numbers cannot describe the problem of cardiovascular disease in its entirety. **Cardiovascular diseases have changed how we think about diseases**, i.e. they have had a sizeable impact on our social psyche.

The present mortality rates from cardiovascular diseases are the consequences of previous (20–30 years ago) exposure to behavioral risk factors such as

inappropriate nutrition, insufficient physical activity and increases tobacco consumption. It is called the 'lag time' effect.

Summary of cardiovascular risk factors[280]

Behavioral risk factors are responsible for about 80% of coronary heart disease and cerebrovascular disease. Over 300 risk factors have been associated with coronary heart disease and stroke. The major established risk factors meet three criteria

- A high prevalence in many populations.
- A significant independent impact on the risk of coronary heart disease or stroke.
- Their treatment and control result in reduced risk.

Most risk factors are planted in childhood (getting addicted to refined carbohydrates like candy and soft drinks) and adolescence (smoking and alcohol). Our cultural beliefs prohibit children from smoking, but not from eating candy, burgers, cola and french fries, which are just as harmful. Add to that the millions of dollars spent on advertising for fast food.

Major modifiable risk factors

1. Hypertension
2. **Dyslipidemia**— ↑ total cholesterol, ↑ LDL and ↑ triglycerides.
3. smoking.
4. Obesity.
5. **Unhealthy diet**— ↓ fruit, vegetable and fish oils, ↑ saturated fat and refined carbohydrates.
6. Diabetes mellitus.
7. Physical inactivity.

Other modifiable risk factors

1. *Lower* socioeconomic classes—As the rich (and developed countries) have become aware of cardiovascular risk, most CVD is happening in middle to lower classess in developing countries.
2. Alcohol (upto one or two drinks a day actually reduced CVD risk, but not more than that).
3. ↑ lipoprotein (a).
4. Psychosocial stress.

Nonmodifiable risk factors

1. Advancing age
2. Family history
3. Male sex.

"Novel" risk factors

1. ↑ homocysteine in blood
2. Abnormal coagulation– ↑ fibrinogen
3. Subclinical inflammation– ↑ C reactive protein.

Ischemic heart diseases

All the knowledge I possess everyone can acquire, but my heart is all my own.

--Johann Wolfgang von Goethe: The Sorrwos of Young Werther

Impairment of heart perfusion compared to its need, due to narrowing of vessels; it may take the form of angina, myocardial infarction, heart failure or sudden cardiac death.

Problem

In most western countries, 30% of all deaths in men, 25% in women are caused by IHDs. **Case fatality** = 25–28% within 28 days (and 55% within first hour). It eats about 3.4–9.4 years of life expectancy of men, and even greater for women. IHDs are a 'modern epidemic', one spread not from person to person but due to a lifetime of bad habits. After sweeping the developed countries, CHD is on the decline (although still very prevalent) there, while affecting more the developing countries.

The ischemic heart diseases have a kind of 'incubation period' of 10 years, i.e. the lag period between behavioral change and onset of disease.

Agent (Fig. 8.1)

Dyslipidemia

Of all lipids, LDL cholesterol is most directly related to IHD. Current recommendation is to screen for blood lipids in all adults over 20. The screen should include a fasting lipid profile (total cholesterol, triglycerides, LDL cholesterol and HDL cholesterol) repeated every 5 years. Apo B and Apo A are probably better indicators than the lipoproteins themselves.

Lipid goal. HDL > 45 mg/dl; LDL < 160 mg/dl; total cholesterol / HDL < 3.5

Smoking

A uniquely human habit, smoking is an important, but reversible risk factor. Smoking cause atherogenesis by releasing carbon monoxide and ↑ sympathetic tone (and possibly also by endothelial damage and setting up microinflammation inside vessels). *Bidis* are safer than cigarettes as they produce no carbon monoxide.

Smoking + cholesterol is a synergistic risk factor, as seen in this graph showing risk of ischemic heart disease with increasing BP in four groups of people.

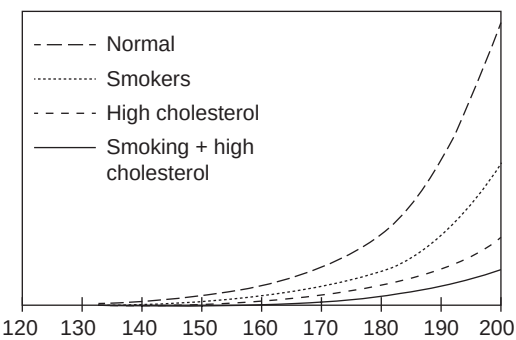

Figure 8.1. Incidence of IHD with increasing BP in four groups of people

Hypertension

The strongest risk factor for IHD. CVD risk doubles for every 10 mm increase in DP or 20 mm increase in SP.

Diabetes mellitus, insulin resistance and the metabolic syndrome

Most patients of diabetes mellitus die of atherosclerosis and its complications. Ageing and rampant obesity in India underlie a current epidemic of Type II diabetes mellitus. The abnormal lipoprotein profile associated with insulin resistance, known as 'diabetic dyslipidemia', accounts for part of elevated cardiovascular risk in patients with Type II diabetes. While diabetic patients often have LDL cholesterol near average, the LDL particles are smaller, denser and more atherogenic. There are also low HDL and high triglycerides. Hypertension often accompanies diabetes and indeed, this cluster of risk factors is now known as the 'metabolic syndrome'. Diabetes mellitus may also cause 'silent' AMI. The target is to keep sugar under 120 at all times and BP < 130/85.

The metabolic syndrome—Any three risk factors of

- Abdominal obesity (waist circumference): Men > 102, women > 88 cm
- **BMI** > 30
- Triglycerides > 150 mg/dl
- HDL cholesterol: Men < 40, women < 50
- Blood pressure > 130 or > 85 mm
- Fasting glucose > 110 mg/dl.

Male sex/postmenopausal state

Decades of observational studies have verified excess coronary risk in men compared with premenopausal women. After menopause, however, both become equal. In this regard, estrogen has been found to increase HDL and reduce LDL.

Dysregulated coagulation or fibrinolysis

Fibrinogen levels correlate with coronary risk; Lp(a), a lipoprotein modulates fibrinolysis, and is a risk factor too.

Risk factors of IHD exclusive of LDL cholesterol

- Smoking.
- Hypertension (BP > 140/90 or anybody on antihypertensive drugs) .
- HDL < 40 mg/dl.
- Diabetes mellitus.
- Family history of premature IHD.
- Age (men > 45, women > 55).
- Obesity (BMI > 30), physical inactivity.
- Alcohol > 75 g/day.
- Male sex/ postmenopause/OCP intake.
- Type A personality (the outgoing ones).
- Lack of dietary fibers, high cholesterol diet, too soft water, deficiency of poly-unsaturated fatty acids, too much salt.

Prevention

Primordial prevention

1. Preserve traditional food habits, implement 'dietary goals' (see the chapter on nutrition).

2. Avoid initiation of smoking, fast foods, colas and candies.
   Schools play the most important role in primordial prevention.

Primary prevention (for everybody in population)

Specific protection. Prudent diet, abstinence from smoking and alcohol, control of stress and hypertension.

Secondary prevention (for those with risk factors)

Screening for hypertension, hypercholesterolemia, diabetes and medical management of such diseases. Screening is recommended each 5 years in all adults over 20. Because the treatment of an acute episode of IHD is costly and not successful in many cases, setting up more ICUs is not an effective community intervention.

Tertiary prevention

Lifelong β blockers and Aspirin, angioplasty, extended benefit under Employees State Insurance.

Hypertension

Definition

Table 8.1. JNC VII definitions on hypertension[281]

	SP	DP
Normal	<120	<80
Prehypertension	120–139	80–89
Stage 1 hypertension	140–159	90–99
Stage 2 hypertension	> 160	> 100

Problem

In most countries, upto 30% of adults suffer from high blood pressure and a further 50–60% would be in better health if they reduced their blood pressure. In India, the average blood pressue in urban men has become 130 mm Hg in 1997, from 120.4 in 1942.[282]

Agent

1. **Primary/ 'essential'/ idiopathic** hypertension—Increases with age, obesity, high salt intake (7–8 g/day), ↓ dietary fibers, psychosocial stress; it is often inherited and is being seen more in lower socioeconomic classes.
2. Renal—Acute/chronic glomerulonephritis; renal artery stenosis.
3. Endocrines—OCP intake, adrenocortical hyperfunction, pheochromocytoma
4. Others—Coarctation of aorta, toxemia of pregnancy, 'white coat' hypertension (i.e. on seeing a doctor).

Prevention

Primary prevention

Population strategy

1. Follow 'dietary goals' by WHO
2. Salt < 5 g/day

3. Weight reduction, physical exercise
4. Changes in lifestyle to adopt to stress.

High risk strategy

Tracking of BP shows people with initial measures closer to threshold have more chance to develop hypertension over years.

People with one or more risk factors should be intensively educated on the consequences and management of hypertension. Provision of 'self-check' BP machines increases their involvement in management.

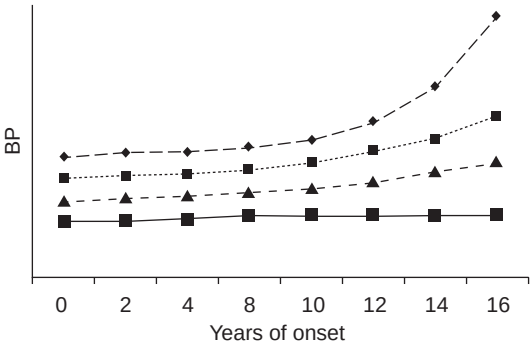

Figure 8.2. People with higher BP at a young age develop higher BP over time

Secondary prevention

Screening for hypertension is not fruitful, as BP varies considerably between observers, observations and even time of the day. If blood pressure of every person who comes in contact with health care is measured, it effectively serves as screening; the following schedule should be followed if hypertension is diagnozed.

1. Measure BP thrice.
2. History—Duration, history of treatment, family history, other diseases, lifestyle.
3. General survey.
4. Systemic examination including fundoscopy.
5. Labs—ECG, urinalysis, serum electrolytes, urea and creatinine, fasting glucose, cholesterol, HDL.

Treatment of hypertension should be sought in a textbook of Internal Medicine.

Cerebrovascular accidents (Stroke)

Rapidly developing clinical signs of *focal/global disruption* of cerebral circulation, lasting > 24 hours *or* leading to death, with no apparent cause other than vascular phenomena.

Stroke carries a high risk of death. Survivors can experience loss of vision and/or speech, paralysis, and confusion. Historically called "apoplexy", "stroke" is so called because of the way it strikes people down.

Agent

- Ischemic stroke—Due to thrombosis/embolism of a vessel.
- Hemorrhagic stroke—Usually due to hypertension.

Host

Aged, usually male, frequently diabetic, smoker, hyperlipidemic; women who use **OCPs** for long time may have a stroke (however, modern low dose OCPs bear little risk of stroke).

Prevention

Even where advanced diagnostic and therapeutic facilities are available, 60% of all those who suffer a stroke either die or live with some disability. Thus curative treatment is not a viable option for reducing deaths or DALY loss from stroke.

Primary prevention

1. Control hypertension—For every ten people who die of stroke, four could have been saved if their blood pressure had been controlled.
2. Control diabetes.
3. Stop smoking.
4. Education regarding risk factors of stroke.

Secondary prevention

Early detection of stroke (slurring of speech, weakness in any limb); often, a major attack is preceded by some transient ischemic attacks (TIA).

Tertiary prevention

Rehabilitation of residual paralysis by physiotherapy, speech therapy.

Rheumatic fever/Rheumatic heart disease

It is the commonest heart disease in 5–30 year age group, but it does not stop there and goes on to produce valcular disease which cause a lifetime of disability. An estimated 6.63×10^6 DALYs were lost due to rheumatic fever in 2000[283] worldwide. In India, the causative organism is very commonly found in children, and 1–3% of all sore throat cases develop rheumatic fever.

Agent

- Group A β hemolytic streptococci—M types such as 1, 3, 5, 6, 14, 18, 19 and 24 have been associated with RF. The tremendous antigenic variation in this bacteria prohibit the development of a vaccine.
- Coxsackie B4 virus.

Host

Commonest in children (of both sexes) between 5–15 years. There are every known types of carriers of Streptococci, which makes eradication an impossibility.

Environment

Poverty, malnutrition, overcrowding, poor housing, lack of awareness often go with rheumatic fever. It is even more distressing that often the disease is allowed to progress to its 'valvular' stage due to misdiagnosis/lack of expertise of doctors.

Clinical course

The WHO 2002–2003 criteria[284]

Table 8.2. Revised Jones' criteria

Major	Minor	Others
Carditis	Fever	↑ PR interval
Sydenham's chorea[a]	Polyarthralgia	↑ ASO titer

Contd...

Contd...

Major	Minor	Others
Subcutaneous nodules	↑ ESR	Positive throat culture
Polyarthritis	↑ WBC count	Rapid antigen test for group A Streptococcus
Erythema marginatum	↑ PR interval in ECG	History of scarlet fever
[a] Greek for 'dance'; a choreographer is one who composes dance.		

Table 8.3. Diagnostic chart for rheumatic fever

2 major[a]	OR	1 major + 2 minor	AND	Evidence of group A streptococci infection	Diagnosis
+		+		+	Primary rheumatic fever
+		+		+	Recurrent rheumatic fever without RHD
		+		+	Recurrent rheumatic fever with RHD
[a] Presence of Sydenham's chorea/ rheumatic carditis (valvular heart disease) without any other criteria is suggestive of rheumatic fever with heart disease. Exclude infective endocarditis and congenital heart diseases.					

A large footnote at the bottom of the WHO diagnostic criteria says—

Patients may present with *polyarthritis* (or with only polyarthralgia or monoarthritis) and with several (3 or more) other minor manifestations, together with evidence of recent group A streptococcal infection. Some of these cases may later turnout to be rheumatic fever. It is prudent to consider them as cases of "probable rheumatic fever" (once other diagnoses are excluded) and advise regular secondary prophylaxis. Such patients require close follow up and regular examination of the heart. This cautious approach is particularly suitable for patients in vulnerable age groups in high incidence settings.

Prevention

Primary prevention

Health promotion. Better housing, alleviating poverty and overcrowding, educating people on hygiene.

Specific protection. Ideally, each member of the community should have undergone a throat swab test. Because it is not actually possible, we concentrate on the high risk groups (schoolchildren) and perform screening. In our country, because of high prevalence of Streptococcal infection, every child with sore throat *must* be given Penicillin (or if he is allergic to penicillin, erythromycin) irrespective of throat swab culture.

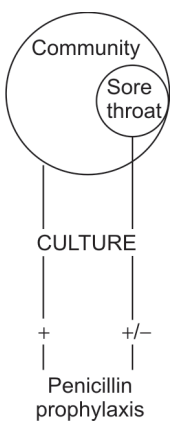

Figure 8.3. Primary prevention of rheumatic fever

Benzathene penicillin 6 lakh (5–10 years) and 12 lakh unit (adults) IM single dose. If valves are involved, continue lifelong.

Secondary prevention

Every case of RF should be given Benzathene penicillin 600000 units in children/1200000 units in adults IM at 3 week intervals till 5 years or the child reaches 18 years of age, whichever is earlier. For rheumatic carditis, treatment must be continued for 10 years (or the patient turns 25). For severe valve involvement, treatment could be continued lifelong.

Because rather than the entire community, we focus only on cases of RF, secondary prevention is much more practicable than primary prevention.

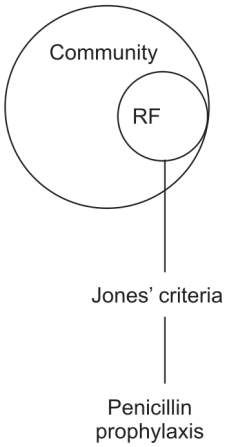

Figure 8.4. Secondary prevention of rheumatic fever

Tertiary prevention

Symptomatic treatment and valve replacement of RHD cases; penicillin must be continued long after surgery.

CANCER

A neoplasm is an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissues and persists in the same excessive manner after the cessation of the stimuli which evoked the change.

Problem

Cancer is a leading cause of death worldwide: it accounted for 7.4 million deaths (around 13% of all deaths) in 2004.[285] However, more than 30% of all cancer deaths are preventable,[286] either by prevention of cancer or by treatment.

In most developed countries, cancer is the *second* largest cause of death after cardiovascular disease, and epidemiological evidence points to this trend emerging in the less developed world. This is particularly true in countries in "transition" or middle-income countries, such as in South America and Asia. Already more than half of all cancer cases occur in developing countries.

Lung cancer *kills* more people than any other cancer—A trend that is expected to continue until 2030, unless efforts for global tobacco control are greatly intensified. Some cancers are more common in developed countries: Prostate, breast and colon. Liver, stomach and cervical cancer are more common in developing countries.

The WHO celebrates **World Cancer Day** each year on 4th February.

Commonest cancers by site

I. Lung—Commonest cancer in the *world* among men
II. Breast—Commonest cancer in the *world* among *women*
III. Colorectal
IV. Stomach
V. Cervix—Commonest cancer in *Indian* women
VI. Prostate
VII. Oral—Commonest cancer in *Indian* men.

Survival

Although breast cancer is second in incidence to lung cancer, it does not cause as much mortality because once diagnozed, it is amenable to appropriate interventions, which lung cancer is often not.

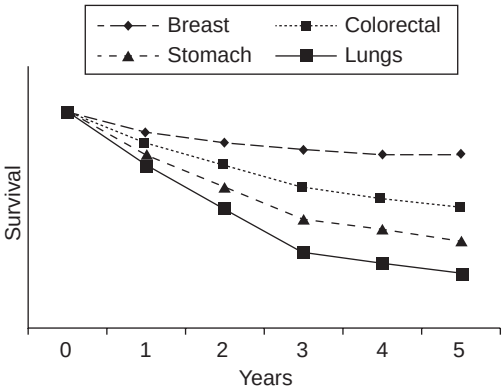

Figure 8.5. Survival rates in cancer

Agent

A carcinogen is any substance, radionuclide or radiation that is an agent directly involved in the exacerbation of cancer or in the increase of its propagation.

- Physical carcinogens, such as ultraviolet and ionizing radiation.
- Chemical carcinogens, such as asbestos, components of tobacco smoke, aflatoxin (a food contaminant) and arsenic (a drinking water contaminant).
- Biological carcinogens, such as infections from certain viruses, bacteria or parasites; such agents are responsible for almost 22% of cancer deaths in the developing world and 6% in industrialized countries.[287]

Table 8.4. Carcinogens

Chemical	
Benzene	Leukemia
Polycyclic hydrocarbons (encountered in industrial and car exhaust)	All cancers
Aromatic amines (those in a cigarette)	Bladder
Occupational exposure to asbestos, chromium, arsenic, nickel, uranium	Many cancers (esp Lung)
Coaltar, X-ray, dyes, heat	Skin
Food	
Ascorbate deficiency	Leukoplakia
↑ saturated fat	Breast, colon
Vitamin A deficiency Betel, areca	Oral, esophageal, colorectal

Contd...

Contd...

Beef Iron deficiency	
Tobacco (either in cigarettes or in its various chewable forms)	Lung, oral
Alcohol	Liver
Smoked fish (a popular dish in Japan), nitrosamines (food additive)	Stomach
Dietary fiber deficiency	Colorectal
Virus	
Hepatitis B and C	Liver
HIV, cytomegalovirus	Kaposi sarcoma, non-Hodgkin's lymphoma
Epstein Barr virus	Burkitt lymphoma, nasopharyngeal carcinoma
Human T lymphotropic virus	T cell leukemia
Human papilloma virus	Cervix
Parasites	
Schistosoma	Bladder
Fungal toxin (aflatoxins), which is produced by the fungus Aspergillus flavus growing on stored grains, nuts and peanut butter	Liver

Host

Age

Ageing is another fundamental factor for the development of cancer. The incidence of cancer rises dramatically with age, most likely due to a buildup of risks for specific cancers that increase with age. The rising incidence of cancer is partly due to the rise of life expectancy.

Sex

It is readily observable that some cancers are 'male' (oral, lung, esophagus, stomach, bladder) and some 'female' (breast, cervix). The difference is attributed to exposure (more men smoke than women) rather than susceptibility. On the other hand, breast cancer is an estrogen dependent neoplasm, which the male body can not sustain, and cervical cancer is frequently caused by human papillomavirus, which causes similar tumors (papillomas) over male genitalia and perianal region.

Genetic factors

'A' blood group	↑ stomach carcinoma
Down's syndrome	Leukemia
Other genetic cancers	Polyposis coli, retinoblastoma

Others

Obesity was found to be a risk factor for cancer in a 2005 study by International Cancer Collaborators, as well as ↓ consumption of fruits and vegetables, physical inactivity and indoor smoke from household fuels.

Prevention

Primary prevention

1. **Control of tobacco and alcohol consumption** through education, legislation against smoking, restriction of smoking in public places. A comprehensive strategy including bans on tobacco advertising and sponsorship, tax increases on tobacco products, and cessation programs can reduce tobacco consumption in many countries. Some countries like Norway have developed ambitious plans to eliminate smoking. India, on the other hand, cannot ignore the unemployment that will be created if smoking or alcohol is banned completely. Each year, these industries are taxed a little more than previous year, but no Government yet has pledged to make India completely smoke free.
2. Maintenance of **personal hygiene** and being in a monogamous relationship prevents HPV infection and carcinoma cervix.
3. Lessen **radiation exposure**, esp. reduce unnecessary X-rays done (which exposes both the patient and the technician to radiation); provide personal protective equipment to workers in radiation plants/ radioactive mines.
4. Lessen **occupational exposure** to carcinogens through both engineering measures and personal protection.
5. **Immunisation**—Hepatitis B vaccine is now slowly being incorporated in National Immunization Schedule; the **human papilloma virus vaccine** is now being used agianst HPV infection.
6. Legislation and surveillance of food additives, drugs and cosmetics.
7. Control of air pollution.
8. Treatment of **precancerous lesions** like cervical tears, polyposis, genital warts, chronic gastritis, chronic cervicitis.
9. Motivate people to recognize 'early signs' of cancer and seek treatment (‘Cancer education’).

Danger signs

- Lump/hard area in breast.
- Sudden change in a previous wart/mole.
- Persistent change in bowel habit (constipation/diarrhea/bleeding).
- Persistent cough/hoarseness.
- ↑ menstrual blood loss/metrorrhagia.
- Blood loss from any natural orifice.
- Swelling/ sore throat that does not heal.
- Unexplained weight loss.

Secondary prevention

Cancer registration

1. **Hospital based registry**—All patients should be registered in a WHO prescribed format (WHO Handbook for Standardized Cancer Registers, 1976). The diagnosis and treatment can be evaluated from hospital registers, but the data cannot be generalized to entire population.
2. **Population based registry**—Optimum population size is 2–7 million. It provides incidence, tools to initiate epidemiological enquiries, surveillance of time trends, planning and evaluation.

Early detection by screening[288]

Cancer screening is possible because

1. Precancerous lesions last for long periods before developing into cancer.
2. All cancers begin as localized growth before spreading.
3. Majority of cancer occur at accessible sites (skin and mucous membranes).

Methods

1. **Mass screening**—Multisite (comprehensive cancer detection by clinicians by a thorough checkup of whole body) or singlesite; based on the existing evidence, mass population screening can be advocated only for breast and cervical cancer, using mammography screening and cytology screening, in countries where resources are available for wide coverage of the population.
2. Selective screening of risk groups.

Treatment

Surgery, radiation, chemotherapy

Tertiary prevention

1. Analgesia—Considered the right of the moribund patient.
2. Rehabilitation (after amputation/laryngectomy/colostomy/facial surgery).

Epidemiology of selected cancers

Oral cancer

The overwhelming majority of oral cavity cancers are squamous cell carcinomas. Almost all of them are readily available for biopsy, but half of them have metastasized by the time of diagnosis, and death ensues within 5 years.

Agent

Oral cancer is almost exclusively caused by chewing tobacco, in synergism with alcohol, amd smoking *bidis*. Unfortunately, most of our lower classes (mostly men and some women) are addicted to one or more forms of chewed tobacco (*khaini, nassi, paan*). A precancerous lesion (leukoplakia or oral submucosal fibrosis) is often the marker of impending disease.

Prevention

1. Stop smoking or chewing tobacco.
2. Early detection of precancerous lesions (oral health can be integrated in primary health care).

Cervical cancer

The cervix uterine houses a squamocolumnar junction, favorite spots of tumor to arise. Through whole of childhood, the cervix is closed and external os inverted. During adolescence, the external os everts a little (ectropion), thus exposing the endocervical columnar epithelium to vaginal acid, stimulating a squamous metaplasia. This zone of transformation around the external os now becomes very metabolically active. In addition, coitus and pregnancy exposes to herpes simplex viruses, papillomaviruses and heads of sperms which may enter these cells and control their metabolism. Thus the physiological metaplasia is converted to dysplasia of the squamous epithelium.

Stages

Sexual activity (early loss of virginity, multiple partners, promiscuous male partner) during teenage and early twenties

↓

Exposure to HPV 16, 18, 31 strains

↓

Cervical intraepithelial neoplasia—Develops in women 25–40 years

↓

Transforms into invasive carcinoma when the woman reaches 40–50 years

Agent

Persistent HPV (16,18,31,33), or herpes simples infection of the transformation zone. **Oral contraceptives** help the growth of the neoplams as it is an estrogen dependent neoplasm.

Host

Carcinoma of cervix occurs most frequently in multiparous women, only between 5% and 8% of patients being nulliparous. All investigators agree that coitus is an important pathogenetic factor. The earlier a woman faces coitus and the more diverse her sexual partners, the more she has the chance of developing the disease. Scarring of the cervix with childbirth and cervicitis is also a risk factor. Because the pathogenesis is essentially viral, the immunocompromised develop the disease early.

Environment

Low socioeconomic conditions and poor hygiene helps spread of the virus.

Prevention

Primary prevention

Health promotion. Safe sexual practices, improvement of hygiene.

Specific protection. The **HPV vaccine** is a bivalent vaccine (against two starins that cause 70% of cervical cancer) which is injected IM in three doses over a period of six months; the National Health Service in UK recommends vaccinating all girls between 12–13 as part of school health services.[289]

Secondary prevention

Early diagnosis (best at the stage of CIN) and treatment.

Screening carcinoma cervix

All women should have PAP smear at the beginning of sexual life and every 3 years thereafter. The problems to screening are

1. Gap in natural history—Not all stages in the progression of carcinoma in situ to frank carcinoma are known.
2. Variable frequency of progression from CIN → Carcinoma
3. The test is only 80% sensitive
4. Response rate (i.e. turning up for the test) is only 60–70%, and least in poor women, who are at the most risk.

Breast cancer

Breast cancer is still much more common in developed countries, and has risen in developing countries only since the eighties.

Agent

Unknown. Fatty diet, OCPs, hormone replacement therapy, radiation and alcohol have all been postulated. Previous benign breast diseases (with epithelial dysplasia) increases risk of cancer.

Host

Rare before 20, incidence increases with age. Incidence is **proportional to duration of reproductive period** (i.e. early menarche and late menopause predisposes to breast cancer), multiparity and late motherhood. Family history is very significant, and carcinoma in one breast increases chance in the other.

Prevention

We do not know enough to prevent breast cancer. However, it can be diagnozed very early and treated effectively.

Screening carcinoma breast

This is very fruitful

1. Self examination—Very useful, the only feasible method in some areas.
2. Clinical examimation.
3. Thermography—No radiation is involved, but the test is not very sensitive.
4. Mammography—It involves radiation exposure which 5 times more dense than an X-ray and requires infrastructure.

Lung cancer

Agent

Tobacco

> And a woman is only a woman, but a good cigar is a Smoke.
>
> *—Rudyard Kipling*

Surprised that such a line could come from him.

Tobacco was introduced to Europe from South America in the 16th century. Although its potential for harm was recognized early, its use was taken up avidly by every society that met it.[290]

Composition. Of about 500 compounds in tobacco, the most active are *nicotine* (acute effect) and *tars* (chronic effect). Smoke of **cigarets** is alkaline and is completely ionized in the alkaline pH of mouth, thus not absorbed from mouth. For effective absorption, it must be inhaled. However, smoke of **cigars and pipes** is alkaline, so that they can be absorbed directly from oral mucosa rather than inhalation. Thus pipe and cigar smokers are more at risk or oral cancer, but less of lung cancer.

The major carcinogens in smoke are polycyclic hydrocarbons, N-nitroso compounds and tars. *Bidis* have a higher content of carcinogens than cigarettes.

Tobacco dependance. Psychoanalysts, in their characteristic approach of any problem, have attributed cigarette smoking to libido.

> 'Getting something orally', one asserts ... 'is the first great libidinous experience in life'; first the breast, then the bottle, then the comforter, then food and finally the cigarette.
>
> *—Scott R B, BMJ 1957; 1: 671*

Initially teenage boys begin to smoke because of psychosocial reasons (self-esteem and status need), but quickly the reason becomes pharmacological. With developing maturity the social demand diminishes (the person wants to quit smoking) but pharmacological demand rises (he cannot quit because of intense withdrawal reactions). He can, unfortunately, develop into a 'chain smoker' whose only objective is to maintain a steady plasma nicotine ($t^{1/2}$ = 2 hrs) concentration.

Others

Occupational exposure to asbestos, ionizing radiation, As, Cr, Ni, Vinyl chlorides (especially factory smoke), air pollution.

Host

Although a 'male' cancer, lung cancer are rising in women after they have started to smoke from a decade or two earlier.

Prevention

1. Control of smoking—See earlier, 'Prevention of Cancer'.
2. Screening by X-ray and sputum cytology is not very effective in lung cancer; treatment is not also very satisfactory.

National Cancer Control Program, 1975–76 and Modified Cancer Control Program, 2005.[291]

Objectives

1. Primary prevention—Health education on cancer.
2. Secondary prevention—Screening, teaching self examination of breasts, strengthen existing treatment facilities.

Figure 8.6. The logo of NCCP

3. Tertiary prevention—Comprehensive cancer rehabilitation and palliative care.

Schemes

1. Strengthening of existing Regional Cancer Centers and development of new RCCs which will act as apex instituted for cancer treatment.
2. Develop oncology wings in Government medical colleges and hospitals.
3. *District cancer control scheme*—The DCCP will be implemented by a nodal agency, which may be a Regional Cancer Center or Government Medical College or Government Hospital with radiotherapy facility. A cluster of 2–3 districts are taken up for prevention, early detection, minimal treatment and provision of supportive cancer care at district levels.
4. Financial assistance to NGOs for undertaking health education and screening activities.
5. Nationwide education and antitobacco campaign from the central level.
6. Research—Training programs, monitoring and publication of manuals from the central level; in 2001, data from all cancer registries and all medical colleges were collated for the "Development of an Atlas of Cancer in India" (www.canceratlas.india.org).

ENDOCRINE

Obesity

It is an abnormal hypertrophy or hyperplasia of adipose tissue. It may be

1. Visceral (central)—Associated with insulin resistance and dyslipidemia
2. Subcutaneous (peripheral).

Definition and assessment

1. Only measurement of *weight* is inadequate to indicate obesity, two standard deviations from median weight-for-height may be held as cut off value.
2. *Skinfold thickness* is more convenient; measure thickness at 4 sites (biceps, triceps, suprailiac + subscapular)—The cut off for summation of these four sites is 40 mm for boys and 50 mm for girls.
3. *Waist circumference* is measured at mid point of rib cage and iliac crest. It is convenient, unrelated to height, correlates closely with body mass index. *Waist hip ratio* (WHR) is perhaps more significant. Men with WC > 102 cm or WHR > 1 and women with WC > 88 cm or WHR> 0.85 are at high risk of metabolic complications.
4. **Body mass index** = weight in kg/(height in meter)^2. A BMI > 25 is overweight, and BMI > 30 is *obese*. BMI provides the most useful population-level measure of overweight and obesity as it is the same for both sexes and for all ages of adults. However, it should be considered as a rough guide because it may not correspond to the same degree of fatness in different individuals (it cannot distinguish between weight contributed muscle and that by fat).

Problem

Globally, in 2005, approximately 1.6 billion adults (age 15+) were overweight and at least 400 million adults were obese. In addition, at least 20 million children

under the age of 5 years are overweight globally in 2005. Once considered a problem only in high-income countries, overweight and obesity are now dramatically on the rise in low- and middle-income countries, particularly in urban settings.[292]

The double burden. Many low- and middle-income countries are now facing a "double burden" of disease:

- While they continue to deal with the problems of infectious disease and under-nutrition, at the same time they are experiencing a rapid upsurge in chronic disease risk factors such as obesity.
- It is not uncommon to find undernutrition and obesity existing side-by-side within the same country, the same community and even within the same household.
- This double burden is caused by inadequate prenatal, infant and young child nutrition followed by exposure to high-fat, energy-dense, micronutrient-poor foods and lack of physical activity.
- And of course, this means that a certain fraction of people are growing obese at the expense of the undernourished fraction due to economic disparity.

Agent

- Genetic—Probably multifactorial.
- Diet— ↑ calorie intake, especially in the form of saturated fat and refined carbohydrates; children are made to be addicted to a plethora of junk food from early childhood, so that they grow obese at a very early age (see the chapter on nutrition).
- ↓ intake of micronutrients.
- Cushing syndrome.
- Hyperthyroidism.
- Ex-smokers gain some weight.
- Alcohol thickens men but thins down women.
- Steroids/ OCPs/ insulin/ β blockers.
- Expression/ anxiety results in a certain pattern of behavior called 'stress eating' when the individual becomes dependent on glucose for mental well-being.

Host

Men are more overweight but women tend to grow more obese with each pregnancy and become maximally obese between 45–49. 30% the obese people have been so since their childhood.

Physical inactivity is not confined to the developed countries anymore, because of increasingly sedentary nature of many forms of work (information technology, call centers, computer accounting), easier modes of transportation, and increasing urbanization.

Complications

1. Almost inevitable—Type II diabetes, gallstones, dyslipidemia, dyspnea, sleep apnea.
2. Likely—Ischemic heart disease, hypertension, osteoarthritis, gout.
3. Probable—Cancer, polycystic ovarian syndrome, low back pain, fetal defects.

Prevention

Aims

1. To maintain BMI within 18.5–24.9 through adulthood.
2. To prevent weight gain > 5 kg anytime during adulthood.
3. For those who are obese already, initial goal is 5–10% weight reduction.

Primary (and primordial) prevention

At the individual level, people can train children (and themselves) from the very beginning on good dietary practices and protect them from junk food marketing campaigns.

- Limit energy intake from total fats and shift fat consumption away from saturated fats to unsaturated fats.
- Increase consumption of fruits and vegetables, as well as legumes, whole grains and nuts.
- Limit the intake of sugars.
- Increase fibers in diet.
- Increase physical activity—At least 30 minutes of regular, moderate-intensity activity on most days.

Although the market is flooded with appetite suppressants and liposuction therapy, their cost and side effects prohibit them to be effective community intervention.

The 'feeling' of being fat and its psychological consequences

Children who "feel fat" have lower self-esteem and feel worse about themselves than children who are actually fat. *Girls* generally overestimate their body weight ("I am too fat!") more often than boys, while the latter frequently underestimate their body weight ("I am too thin, I need to be more bulky!"). This is because children of today are bombarded with images of super trim 'size zero' female models and muscular 'six pack' male models, on the television, newspaper, hoardings and internet. It is only natural that some 'normal' children (and teens) will feet fat / thin relative to these 'ideal' figures, and become victims of depression. There has only been a recent change in trends by inclusion of normal, 'plus size' models in covers of fashion magazines.

Diabetes mellitus

It is a state of chronic hyperglycemia due to an absolute/relative deficiency of insulin.

Definition[293]

After at least 8 hrs of fasting, the venous glucose is measured. Now the patient is fed 75g glucose (300 kCal)—For children or anybody below 50 kg, the dose is adjusted as 1.75 g/kg. After 2 hrs of the meal, a postprandial venous glucose is measured.

Condition	Venous plasma glucose
Diabetes mellitus	Fasting > 126 Or postprandial > 200 Or random > 200
Impaired fasting glucose	Fasting 110–125
Impaired glucose tolerance	PP 140–199

Problem[294]

More than **220 million** people worldwide have diabetes. In 2005, an estimated 1.1 million people died from diabetes. Almost 80% of diabetes deaths occur in low- and middle-income countries, and 55% of diabetes deaths are in women. It is no wonder that diabetes, along with ischemic heart disease has emerged as the epidemic of our times.

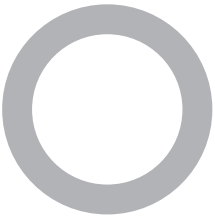

Figure 8.7. World Diabetes Day logo: The blue circle

The International Diabetes Federation celebrates **World Diabetes Day** on 14th November.

Types

1. **Type I**—Autoimmune, early onset, fatal without treatment, ketosis is common.
2. **Type II**—Insulin resistant, slow process, associated with the metabolic syndrome, complicated by other diseases.
3. **Gestational** (diabetes in pregnancy)—Due to growth hormone, progesterone, cortisol and prolactin excess.
4. **Maturity onset diabetes in the young** (MODY)—Genetic β cell malfunction.
5. **Lipotrophic**—Genetic defect in insulin action.
6. Diseases of exocrine pancreas.
7. **Endocrine diseases**—Pheochromocytoma, Cushing syndrome, acromegaly, hyperthyroidism.
8. **Drug-induced**—Steroids, thiazides, phenytoin, niacin.
9. **Congenital diseases**—Down's, Klinefelter's, Turner syndrome, Friedreich's ataxia.

Type I diabetes mellitus

Type I diabetes has a rapid, discernible onset, usually before 30 years of age. It is usually autoimmune in origin, insulin dependant, and develops ketoacidosis if untreated.

Agent

1. Infections—Mumps, coxsackie, CMV, rubella
2. Nutrition—Early introduction of cow's milk
3. Autoimmunity—Against islet cells, glutamic acid decarboxylase
4. Genetic—Associated with HLA DR3 and DR4.

Type II diabetes mellitus

Agent

Insulin resistance seems to be the primary pathology. Upto a certain age, this resistance is countered by increasing secretion, until β cell dysfunction sets in and insulin production drops. Insulin resistance is brought on in the first place by **rapid rise of blood glucose** after regular meals of refined sugars (burgers, colas, candies) alongwith obesity.

Host

1. Age—Type II diabetes usually comes to light only after 30, and many people in the world live with it without being aware of it.
2. Sex—In India Type II diabetes is male dominant.
3. Genetic—Family history of Type II diabetes is a risk factor.
4. Central obesity—Waist circumference > 107 cm in men and > 88 cm in women carries a high risk of Type II diabates.
5. Habitual physical inactivity.
6. Maternal diabetes.

Environment

1. **Diet**—Excess of saturated fatty acids, refined carbohydrates, inadequate intake of dietary fibers and polyunsaturated fatty acids; Indian children (and teenagers) have acquired all the bad food habits of the west (see the chapter on nutrition) since the last two decades, and Type II diabetes is on the rise in children.
2. **Stress**—Physical (trauma, surgery) or mental stress may bring out latent diabetes.

Table 8.5. Differences between Types I and II diabetes mellitus

	Type I	Type II
Onset	< 30	> 40
Ketosis	Common	Uncommon
Body weight	Weight loss	Occurs more in obese
Prevalence in India	0.2–0.3%	5%
Genetic	90%	50%
Circulating antiislet cell antibody	+	–
Associated autoimmune diseases	+	–
Treatment	Insulin	OAD and insulin
Insulin level	↓↓	↓/ ↑
Insulin resistance	Uncommon	Usual

Complications[295]

1. Diabetes increases the risk of **heart disease and stroke**. 50% of people with diabetes die of cardiovascular disease (primarily heart disease and stroke).
2. Combined with reduced blood flow, neuropathy in the feet increases the chance of foot ulcers and eventual limb amputation.
3. **Diabetic retinopathy** is an important cause of blindness, and occurs as a result of long-term accumulated damage to the small blood vessels in the retina. After 15 years of diabetes, approximately 2% of people become blind, and about 10% develop severe visual impairment.
4. Diabetes is among the leading causes of **kidney failure**. 10–20% of people with diabetes die of kidney failure.

5. Diabetic neuropathy is damage to the nerves as a result of diabetes, and affects upto 50% of people with diabetes. Although many different problems can occur as a result of diabetic neuropathy, common symptoms are tingling, pain, numbness, or weakness in the feet and hands.

The overall risk of dying among people with diabetes is at least *double* the risk of their peers without diabetes.

Primary prevention

Population strategy

Type I diabetes is not practically preventable, except for genetic counseling. We can only prevent Type II diabetes through a sea change in our lifestyle, habits and activity. The 'dietary goals' as enunciated by WHO must be followed. But diabetes cannot only be controlled by dieting. The cosmopolitan India must rethinks its priorities of life, allow himself to slow down, if just a little, in the rat race, and destress once every often.

High risk strategy

Concentrate to eliminate risk factors in

1. First degree relatives of a diabetes patient
2. Obese
3. Those with impaired glucose tolerance.

Secondary prevention

Screening

Target population for screening for diabetes is

1. People > 40
2. Family history
3. Obese
4. Women with babies > 4.5 kg at birth
5. Excess weight gain in pregnancy
6. Premature atherosclerosis.

There are two methods for screening:

1. **Glycosuria** after 2 hrs of a meal—This test is neither very sensitive nor specific.
2. **Oral glucose tolerance test**.

Oral glucose tolerance tests

The patient should take good carbohydrate meal three days prior to the test, and should avoid all kind of drugs/exercise that will affect blood glucose level. Further, he should consume 30–50 g of carbohydrate the prior evening. Next, he should do at least 8 hrs fasting before the test.

↓

Blood is collected at the fasting state from a vein and the glucose is estimated. Normally, it should be 75–110 mg/dl

↓

The subject is fed 75 g pure (anhydrous) glucose in 250–300 ml of water.

↓

Blood and urine are collected at ½ hr intervals for next 2 ½ hrs. After 2 ½ hrs, blood glucose in a normal person should be 110–140 mg/dl.

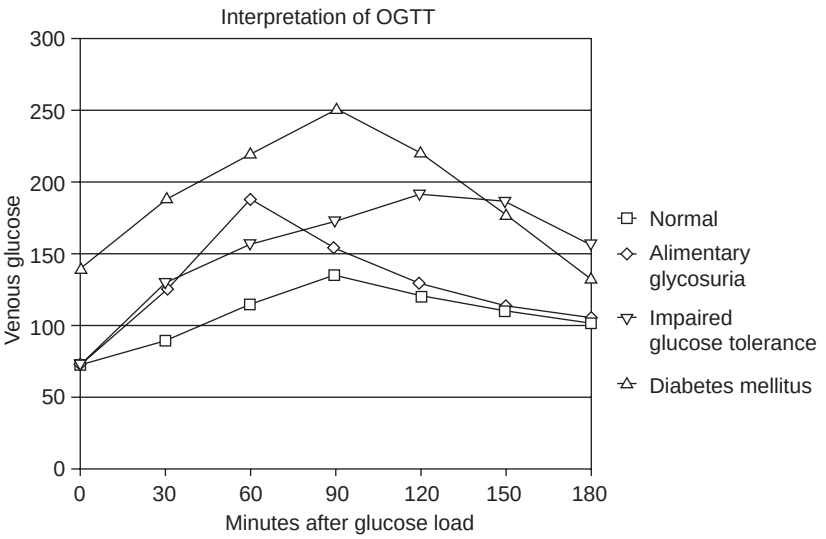

Figure 8.8. Interpretation of OGTT

Treatment of diabetes

Consult a textbook of internal medicine.

Monitoring of therapy

- Blood sugar
- Urine for sugar/protein/ketone
- Blood pressure.
- Visual acuity.
- Weight.
- Examination of vascular supply of feet (diabetic gangrene is very common in lower limbs).
- Glycosylated hemoglobin—Indicator of long-term glycemic control.

Most of the monitoring can be done by the patients themselves through appropriate self care devices.

Tertiary prevention

Save the 'diabetic foot', eyes and prevent other disabilities (ischemic heart disease, renal failure). The patient can still be lead a good life (Wasim Akram is a human, after all).

EYES

Blindness

Definitions

National Program for Control of Blindness

Visual acuity < 6/60 in better eye with *available* (i.e. the maximum the person or health system can afford) correction

Or

Visual field restricted to ≤ 10° around fixation.

WHO

Visual acuity < 3/60 in better eye with *best* possible correction (walk about vision).

WHO	Can't see	Can see	NPCB
Normal vision	-	6/18	Normal
Low vision	6/18	6/60	Low
	6/60	3/60	Economic blindness
Blindness	3/60	1/60	Social blindness
	1/60	PL	Manifest blindness
	PL	Nothing	Absolute blindness

Problem

Blindness, in developing countries, is due to cataract in maximum cases, while in developed countries it is age related macular degeneration. About 80% of all blindness is *avoidable* (preventable or curable).

India[296]

1. Prevalence of blindness—0.7% of population
2. Blindness caused by cataract—77%
3. Vitamin A deficiency is major cause of blindness in childhood
4. Cataract surgeries are increasing.

Main causes of blindness are as follows—Cataract (62.6%), refractive error (19.70%), corneal blindness (0.90%), glaucoma (5.80%), surgical complication (1.20%), posterior capsular opacification (0.90%), posterior segment disorder (4.70%), others (4.19%). Estimated National Prevalence of Childhood Blindness /Low Vision is 0.80 per thousand.[297]

Blindness is *increasing* on the whole in India, due to ↑ life expectancy, ↑ geriatric population, poor access to health service, misconception about cataract surgery. Sadly, **uncorrected ametropia** (refractive error, which is easily corrected by glasses) is an *accepted* cause of blindness in India, because many people cannot afford glasses.

Agent

The National Survey on Blindness (2001–02) survey on blindness shows

1. **Cataract (62%)**—Cataract sets in at an earlier age in India than in the west
2. **Ametropia (20%)**
3. **Vitamin A deficiency (0.9%)**, precipitated by measles, diarrhea and PEM
4. Trachoma
5. Glaucoma (6%)
6. Diabetes
7. Trauma.

Host

Age. 30% of blind in India are under 20, mostly due to trachoma and vitamin A deficiency. However, blindness increases rapidly with age.

Sex. Blindness is female dominant.

Occupation. Workers of cottage industries, fumes, quarries are susceptible to eye injuries; those working with radiation an electrical flash may develop premature catarct.

Social factors. Quack doctors and bad family practices often make a person unnecessarily blind.

Prevention

The 'eye care revolution' is simply the integration of ophthalmology in the three tier health care system, using epidemiologic approaches and teamwork (inclusion of public health workers, rather than only ophthalmologists) in eyecare, and setting up a national program for control of blindness.

Primary level

1. Treatment of common ailments like conjunctivitis, opthlamia neonatorum, trachoma, superficial foreign bodies by village health guides and MPHWs.
2. Checking refraction and provision of cataract surgery at some PHCs.

Secondary level

1. **Eye camps/mobile ophthalmologic units**—Not for therapeutic but for screening blindness only; doing cataract surgery in camps may lead to eye infection.
2. Initiation of **cataract surgery** in some district hospitals; patients are *transported in* government run vehicles; extracapsular cataract surgery is preferred and a spectacle, or better, an intraocular lens is mandatory.

Tertiary level

1. **Upgrade eye departments** to operate retinal detachments, corneal grafts and cataracts; arrange for separate beds for such.
2. Eye banks and hospital corneal retrieval program.

Specific programs

1. National program for control of blindness.
2. School eye health services.
3. Vitamin A prophylaxis.
4. Occupational eye care.

Long-term measures

Water sanitation

National Program for Control of Blindness, 1976

National Program for Control of Blindness was launched in the year 1976 as a 100% centrally sponsored scheme with the goal to reduce the prevalence of blindness from 1.4% to 0.3%. As per survey in 2001–02, prevalence of blindness is estimated to be 1.1%. Target for the 10th plan is to reduce prevalence of blindness to 0.8% by 2007.

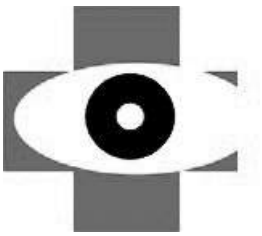

Figure 8.9. The NPCB logo

Objectives

1. To reduce the backlog of blindness through identification and treatment of blind
2. To develop eye care facilities in every district.
3. To develop human resources for providing eyecare services.
4. To improve quality of service delivery.
5. To secure participation of voluntary organizations in eye care.

Organization

National Institute of Ophthalmology (Dr Rajendra Prasad Center at AIIMS)
↓
10 Regional Institutes of Ophthalmology
↓
82 upgraded eye departments in Medical Colleges
↓
166 eye banks
↓
District unit: DISTRICT BLINDNESS CONTROL SOCIETY
Chaired by magistrate, run jointly by government, NGO and private agencies
↓
445 district hospitals with cataract surgery + ophthalmologist

Activities

1. Establishment of 10 regional institutes of ophthalmology under Dr Rajendra Prasad center.
2. Upgrading 82 eye departments in medical colleges.
3. Mobile eye units.
4. Recruitment of manpower and provision of services.

Revised Strategies

1. Treatment of corneal diseases and glaucoma alongwith cataract.
2. Integration of ophthalmology in school eye services, training of teachers to recognize early eye disease, provision of free glasses for poor children.
3. Camps for screening cataract and correcting ametropia; all cataract surgery must be institutional, ensuring mandatory lens implants (by 2007–2008, 92%

of all cataract surgery patients have been provided intraocular lens implants rather than glasses).

4. Focus on underserved areas, preparation of village blind registers and preference to bilaterally blind.

Eye (cornea) banks

Banks to procure, preserve and utilize cornea. They must provide

1. 24 hrs service through telephone.
2. Correct procurement and transport of cornea.
3. Preservation—Cornea can be preserved in a moist chamber in refrigerator for 24 hrs or in the McCarey Kaufman media for 96 hrs at 4°C or KSOL/ DEXOL medium for 2 weeks or cryo preservation for months to years.
4. Equitable cornea transplants—Maintaining a 'waiting list' of patients who need transplantation and disbursement of cornea according to priority.

Currently, nearly *twenty thousand* donated eyes are collected per annum in India. Hospital retrieval program is the main strategy for collection of donated eyes, which envisages motivation of relatives of terminally ill-patients, accident victims and others with grave diseases to donate eyes. Eye donation fortnight is organized from 25th August to 8th September every year to promote eye donation/ eye banking.

Educational activities

IEC activities are undertaken at Central, State and District Blindness Control Societies level. Special campaigns for mass awareness were undertaken during Eye Donation fortnight (25th August to 8th September) and **World Sight Day (2nd Thursday of October)**. At the Central level, prototype IEC material is produced and disseminated to the States.

Vision 2020 (WHO, 1999)

"To eliminate avoidable blindness by 2020, to give the millions of needlessly blind, the right to sight," namely – cataract, trachoma, onchocerciasis, childhood blindness, ametropia, diabetes, glaucoma, corneal blindness.

Actions

1. **Cataract**: To eliminate backlog by 2020 and providing good surgery with IOL.
2. **Trachoma**: SAFE strategy (surgery for trichiasis, antimicrobials like azithromycin, facial cleanliness, environmental improvement).
3. **Childhood blindness**: Vitamin A prophylaxis, measles vaccination, preventing neonatal conjunctivitis, pediatric eye care (congenital cataract, retinopathy of prematurity), low vision aids.
4. **Ametropia**: Correction of refraction at PHCs, vision screening, low cost glasses.
5. Control of onchocerciasis.
6. Human resource development.

ACCIDENTS

"Unpremeditated event resulting in recognizable damage". Accidents are a man-made epidemic and no longer considered 'accidental'.

Problem

All the benefits industrialization, increase in transport facilities, overcrowding, urbanization and rural electrification have also brought a number of 'new' accidents. In earlier times, when you walked down the road, the most you get hit by was a bullock cart. Nowadays, the offending vehicle may range from a bicycle to a superfast express train. In those medieval days, the maximum damage you could do to yourself while in your home was to cut yourself/ scald yourself in boiling water/ set fire to the house. Nowadays, you can be electrocuted, be burnt from a gas leak, spill acid (those used in cleaning toilets) over yourself, put your finger in the mixer grinder, get your clothes stuck in the vaccum cleaner, fry your hand over the microwave, ingest some rat poison or kerosene ... there is an ever increasing number of potential accidents.

Among accidents in India, road traffic accidents top the list, followed by occupational accidents, burns, violence and poisoning.

Agent

Accidents and injuries are commonly classified based on "intentionality". Most road traffic injuries, poisoning, falls, fire and burn injuries, and drowning are unintentional. *Intentional* injuries include interpersonal violence (homicide, sexual assault, neglect and abandonment, and other maltreatment), suicide, and collective violence (war).

Host

- Poisoning, drowning, burns, and maltreatment by caregivers affect primarily small children.
- Road traffic accidents, interpersonal violence and sports injuries tend to affect older children and adolescents.

In addition, injuries tend to be more prevalent in boys.

Environment

- In the *rural* areas injuries mainly to farming activities, pesticide poisoning, and drowning.
- In the urban areas, most injuries are traffic related, or linked to gadgets and electrical appliances, falls or poisonings resulting from household chemicals and drugs ingested by small children.

The environmental factors leading to injury may also be associated with social factors, such as family stress and critical life events (e.g. hospitalization or chronic disease of a parent, or change of residence).

Road traffic accidents

Many road traffic accidents (RTA) goes under reported, especially the minor injuries.

Indicators of traffic accidents

- Proportional mortality due to traffic accidents.
- Deaths (i.e. killed overnight or within 30 days)/million population.
- Deaths per 1000 vehicles per year.

- Number of accidents per vehicle per km gives incidence of accidents with respect to distance covered.
- Number of accidents per driver per hr gives incidence of accidents with respect to man hrs.

Agent

1. Motorized and nonmotorized vehicles moving in the same tracks; there is no segregation of vehicles in most Indian streets.
2. Pedestrians are not effectively segregated from traffic; there are footpaths, but in many cities in India they are blocked by street hawkers and vendors.
3. A high number of old vehicles.
4. Large proportion of two wheelers, which are unstable by design.
5. Too many overloaded vehicles running to speed past each other.
6. Disregard of traffic rules.
7. The practice of crossing the road whenever you want without waiting for the signal.

Host

Young men are involved in most road traffic accidents (rather than older, experienced drivers), especially after they have had a binge of alcohol.

Environment

1. The condition of Indian roads, the quality of traffic regulation and bad lighting contribute to accidents.
2. The **economics** of bus accidents is interesting; bus drivers will always be racing against other buses if their chief income is from *commissions* (depending on number of passengers picked up) rather than a fixed salary from state.

Prevention

The Haddon matrix lists all possible interventions on the part of the driver, the vehicle and the environment to avoid an accident/minimize damage.

	Precrash	Crash	Postcrash
Driver	Education regarding safe driving Enforce a maximum alcohol limit 80 mg% in blood to be safe for driving Periodic driving tests and medical fitness examinations	Personal protection during crash (air balloons, helmets, seat belts) to be made mandatory	Emergenecy management and rehabilitation
Vehicle	Regular checkup of vehicles Setting speed limits in busy areas	Crash protective design	The vehicle should have an emergency exit should not be inflammable
Environment	Improved roads, lights	The boundaries and railings of the road should be collapsible (so that damage is minimum during impact)	Trauma care center

Preventing accidents

Primary prevention

1. **Data collection** of all accidents, and possible *reconstruction* of how the accident happened.
2. **Safety education** that must begin with children, and end with drivers; the education must be delivered through schools and include traffic rules, common hazardous substances in the households, safe use of electrical devices, etc.
3. Use of **personal protective equipments**—Seat belts, helmet, leather clothing and boots, steering balloons.
4. **Improve environment**, i.e. Roads, lighting, marking of danger points, provision of fire guards, to store toxic substances safely and out of reach of children, etc.
5. **Laws** to ensure that only good quality drivers (who are medically fit) and vehicles get on the street, speed limits are not crossed and alcohol limit in blood/ breath stays below threshold.

Secondary prevention

Emergency care after accidents, with rehabilitation services must be integrated into primary health care.

9 Communicable Diseases

KEY FEATURES

- EPIDEMIOLOGY OF INFECTIOUS DISEASES
  - The Chain of transmission
  - Investigation of epidemic
- INTESTINAL INFECTIONS
  - Acute diarrheal disease
  - Cholera
  - Enteric fever
  - Acute bacterial gastroenteritis (Food poisoning)
  - Dracunculiasis
  - Poliomyelitis
  - Hepatitis A
  - Teniasis
  - Hydatid disease
- PARENTERAL ROUTES
  - Hepatitis B
  - Hookworm infestation
  - Malaria
  - Dengue
  - Lymphatic filariasis
  - Yellow fever
  - Kyasanur forest disease
  - Chikungunya fever
  - Japanese encephalitis
  - Leishmaniasis
  - National Vector Borne Disease Control program
  - Plaque
  - Rabies
  - Tetanus
- RESPIRATORY INFECTIONS
  - Smallpox
  - Chickenpox (Varicella)
  - Diphtheria
  - Pertussis
  - Influenza
  - Mumps
  - Rubella
  - Severe acute respiratory syndrome (SARS)
  - Measles
  - Acute respiratory infections
  - Tuberculosis
  - Leprosy
- SEXUALLY TRANSMITTED DISEASES
  - Reproductive tract infections
  - HIV infection

EPIDEMIOLOGY OF INFECTIOUS DISEASES

We direct our special attention to the foremost category of disease prevalent in our country, and most developing countries.

The chain of transmission (Fig. 9.1)

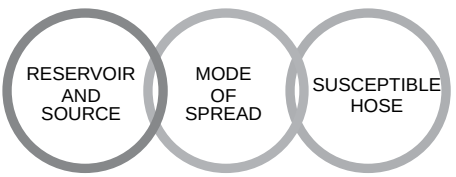

Figure 9.1. Chain of transmission

Source

The source of the micro-organism is usually some host or object from where an the organisms directly perpetrates into another host. The word 'direct' needs some explanation here. If any inanimate object can spread the micro-organism on its own, without actually needing a host, it may be considered a source. Because droplet nuclei of sputum (dessicated droplets that float freely in air) from a tuberculosis patient spread the bacilli long after the patient is gone, sputum may be considered an independent source of tuberculosis. However, this is not true for measles, because the measles virus do not survive long enough in droplets so that they can disseminate as droplet nuclei.

Nosocomial sources

Nosocomial infections are

- *New* infections other than the existing disease
- Acquired after *2 days of the stay* in the hospital
- Except wound infections.

Nosocomial infections may affect patients, medical care staff and people visiting the patient. Commonest nosocomial infections are urinary tract infections, pneumonia, wound infections and sepsis. More interventions you try on the patient (catheters, IV cannulas, intubation, general anesthesia) – more the risk of infection. Because the medical culture often demands doctors to be on duty, however sick, they themselves may be, a patient can catch a cold from a doctor! (And if the patient is diabetic, that cold could kill him).

Nosocomial infections are usually the ‘resistant’ type and it is best to avoid them by:

1. Isolation of contagious patients, barrier nursing.
2. Placing invasive devices only on need and remove such promptly.
3. Limit surgical antimicrobial prophylaxis (to stop development of undue resistance); use narrow spectrum antimicrobials for known pathogens.
4. 'Universal precautions' (Fig. 9.2).
5. Surveillance by infection control team.

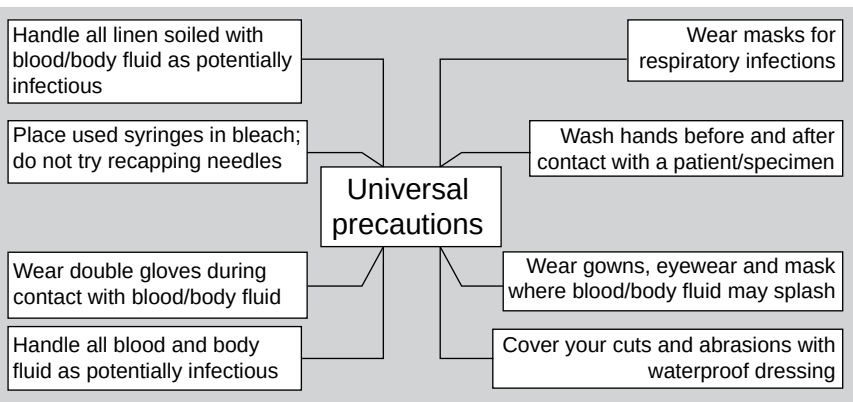

Figure 9.2. Universal precautions

How to wash your hands[298] (Fig. 9.3)

A regular soap or alcohol based hand rub will do a satisfactory job. Washing your hands properly takes about as long as singing "Happy Birthday" twice.

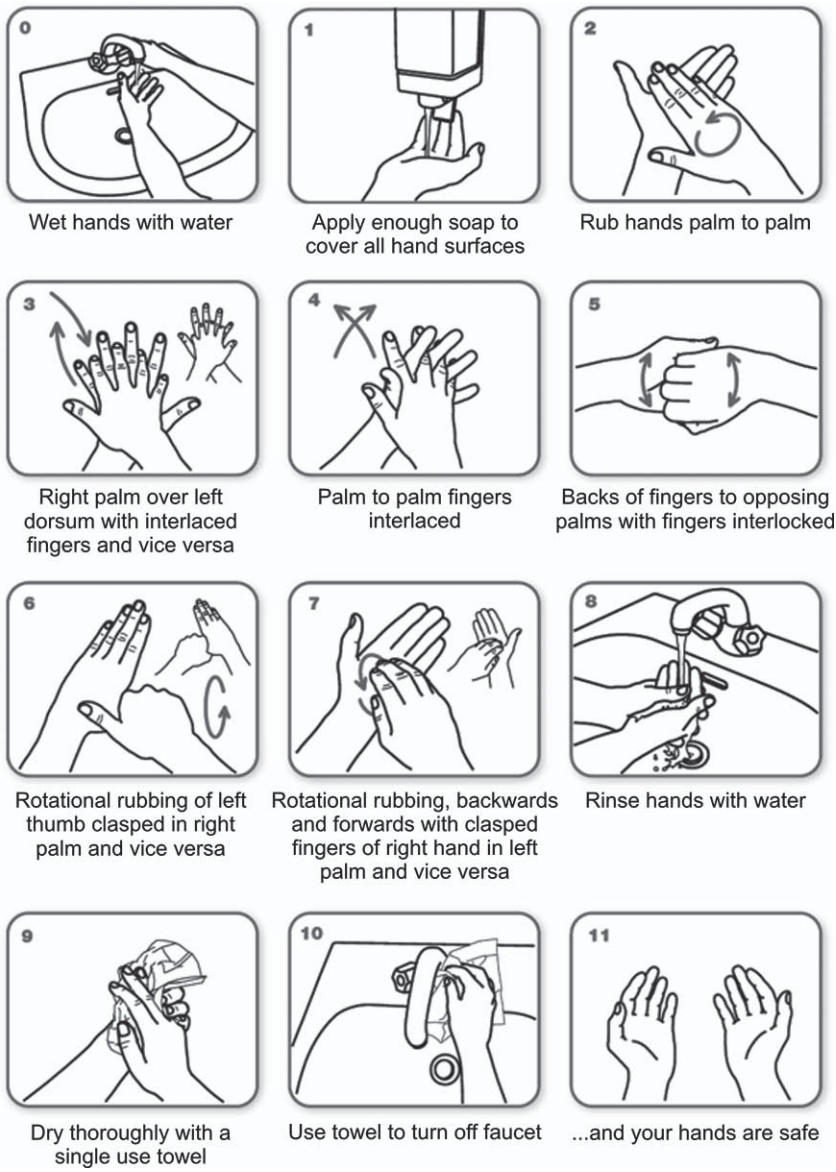

Figure 9.3. Proper hand washing

Bio safety levels

In this age of increasing bio hazards and bio terrorism, it is only imperative that dangerous microbes be dealt in the most stringent manner possible. A **bio safety**

level is the level of the containment precautions required to isolate dangerous biological agents in an enclosed facility, as prescribed by the CDC[299] or the European Union. There are four levels of bio safety, each more rigorous than the earlier, to handle progressively dangerous agents. In India, BSL-4 laboratories are present at AIIMS, and High Security Animal Disease Laboratory (HSADL) Bhopal.

Reservoir

It is the host/environment where the agent lives and multiplies, depends primarily for survival, and from where it reproduces in such a manner that it can be transmitted to a susceptible host.

1. *Homologous* reservoir—*Man* is the reservoir for many infections (i.e. cholera, amoebiasis, hookworms) that infect another man.
2. *Heterologous* reservoirs—*Birds* infected by Salmonella or Influenza virus, *soil* rich in *Clostridium tetani*, *armadillo* harboring *Mycobacterium leprae* could all transmit the micro-organism to man.

Human reservoirs

Cases

A person having the disease is a *case*, if his illness matches with the case definition according to International Classification of Diseases.

1. *Clinical cases* are graded subjectively as mild, moderate, severe or typical/ atypical. *Mild* clinical cases are equivalent to carriers, as they do not care about their ailments and go on spreading the disease.
2. *Subclinical* **cases** have the *infection* but *no illness* and spread the disease[300] (poliomyelitis, hepatitis B, enteric fever, mumps, rubella); **latent** infections are those that neither cause a disease nor spread it (i.e. the varicella zoster virus remains in the dorsal root ganglia for indefinite periods of time without doing anything at all, the tuberculosis bacilli could lie dormant in Ghon's focus inside the lungs without ever surfacing).
3. *Primary* **case** is the 1st case of a communicable disease introduced in a population.
4. *Index* **case** is the 1st case seen in an epidemic by the epidemiologist.

Carriers

A *carrier* is an host that harbors the agent but has no clinical illness and thus he/ she serves as potential reservoir of infection. Carriers are classified as follows.

Stage of disease. *Incubatory* **carriers** begin to spread the disease even before symptoms appear (measles, mumps, polio, pertussis). *Convalescent* **carriers** (typhoid, dysentery, cholera, diphtheria) spread the disease while recovering from the illness. People who have never had any symptoms but still spread the disease are *healthy* **carriers** who have actually emerged from subclinical infections (i.e. they were not aware that they were infected, took no treatment, and are now spreading the agent).

Duration. *Temporary* carriers are communicable for variable periods of time after cure, but chronic carriers (HBV, typhoid, dysentery) are usually communicable more than a year.

Route of spread. The organism may disseminate through bile, feces, urine or blood.

Animal reservoirs

Table 9.1. Some important animal reservoirs

Influenza	Pigs, birds
Chlamydia	Ducks
Japanese encephalitis	Pigs
Histoplasma	Birds

Other reservoirs

Clostridium tetani and *Bacillus anthracis* and many fungi live in soil.

Modes of transmission

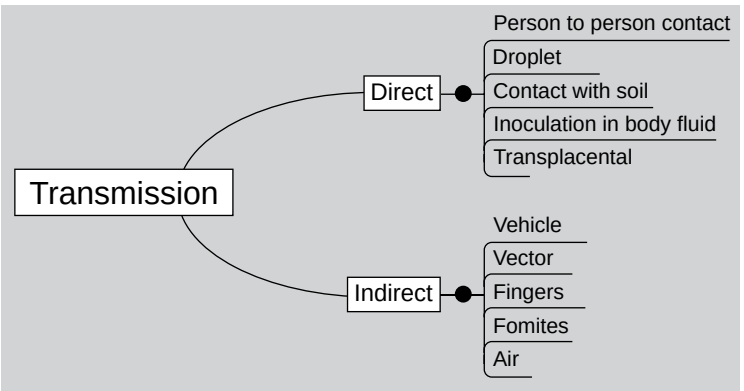

Figure 9.4. Modes of transmission

For a micro-organism to invade one host from another, there must be a *physical portal* between the two hosts. Bugs can not be transmitted by electromagnetic, telepathic or spiritual media. You can *not* fax an anthrax bacteria your enemy, or e-mail the plague bacilli to all of your contacts (even if you dearly wish to). You have to deliver it yourself, or post it in a parcel. The very fundamental of preventive medicine is finding out these physical portals.

Direct

If a certain person gets infected *in the presence of* the reservoir, i.e. the organisms jumps from one host to another without any intervening period, then the transmission is said to be direct.

1. **Person to person contact**—The organism spreads by contact with denuded epithelium. Contrary to what people think, most sexually transmitted diseases can also be transmitted by any kind of contact which involves an epithelial breach. The rate of microerosions is, however, much more in sexual intercourse (and even higher in anal sex) than other kinds of activities.

2. **Droplets**—Droplets microparticles of respiratory secretions, which, when coughed or sneezed out, are blasted into air with tremendous velocity[301] and inhaled by anybody in vicinity. Those particles < 5 μm manage to reach the alveoli and cause infection. Most respiratory infections (influenza, diphtheria, tuberculosis) are spread by droplets. Such infections are difficult to control in an overcrowded population like our country, specially when people believe spitting phlegm (often mixed with betel juice) over the streets is their birthright.
3. **Contact with soil**—The contact may be deliberate (putting soil or cow dung over the cord stumps of newborns, which introduces the tetanus bacilli) or accidental (carrying out deliveries over an unclean surface, which has the same effect); farmers who walk bare feet in open field are infested by hookworms through their feet.
4. **Inoculation in anybody fluid**—This gives the organism the most direct access to circulatory system. For example, hepatitis B and HIV are secreted in all body fluids, and if the body fluids of a patient (blood/ serum/ CF/ saliva/ semen/ vaginal secretion) come in contact with anybody fluid of a healthy person (through epithelial abrasions), he gets infected.
5. **Transplacental**—The blood of fetus and mother are separated throughout the pregnancy except during labor, when the two get mixed, and some organisms can infect the fetus from mothers blood during this period (HIV, hepatitis B). However, some organisms can cross the placental barrier in early pregnancy and usually cause chorioamnionitis, resulting in fetal malformation or abortion (rubella, cytomegalovirus, chickenpox).

Indirect

Indirect transmission implies a stage of *external survival* of the agent between two hosts, so that it can infect a person sometime after the reservoir is gone from the site. Thus the factors which control indirect transmission are:

1. Viability of the agent—How long can it survive without any host to sustain it.
2. Virulence of agent—How dangerous can it still remain after the period of external survival.
3. Environment—It is the environment suitable for survival of the organism.

The methods of indirect transmission are as follows.

Vehicle

Inanimate objects carrying the organism around (i.e. water, food, blood). Often a vehicle is responsible for a **point source epidemic**, for example, a certain contaminated water source causing diarrhea in everybody who drank it. The features of such an epidemic are—

1. A sudden rise in degree of contamination causes the outbreak.
2. Cases are usually confined to exposed population (nobody who has not drank water from that source falls ill).
3. Secondary cases seldom occur after the primary case are obscured (person to person infection does not usually take place).

4. May spread over wide distance (as people go to their homes after exposure).
5. Isolation of agent from the vehicle is not always possible.
6. Epidemic dwindles if the vehicle is withdrawn.
7. Common source of infection (i.e. the contaminated water source) is usually traceable from history of the cases.

Vectors

An intermediate organism which transports a micro-organism between two hosts. Examples include

- Man—*mosquitoes*—man (malaria, dengue, filaria)
- Rat—*flea*—man (plague)
- Man—*sandly*—man (kala-azar).

Flies only carry the agent, and are in no way *essential* for the organism to carry out its life cycle, and thus flies are *mechanical* vectors. *Biological vectors* harbor a part of the life cycle of organism inside them, rather than just carry it. Biological vectors may be classified as follows:

1. **Cyclo developmental**—The agent only *develops* into successive life stages in the vector, but does not multiply (microfilariae in mosquitoes).
2. **Cyclo propagative**—The agent *both* develops and multiplies in the vector (plasmodium in mosquitoes).
3. **Propagative**—The agent only *multiplies* in vector, does not develop (**Y***ersinia pestis* in rat flea).

Factors relating to vector borne transmission

1. Feeding preferences of the vector (*Culex* mosquitoes feed at night when microfilariae come out in blood, which makes the Culex an efficient vector for microfilariae).
2. *Infectivity* of vector (i.e. all mosquitoes do not cause malaria all the time, but only those who have *sporozoites* in their salivary glands at a particular time); the time for a vector to become infective after it has acquired the micro-organism, is the **extrinsic incubation period**; if the vector can be killed within this period, it will have failed to infect any other host with that micro-organism.
3. *Susceptibility* of host—Whether the host makes himself available to vectors (i.e. does not sleep with mosquito nets).
4. *Viability* of micro-organism within the vector; if the micro-organism can not sustain itself within the vector, it won't spread from that vector.

Can HIV be transmitted by mosquitoes?

NO. When an insect bites a person, it does not inject its own or a previously bitten person's or animal's blood into the next person bitten. Rather, it injects saliva, which acts as a lubricant so the insect can feed efficiently. Diseases such as yellow fever and malaria are transmitted through the *saliva* of mosquitoes, NOT their blood. However, **HIV lives for only a short time inside an insect**, not enough time to infect the salivary gland of the animal and enter the next human bitten, because the mosquito will usually *rest* after a blood meal, whilst the virus will die.[302]

5. *Domesticity* of vector (degree of association with man)—The reduviid bug lives in remote forests and thus sleeping sickness is not such a major problem; however, because mosquitoes live close around human habitat, malaria remains a major problem.
6. Number of vectors—The rise of malaria in monsoon is due to the increase in number of mosquitoes.

Airborne route

1. *Droplet nuclei* are microparticles of dried respiratory secretions; not all organisms that spread through *droplets* survive in droplet nuclei. The dried sputum of a tuberculosis patient remains infective long after the patient is gone, because the bacilli survive the dessication and begin to float freely in air in microparticles (nuclei) of sputum. Similarly, the droplets sneezed out by an influenza patient into his bed, remains infective and the virus take the airborne rout when the droplets have dried out.
2. *Dust* carries with it the Streptococcus bacilli, fungal spores, and tuberculosis bacilli; because dust is a frequent cause of nosocomial infections, hospitals advise *wet mopping* of the floors rather than dusting with a broomstick.

Fomites

Fomites are any inanimate object except food and water (like clothes, utensils and personal belongings of a patient) that bear germs and spread the disease. Suppose you have caught a cold and you sneeze into your towel when taking a bath. The virus which are deposited in the towel, if they manage to live long enough, will infect the next person who uses the towel. Or you could blow your nose with your hands, wash it off in a basin and close the *tap* with that hand. The tap then becomes the *source* of infection for the next person who touches the tap and then touches his nose/mouth. A similar chain of events could also be perpetuated by a doorknob, pens and pencils (kids frequently chew one anothers pencils), handle of a toilet flush (specially those in public toilets).

Fingers

The importance of clean hands can not be overstated, specially after the *swine influenza* epidemic. Most eye infections are *hand to mouth*, i.e. you have conjunctivitis → you rub your eyes → your hands get soaked with virus → you *handshake* with someone → he touches his eyes (for no particular reason, just because of habit) → he gets conjunctivitis.

Susceptible Host

There are four stages of successful parasitism:

1. Entry in a host (exposure)—Through any of the said modes of transmission; most organisms will be killed instantly by our natural immune barriers after getting entry, only occassionally does one survive to pass on to the next stage, that is.
2. Infection (multiplication and colonization within the host); mere exposure to an organism does not imply infection; to successfully infect a host, an organism must find a way through its natural barriers. Again, all infections

are not necessarily pathogenic. A myriad of organisms have infected (colonized) our gastrointestinal tract; they depend on us for nutrition, and we depend on them for several important processes (like vitamin K absorption, digestion of fatty acids, maintenance of normal gut motility); such a symbiotic relationship is said to be **commensalism**.

3. **Exit** from the host—For the purpose of infecting another host, some of the progeny of the original micro-organism must find a way to get out of the host through one of its natural outflow tracts, i.e. either through GI tract (salmonella, cholera, rotavirus, poliovirus), or respiratory tract (tuberculosis, influenza, chickenpox) or renal tract (salmonella). Organisms which are confined to blood of the host and can not invade one of these tracts have developed intricate evolutionary mechanisms to get picked up by a *vector* from the blood. Still, there are some organisms which fail to do any of these, and thus some disease like *hepatic* amoebiasis (not intestinal amoebiasis), hydatid cyst, tetanus or bubonic (not pneumonic) plague, there is no exit for the micro-organism and the human host is a *dead end* for it. For some other diseases like *rabies*, the virus may escape through the saliva of the case but humans do not ordinarily bite each other, so the virus is effectively locked in within the case.
4. **Survival** in external environment—For the micro-organism to be picked up by another host, it must survive in the external environment for some period. The 'external environment' could be water, soil, food, droplets or a vector.

Incubation period

The interval between exposure and first clinical manifestation is called the incubation period. A standardization of the incubation period is the *median incubation period*, i.e. the period until 50% of cases show first clinical manifestation. The time between exposure and *first case* is the **minimum incubation period**, and the last case, **maximum incubation period**.

The factors which influence the length of the incubation period are

1. Fertility of agent—How quickly it multiplies and reaches the quorum for causing a clinical disease; slow growers such as *Mycobacterium leprae* (with a long generation time) surface only after years have passed together
2. Route of entry—The incubation period of *inhaled* measles virus is 10–14 days, but when it is artificially injected, it causes disease in 7 days.
3. Immunity of the host—Clinical manifestations will only surface when the micro-organism has overcome the immunity mounted by the host.
4. Infective dose—A higher dose of micro-organisms will shorten the incubation period.

Short incubation period (hours – days)	S *aureus* food poisoning, Cholera
Medium incubation period (days – weeks)	Typhoid, Chickenpox, Mumps, Measles
Long incubation period (weeks – years)	Hepatitis A and B, Rabies, Leprosy

Usually, infectious diseases are not communicable during incubation period, **except measles, whooping cough, hepatitis A, mumps and chickenpox**.

Importance of the incubation period

1. Tracing the source of infection (plausible in diseases of very short incubation period)—If two persons have both developed acute gastroenteritis after 6 hours of a feast, the source of infection will most probably be found in anyone of the items served in that feast.
2. The period of surveillance and quarantine for a disease = maximum incubation period; if a person does not develop clinical disease within maximum incubation period, we deduce that he is not infected.
3. Postexposure prevention with immunoglobulins is logical *only* within one incubation period; after the incubation period, the organism will already be many innumber and bound to tissue receptors, so immunoglobulins will fail.
4. To know whether an epidemic is *point source* (all cases occur in one incubation period) or *propagated* (cases occur over a much longer time than one incubation period).
5. In some diseases (rabies), shorter incubation period means worse prognosis.

Primary and secondary cases (Fig. 9.5)

Primary case is the 1st case in a study population, and secondary cases all others *within one incubation period* after the primary case (i.e. they have acquired infection from the primary case). The interval between surfacing of primary case and 1st secondary case is the **serial interval**.

Communicable period

The time interval during which an agent may be transferred directly and indirectly from an host to another (not necessarily of same species). *Generation time* is the interval between exposure and maximum infectivity of the host. It is almost never equal to the incubation period but close, and signifies the height of communicable period. Some diseases have a generation time *shorter* than infective period (mumps, chickenpox), i.e. they are maximally infective before onset of symptoms.

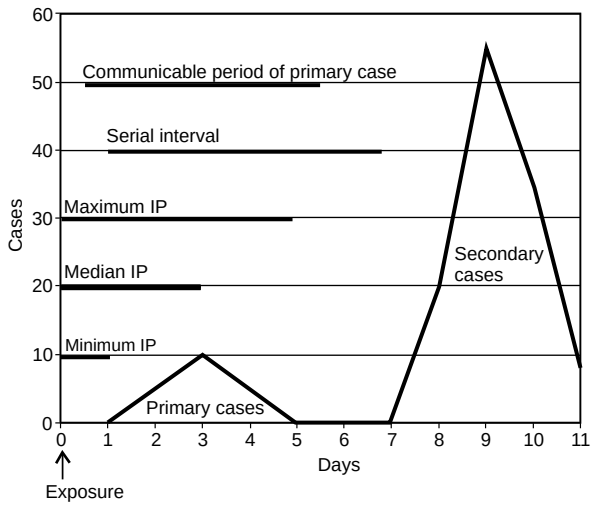

Figure 9.5. Primary and secondary cases

Measuring communicability – secondary attack rate

It is the **number of exposed persons developing the disease within one incubation period/number of susceptible contacts (population at risk)**. The SAR measures the communicability of a disease. Also, it allows comparing between attacks rate in vaccinated and nonvaccinated individuals.

But the SAR

1. It is applicable where the primary case is infective for a very short period. When communicable period is longer, we have to use person-time as denominator.
2. It is not applicable in disease where 'susceptibility' is undefined (i.e. influenza).

For example, if there are 100 students in a class, 60 of them are vaccinated against measles, only 40 are susceptible. Suppose one of these 40 develops measles (primary case). Then number of remaining susceptible = 40 – 1 = 39. If 26 students are infected from this one primary cases, number of secondary cases = 26 and SAAR = 26/39 = 66%.

Immunity

Because this is no place for a wholesome discussion of immunology, please refer to some good books on immunology out there (or your pathology book) if you are *really* into it.

Active immunity

Active immunity (the power to resist infections on your own) develops from

1. A clinical infection (as in measles)
2. A subclinical infection (as in poliomyelitis)
3. Vaccination.

The first time an antigen is encountered in life, the immune system of the individual spends sometime figuring what it is up against, and then mounts a feeble immune response by producing a small titer of *predominantly* IgM antibodies. This is **primary response** which lasts only as long the antigen lasts in blood. However, the second time that particular antigen is encountered, the immune system recognises it instantly (due to **immune memory**, a property of cell mediated immunity) and produces a plethora of *predominantly* IgG antibodies for a much longer period, which is known as **secondary response**. This is what give the individual immunity to a previously exposed agent.

Primary response	Secondary response
Depends on dose of antigen, nature of antigen, route of administration, use of adjuvants, host nutritional status. Primary response produces immune memory (remembering how to deal with a particular antigen, the knowledge which comes of use in secondary response)	Shorter latent period Rapid and increased production of antibody Chiefly IgG mediated Antibody response maintained for long periods Secondary response is the underlying principle of **booster** dosing.

Passive immunity

Passive immunity (lending somebody else's antibodies) is acquired by:

1. Passive immunization (injection of ready made antibodies).

2. Maternal IgG transfer through placenta, which persists in child upto 6 months.
3. Maternal IgA transfer through milk.

Passive immunity is

1. Rapid
2. Temporary
3. Bereft of immune memory
4. Expensive (vaccines are cheaper than antibodies).

Immunodeficiency

The concept of immunity is relative to the infective dose, i.e. there is a limit to the capabilities of the immune system and most normal people will be overwhelmed by an astoundingly large dose of microbes (i.e. transmission of a virus through blood transfusion). However, there are times when the body can not even handle the day-to-day load of microbes.

Congenital. Digeorge disease, Bruton's diseases Thymic hypoplasia, Lazy lymphocyte syndrome, Leukocyte adhesion defects.

Acquired. Malnutrition, sudden changes in diet, fatigue, drugs (anticancer, anti retroviral, steroids), radiation, infection (HIV), nephrotic syndrome.

Herd immunity

It is the l*evel of resistance of a community* to a disease. It is built up by

1. Clinical/ subclinical infections
2. Immunization of individuals
3. Herd structure (hosts, vectors, reservoirs, environment).

When swine influenza first hit the world, it spread like wildfire. With time however, as our *herd immunity* has grown, it is turning more and more benign. Because there are no subclinical infections of *rabies* or *tetanus*, and preexposure vaccines are not used, there is no herd immunity to these diseases. Before the advent of sanitary latrines, we used to have herd immunity against poliomyelitis, as most kids acquired a subclinical infection during childhood which gave immunity. However, with sanitary systems taking over, most kids are nowadays protected from the virus, and when they encounter it, they often develop clinical disease.

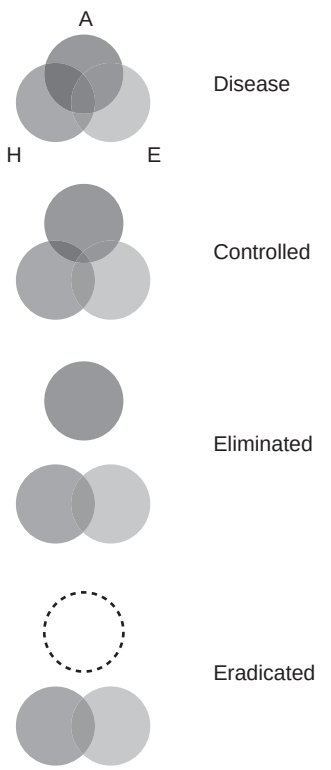

Figure 9.6. Three levels of struggle

Control of communicable disease

Prevention is protecting an *individual* from a disease, while *control* is protecting the entire community (Fig. 9.6).

Controlling the reservoir

Ideal but often impracticable.

1. **Early diagnosis** of the reservoirs by initiation of investigation. Start treatment immediately.
2. **Notify** to local authorities (for more help) and WHO (for plague, cholera, yellow fever).
3. **Isolation** of the reservoir is not always possible or effective; go for something like 'Ring immunisation' (isolate the reservoir surrounded by immune individuals) or isolate *chemically* by adequate treatment. Physical isolation is useful only in those disease which spread during clinical phase (and not before diagnosis) – like diphtheria and cholera. It is not effective for diseased which spread in preclinical stage, i.e. mumps.
4. **Quarantine** (limit movements) of reservoirs (man or animals) and exposed persons. Maintain quarantine till maximum incubation period. The difference with isolation is that those who are *isolated* are diseased, and those who are *quarantined* are yet healthy.
5. **Treatment** - Lt reduces communicability of the reservoir, reduces chance of becoming a carrier; but treatment alone is ineffective in control of diseases.
6. **Disinfection** of all the fomites of the patient is necessary
7. **Surveillance** of cases and carriers and the health situation in general is important.

Interruption of transmission

A major part of community treatment involves breaking the chain of transmission. This is best called, in a broad sense, the sanitation barrier.

Protecting the host

1. Health promotion (nutrition, etc.)
2. Specific protection (immunization, nutritional prophylaxis, chemoprophylaxis, occupation safety measures).

Immunization schedules

Because no two community have exactly the same health problems, each country (and each community) needs a different set of vaccines to be protected. An **immunization schedule** is the time-table of taking vaccines, beginning from birth. A good immunization schedule should be

1. Relevant, i.e. vaccines against major public health problems must be included.
2. Effective—To be effective the child needs to get vaccinated *before* exposure to the disease, the vaccines themselves need to be effective (live vaccines are preferred over killed vaccines), and they need to be given maintaining a minimum interval.
3. Accessible to mothers; you can not count on the compliance of mothers, who have a hell lot of other things to do than take their children to health center; try to combine vaccines as much as possible, so as to reduce the total number of visits. Carry out immunization clinics between 11 am–2

pm, when mothers will be somewhat free of housework. Do not make them wait in a long queue outside the clinic. Remember that they are the 'customers'[303], and the customer is always right; *you* are the one who must adapt to them, not the other way around; possible combinations—DPT, MMRV, typhoid tetanus.

4. *Feasible*—Because funds are always limited, you can NEVER arrange for ALL the vaccines and ALL the equipments; prioritize which vaccines are the most needed in the community and spend money on those.
5. *Acceptable*—The vaccines should not cause severe adverse reactions (otherwise it will be the last time you will see that particular mother and child), injections should not be *too* painful, or should not cause significant damage to the baby (Indian families are suspicious about immunization anyway, do not provoke them). Ensure that you harm nobody's spiritual beliefs (one mother refused giving OPV to her baby on the ground that members of family, including the baby, does not touch 'outside food' on that particular day of week; in such a case, tell her to come the next day rather than force her).

National immunization schedule

Table 9.2. National immunization schedule

Time	Name	Dosage
Birth	BCG OPV-0	0.1 ml intradermal just over insertion of left deltoid 2 drops oral (sensitizing dose)
6 weeks	BCG, if not given at birth OPV-1 DPT-1 Hepatitis B 1	 2 drops oral 0.5 ml IM left anterolateral thigh 0.5 ml IM left anterolateral thigh
10 weeks	OPV-2 DPT-2 Hepatitis B 2	2 drops oral 0.5 ml IM left anterolateral thigh 0.5 ml IM left anterolateral thigh
14 weeks	OPV-3 DPT-3 Hepatitis B 3	2 drops oral 0.5 ml IM left anterolateral thigh 0.5 ml IM left anterolateral thigh
9 months	Measles	0.5 ml SC left anterolateral thigh
16–24 months	OPV-b DPT-b	2 drops oral 0.5 ml IM left anterolateral thigh
5–6 years	DT	0.5 ml IM left deltoid
10 years	T	0.5 ml IM left deltoid
16 years	T	0.5 ml IM left deltoid
For pregnant women		
At registration (12–16 wk)	T1	0.5 ml IM left deltoid
4 weeks further	T2	0.5 ml IM left deltoid

WHO Universal Immunization Program

The UIP includes, in addition to those in our national immunization schedule, the *Haemophilus influenzae B* vaccine to be given at 6, 10 and 14 weeks and the yellow fever vaccine at 9 months.

Recommended schedule by Indian Academy of Pediatrics

Some vaccines which have been omitted in the National Immunization Schedule/ UIP due to financial constraints are recommended by IAP for those who can afford it.

Table 9.3. Immunization schedule—IAP

BCG	Between birth and 2 weeks
OPV	Birth; 6, 10 and 14 weeks, 16–18 months, 5 years
OPV	6, 10 and 14 weeks, 16–18 months, 5 years
Hepatitis B	Birth; 6, 10 and 14 weeks
Haemophilus influenzae B (Hib) conjugate	6, 10 and 14 weeks
MMR	15 months
Typhoid	2, 5, 8 and 12 years
TT or dT[a]	10 and 16 years
TT	2 doses one month apart in pregnant woman, or a booster dose if previously immunized
Vaccines that can be given after discussion with parents	
Chickenpox	After 1 year of age
Hepatitis A	For high risk infants at 18 and 24 months
Pneumococcal conjugate	6 weeks
Influenza vaccine	For high risk infants at 6 months of age
[a] The "adult" type diphtheria tetanus combination, which contains a lot less diphtheria toxoid than the pediatric one, thus a small 'd'	

Vaccines

Vaccines are antigens to induce specific active immunity against a disease.

Live attenuated vaccines

Live attenuated vaccines (LAV) are prepared by attenuation of live organisms by heat, subculture (BCG, OPV, MMR). Live vaccines are more potent because the organisms multiply in body, express *all* the antigens of that particular organism and often leave a residual immunity after they have been excreted by the body (i.e. mucosa immunity provided by oral polio vaccine).

ALLLIVEVACCINESARECONTRAINDICATEDINIMMUNODEFICIENT AND PREGNANCY. Also, there must remain a gap of AT LEAST 3 WEEKS between administering a live vaccine and any other vaccine. Or they may be given on the same day.

Live vaccines are single dose (except polio, which is given thrice because seroconversion occurs one strain at a time). Because live vaccines contain living organisms, they require a stringent cold chain.

Killed vaccines

Killed vaccines: Organisms killed by heat/chemical methods (typhoid, pertussis, HAV, influenza, cholera, plague) may also induce active immunity, because they still retain the antigens. Killed vaccines are:

- Safer than live vaccines (the organisms are already dead)
- Less potent
- Require booster doses (Fig. 9.7)
- Cannot be given orally (they'll be readily digested by enzymes).

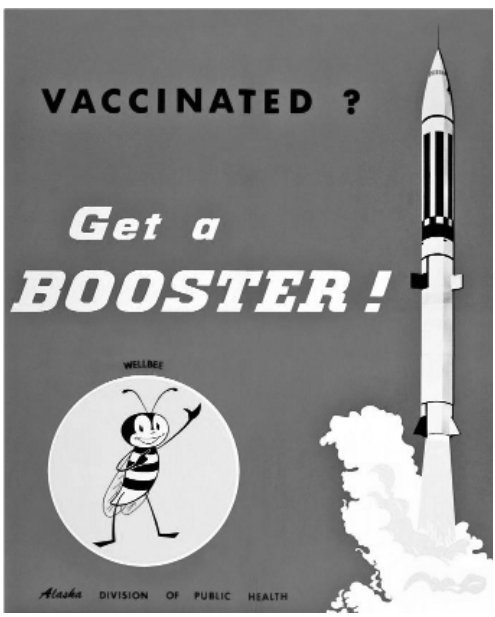

Figure 9.7. This 1964 poster featured what at that time, was CDC's national symbol of public health, the "Wellbee", who here was reminding the public to get a booster vaccination; the image reflects the parallel development of immunology and space sciences during the sixties [CDC Public Health Image Library].

Subunit vaccines

Subunit vaccines/cellular fractions/toxoids are particular proteins from the agent which, after detoxication, may be used for immunization (HBsAg, meningococcal cell wall antigen, *Haemophilus influenzae B* capsule, diphtheria and tetanus toxoids).

Adjuvants

An adjuvant is a compound that are added to potentate a vaccine (they produce a local granuloma to retain the antigen and decelerate its release) i.e. aluminium phosphate, aluminium hydroxide, water in oil.

Freeze dried vaccines

Freeze drying (also known as **lyophilization** or cryodesiccation) is a dehydration process typically used to preserve a perishable material or make the material more convenient for transport. Freeze-drying works by freezing the material and then reducing the surrounding pressure and adding enough heat to allow the frozen water in the material to sublime directly from the solid phase to the gas phase.

Freeze dried vaccines (BCG, measles, yellow fever) are powdered vaccines prepared in such a way as to increase shelf life of vaccine. They are reconstituted in a suitable liquid (BCG in saline, Measles in double filtered pyrogen free water. In addition, preservatives like thiomersal are often added.

Immunoglobulins

Normal human Ig

They are antibody rich plasma fractions obtained from a pool of at least 100 donors. Such 'passive immunisation' has been devised for measles (for highly susceptible individuals) and HAV (for travelers). Because normal human Ig contains many other antibodies other than the one wanted, *live vaccines given within 12 weeks of normal human Ig will get inactivated* by those immunoglobulins. On the contrary, normal human Ig may be given after 2 weeks of a live vaccine.

Specific human Ig

It is made from plasma of *single* convalescents patients, and contains antibodies at least in five fold concentration. Such Ig's have been devised for chickenpox, rabies, HAV, HBV, rubella, tetanus, Rh-isoimmunization. They are more specific, concentrated for intramuscular use and do not interfere with live vaccines.

Adverse reactions to immunoglobulins

1. Local—Pain and sterile abscess.
2. Systemic—Flushing, flank pain, rigor, dyspnea, shock, urticaria, arthralgia, fever, diarrhea.

Antisera/ antitoxin

They are crude preparations of animal serum (diphtheria, tetanus, gas gangrene, AVS, rabies), slowly but surely being obsoleted.

The 'cold chain'

It is the combination of *machines*, *personnel* and procedure responsible for storing vaccines at recommended temperature (0–8°C) from *production to use*. Arranging the vaccines by heat sensitivity, we have OPV > Measles > Pertussis and mumps > Hepatitis B > DPT > DT > BCG > DPT > TT. The T series vaccines and hepatitis B vaccine are also 'freeze sensitive' and should never be exposed to subzero temperatures.[304]

REGIONAL CENTERS
Store > 3 months in cold rooms (< 0°C)
Transported to PHCs in cold boxes with ice packs;
cold boxes may also be used for emergency storage

↓

PHC
Store for < 1 month in ILR (2–8°C)
or for a longer duration in deep freezer (< 0°C)

↓

SUBCENTER
Distribute by vaccine carriers containing 4 fully frozen ice packs (2–8°C);
Unused vials are returned to PHC, marked and restored in ILR;

What to do with vials that have been opened but not used?

Global policy on this matter used to be that opened vials of all vaccines were discarded at the end of each working day. In 1995, WHO recommended a changed global policy on the use of opened vials of vaccine as follows.

1. Opened vials of OPV, DPT, DT, TT and hepatitis B vaccines may be used in subsequent immunization sessions until a new shipment of vaccine arrives, provided that each of the following 3 conditions are met
   - The expiry date has not passed
   - The vaccines are stored under appropriate conditions (0 to +8°C)
   - Opened vials of vaccine which have been taken out of the health facility for immunization activities (e.g. outreach, National Immunization Days) are discarded at the end of the day.
2. Opened vials of measles, yellow fever and BCG vaccines must be discarded within six hours.
3. An opened vial must be discarded immediately if
   - sterile procedures have not been fully observed, or
   - there is even a suspicion that the opened vial has been contaminated, or
   - there is visible evidence of contamination, such as a change in appearance, floating particles

Cold chain equipments

Walk in cold rooms. A cold room where people walk in and get the vaccine (as simple as that, does it need any more explaining?)

Deep freezers. Deep freezers create sub zero temperatures. They are suitable

1. to store for vaccines > 3 months
2. to make ice packs which are used in vaccine carriers.

The deep freezer should be placed in a well ventilated room at least 10–20 cm from the wall, and should not tilt on any side (should be perfectly horizontal). The power cable should be fixed PERMANENTLY to the power line and not through a plug (which could come open anytime). Use a voltage stabilizer so that the freezer is not exposed to fluctuating current. The power switch should be TAPED in the on position so that nobody can turn them off accidentally. Its temperature should be recorded every morning and evening.The lid of the deep freezer should be LOCKED when not in use. The device should be cleaned when ice has grown 4–6 mm thick over the inner walls (during the period of defrosting, store the vaccines in a cold box). After defrosting, clean the freezer and make it dry before loading vaccines again.

What not to do with a deep freezers

- Do not store food and drinks
- Do not open the lid too often
- Do not store DPT, DT, TT or BCG vaccine in a deep freezer
- Do not store diluent for vaccines in deep freezer
- Do not keep the deep freezer and ILR in contact with each other.

One person should be responsible for maintenance of the freezer.

Ice lined refrigerators. They maintain a temperature of 2–8°C; vaccines can be stored up to a month in ILR. The same precautions as the deep freezer apply also to the ILR. However, the T series vaccines and BCG can be kept in ILR (Fig. 9.8).

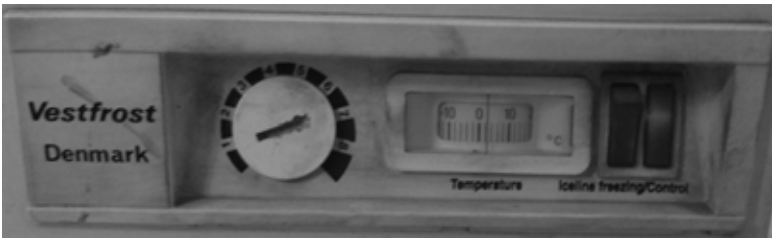

Figure 9.8. The temperature reader of an ILR

Cold boxes. Cold boxes, packed with ice packs, are used for regional transportation of vaccines.

Vaccine carriers. These are packed with 4 fully frozen ice packs on the day of vaccination. They can maintain the vaccine for 48 hours in 2–8° if not opened (Fig. 9.9).

Figure 9.9. A vaccine carrier, with a prohibitive warning

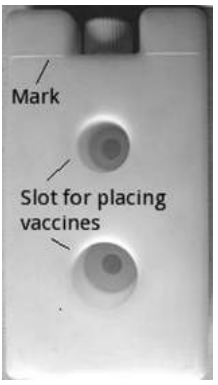

Figure 9.10. An ice pack

Ice packs. They are filled with salt free water (salt reduces freezing point of water) up to a preset mark (if we fill up to the brink, water will *expand* when frozen and crack the pack) and frozen in a deep freezer (Fig. 9.10).

In all these devices, the T series vaccine and hepatitis vaccine should never be placed in direct contact with ice.

Vaccine vial monitor (Fig. 9.11)

It is a heat sensitive label to monitor *cumulative* heat exposure. It consists of round piece of blue material inside which lies a heat sensitive square of lighter hue. The square changes to darker shades with

1. high temperature, short exposure or
2. lower temperature, long exposure or
3. high temperature, long exposure

The vaccine is usable only until the inner square is lighter than the outer circle. The VVM is initiated with the OPV as it is the most heat sensitive vaccine.

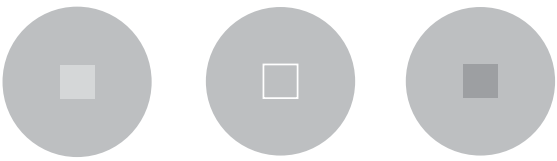

Figure 9.11. Vaccine vial monitor

DT and TT vaccine shipping indicators

This is another type of indicator, which travels with the vaccines from manufacturer to central store and is included with each 3,000 doses of DT, DPT and TT procured through UNICEF. This indicator has a temperature sensitive dot that irreversibly change from silver-gray to black at temperatures above +48°C, temperatures which may be reached if vaccines are left in the sun or in poorly ventilated places.

The shake test

Before administering a T series vaccine, it is a must to shake it and see a uniform mixture; any granules indicate freezing at some point of time, and the vial is discarded.

WHO recommendation on vaccine storage temperatures

To summarise, if you work,

At the national level. Keep your vaccines for a maximum of 6 months

- Store OPV, measles, and mumps vaccines at –15 to –25°C
- Store hepatitis B, DPT, DT, TT and BCG at 0 to +8°C
- Send vaccines to regions in insulated containers at 0 to +8°

At the regional level. Keep your vaccines for a maximum of 3 months

- Store OPV, measles, and mumps vaccines at –15 to –25°
- Store hepatitis B, DPT, DT, Td, TT and BCG at 0 to +8°
- Send vaccines to districts in insulated containers at 0 to +8°

At the district level. Keep your vaccines for a maximum of 1 month,

- Store OPV, measles, and mumps vaccines at –15 to –25°C, if possible
- Store hepatitis B, DPT, DT, Td, TT and BCG at 0 to +8°C
- Send vaccines to health facilities in insulated containers at 0 to +8°C

At the health facility level. Keep all your vaccines for a maximum of 1 month: Store all vaccines at 0 to +8°C.

Adverse reactions to immunization

Adverse reactions to immunization can be classified as those due to the vaccine itself (vaccine reactions), those due to program error (faulty production or administration of vaccines), those due to anticipation and anxiety of the impending injection (injection reactions) and incidental events.

Table 9.4. Vaccine reactions

Reaction	Vaccine responsible	Treatment
Acute flaccid paralysis	OPV	
Disseminated BCG infection—Widespread infection by *M. bovis* (used in BCG) within 1–12 months of vaccination, usually in immunocompromised subjects	BCG	Antitubercular drugs
Lymphatic swelling and local ulcer within 2–6 minutes	BCG	Firm–No treatment Soft–Aspiration Persistent sinus after 12 weeks—INH 5 mg/ kg × once daily x 3 months
Incessant cry within 48–72 hours	DPT	Analgesic Feeding
Encephalopathy—Seizures, sensory and behavioral alternation within 48 hours of DPT pr 7–12 days after measles vaccination	DPT, measles, MMR	
Seizures (24–48 hours)	DPT Measles	Diazepam, antipyretic, IV fluids
Fever between 12–24 hours of vaccination	DPT Measles	Antipyretic sponging, paracetamol
Toxic shock syndrome	Measles (caused by contaminating Staphs)	IV fluid, cloxacillin, steroids, antipyretic
Sterile abscess within 72 hours of vaccination	T series HBV Typhoid	Drainage if needed
Sciatic injury (gluteal injection)	Any	Antimicrobial, antipyretic, drainage of abscess if any
Local reaction (nonfluctuant swelling/redness)	Any	Paracetamol
Anaphylaxis	Any	Adrenaline, cardiopulmonary resuscitation, IV dextran, cortisol, oxygen
Hypotension	DPT	IV fluids, dexamethasone, Oxygen

Assessment of immunization status in community

Cluster sampling—The community is divided in 30 sectors, and 7 children who are 1st child of their parents and belong to 12–23 months age group are seen from each sector for immunization status.

A **fully immunized child**

1. has completed primary doses before 1 year
2. has taken vaccines at least at 4 weeks interval
3. has taken no vaccine *before* time

Postexposure prophylaxis

It is the combined administration of vaccine + immunoglobulin and AMA if necessary immediately after exposure to the agent (i.e. bitten by a dog, pricked by a needle).

Chemoprophylaxis

Table 9.5. Chemoprophylaxis

Disease	Drug	Indication
Cholera	Tetracycline	All contacts
Bacterial conjunctivitis	Erythromycin ointment	All contacts
Diphtheria	Erythromycin	All contacts
Influenza	Amantadine	For the elderly
Malaria	Chloroquine	Travelers, pregnant women
Meningococcal meningitis	Sulfadiazine, rifampicin	All contacts
Plague	Tetracycline	Contacts of pneumonic case

Investigation of epidemic

What people tell you

That a substantial number of people are suffering from a common ailment of recent onset (i.e. passing loose stools frequently associated with nausea, vomiting, abdominal cramp, fever). In countries like ours, where most infectious diseases are endemic, we don't mind a few dozens of malaria patients flooding our wards at a time. However, it is imperative to treat *every* occurrence of an infectious disease as potentially disastrous, and investigate accordingly.

What you need to know

Verify diagnosis

1. Confirm case definition—Clustering of cases *per se* is not conclusive of an infectious disease; for example, several cases of postoperative hepatitis in a hospital, which was initially suspected to be due to hepatitis B (which could have spread through blood transfusion during hospital stay), were later found to be due to use of halothane during anesthesia.
2. Identify whether it is a NEW disease.
3. If the disease is very contagious and severe (such as the H1N1 influenza of 2009), ask for international collaboration for such a disease.

The method of confirmation of diagnosis is to serach for suggestive history of exposure in every case. First, get the *index case* to sit before you and ask about

1. His personal characteristics (for later identification).
2. Food history of 48 hours (what, when, how).
3. His recent activities (unsafe sex, handling dangerous micro-organisms, working in a mosquito dense area, etc.)

Every case must be interviewed similarly; *sampling* is NOT allowed in investigation of epidemic (and also, if you remember, in census). Go for laboratory diagnosis of cases (at least a few cases). Lastly, *treat* the cases appropriately.

Check whether it is really an epidemic

Define the population at risk. Map the geographical expanses of the community involved, and obtain census data of that region. Mark down high risk individuals for the disease. The denominator for all epidemiological calculations should be this 'population at risk'.

Confirmation of epidemic. If a disease is *endemic* in a community, it is necessary to estimate its previous frequency and thereby confirm an increase in incidence *above the normal endemic level*. *Pseudo epidemics* may arise from sudden increases in doctors' or patients' awareness of a disease (more people begin to report previously ignored symptoms). When the endemic level has been defined from incidences over previous weeks, months, or years. Usually, if the incidence of the disease exceeds mean (endemic) incidence by two standard errors, it is said to be an epidemic.

Propagated epidemics emerge gradually whereas **point source** epidemics, arise abruptly.

Search for cases

Typically, only a fraction of total cases will report to you. You have to find the rest of the cases yourself. This has two purposes.

Determining the cause of the epidemic. Map their distribution of cases according to

1. **Time**—Prepare an epidemic curve (see the chapter on epidemiology), which will you what kind of epidemic it is (point source/propagated), what is the incubation period and how quick is its rate of spread.
2. **Place**—Prepare a spot map of cases, which again tells you the nature of the epidemic.
3. **Persona**—Analyze the personal characteristics of the cases (do they have some activity and exposure in common, which might have caused the disease).

Find attack rates for each suspected determinant you suspect from these three categories. For example, suppose in a community feast attended by a thousand people, several have fallen sick. From analysis of cases, you find that most of the people who have eaten either the salad or the curd have fallen sick. You have too now determine the attack rate of salad (people who have eaten salad and fell sick/ total number of people who have eaten salad) and curd. If attack rate for salad is greater than curd, you have a stronger suspicion. The next logical step would then be to ask everybody who has eaten curd—"Did you also eat salad?" It may be that salad is the real cause of disease, and it is only by chance that most of whom have eaten salad have also relished the curd.

Evaluate the environment

Check and identify any sanitation breakdowns (i.e. a leaking sewage pipe, a factory producing toxic waste, abundance of mosquito breeding places, unhygienic cooking practices) in the alleged locality.

Now decide ***what caused this epidemic***

Make a hypothesis

Your hypothesis should look something like this—"eating from the year end feast has caused this epidemic of food poisoning" or whatever the case may be. In total, you must try to identify the **chain of transmission**, i.e. what is the reservoir of infection → how is it transmitted → who are being affected?

Test the hypothesis

Compare attack rates of every plausible factor for a reasonable deduction. A *retrospective cohort study* is ideal in such cases. See the chapter on statistics on how to test hypotheses.

Do the formalities

That is, write a nice report with the summary of the epidemic, the control recommendations and evaluation of strategies.

Continue investigation

…up to last case + 2 × incubation period. The follow up includes all laboratory investigation of cases to know more about pathogenesis of the disease.

INTESTINAL INFECTIONS

Acute diarrheal disease

Acute diarrhea is passage of loose stools with recent change in consistency and frequency usually > 3 per day. *Persistent diarrhea* is that which lasts for 2 weeks. *Dysentery* is diarrhea with blood.

Excluded from the definition of diarrhea are the following,

1. Frequent, formed pasty stool in older children and adults
2. Defecation just after or during feeding (gastrocolic reflex)
3. Loose greenish yellow stool on 4th day of life (transitional diarrhea)
4. Semi loose stools in breastfed infants.

Diarrhea, along with ARI, is the major cause of morbidity, mortality and malnutrition in under five children. IT imposes two great dangers - fluid and electrolyte loss, and malnutrition (Fig. 9.12).

Figure 9.12. The cholera bringing death: drawing from the middle ages, from Le Petit Journal[305]

Agent

Table 9.6. Causes of diarrhea

Noninvasive (i.e. act through toxin, do not infest the GI tract) food borne bacteria	*Staphylococcus aureus, Bacillus cereus, Vibrio parahemolyticus*
Noninvasive water borne bacteria	Enterotoxigenic *E coli, enterohemorrgahic E coli, Vibrio cholerae, Vibrio parahemolyticus, Shigella dysentery* type I, Clostridum difficile, Clostridium welchii type A2
Invasive water borne bacteria	*Shigella dysenteriae type I, Salmonella* (all species except *S typhi*), Campylobacter jejuni, enteroinvasive *E coli, Yersinia enterocolitica, Yersinia* pseudotuberculosis
Invasive food borne bacteria	*Clostridium welchii, Bacillus cereus*, Salmonella (all species except S typhi), Shigella
Virus	Rotavirus (major cause of diarrhea between 6m – 2yrs) , Adenovirus, Norwalk virus, HIV
Protozoa	Giardia intestinalis, *Cryptosporidum parvum*
Fungi	*Candida albicans*
Food	Lactose intolerance Allergy
Systemic diseases	Renal disease Endocrine disorders
Organic bowel diseases	Appendicitis, short bowel syndrome, intussusception
Drugs	Amoxicillin, ampicillin, azithromycin
Neural	Encopresis

Host

Diarrhea affects predominantly children of 6 months – 2 years, especially those who are malnourished.

Environment

Like most water borne infection, diarrhea flares up in warm and rainy seasons. Faulty breastfeeding and insanitary complementary feeding are mostly responsible for such seasonal spruce up.

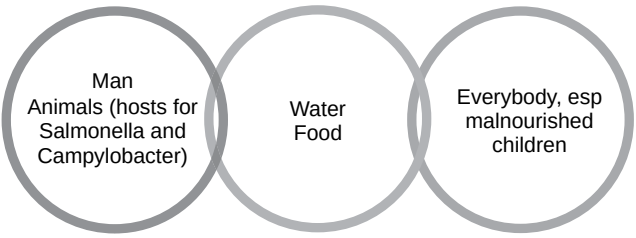

Figure 9.13. Chain of transmission of diarrheal diseases

Control

Controlling the reservoir

Usually cases are the only reservoirs. Appropriate case management (early diagnosis, notification, isolation and treatment) should follow quickly. Three principles of diarrheal management are,

1. Oral rehydration therapy
2. Correct feeding practice during illness
3. Avoid unnecessary antimicrobial drugs.

Break the chain of transmission

Establishing a sanitation barrier between feces and water (see 'Health and environment').

Protect the host

Health promotion. Better mother and child care in the form of

1. Improved nutrition during prenatal and postnatal period
2. Exclusive breastfeeding
3. Appropriate complementary feeding
4. Supplementary nutrition.

Measles vaccination may be required to prevent malnutrition, as co-infection with measles seriously hampers the nutritional status of the child. Also, education on diarrhea, its causes, home management and proper use of ORS is necessary.

Diarrheal disease control program, 1980

Aims

Reduce mortality due to diarrheal diseases (including cholera) through oral rehydration therapy.

Table 9.7. Types of fluids for oral therapy

Home available fluid (HAF)	Plain water, coconut oil, dal water without salt, lemon water + ADEQUATE FOOD/ BREASTFEEDING
WHO ORS (**311 mmol/L**)[a]. Dissolve one sachet in 1 l water (for infants < 2 months of age, dilute in 1½ l water as they have poor renal function). **Shelf life** is 24 hours; can't be kept overnight after preparation	**Composition**. NaCl 3.5 g, Na_3 citrate dehydrate 2.9 g, KCl 1.5 g, glucose 20 g **Ionic composition**. Na+ 90 mEq/l, K+ 20 mEq/l, Cl- 80 mEq/l, HCO_3–30 mEq/l (the citrate produces bicarbonate in vivo)
Home made fluid. The once practiced salt sugar solution is NOT recommended, because mothers frequently put extra salt and sugar in the solution, so that it becomes hypertonic and draws even more water from the GI tract)	Salt 5g Sugar 20 g Water 1l
Rice ORS. 50 g puffed rice in water **Amino acid** ORS. Not very much in use	More effective in cholera

Contd...

Contd...

Low osmolarity ORS (**245 mmol/l**). Since 2004, UNICEF and WHO have changed the composition of ORS for better efficacy. More effective in noncholera diarrhea.**ReSoMal**, (resuscitation solution for malnourished) is ORS specially developed for severely malnourished children	**Composition**. NaCl 2.6 g/l, glucose 13.6 g/l, KCl 1.5 g/l, Na_3 citrate dehydrate 2.9 g/l **Ionic composition**. Na+ 75 mEq/l, Cl- 65 mEq/l, glucose 75 mmol/l, K+ 20 mEq/l, citrate 10 mmol/l
[a] The ORS was developed by analysis of composition of cholera stools. The credit of the invention usually goes to **Dr Dilip Mahalanabish**, who thought of it during the cholera epidemic in Bangladeshi refugee camps in their freedom fight of 1971.	

Rationale

Loss of water and electrolytes is the usual cause of death (not inflammation or sepsis) in diarrhea. It is thus useful to classify patients on basis of dehydration and formulate a treatment plan (Fig. 9.14).

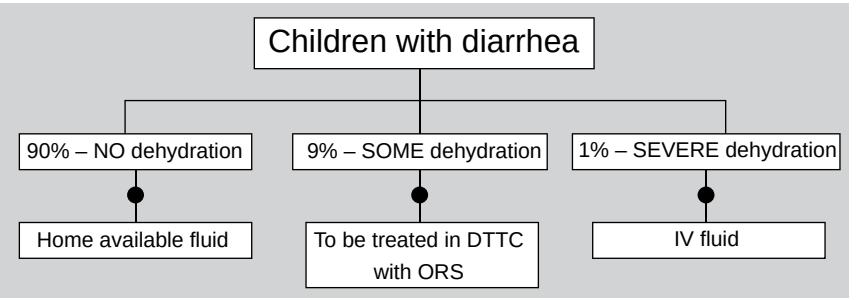

Figure 9.14. Categories of diarrhea patient

Coverage

1. Correct case management at home and health facility
2. Improve ORT use rate to 60% by 2000.

Strategy

1. Correct case management
2. Ensure supply of ORS by health functionaries and village level workers
3. Eliminate irrational drugs
4. Educate mother about danger signs.

Implementation

Assess nature of dehydration

History. Duration of illness, consistency of stool, presence of blood, convulsion, fever, associated illness, feeding practice, drug history.

Examination.

Table 9.8. Treatment plans for diarrhea patients

	Plan A	**Plan B**	**Plan C**
Condition	Alert	Restless, irritable	Lethargic, unconscious
Eyes	Normal	Sunken	Very sunken and dry
Tears	Present	Absent	Absent
Mouth and tongue	Moist	Dry	Dry
Thirst	Normal	Thirsty	Very thirsty but drinks poorly
Skin pinch return time	<2s	>2s	>>2s
Conclusion	No dehydration	Any two signs including one in **bold** = SOME dehydration	Any two signs including one in **bold** = SEVERE dehydration

Plan A: oral rehydration with home available fluids

Plan A was devised to treat diarrhea with no dehydration totally at home.

1. PREVENT DEHYDRATION. Administer HAF more than usual. If child vomits, it gives small sips frequently. If it doesn't improve in 2 days, call for a health worker.

Table 9.9. Plan A for no dehydration

Age	**HAF to be given after each stool**	**HAF per day**
<6m	50 ml	
6m–2y	50–100 ml	500 ml
2y–5y	100–200 ml	1l
6y–10y	100–200 ml	1l
>10 y	As much needed	2l

2. PREVENT MALNUTRITION. Give plenty of food. Continue breastfeeding wherever applies. If the child is taking solid food, give cereals, pulses, vegetable, and bananas (for K+ ions). See that food is well cooked. Encourage feeding and push the child to eat as much as 6 *times* a day.
3. PREVENT PROGRESSION: Watch for signs of some or severe dehydration and give ORS if it develops or come to a health worker.

Plan B: ORS

Plan B is applies for patients with *some* dehydration. They should ideally be treated in **Diarrhea Treatment and Training Centers** (DTTC) where they are temporarily admitted, treated for the duration of their illness and discharged quickly.

CORRECT DEHYDRATION. Intensely feeding ORS in 1st 4 hours; amount of ORS in ml = **patient's weight in grams** × **0.075**. Check both for dehydration and over hydration regularly. After 4 hours, assess again and recategorize.

Table 9.10. Plan B for some dehydration

Age	Amount of ORS in 1st 4 hours
< 4m	200–400 ml
4m–11m	400–600 ml
12m–23m	600–800 ml
2y–4y	800–1000 ml
5y–14y	1200–2200 ml

If the condition remains same after 4 hours of ORS → continue for another 4 hours → if it improves, continue ORS at maintenance dose (plan A) after each stool; if it shows no improvement however, switch to plan C.

Plan C: IV rehydration

Indications

- Severe dehydration
- Shock
- Plan B failure
- Abdominal distension due to hyponatremia
- Vomiting continuing > 3 hours
- Failure to drink ORS deranged sensorium

EMERGENCY REHYDRATION with 100ml/kg fluid is the rule. Use either,

1. Ringer lactate (the lactate yields bicarbonate which corrects acidosis)
2. Normal saline.

Table 9.11. Hourly plan for iv rehydration

Age	**First give 30 ml/kg in**	**Then give 70ml/kg in**
Infants	1 hour	5 hour
Older	½ hour	2 ½ hour

Initial rehydration should be done fast until pulse is easily palpable

↓

After 1–2 l has been infused, slower the rate

↓

From the point when patient can *drink*, give ORS 5 ml/kg/hr (may also be given by nasogastric tube)

↓

After 4–6 hours of treatment if pulse is still weak or oliguria remains then repeat the infusion

↓

As hydration improves follow plan B and A subsequently

↓

After 6 hours of good hydration, release the patient with all necessary advice to mother

Zinc supplementation

WHO and UNICEF have recently recommended zinc tablets for management of diarrhea (10 mg for children under 6 months, 20 mg for older children) × 10–14 days. Zinc tablets are now made available through subcenters and PHCs.

Cholera

We've got the cholerer in camp -- it's worse than forty fights;
We're dyin' in the wilderness the same as Isrulites;
It's before us, an' be'ind us, an' we cannot get away,
An' the doctor's just reported we've ten more to-day!
—Rudyard Kipling, "Cholera camp"

Cholera is the prototype of diarrheal diseases, and often the two terms are used as synonyms. Although amenable to very simple treatment, cholera has caused *seven* pandemics, most of which have originated from India. However, in the developed world, due to nearly universal advanced water treatment and sanitation practices, cholera is no longer a major health threat. The last major outbreak of cholera in the United States occurred in 1910–1911.[306]

Agent

Vibrios are comma shaped bacteria are a group of comma shaped Gram negative, described by Robert Koch as moving 'fish in stream'. Their classification is a point of confusion for many, so I do a little taxonomy here. Most enterobacteria (including vibrios) have two common antigens

- their flagella (H antigen).
- a phospholipid-protein-polysaccharide complex which forms the endotoxin (O antigen).

Depending upon variability of these antigens, vibrios are classified as follows.

Table 9.12. Classification of Vibrios by Gardner and Venkataraman

H Serogroup	O serogroup	Biotypes	Minor antigen serotypes
A (Cholera vibrios)	0, 1 (causes cholera)	Classical	Ogawa, Inaba, Hikojima
		El Tor	
	2–139 ('nonagglutinable' vibrios)		
B (heterogenous H antigen)			

While all isolates from cholera belonged to serogropus 0–1, not all members of these groups were seen to create cholera. Gouttschlich (1905) isolated some vibrios from Haj pilgrims in *El Tor* quarantine station. The pilgrims had not died of cholera but gangrene of colon. The bacteria came to be known as *El Tor* vibrios. However in 1961, *El Tors* caused the seventh cholera epidemic and were thus classified under cholera vibrios. Nowadays, the *El Tor* biotype has almost replaced classical cholera.

Table 9.13. Differences between classical and *El Tor* vibrios

Feature	Classical	El tor
Sheep blood hemolysis	–	+
Chicken RBC agglutination	–	+
Polymyxin B sensitivity	+	–
Phage IV susceptibility	+	–
Phage V susceptibility	–	+
Clinical picture	Severe watery diarrhea	Most cases are mild/ asymptomatic; prone to produce more carriers, less secondary attack rate

A new strain, the 0–139 Bengal strain was isolated in 1987, and was a causative agent in 1992 epidemic in Bangladesh, India and Pakistan.

Pathogenesis

The vibrios produce **enterotoxin**, which is a ligand for a GM1 ganglioside receptor present in the enterocytes, resulting in secretion of water, bicarbonates, sodium and potassium.

Host (Fig. 9.15)

Cases

Most infections in El tor cholera are mild or asymptomatic, and often get missed.

Carriers

Cholera harbors every type of carriers—Incubatory, convalescent, healthy and chronic carriers. Carriers do not have diarrhea and pass very few vibrios in their stools. For identification, they must be purged by administration of oral $MgSO_4$, and their stool must be cultured in an *enrichment* media (a liquid media where bacteria will multiply).

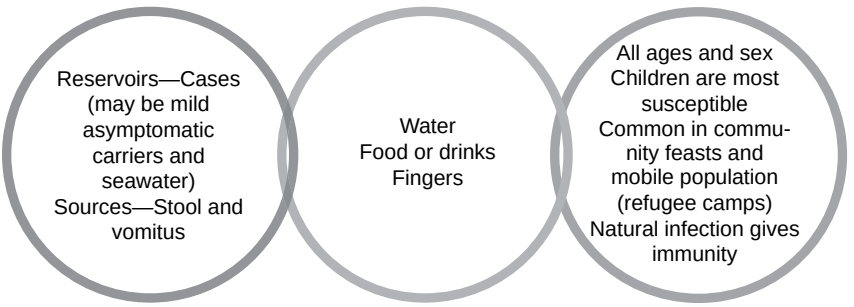

Figure 9.15. Chain of transmission of cholera

Incubation period

Few hours ↔ 5 days

Clinical course

Rice watery stool 30–40 times a day with mucus flakes, spontaneous explosive vomiting, severe abdominal cramps, usually no fever; death can ensue due to shock very quickly.

Labs

Sample

Stool is usually taken as sample.

1. A lubricated catheter is introduced in rectum and stool is collected in a screw capped container. Usually the mucous plaques that come out are observed. Each ml of stool has 10^6–10^9 bacilli.
2. **Rectal swabs** may be taken, which absorb about 0.1–0.2 ml fluid. They are useful in diagnosing convalescent patients who no longer have watery diarrhea.

The **drinking water** in the locality should also be collected as sample and transferred in peptone water medium.

Transport

Cholera stools are ideally transported in 4°C in either **Venkataram Ramakrishnan**/ Cary Blair medium. If the laboratory is within 1 hour distance, the stool may be transported in alkaline peptone water. If a selective medium like **Monsur's GTTTA** (Gelatin Taurocholate Trypticase Tellurite Agar) is used for transport, it automatically kills other bacterium and preserves only vibrios which produce grayish black colonies. Thus there is no need for further isolation in laboratory.

If **no transport media** are available, strips of blotting paper soaked in stool are sent packed in airtight plastic bags.

Motility study

The vibrio show 'fish in stream' motility in the mucous plaques. The characteristic motility can be seen in phase contrast/dark field microscope.

Culture

On arrival at laboratory, the vibrios are incubated in some **enrichment media** (alkaline peptone water tellurite) for 6–8 hours, so that their numbers amplify. They are now streaked in different media.

Nonselective media. Bile salt agar/MacConkey media are streaked. After overninght incubation, the colonies are tested for **agglutinability with cholera antiserum** (serogroup 0–1 antiserum). It can also be tested for chick RBC agglutination to distinguish between classical and *El tor* biotypes.

Selective media. *Vibrio cholerae* produces yellow colonies in **TCBS** (thiosulfate citrate bile salt sucrose) agar, and *Vibrio parahemolyticus* forms green colonies.

Biochemistry

Vibrio cholerae ferments glucose, sucrose, mannose (but *not* arabinose).

Investigating a cholera epidemic

For general guidelines, see 'investigation of an epidemic' at the beginning of this chapter.

1. **Verify** that the diarrhea is being caused by a *Vibrio cholerae* O1 or O139 strain.
2. **Notify** the incidence of cholera to health authorities and WHO within 24 hours of occurrence.
3. Case finding.
4. Treatment—See treatment of acute diarrheal diseases; if the case is diagnosed to be cholera, antimicrobials can be used to reduce severity and duration of the disease (Doxicycline 300 mg single dose for adults, Tetracycline 12.5 mg/kg × 4 × 3 days or Cotrimoxazole (with 5 mg/kg trimethoprim) × 2 × 3 for children. In pregnant women, Furazolidone must be used.
5. Investigation of local water supply, excreta disposal methods, food sanitation; to set up a 'sanitation barrier' between human excreta and drinking water (see the chapter on environment).
6. Disinfection of fomites of every case with cresol/bleaching powder.
7. **Chemoprophylaxis** to household contacts of a case with Doxicycline 300 mg or 6 mg/kg single dose.
8. Vaccination.

Cholera vaccines

Oral vaccines

1. **A killed whole-cell Vibrio cholerae O1 in combination with purified recombinant B subunit of cholera toxin**.
2. **A live-attenuated live oral cholera vaccine**, containing the genetically manipulated *Vibrio cholerae* O1 strain CVD 103-HgR.

Parenteral vaccine

It contains a mix of phenol killed vibrios to be given IM. The injection is painful and gives protection for 3–6 months, but not very effective. The CDC does not recommend cholera vaccination for prophylaxis.[307]

Enteric fever

Typhoid fever, also known as enteric fever, or commonly just typhoid (the rashes were thought to be similar to typhus), is an illness caused by the bacterium *Salmonella enterica serovar Typhi*.

Agent

Salmonella typhi, Salmonella paratyphi (A,B); the bacilli survive in polluted water and soil for 70 days (and in ice cream, unfortunately, for several months); they are readily destroyed by heating at 60°C for 15 minutes, or by pasteurization.

Host

1. **Age**—Most cases occur during adolescence (5–19 years) due their food habits. People over 60 have greater chance to become carriers.
2. **Sex**—Incidence is same in both sexes, but females tend develop into carriers Neither natural infection nor vaccination may give lifelong immunity.

Environment (Fig. 9.16)

Peak of cases occur during monsoon (July-September); unhygienic milk, and contamination of water with feces help the dissemination of the bacilli.

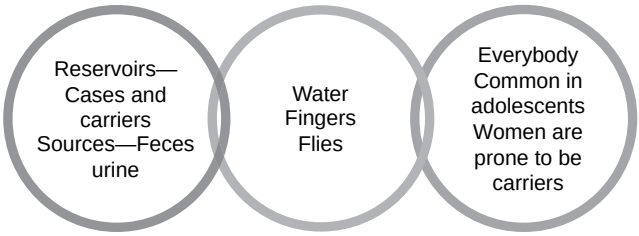

Figure 9.16. Chain of transmission of enteric fever

Enteric fever exhibits the whole gamut of carriers (Fig. 9.17).

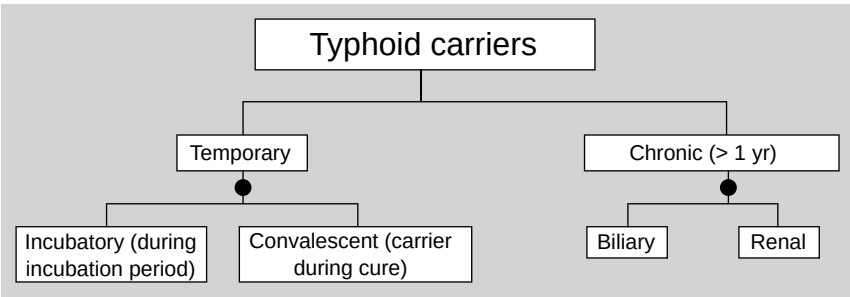

Figure 9.17. Carriers of typhoid

Clinical course

Incubation period = 10–14 days. Typhoid fever is characterized by a sustained fever as high as 40°C (104°F), profuse sweating, gastroenteritis, and nonbloody diarrhea. Less commonly a rash of flat, rose-colored spots may appear.

First week	Second week	Third week	Fourth week
• Slowly rising ('step ladder') temperature with bradycardia, malaise, headache and cough • Epistaxis is seen in a quarter of cases • Leukopenia, eosinopenia and relative lymphocytosis	• Patient lies prostrated with *high fever and bradycardia* ('Sphygmothermic dissociation'), classically with a dicrotic pulse wave • Delirium, **rose spots** appear on the lower chest and abdomen which *blanch* with pressure • Abdominal pain, greenish diarrhea	• A number of complications • Intestinal hemorrhage (due to bleeding in congested Peyer's patches) • **Perforation** in distal ileum (usually not detected right at incidence, but when peritonitis sets in, and	By the end of third week the fever has started reducing (defervescence) This carries on into the fourth and final week

Contd...

Contd...

First week	Second week	Third week	Fourth week
• Blood cultures are show *Salmonella typhi* or paratyphi • Widal test: **negative**	with a characteristic smell of 'pea-soup' • Constipation may also occur • Hepatosplenomegaly, elevation of liver transaminases • Widal reaction: **strongly positive with antiO and antiH antibodies**. Blood cultures are sometimes still positive at this stage	frequently fatal) • Paralytic ileus, encephalitis, metastatic abscesses, cholecystitis, endocarditis and osteitis	

Diagnostics

Blood culture

Inoculation of blood in MacConkey agar forms colonies within 10 days, which fermet glucose but not mannitol or lactose. H_2S is a by product of permentation and can usually be diagnosed by the black color in iron containing media (i.e. TSI media)

Widal test

This is a serological test to detect H and O antibody developed in patients of *S. typhi* and *S. paratyphi* infection.

Equal volumes (0.4 ml) of serial dilutions of serum (1/10, 1/20, 1/40, 1/80, 1/160, 1/320, 1/640) is taken test tubes and incubated in water bath 50–55°C for 2 hours

↓

Control tubes containing the antigen and normal saline are checked for auto agglutination

↓

H, O antigens of *S. typhi* and H antigens of *S. paratyphi* A and B are added in separate series. The paratyphoid O antigen is NOT added as it cross reacts with typhoid O antigen

↓

Agglutination titers are read. H agglutination forms *clumpy* coagulum, while O agglutination is seen as a disk like pattern below the tube

How reliable is Widal test

1. Antibodies appear in blood at the end of first week, reach peak at 3–4 week, and then fall.
2. Demonstration of a *rising titer of antibodies* on separate occassions is more reliable than a single test.

Contd...

Contd...

3. **Titers > 1/160 for O antigens and >1/160 for H antigens** is significant.
4. H antibodies persists longer than O.
5. Antibodies may be present beforehand due to previous infection/ immunization.
6. Patients who have been previously immunized may develop an anamnestic (i.e. 'unable to forget') antibody response with a fever unrelated to typhoid. But this response will be transient, while a true widal response will be retained after a week.
7. Bacteria used to prepare the antigens should be free of fimbriae.
8. **Antimicrobial treatment** drastically reduces antibody titer in blood.
9. **Carriers** will be widal negative.

Specific antibody tests

The Typhidot® test detects specific is a very rapid (3 hours) test to detect IgM antibodies against lipopolysaccharide membrane of *S typhi*.

Control

Controlling the reservoir

Cases

- Early diagnosis
- Notification
- Isolation in hospital until 3 negative stool samples are found on separate days
- Drugs: Ciprofloxacin 15 mg/kg × 7 days is the choide for uncomplicated disease; if the bacteria are quinolone resistant, try Azithromycin 8 mg/kg × 7 days or Ceftriaxone 75 mg/kg × 10–14 days
- Follow up
    1. 3–4 months after discharge
    2. After 12 months
- Disinfection of fomites
    1. 5% cresol
    2. 2% chlorine + steam.

Identification of carriers

1. Culture of stool, urine, duodenal aspirate
2. Serology (Vi antibody)
3. Sewer swab technique (samples are taken from house sewers and cultred; if *S typhi* grows, it indicates that there is a case or carrier in the house)

Treatment of carriers. Ampicillin 4–6 g + Probenecid 2 g × 6 weeks usually achieves good cure because it is eliminated mostly by bile; **cholecystectomy** may be required if gallbladder is irreversibly damaged.

Surveillance. The carriers should be prohibited from handling food or water for others. Intensive education on hygiene (wash hands after defecation, urination and before handling food) is necessary.

Breaking the chain

Water sanitation, cover food and water from flies, use of sanitary latrines.

Protecting the host

The older heat killed phenol extracted whole cell vaccines are longer recommended for use, because of pain and inflammation at the site of the injection.

Purified Vi polysaccharide. Injected IM or SC at a dose of 25μg in a single dose, additional doses every 3 years are needed to maintain protection. It is to be used only in children aged > 2 years.

Oral – Live vaccines. The vaccine is most commonly used to protect travelers to endemic countries, but there is no reason why the vaccine could not be used in large scale public prevention programs. The vaccine is given by mouth, either as capsules or a liquid suspension. The vaccine must be stored at 2–8°C, but will retain its potency for 14 days at 25°C. In the US and Canada, an initial course of **4 doses on alternate days** is recommended. Full protection is achieved 7 days after the last dose. A booster dose is recommended after 5 years.

Indications

The WHO recommends two vaccines: the live, oral Ty21a vaccine and the injectable Typhoid polysaccharide vaccine. Both are between 50–80% protective and are recommended for travelers to areas where typhoid is endemic.[308]

There is no indication of including typhoid vaccine in the universal immunization program, because the vaccines are only partially effective, and sanitation is a much better measure to control typhoid. Vaccination is indicated only in

1. Household contacts of typhoid case
2. School children during an outbreak
3. Hospital staff
4. Travelers to endemic area
5. People attending mass communions, where food is likely to be served.

Contraindications

Children below 1 year cannot handle a typhoic vaccine (Peyer's patches do not develop). Antimalarial drugs must be stopped 3 days before the ingestion fo Ty21 vaccine, or else the 'live' bacteria may die.

Investigation of typhoid sporadic

If you still remember, *sporadic* means irregular, infrequent cases widely separated by space and time, but may herald an epidemic.

Verify diagnosis

1. Clinically
2. Serologically
3. By blood culture (if needed)

Treat these cases as per schedule.

Collect data from

Institutes (where sporadics have occurred), surroundings (water sources, distribution, waste and excreta disposal methods, restaurants/communions where the affected people have ate).

Identify source of infection

Sources of infection are frequently contaminated water, ice, milk, milk products, vegetables, salad, shellfish. Food is usually contaminated during preparation or after preparation or during 'freshening' with contaminated water. Also, fruits and vegetables grown in 'sewage farms' are contaminated at their very origin.

1. Inside the institution: On the spot survey, sanitary survey (water supply an storage system, kitchen, latrine)
2. Outside the institution: History of recent eating out (feast/restaurants); consumption of processed food brought from outside; similar cases in locality.

Identify environmental breakdowns

Has any construction work nearby that has broken into water supply network/ sewage pipes? Take an engineering team with you (they know what lie in the undergrounds) and get to know the possible site of feco-oral contamination.

Identify reservoirs

1. Cases (inmates of institution, cooks) – Widal test (single cut off point may be set on area basis); a fourfold rise in H and O titer in a gap of 7 days is diagnostic than a single test. The rapid diagnostic tests may also be used.
2. Carriers (inmates/cooks)—Identify them by repeated stool examination (direct demonstration of bacilli in stool/urine/duodenal aspirate) and indirect Vi agglutination test (usually positive in carriers).

Prevent and control

1. Control reservoirs: Isolate cases, carriers and treat them
2. Break the chain of transmission: Repair sanitary systems
3. Protect the host: Typhoid vaccine, IEC.

Acute bacterial gastroenteritis (Food poisoning)

Food poisoning is an acute gastroenteritis caused by many bacteria/ toxins/ chemicals (fertilizers/ pesticides, Cd. Hg)/ vegetable or animal poisons in contaminated food or drink. We will chiefly be concerned with bacterial food poisoning.

Agent

Staphylococcus aureus (enterotoxin), Bacillus cereus (spores), *Clostridium welchii*, Salmonellae (typhimurium, choleraesuis, enteritidis), *Clostridium botulinum* A, B, E (botulinum toxin).

Table 9.14. Agents causing food poisoning

Name	Source	Mechanism and clinical course	Incubation period
Staphylococcus aureus	Found in everybody's skin, nose, throat (pyoderma of food handlers or mastitis of	Preformed toxin in food, which is heat resistant and acts directly on GIT →	1–6 hours (shortest)

Contd...

Contd...

Name	Source	Mechanism and clinical course	Incubation period
	milking cow is a good source); milk and milk products, custards, Russian salad (i.e. where milk is added) are very prone to contain staphylococci	sudden onset nausea, vomiting, diarrhea; fever and death are rare	
Bacillus cereus	Found in soil and everything that is borne out of soil (cereals), raw/dried or processed food	Heat stable spores who germinate in reheated food and form toxin in food; there are two kinds of manifestation • emetic form • diarrheal None of the two forms present with fever.	Emetic:1–6 hours Diarrheal: 12–24 hours
Clostridium welchii	Primarily spread through fecal contamination of food (meat and poultry)	Heat stable spores who germinate and 30–50°C (especially reheating) and liberate enterotoxins α and theta → cramps and diarrhea; NO FEVER OR DEATH	6–24 hours
Salmonellae	Salmonellae are spread though feces and urine of humans (carriers) , rats and mice; they are abundantly found in farm foods, poultry (especially egg with cracked shell), meat, milk products	Invasive colitis → sudden onset CHILL and FEVER, nausea, vomiting, watery diarrhea; 1% mortality	12–24 hours
Clostridium botulinum	Primarily resides in solid, makes it way in home preserved food (pickles)	Preformed toxin in food which is anticholinergic → dysphagia, diplopia, ptosis, quadriplegia, heart failure (but only minimal GI symptoms); about 50% mortality	12–36 hours

Host

Everyone is susceptible. People attending a mass feast falling ill are prime suspects.

Environment

Breakage of hygiene in processing/cooking of food is the root of food poisoning.

Investigation

Suggestive clinical features

↓

VERIFICATION OF DIAGNOSIS

Find index case and secondary cases; ask for personal characteristics, food history (what – when – where). Go for laboratory diagnosis if possible. DO NOT forget to treat these patients, they are not only academic interests

↓

VERIFICATION OF EPIDEMIC

Active search for new cases (for treatment and epidemiological characteristics) amd defining the population at risk (i.e. how many people ate at that particular feast) – remember, enumerate ALL the cases, NO SAMPLING is allowed

↓

FILL UP EPIDEMIOLOGICAL CASE SHEET

Ask all the population at risk to fill up personal characteristics and food history

↓

PREPARE THE EPIDEMIC CURVE

This will give you the INCUBATION PERIOD, and the probable causative agent

↓

EVALUATE THE ENVIRONMENT

Is the kitchen clean

Do the cooks have pyoderma/carriers of salmonellae? If they have, does the bacteria in them is the same as that in cases?

↓

DATA ANALYSIS

Formulate cause specific attack rates (i.e. how many people ate which item and how many of such fell ill)

↓

FORMULATION OF HYPOTHESIS

Construct the chain of transmission, i.e. reservoir (cook/soil) > mode of entry (into the food items) > susceptible hosts (i.e. who ate that food)

↓

TEST HYPOTHESIS

Do an analytical study (Case control study/ Retrospective Cohort study) to compare attack rates in people who have and have not ate a particular food item

If possible, do some serology to substantiate the biological plausibility; both cases and sources(cooks) will be +ve

↓

REPORT

Summary, Recommend control, Evaluate current strategy

Dracunculiasis

Dracunculiasis was declared 'eradicated' in February 2000, our millennium achievement, after the last case occurred in 1996.

Agent

Dracunculus medinensis infesting subcutaneous tissues.

Host

No particular preponderence to any age or sex.

Environment

1. Bathing in surface water.
2. **Step wells** help disseminate the eggs of the worms, as people literally 'step into' the well and the worm gets contact with water.
3. Water source contaminated with cyclops (a crustacean which is the host of the worm).
4. **Season**—In the dry months, the worm spreads through step wells; in the monsoons, it prefers ponds.
5. **Temperature**—25–30°C.

Chain of transmission

Reservoir/source

Human cases harboring gravid female in their feet.

Mode

Ingestion of water infested with cyclops. The worm nests in the subcutaneous tissue, particularly in those parts of the body which comes in contact with water (feet, hands). When the person dips his feet into water (as in bathing in a step well), the worm liberates eggs into the water which infects the cyclops.

Control

Controlling the reservoir

Treatment with niridazole/mebendazole; surveillance

Breaking the chain

1. Provision of piped drinking water; abolition of step wells.
2. **Control cyclops** by physical methods (straining and boiling), chemical methods (chlorine, lime, abate) or biological methods (Barbel fish, gambusia); see the chapter on entomology.

Poliomyelitis[309]

Problem

Although eliminated from the developed world due to vaccination, poliovirus is endemic in India, Pakistan, Afghanistan and Nigeria. The WHO has resolved in 1988 to *eradicate* the poliovirus, and the **Global Polio Eradication Initiative** [www.polioeradication.org] was formed in joint effort of WHO, Rotary International, the US Centers for Disease Control and Prevention (CDC) and UNICEF.

India

There were 50284 cases of acute flaccid paralysis in 2009 all over India, of which **741** was confirmed to be poliomyelitis, mostly contributed by two states—Bihar and UP. Notably, **21 cases** were due to **vaccine derived poliovirus** (VDPV). **At the time of writing (March 2010), there has already been 16 cases of poliomyelitis in 2010**.[310]

Poliomyelitis is undergoing certain epidemiologic changes,

1. Affecting higher age groups.
2. Shifting from temperate to tropical zones.
3. The **Type III** poliovirus has taken over Type I as the major wild strain, mostly due to the use of *monovalent* vaccine against Type I poliovirus as supplementary vaccination since 2005.
4. The **vaccine derived poliovirus** (VDPV) has emerged as a serious challenge to polio eradication; it has become emergent in those areas where there is a gap in immunization coverage. Because VDPV is the attenuated virus that is given in the oral polio vaccine, at some point of time we have to stop oral polio vaccination to prevent the rise of VDPV.
5. Has become confined to certain 'reservoirs' (UP and Bihar in India).

Persistent *pockets* of polio transmission in northern India, northern Nigeria and the border between Afghanistan and Pakistan are the current focus of the polio eradication initiative. **As long as a single child remains infected, children in all countries are at risk** of contracting polio. Between 2003 and 2005, 25 previously polio-free countries were reinfected due to imports of the virus.[311]

Agent

Poliovirus Types I, II, III; the virus was discovered by none other than **Karl Landsteiner**[312] himself.

	Type I	Type II	Type III
Paralysis	+++	+	++
Epidemic potential	+++		
Produces VDPV	+	++	+++
Eradication sequence	3rd	1st	2nd

Poliovirus Type III is the now the prevalent cause of paralytic poliomyelitis. The last case of Type II poliomyelitis occurred in India in 1999, and it is on its way to be the first to be eradicated among the three. Type I polio still occurs but with decreasing frequency.

Resistance

Resistant to phenol, chlorine and iodine, but heat sensitive and is killed by pasteurization; survives 4 months in water and 6 months in feces.

Host

Under five children, especially those between 6 m – 3 y. Males are affected more than females. *Immunity* is type specific, and is conferred by both clinical/

subclinical infection and vaccination. In immune individuals, IgA antibodies against poliovirus are present in the tonsils and gastrointestinal tract and are able to block virus replication; IgG and IgM antibodies against PV can prevent the spread of the virus to motor neurons of the central nervous system.[313] Immunodeficiency, tonsillectomy, trauma, fatigue or IM injections during an epidemic makes the child vulnerable to poliomyelitis. Though this connection between injections and paralysis has repeatedly been demonstrated, "the mechanism by which this phenomenon occurs is not well-understood. (However) the best evidence to date suggests the trauma initiates a reflex dilation of blood vessels at the corresponding spinal cord level and facilitates entry of the virus" [LaForce, 1983, p. 30]. *Pregnancy* seems to be a risk factor.

Environment

Poliomyelitis is a disease of monsoon disease (July-September), due to breakage in sanitation barrier and because the virus survives better in a cooler environment. Overcrowding and poor sanitation help the spread of the virus.

Chain of transmission

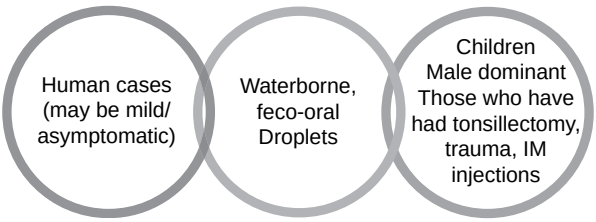

Figure 9.18. Chain of transmission of poliomyelitis

Communicable period is 7–10 days before symptoms ↔ 7–10 days after. **Maximum discharge in stool** occurs 2 weeks after paralysis, which may continue for several more weeks.

Is polio a by product of sanitation?

Poliomyelitis has had an rise in incidence in developed countries *after* introduction of modern sanitation systems, and have only been recently controlled by way of vaccination. One theory postulates that before the advent of modern sanitation, virtually all individuals were exposed to the poliovirus early in their lives when they were at least partially protected by maternal antibodies. Thus, they developed mild, nonparalytic infections, probably during infancy, which provided them with lifelong immunity. However, after we built sanitation systems, these early infections were not acquired, immunity did not develop, resulting in greater susceptibility to paralytic polio.

> Put simply, paralytic polio was an inadvertent by-product of modern sanitary conditions. When people were no longer in contact with the open sewers and privies that had once exposed them to the poliovirus in very early infancy when paralysis rarely occurs, the disease changed from an endemic condition so mild that noone knew of its existence to a seemingly new epidemic threat of mysterious origins and terrifyingly unknown scope.
>
> *—Patenting the Sun: Polio and the Salk Vaccine, Jane Smith, 1990, William Morrow & Co*

Clinical course

Incubation period = 7–14 days.

Inapparent disease (90–95%)

No symptoms, only get immunity from infection.

Abortive (4–8%)—Because it 'aborts' midway without entering the CNS

Non specific respiratory (fever, sore throat) and gastrointestinal (nausea, vomiting, abdominal pain) symptoms.

Nonparalytic 'aseptic' meningitis (1–2%)

No sensory alteration but Kernig's sign, neck rigidity, Brudzinski sign are found. Child returns to normalcy with immunity to wild poliovirus. On CSF examination, neutrophil count is normal (thus called 'aseptic') and virus is not easily isolated.

Paralytic (0.5%)

Acute flaccid paralysis = sudden onset weakness and floppiness in,

1. Any part of the body of a child < 15 years
2. A person of any age in whom poliomyelitis is suspected

Include all cases with

1. Current flaccid paralysis (occurring within 5 weeks)
2. Past flaccid paralysis
3. Ambiguous cases.

Paralytic polio is the rarest situation where the virus gets to lodge in anterior horn cells. Incidence of 1 clinical case of paralysis indicates at least a 1000 subclinical cases in the locality. The initial paralysis may not be the picture of the final outcome. **Early symptoms** of paralytic polio include high fever, headache, stiffness in the back and neck, asymmetrical weakness of various muscles, sensitivity to touch, difficulty swallowing, muscle pain, loss of superficial and deep reflexes, paresthesia (pins and needles), irritability, constipation, or difficulty urinating. Paralysis generally develops one to ten days after early symptoms begin.

1. **Spinal disease**—Affects lower limb preferentially (quadriceps, peronei, tibialis anterior), upper limb (deltoid, biceps, triceps), abdominal muscles (localized bulging – 'phantom tumor'); the paralysis is descending type, asymmetrical, causes no sensory loss and diminishes deep reflexes; death is caused by intercostals/diaphragm paralysis.
2. **Bulbar disease**—Affects IX and X (dysphagia, nasal intonation/ regurgitation, aspiration, dysphonia), death is caused by involvement of respiratory and cardioinhibitory center.
3. Bulbospinal disease.
4. **Encephalitis**—Headache, confusion, change in mental status, rarely seizures and spastic paralysis.

Overall, 5–10% of patients with paralytic polio die due to the paralysis of muscles used for breathing. The mortality rate varies by age: 2–5% of children and up to 15–30% of adults die.[314] Those who survive often live the rest of life with the stigma of one (or more) atrophic limbs, contractures, deformity.

Prevention

Primary

Health promotion. Personal hygiene, water sanitation, proper excreta disposal, good housing, IEC

Specific protection. Vaccination (routine, supplementary, outbreak response, mopping up).

Secondary

Early diagnosis. AFP surveillance, virus isolation from stool and virological classification from stool, serology (which, however, can not distinguish VDPV from wild virus and can not identify types of virus)

Treatment. Paralytic polio has no treatment.

Tertiary

Disability limitation. Correct positioning of limb (hip flexed, knee flexed, support); passive and active movements in sequence; hot water fomentation

Rehabilitation. Medical (calipers/crutches/abdominal support), vocational (occupational therapy), social (activities and normal schooling), mental (family support)

Control

Controlling the reservoir

Isolate cases of poliomyelitis and dispose their excreta safely.

Breaking the chain

Water sanitation, safe excreta disposal.

Protecting the host

Inactivated polio vaccine (IPV)

The first inactivated virus vaccine was developed in 1952 by Jonas Salk, and announced to the world on April 12, 1955.[315] The Salk vaccine, or inactivated poliovirus vaccine (IPV), is based on poliovirus grown in a type of monkey kidney tissue culture (Vero cell line), which is chemically inactivated with formalin. After two doses of IPV (given by injection), 90% or more of individuals develop protective antibody to all three serotypes of poliovirus, and at least 99% are immune to poliovirus following three doses.

Oral poliomyelitis vaccine

The OPV was introduced by Albert Sabin in 1957, produced by the repeated passage of the virus through nonhuman cells at subphysiological temperatures. It is a trivalent vaccine, i.e. bears all three types of virus grown in primary monkey kidney/human diploid cells.

Dosage. *2 drops* oral at birth, 6, 10, 14 weeks. It is important to be completed within 6 months. A booster may be given between 16–24 months.

Method. Tilt the head of the child, open its mouth by pinching the cheeks and drop the vaccine directly into tongue.

Herd immunity. The OPV infect the GI tract of the child if given sufficient time (OPV given to a child with diarrhea does not count as vaccination, because the child will egest the virus very quickly), and is passed in the child's feces into the environment. Administering the vaccine to all children within a short period will flood the environment with the vaccine virus and effectively eliminate the wild virus from its niche. **Thus OPV is not just vaccine for an individual**, **but for the community**. A certain **herd immunity** to poliomyelitis will develop only if 66% of community is vaccinated with OPV (Fig. 9.19).

Figure 9.19. We meet "Wellbee" again in this poster from 1963, who urges to receive the OPV. Note the first point she makes for the vaccine [CDC Public Health Image Library]

Contraindications. Acute infections, fever, diarrhea, dysentery, immunosupressed children.

Storage. Ideally at -20°C in a deep freezer; during transport, keep over dry ice; while administering the vaccine, keep the vials in ice packs. Recent stabilized vaccines can be stored at 4°C for a year.

Table 9.15. Differences between oral and inativated poliomyelitis vaccines

	OPV	**IPV**
Type	Live attenuated vaccine (trivalent)	Killed formolised vaccine (trivalent)
Route	Oral	SC/ IM
Immunity against clinical disease	Quick Systemic + GI immunity (so that the virus can not reside in GI tract and spread through feces) Long duration	Some weeks Only protects against paralysis (the virus will still manage to infect GI tract and spread through the feces of the child) Uncertain duration
Reinfection by wild virus	Impossible (due to GI immunity)	Possible
Useful in control of epidemic	Yes (vaccine virus replaces wild virus in environment)	No
Manufacture	Easy	Hard
Cost	↓	↑
Administration	Easier	Skilful (needs injection)
Storage	Stringent cold chain needed (it is the most heat sensitive vaccine)	Not need of stringest cold chain
Complication	Vaccine Associated Paralytic Poliomyelitis	None
Contraindications	Immunocompromised subjects Developed countries (where the wild virus has long been eradicated and the vaccine virus can start an epidemic of its own) Pregnancy People over 40 who have never been exposed to poliovirus/OPV earlier	Allergy

Polio eradication

Feasibility

Technical

1. No animal reservoirs.
2. No chronic carriers.
3. The virus half life of the virus in sewage, when it can spread, is only 48 hours.
4. Effective vaccine—OPV, which is cheap, easy to administer, gives intestinal immunity, suitable for outbreak control or mass campaign, provides herd immunity, delivered routinely and in 'pulses'.
5. Easy diagnosis from stool specimens.
6. AFP surveillance is a sensitive tool for monitoring poliomyelitis.

Operational

1. Polio is universally accepted as being of sufficient importance.
2. Social, political commitment and intersectoral coordination have already been obtained.
3. Social mobilization campaign and comprehensive plans for advocacy are on the go.

The following strategies are necessary for eradication of poliomyelitis.

Maintenance of high routine immunization coverage

Immunization coverage is obtained from reports from health centers/coverage evaluation surveys. Target is to immunize at least 90% of under five children.

National immunization days

From 1995 onwards, India has been observing NIDs in the form of pulse polio immunization (a 'pulse' as in a sudden push or thrush). It is the *supplementary* immunization (in addition to routine immunization) to all target children (< 5 years) irrespective of their immunization status, within a short period so as to flood the environment with vaccine virus (to replace wild strain), during low transmission seasons (winter). The aim of pulse polio is not confined to protecting that particular child but the entire community.

Intensified PPI

Initially, PPI was a single day activity run from particular polio 'booths', all throughout the country. With progression of eradication, PPI has been 'intensified' by extra 2 days of activity.

1. Day 1—Immunize children who come to booths
2. Day 2 and 3—Door to door search for nonimmunized children

With wild polio being focused in some areas, **subnational immunization days** (i.e. immunization only in endemic areas) have also been introduced. Depending on the type distribution of viruses, *monovalent* oral vaccines for Type I and Type III viruses are being used in different areas since 2006.

AFP surveillance

Aims of detection

Even if polio is eradicated, AFP will still have an incidence of 1 case/10000 under 15 children (background rate) due to other causes (Guillain-Barré syndrome,

transverse myelitis, etc.). This is the aim of detection, and the indicator for sensitivity of AFP surveillance.

Reporting

AFP reporting unit
For initial identification of AFP cases in hospitals
Report IMMEDIATELY by telephone, followed by a WRITTEN report
Even if no case is found, weekly reports must be sent
All private practitioners/homeopath/ ayurved practitioners must also report any case of AFP
↓
District immunization officer (District maternal and child health officer) or Surveillance Medical Officer (SMO)
↓
Center: National Polio Surveillance Project Register

Polio is also *notifiable to WHO* under the International Health Regulations.

Investigation

Initial investigation by DIO within 48 hours of notification and filling out case investigation form
↓
Adding the case to the **line list** of all AFP cases in that district in that year (to avoid ambiguity and duplication of cases)
↓
Stool collection and shipment to WHO accredited laboratories

Adequate specimens of stool

1. 2 specimens, at least 24 hours apart collected in a wide mouthed plastic/glass bottle with external screw cap
2. Collected within 14 days (i.e. period of maximum virus excretion) of onset of paralysis
3. Each of 8–10 g
4. Sent to DIO on same day; DIO will send the sample to laboratory within 2 days
5. Arriving at a WHO certified laboratory in good condition (not dessicated, no leak, well documented to prove reverse cold chain[316] maintenance); should be carried in specimen carrier/vaccine carrier with ice packs.

The requisition for examination should contain
1. Date of collection
2. EPID number (IND WB MSD 06 01 – to denote country, state, district, year and sequence of case, respectively)
3. Case identification data (name, age, sex, address, Doctor's particulars)
4. To whom the laboratory report should be sent
5. Date of onset of paralysis
6. Number of OPV does received
7. Date of last OPV dose.

The container should be labeled correct identification (specimen no. and EPID no.). The report must reach back to DIO in 28 days. The DIO should follow up the case after 60 days of paralysis. The finding of residual weakness on follow up after 60 days, is suggestive that the case may actually be polio, and this information is taken into account during final virologic classification.

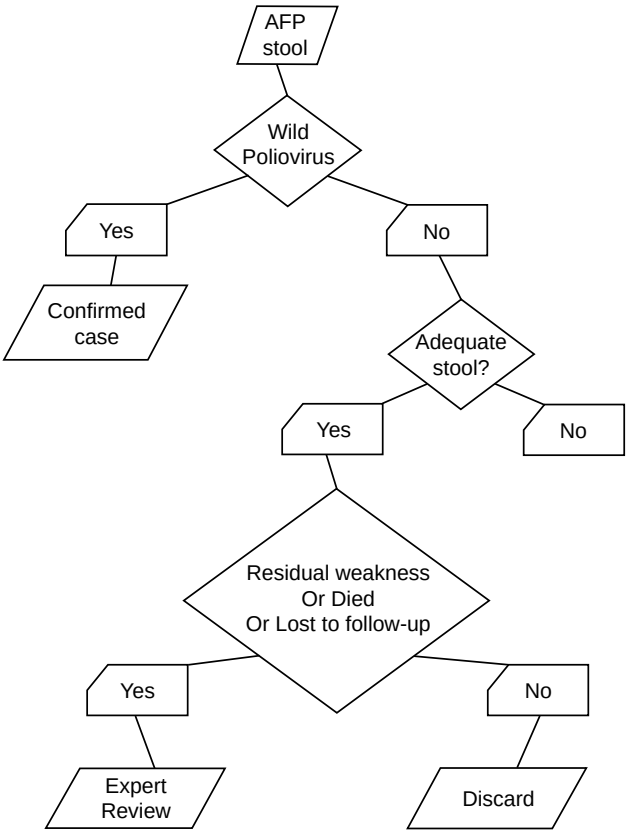

Figure 9.20. Virologic classification of stools

Control

A single AFP constitutes a polio epidemic. Control measures to be taken by DIO are

1. Active search to find additional cases(1 case = 1000 subclinical cases); look for all under 15 children who have developed flaccid paralysis within 60 days before paralysis of this particular patient
2. **Outbreak response immunization**—OPV to 500 under five children encircling that area/ all under five children in that village or ward.

Indicators of AFP surveillance

1. **Nonpolio AFP rate (background rate)** should be at least **1 per 100000** under 15 children; it indicates the *sensitivity of reporting* of AFP cases; in India, it is 10.72 on 20th February, 2010.
2. Proportion of AFP cases from where *adequate specimens* (see earlier) was collected – at least **80%**; upto 20th February 2010, adequate specimens were collected in 83% AFP cases in India.
3. Proportion of nonpolio enteroviridae should be found in 10–15% stool samples.

Mopping up immunization campaigns

When wild virus will be localized to pockets, house to house OPV campaigns to vaccinate all leftovers, will finally eradicate polio.

Global Polio Eradication Initiative Strategic Plan (2004–2008)

At present, most of the world does not use polio vaccines anymore because polio has been eliminated from those countries a long time ago. If somehow the virus escaped from its 'reservoirs' like UP and Bihar into those unprotected populations, it will cause a disasterous outbreak. Thus the modified plan concentrates

1. Eliminating polio in these reservoir areas through routine and supplementary immunization.
2. Improve quality of surveillance in these regions.
3. To develop a plan for safe cessation of OPV (otherwise, we will eradicate wild poliovirus only to face VDPV).
4. Integrate the human resource and infrastructure that has already been developed for eradication of poliomyelitis, into control of *other* diseases.

Hepatitis A

Agent

Enterovirus 72 or HAV, family Picornaviridae. It survives almost 10 weeks in water, and resistant to heat, acid and usual levels of chlorination. NaOCl has been recommended as a disinfectant for fomites of hepatitis A patients.

Host

Children have mostly mild or subclinical infections, but adults show severe disease. *Immunity* may be natural or vaccine induced, both long lasting (due to persistent IgG antibody).

Environment

Poor water sanitation, overcrowding helps the spread of this feco oral infection. Paradoxically, the same phenomenon that happened with poliomyelitis is now being seen with this infection, i.e. hepatitis A is rising in both incidence and severity where sanitary latrines have just been introduced.

Chain of transmission

Reservoir/ source

Cases (clinical/subclinical).

Mode

Feco oral (through food, fingers or water) parenteral (rare), venereal (common in homosexuals due to the combination of oral and anal sex). **Communicable period** is 2 weeks before jaundice ↔ 1 week after. Shedding of virus in stool reduced rapidly after onset of jaundice.

Clinical course

Incubation period = 15–45 days depending on infective dose. Nonspecific symptoms like malaise, vomiting, anorexia are much common than jaundice. In endemic regions, all cases of clinical icterus may be suspected as viral hepatitis.

Confirmation of hepatitis A

Acute onset fever and jaundice (fever preceding jaundice), malaise, anorexia, hepatomegaly, SGPT > 8 times of normal and serum bilirubin > 2 mg%. *Serology* gives more important evidence. Epidemic clustering of cases point towards diagnosis.

Lab

1. LFT to show bilirubin > 2 mg % and rise of SGPT
2. Demonstration of HAV particles or antigen in feces
3. Serology.

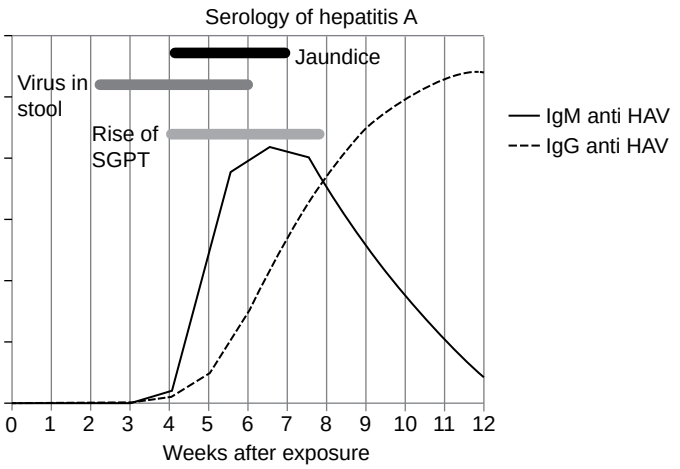

Figure 9.21. Hepatitis and serology

Control

Controlling the reservoir

It is very difficult because

1. Period of maximum infectivity (fecal shedding of virus) is during incubation period, when the patient would not normally come to the doctor.
2. Great number of subclinical cases.
3. There is no definitive drugs to render cases noninfective.

Breaking the chain

Water hygiene (especially boiling the water during an epidemic) with residual chlorine 1 mg/l (more than the usual 0.5 mg/l), hand washing, sanitation barrier.

Protecting the host

AntiHAV immunoglobulin is given to

1. Travelers to endemic area
2. Close personal contacts of cases in households, day care centers, epidemics (exposed within past 2 weeks)
3. To control outbreaks in institutes

A killed vaccine has been devised to be given IM twice at 6–18 months interval, in children more than 1 year of age.

Teniasis

Teniasis is a disease of pigs and cattle which is transmitted to man (zoonosis) as part of its life cycle. It is classified as a 'cyclozoonosis' because it needs at least to vertebrate hosts to complete its life cycle. The **definitive host** for both these parasites is man, and the **intermediate host** is pig or cows depending on the species (however, man can also become an **intermediate host** of *Tenia solium*, a condition known as cysticercosis). It is not a zoonosis in the sense hydatid disease is, where dogs are the definitive host and is only *accidentally* transmitted to man.

Agent

1. Taenia solium (pork tapeworm)—It causes intestinal teniasis (infestation by the adult worm) or cysticercosis (infestation by the larva) anywhere in the body.
2. Taenia saginata (beef tapeworm)—Not a major public health problem in India; in regions where beef is staple diet, it is very prevalent.

Chain of transmission

Reservoir/source

Cysticerci in undercooked pork, adult worm in human.

Mode

Ingestion of cysticerci/ova. In case of ingestion of cysticerci, the larva changes to adult in the intestine and inhabits the gut. When ova are consumed (specially in undercooked vegetables), they may be develop into larva and absorbed in blood → transported to various organs (specially brain) where cysticercosis develops.

Clinical course

Incubation period = 8–14 weeks. There is abdominal discomfort, chronic ingestion and anorexia. **Cysticercosis** (presence of larva in body tissue) presents with varying array of symptoms depending on the organ. In its most common form, it inhabits the brain (**neurocysticercosis**) and presents with epilepsy.

Control

Controlling the reservoir

Early diagnosis. Stool examination for ova
Prompt treatment. Praziquantel 15 mg/kg, repeat after 2 weeks or Niclosamide 60–80 mg/kg for one week.

Breaking the chain

Proper sewage treatment, prohibition of sewage farming (i.e. using sewage as manure), regular inspection of beef and pork vendors.

Protecting the host

Education on dangers of undercooked pork and beef; to avoid undercooked vegetables (especially the 'salad' served by many roadside vendors and restaurants), to learn proper cooking.

Hydatid disease

Agent

Metacystodes of *Echinococcus granulosus*, *Echinococcus multilocularis*, *Echinococcus oligarthus*, *Echinococcus vogeli* (polycystic hydatid cyst), all of which are small *cestodes* (same group as that of tapeworms).

Host

The **definitive host** is a number of canines (dogs, wolves); many species of vertebrates (commonly sheep, and accidentally men) may serve as **intermediate host**. Shepherds (who usually have a pet dog to keep watch over sheep and shoemakers (who handle animal hide) are the most susceptible.

Environment

Contact with dog (or more precisely, hand to mouth transmission of dog feces), uncontrolled slaughter and consumption of sheep, etc.

Chain of transmission

Reservoir/source

Eggs in dog feces, vegetables, household dust.

Mode

Ingestion of eggs (from dog feces or household dust), or ingestion of larva from flesh of sheep.

Clinical course

Incubation period = months to years. After ingestion of eggs/larvae, the larvae can migrate to any organ (commonly the liver) to form several generations of larvae in a fluid filled ball called a **hydatid cyst**. Hydatid cysts have the ability to grow quite large; cysts the size of golf balls are not uncommon, and cysts the size of basketballs are reported on rare occasions.

The only way a *dog* can get infected is by *ingestion* of the hydatid cyst. Because the worm normally runs a dog-sheep cycle, this is usually no problem (the dog/wolf can just eat the sheep). However, because humans cannot ordinarily be *eaten* by dogs, the human host is a dead end for the worm.

If a hydatid cyst breaks open, the fludi inside leaks out and produces a severe (sometimes fatal) anaphylaxis. A sharp blow to the abdomen might rupture a cyst in the liver (or a bear hug).

Labs

Casoni's reaction

Intradermal injection of hydatid fluid (sterilized by Sietz filter) produces a large wheal in half an hour with multiple processes in the sensitized individual (i.e. those exposed to the worm). This test is very nonspecific.

Serology

Complement fixation, hemagglutination, flocculation and ELISA test are now available.

Treatment

Surgery

Control

1. Meat inspection at slaughter house
2. Check stray dogs for infection and treat with praziquantel
3. Ensure that dogs do not eat sheep/do not gain access to sheep carcass
4. Education to shepherds, butchers, dog owners and shoemakers.

PARENTERAL ROUTES

Hepatitis B

Hepatitis B is a global problem characterized by its preponderence to cause cirrhois, liver failure and hepatocarcinoma, its long duration of illness, and production of *carriers*. It is 50–100 times more infectious than HIV, and an important occupational hazard for health workers.[317]

Agent

The hepatitis B virus (HBV) is one of the family *Hepadnaviridae* and genus *Orthohepadna*, a DNA virus with a reverse transcriptase enzyme. It survives in external environment for some days; destroyed by NaOCl and autoclaving (Fig. 9.22).

Antigens

1. The **surface antigen** (HBsAg) which is the marker of infection.
2. The **core/'insoluble'** antigen (HBcAg) present in the capsid of the virus; it is only expressed in the surface of liver cells after infection, and never found in blood.
3. The '**soluble**' antigen (HBeAg), a component of nucleocapsid, is secreted in blood and is an indicator of **ongoing virus replication**.

Host

Any age and sex is susceptible; *neonates* may acquire infection by placental transmission and they are very prone to become *carriers*. Recovery from disease gives life long immunity.

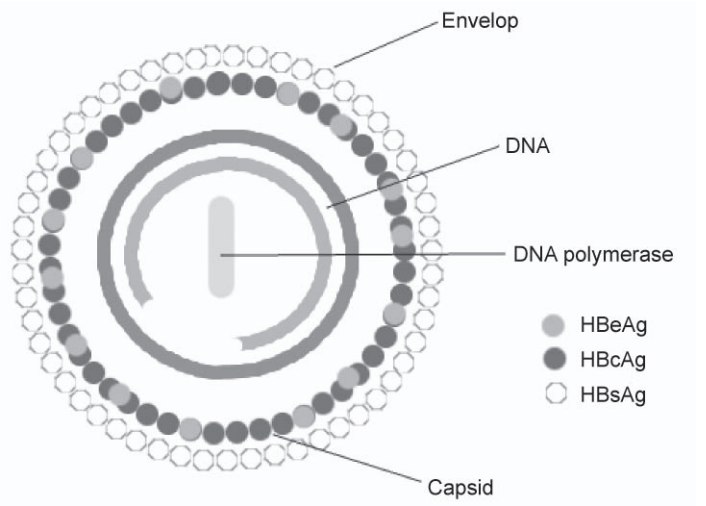

Figure 9.22. The hepatitis B virus

High risk group

1. Medical personnel
2. Laboratory workers
3. Homosexuals (anal sex causes more epithelial damage than vaginal sex)
4. IV drug users
5. Infants of infected mothers (becomes a chronic carrier)
6. Patients of repeated blood transfusion.

Environment

Nothing very particular. It is the 'social environment' that matters, not physical environment.

Chain of transmission (Fig. 9.23)

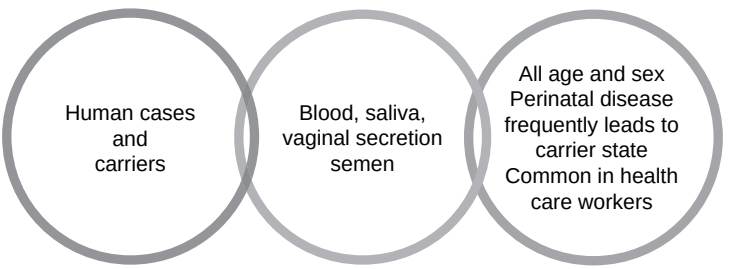

Figure 9.23. Chain of transmission of hepatitis B

Communicable period is 1 month before onset of jaundice ↔ appearance of antiHBs in blood. There are 4 main modes of transmission:

- Blood, blood products, contaminated syringes and needles, skin pricks, tattoing, body piercing, accidental inoculation by razors while shaving ...

- Sex (specially anal sex)
- Transplacental
- By physical contact from child to child (throuch cuts, abrasions and grazes).

Clinical course

Incubation period = **50–180 days**, **extremely variable**. After an exposure, two-thirds of cases develop an **acute hepatitis**, showing a flu like syndrome and jaundice, and the rest one third only have a subclinical infection.

Prognosis

1. About 90–95% patients (who have good cell mediated immunity) recover with resolution of liver in 1–2 months and eliminate the virus entirely from the body in 6 months.
2. 0.5% of patients with hypersensitivity (and coinfection with hepatitis D) develop **fulminant hepatitis**, often fatal.
3. 5–10% of patients with limited cell mediated immunity progress to chronic hepatitis B (HBsAg persisting in blood for more than 6 months).

Carriers

People with deficient cell mediated immunity usually becomes a carrier of HBV. Simple carriers bear either HBsAg or HBeAg in their blood. Super carriers bear both in blood.

Complications

Hepatic complications. Chronic hepatitis, hepatocarcinoma, fulminant hepatitis
Extrahepatic. Serum sickness like reaction, arthralgia, acute glomerulo nephritis, myalgia (due to immune complex deposition).

Labs

Serology gives the definitive diagnosis (Fig. 9.24).

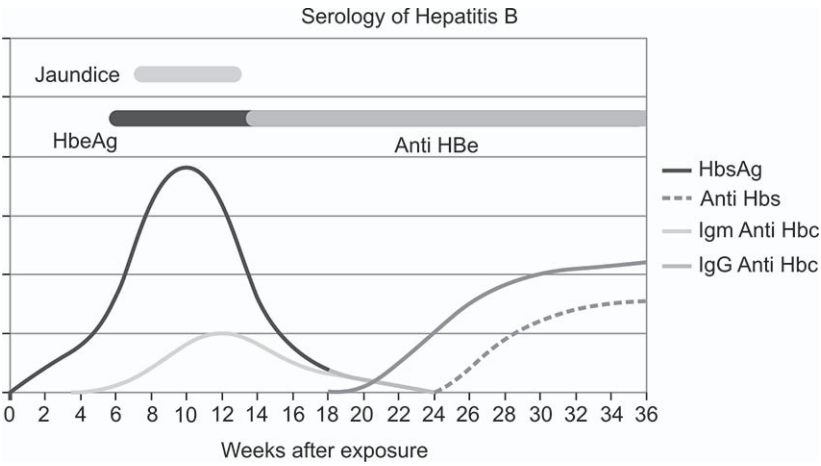

Figure 9.24. Hepatitis B serology

HBeAg is marker of *replication* (infectivity) and AntiHBs is the marker of *recovery*.

Table 9.16. Serologic diagnostics of hepatitis B

Situation	HBsAg	AntiHBs	AntiHBc	HBeAg	AntiHBe	Remarks
1	+	–	IgM	+	–	Acute infection, high infectivity
2	+	–	IgG	+	–	Chronic infection, high infectivity
3	+	–	IgG	–	+	Late acute or chronic infection, low infectivity
4	+	+	+	+/–	+/–	HBsAg of one serotype and anti-HBs of other serotype or, process of seroconversion (rare)
5	–	–	IgM	+/–	+/–	Window period (the gap where neither HBsAg or antiHBs can be found in blood)
6	–	–	IgG	–	+/–	Low level carrier Or, remote infection
7	–	+	IgG	–	+/–	Recovery
8	–	+	–	–	–	Immunized with HBsAg subunit vaccine or, remote infection/false positive

Prevention and control

Controlling the reservoir

- Treatment of chronic hepatitis cases with Adefovir dipivoxil, interferon alfa-2b, pegylated interferon alfa-2a, lamivudine, and entecavir.
- Advice chronic hepatitis cases and carriers not to donate blood.

Breaking the chain

Better quality of blood transfusion; to screen all blood fonors for hepatitis B infection; universal protection among health care workers.

Protecting the host

Education to risk groups, vaccination.

Hepatitis B vaccine (Fig. 9.25)

The hepatitis B vaccine is a **subunit vaccine** constituting of isolated HBsAg, which may be obtained from two sources.

- Plasma of human carriers (which is a scare resource)
- Recombinant DNA of yeast—This vaccine is more immunogenic and can be produced en masse.

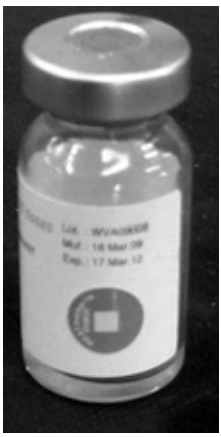

Figure 9.25. The hepatitis B vaccine: Note the vaccine vial monitor

Dosage. 1 ml vaccine (20 μg of HBsAg) IM stat (in the deltoid of adults and anterolateral thigh of infants, NEVER in the buttoks), 1 month and 6 months. A booster may be given after 3–5 years for the immunocompromised. The vaccine is being given to all children as a pilot program in some places. If an additional dose is given at *birth*, it protects against perinatal infection. The vaccine is freeze sensitive and should be stored between 2–8°C. When hepatitis B vaccine has been administered at the same time as other vaccines, no interference with the antibody response of the other vaccines has been demonstrated.

Who should get vaccinated? The CDC recommends vaccination for

- All babies, at birth
- All children 0–18 years of age who have not been vaccinated
- People of any age whose behavior or job puts them at high risk for HBV infection.

Pregnancy, lactation and immunocompromised state are NOT a contraindication for the vaccine.

Duration of protection. Immunologic memory remains intact for at least 23 years[317] and confers protection against clinical illness and chronic HBV infection, even though antiHBs levels might become low or decline below detectable levels. Protection could also extend lifelong. However, the vaccine is most effective only when given to infants, children and young adults.

Postexposure prophylaxis

For surgeons, nurse, laboratory workers, newborn of infected mother and sexual contact of acute patient.

HBIg 0.05–0.07 ml/kg × 2 at interval of 30 days. 1st should ideally be given within 6 hours of exposure. The subject should also be tested for HBsAg and if negative, a full course of vaccination is to be implemented.

Combined

HBIg within one day and vaccine within 7 days of exposure does not interfere, but help each other.

Hookworm infestation

Agent

Ancylostoma duodenale, *Necator americanus*. The infective form is the 3rd stage larva, which survives a month in soil, infects percutaneously and resides in intestine. Each adult worm survives 1–4 years.

Host

Agricultural workers working bare feet are most susceptible. The manifestations are also greater in malnourished people. In endemic areas, there may be few clinical signs due to a host parasite 'equilibrium', i.e. both have learnt to live together without damaging either.

Environment

1. Sandy, friable, damp soil with decaying vegetables provide ideal habitat for the larva
2. Temperature—24–32°C
3. Oxygenation of soil
4. Moisture
5. Indiscriminate 'open field' defecation discharges the worm in the environment
6. Sewage farming (worms in stool thus gain access to soil).

Chain of transmission

Reservoir

Cases

Source

Soil contaminated with feces 5–10 days ago (i.e. the time taken for the ova to devvelop into 3rd stage larva).

Mode

1. Skin > blood > lungs > trachea > esophagus (retrograde swallowing) > intestine. The **infective period** continues as long one passes the eggs in stool.
2. An *oral* infection may be caused by eating contaminated vegetables, which take the same course as a skin infection.

Clinical course

Incubation period = 7 weeks (a duodenale) and 5 weeks–9m (*N americanus*). To begin with, there is a *ground itch* at the site of entry, following 2 weeks of which

there is pneumonia. Most people are asymptomatic in regards to tract, but some show peptic ulcer like symptoms. Anemia is the chief pathology. Worms are an important cause of malnutrition, especially important in pregnancy.

Endemic index

Chandler's index = number of eggs/g of stool for entire community; if the index > 300, it indicates a dangerously high prevalence of hookworms.

Control

Controlling the reservoir

Deworming. Mebendazole 100 mg × 2 ×; 3 or Albendazole 400 mg single dose

Treatment of anemia. Iron folate tablets (100 + 500) × 2 × 3 months or when Hb reaches 12g%.

Breaking the chain

Sanitation barrier, health education.

Malaria

This day relenting God
Hath placed within my hand
A wondrous thing; and God
Be praised. At his command,
Seeking his secret deeds
With tears and toiling breath,
I find thy cunning seeds,
O million-murdering Death.
I know this little thing
A myriad men will save,
O Death, where is thy sting?
Thy victory, O Grave?

— Sir Ronald Ross, in a letter to Sir Patrick Manson, on Aug 22 1897, a day after he discovered the malaria parasite in mosquitoes

'Malaria' (a term coined in 1753) means 'bad air'. It is interesting to note that the treatment of the disease became well known before the etiology. **Charles Laveran** discovered the malaria parasite in an unstained preparation of fresh blood (1880). **Sir Ronald Ross**, in his will laboratory at the SSKM Hospital Kolkata, discovered the transmission of the parasite by mosquitoes.

Malaria is primarily a fever with chill and rigor with variably, jaundice and splenomegaly. The distinction of malaria has always been its **periodicity** of attacks.

The problem

World[318]

Globally, **one child dies of malaria every 30 seconds** (malaria stubbornly remains in the top killers list even in this age of noncommunicable diseases). There were

247 million cases of malaria in 2006, causing nearly one million deaths, mostly among African children. Approximately **half of the world's population is at risk of malaria** (and we thought we were close to eradicating it!). Travellers from malaria-free areas (who have no herd immunity) to disease "hot spots" are especially vulnerable to the disease. Malaria takes an economic toll - cutting economic growth rates by as much as 1.3% in countries with high disease rates.

India

Although the number of cases of confirmed malaria have decreased between 200–2008,[319] 27% of Indian population are still at high risk of malaria transmission, especially the north eastern states, where

1. Malaria is transmitted all the year
2. Falciparum predominates
3. Drug resistance is rampant.

The prevalent epidemiologic types of malaria in India are

- Tribal malaria—More due to lack of health facilities than disease propensity
- Rural malaria—*Anopheles culicifacies* is the chief vectors, vivax malaria predominates with occassional exacerbations of falciparum malaria
- Urban malria—Related to overcrowding, unplanned growth, overloading of water and sanitary systems
- Malaria in project areas (i.e. construction sites where many laborer have gathered to stay temporarily)—Limited health facilities result in chloroquine resistant malaria
- Border malaria—Related to immigration and mixing of populations.

Agent

1. *Plasmodium vivax* (75% in India)—Benign tertian (48 hour cycle) malaria
2. *Plasmodium falciparum* (25% in India)—Malignant tertian malaria
3. *Plasmodium malariae* (limited in south India)—Quartan (72 hour cycle) malaria
4. *Plasmodium ovale*—Not found in India

The definitive host of the parasite is mosquitoes, and man is the intermediate host.

Host

All ages and sexes are susceptible, children and elderly show milder symptoms but ↑ chance of death. The risk groups are

1. Farmers.
2. Mobile population in cities.
3. Travelers from malaria-free regions.
4. Nonimmune pregnant women—The illness can result in high rates of abortion and cause over 10% of maternal deaths.
5. Semi-immune pregnant women risk severe anemia and impaired fetal growth even if they show no signs of acute disease.
6. Lower socioeconomic groups, living in overcrowded houses, in insanitary environments, often without mosquito nets.

Immunity is acquired by infection but only partial; in regions where malaria is endemic for many years, people develop a certain degree of tolerace to the parasite and show no great clinical signs. *Sickle cell disease or trait protects against malaria*; Duffy –ve individuals have low risk against vivax malaria.

Environment

Factors which promote the growth of mosquitoes, i.e. monsoon, temperature 20–30°C, humidity > 60%, tropical and subtropical regions, inadequate irrigation and drainage system, ill ventilated, ill lighted housing, not using mosquito nets and plastering the walls *after* residual DDT spray has been applied.

Chain of transmission (Fig. 9.26)

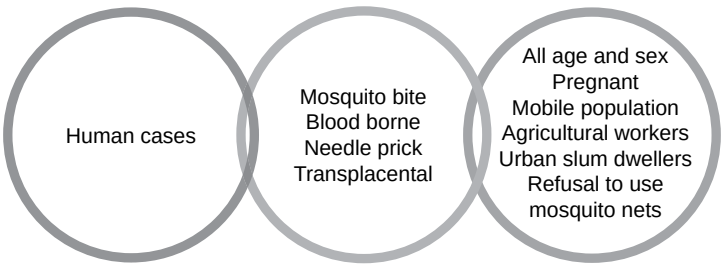

Figure 9.26. Chain of transmission of malaria

Reservoir/source

Human cases (may harbor multiple species of parasites). For the host to be infective (successful parasitism),

1. Must harbor both sexes of gametes
2. Gametes must be viable and mature
3. Gametes must be present > 12/mm^3 of blood.

The **communicable period** is as long as the blood contains gametocytes.

Mode

Vector

Infected female Anopheles mosquitoes. The **extrinsic incubation period is about 8–10 days**, and if the mosquitoe can be killed within this period, malaria may be prevented. The sporozoites develop and reach the salivary glands 8–10 days after the mosquito has bitten an infected individual. These **sporozoites are the infective forms**.

Table 9.17. Vectors of malaria

Vector	Distribution	Breeds in
Anopheles culicifacies	Rural	Fresh water (rice fileds)
Anopheles stephensi	Urban	Tanks, cisterns, tyres, gutters, coconut shells
Anopheles fluviatilis	Foothills	Moving water
Anopheles sundaicus	Coastal	Brakish

Malaria transmission rates can differ depending on local factors such as rainfall patterns, the proximity of mosquito breeding sites to people, and types of mosquito species in the area. Some regions have a fairly constant number of cases throughout the year—These countries are termed "malaria endemic". In other areas, there are "malaria seasons" usually coinciding with the rainy season.

Induced malaria

Spread through body fluids, i.e. blood transfusion (the parasite survives for 14 days in stored blood), needle prick, organ transplant. Notably, the infective form in this case is **trophozoites** who infect RBC directly and there is **no hepatic cycle** in such malaria.

Transplacental

Malaria can be disastorous in pregnancy as it can cause LBW, abortion and maternal deaths.

Clinical course (Fig. 9.27)

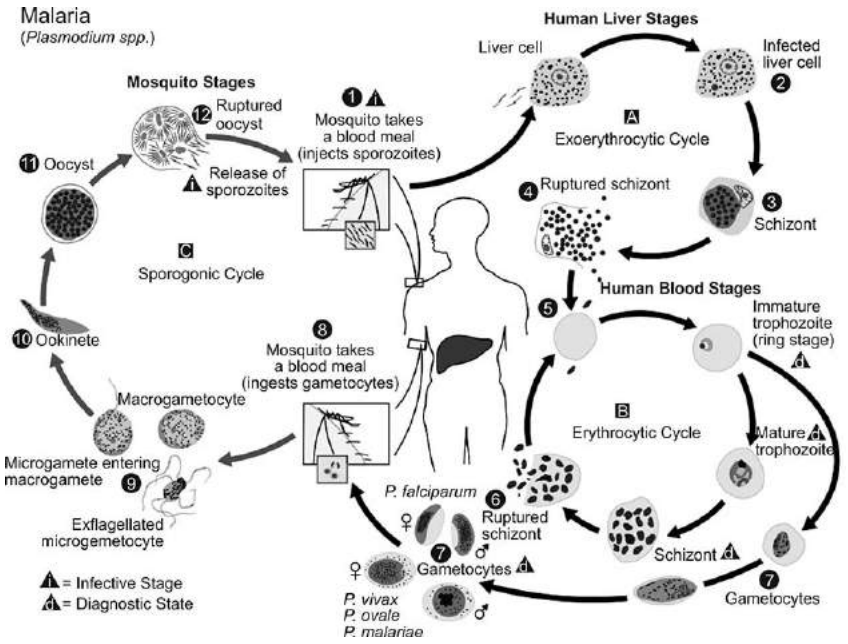

Figure 9.27. The life cycle of malaria parasite[US CDC Public Health Image Library]

Incubation period

It is the time taken for appropriate number of merozoites in blood as to cause fever after inoculation.

P vivax *P ovale* *P falciparum*	10–14 days
P malariae	18 days–6 weeks

Plasmodium malariae has the longest incubation period, and the others intermediate. People with partial immunity show **asymptomatic parasitemia**. The rest have fever (tertian – 48 hours or quartan – 72 hours; may be quotidian – 24 hours if **two generations** of parasites have infected), which may be associated with chill, rigor, jaundice and splenomegaly.

Table 9.18. Differences between vivax and falciparum malaria

	P vivax	***P falciparum***
Pre erythrocytic schizogony	6–8 days	6–7 days
Incubation period	12–17 days	9–14 days
Primary attack	Mild to severe	Severe
Duration if untreated	1.5–5 years; **relapse** is frequent (parasite is maintained in liver cycle for long time)	1–2 years; **recrudescence** may occur (parasite is maintained in RBCs for long time)
Complications and death	Does not occur	Occurs
Anemia	Uncommon	Common

Stages

Initial chill. It lasts from 15–60 minutes (sometimes causing convulsions)—It is due to relase of merozoites from RBC into blood. Nausea, vomiting and headache are common at this stage.

Fever. It lasts for several hours, can rise upto 40°C. In this stage, the merozoites break out from RBCs and invate new RBCs (Fig. 9.28).

Sweating. The patient falls asleep and later awakes feeling relatively well.

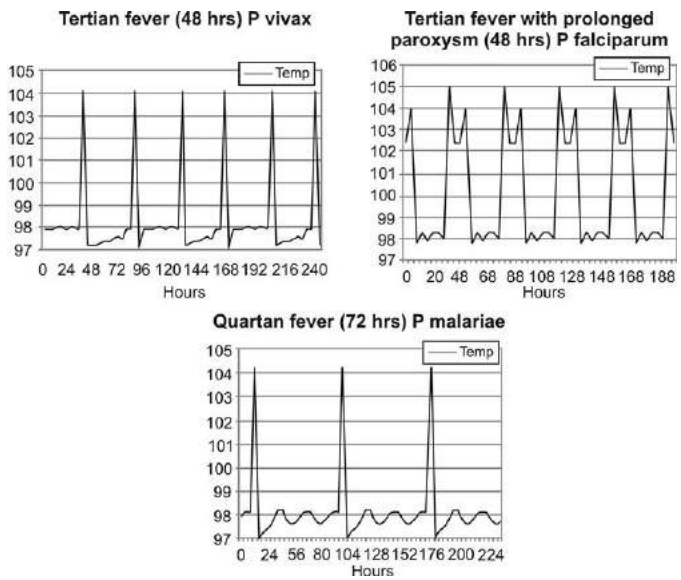

Figure 9.28. Types of fever in malaria

This 'classical pattern' is now being seen increasingly rarely.

Labs

The recommended method is blood smear examination for parasites. The blood is taken anytime during fever, and two smears, one thick (to identify parasite) and one thin (to know species) is made. Usually when the patient presents at subcenter, he is given presumptive treatment + smear taken by MPHW (male) and sent to PHC for examination.

Malariometry

Epidemiologic indices

Proportional case rate

Yearly malaria cases/total cases × 100

Spleen rate

Percentage of children of 2–10 years with enlarged spleen. It indicates the prevalence of malaria in children. It was the chief indicator in pre eradication era for classifying areas (< 10%—No malaria; 10–25%—Hypoendemic; 25–40%—Endemic; > 40%—Hyperendemic).

Infant parasite rate

Number of infants whose blood smear +ve / total number of infant blood slides examined × 100; it is the **most sensitive index for recent transmission of malaria**; if it remains 0 for 3 consecutive years, there has been no transmission of malaria.

Children parasite rate

As above, for children 2–10 years

Annual blood examination rate (ABER)

Number of blood slides examined/ total population × 100; it gives the **efficacy of surveillance**; a minimum of 1% of population should be examined monthly, or 10% of population in a year.

Annual parasite incidence (API)

Number of smear +ve cases in one year / total population in surveillance × 1000; it indicates incidence and is the current choice for classification of areas.

Slide positivity rate (SPR)

Positive smears in one year/total slides examines in one year × 100

Slide falciparum rate (SFR)

Percentage of slides in a year that disdplay *Plasmodium falciparum.*

Annual falciparum rate

Positive slides for falciparum in one year/population under surveillance × 100

Entomological indices

Vector density

Number of mosquito catch/ person/ hour; the **critical density** is the minimum number of moquitoes needed for transmission of malaria; for *Anopheles*

culicifacies it is 3.3/man/hour, for *Anopheles fluviatilis*, 1.3/man/hour (it is a much more efficient parasite).

Sporozoite rate

Proportion of mosquitoes which are infected (i.e. found to bear sporozoites on dissection).

Human blood index

Proportion of mosquitoes found to carry human blood on dissection (indicates anthropophilism).

Bite rate

Average number of anopheles bites per person per day; measured by the number of anopheles mosquitoes caught in a day by a human bait.

Inoculation rate

Bite rate × sporozoite rate (not all mosquitoes are loaded with sporozoites).

Indicators of antimalaria coverage[320]

Insecticide treated net (ITN) coverage

Number of ITN distributed in one year divided by two (assuming one net covers two people)/population at risk for malaria.

Artemisinin combined therapy (ACT) coverage

Number of ACT courses given/number of reported malaria cases.

Indoor residual spray (IRS) coverage

Percentage of at risk population protected by IRS.

Coverage of parasite based testing

Percentage of suspected malaria cases tested by microscopy or rapid diagostic tests.

Prophylaxis in pregnancy

Percentage of pregnant women in endemic areas receiving chemoprophylaxis.

Treatment[321]

Some pharmacology

There are six categories of antimalarial drugs

- **True prophylaxis** kills sporozoites as they enter the body; no drug is reliable in this regard, but chloroquine has been tried.
- **Causal prophylaxis** stops the hepatic cycle (primaquine, proguanil).
- **Suppressive prophylaxis** suppresses the RBC cycle so that clinical attacks are prevented (chloroquine, proguanil, mefloquine—All in low doses).
- **Clinical cure** clears parasite from blood (chloroquine, quinine, mefloquine, atovaquone, artemisinin derivatives, halofantrine, lumefantrine, doxycycline, pyrimethamine, sulfadoxine).

- Radical cure clears both blood and liver (primaquine).
- Community prophylaxis kills gametes in blood (primaquine, chloroquine, artemisinins).

Chloroquine

Chloroquine is the first line drug for uncomplicated malaria; it is RBC schizonticidal + gametocidal, but cannot be given in empty stomach. It is a cumulative drug, thus cannot be used continuously for > 3 years. If total chloroquine accumulation in body exceeds 15 g, it may cause *amblyopia* (loss of vision without any organic pathology in visual pathway).

Primaquine

Primaquine is used for preventing relapse and community prophylaxis; it is hepatic schizonticidal and gametocidal. It is *contraindicated* in infants, pregnancy and in G6PD deficient subjects (look for cyanosis for ½ an hour after dosing).

Choice of diagnosis and therapy (Fig. 9.29)

1. All fever cases should preferably be investigated for malaria by Microscopy or Rapid Diagnostic Kit (RDK).
2. The first line of treatment is chloroquine and the second line is ACT (artemisinin combination therapy), preferably artesunate + sulfadoxime + pyrimethamine combination. In severe malaria, quinine remains the drug of choice.

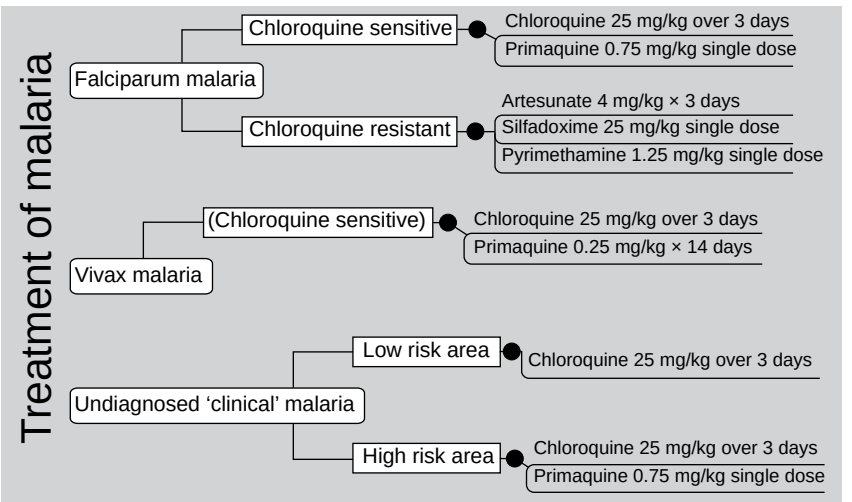

Figure 9.29. Treatment of uncomplicated malaria

Dosage for chloroquine sensitive malaria

Microscopically, positive falciparum cases. Chloroquine 25 mg/kg over 3 days (10 + 10 + 5) + single dose Primaquine 0.75 mg/kg on the first day

Microscopically, positive vivax cases. Chloroquine 25 mg/kg over 3 days + Primaquine 0.25 mg/kg × 14 days under medical supervision (primaquine is added to stop liver cycle and prevent relapse).

If only RDK for Pf is tested, negative cases showing sign and symptom of malaria without any other obvious causes should be considered as 'clinical malaria' and treated with chloroquine 25 mg/kg body over three days.

In situations where **diagnosis by microscopy or RDK is not possible**, cases showing sign and symptom of malaria should be considered as 'clinical malaria' and treated with chloroquine 25 mg/kg over 3 days in low-risk area (see later). In high-risk area single dose of primaquine 0.75 mg/kg should also be given on the first day.

Dosage for chloroquine resistant malaria

ACT is the first line of antimalarial drug for treatment of *P. falciparum* in chloroquine resistant areas. The dose is artesunate 4 mg/kg × 3 days orally and sulfadoxime/sulphalene 25 mg/ kg + pyrimethamine 1.25 mg/ kg on the first day. ACT should be given *only to confirmed* falciparum cases, and full compliance is to be assured (otherwise artemisinin resistance will develop soon). Primaquine may not be given with ACT combination as artesunate reduces gametocytes too.

Artemisinin should never be used alone, otherwise resistance will develop to this critical class of antimalarials.

Antimalarial drug preparations

Chloroquine. 150 mg tablets
Primaquine. 2.5 mg and 7.5 mg tablets
Artesunate. 50 mg tablets
Sulfapyrimethamine. 500 mg sulfadoxime/sufalene + 25 mg pyrimethamine

Indications to switch to ACT therapy

1. The area/PHC showing a treatment failure more than 10% (both early and late) to chloroquine in the minimum sample of 30 cases.
2. Change to ACT therapy in a cluster of PHCs around the resistant foci after taking into consideration the epidemiological trend of *P. falciparum* (*Pf* > 30%) and clinical response in these areas and approval of Directorate of NVBDCP.
3. If in spite of full treatment with no history of vomiting, diarrhea (i.e. clinical improvement has occurred) patient does not respond within 72 hours parasitologically.
4. In areas with high disease burden, high proportion of *Pf*, inadequate facilities for laboratory diagnosis and the inaccessibility and relatively poor communication facilities and the *Pf* chloroquine resistant pockets, ACT may also be given on clinical diagnosis of malaria by a trained medical officers or trained paramedical personnel.

What if the parasite is resistant to ACT?

Oral quinine with tetracycline or doxycycline.

Severe/ complicated falciparum malaria

One of

- Quinine IV 10 mg/kg in 5% dextrose, to be run in four hours × thrice daily; switch over to oral dose as soon as possible; total duration of therapy should be 7 days.

- Parenteral artemisinin derivatives (for adults and nonpregnant women only), i.e. Artemether 1.6 mg/kg IM × 2 × 3 or Artesunate 2.4 mg/kg IV + 1.2 mg.kg IV after 12 hours + 1.2 mg/kg IV for four days.
- In case of nonavailability of the above drugs, Chloroquine 10 mg/kg in isotonic saline should be infused over 8 hours followed by 15 mg/kg in the next 24 hours.

Prophylaxis

Chemoprophylaxis is useful for travelers and pregnant women (2nd trimester); chemoprophylaxis is contraindicated in children (chloroquine hampers development of natural immunity and quickens drug resistance).

The general scheme is chloroquine 10 mg/kg stat + 5 mg/kg once a week from 7 days before entry in endemic area ↔ 1 month after leaving. For chloroquine resistant malaria use chloroquine + daily dose of proguanil.

Control of malaria

Controlling the reservoir

1. **Case detection**—All fever cases should be presumed to be malaria unless proved otherwise, and presumptive treatment given. Diagnosis is done by blood smear examination and Rapid (antigen based) Diagnostic Kits.
2. Treatment.

Breaking the chain

Vector control is the keystone to control malaria.[322]

1. Antiadult—Indoor residual spray with DDT[323] 1–2 g/m_2 from 2 ft above floor; space spray with pyrethrum (for closed rooms), malathion (ultra low volume spray).

What is the rationale of indoor residual spray in malaria?

   a. Anopheles mosquitoes are endophilic (i.e. tend to stay within human habitats) and rest especially on walls, mostly 2 under ft height from floor
   b. The residual spray is effective for 6 months
   c. It is the simplest adult mosquitoe control technology, and feasible (both economically and logistically) – In short, it is an **appropriate technology** (one of the principles of primary health care).
2. Antilarval—*Source reduction* (drainage, filling of stagnant water, flushing, change of salinity of water); *larvicides* (paris green, temephos)–Must be applied at frequent intervals; *biological control* (Gambusia fish); importantly, *mineral oils* are not effective against anopheles.

Protecting the host

1. Mass distribution of mosquito nets (pretreated with insecticide, diameter of holes < 0.0475 inch), screens, repellants (diethyltoluamide); education on protecting oneself from mosquitoes.
2. Chemoprophylaxis—Important for travelers and pregnant women, little role in endemic countries (people are semi-immune anyway).

A history to learn from – the control of malaria in India

National malaria control program, 1953

So long a Cinderella, the **National Malaria Control Program** was launched in 1953 which had basically two strategies,

1. Active and passive case detection and treatment
2. Indoor residual DDT spray (1g/m_2) twice a year where spleen rate is >10%.

Encouraged by its success, a hasty decision changed it to National Malaria *Eradication* Program (bypassing the elimination step), which shared some early success till 1961, with these strategies,

1. 2 rounds DDT spray in all areas
2. Active and passive surveillance
3. Presumptive and radical treatment.

The first setback occurred in 1970, and there came to be the resurgence of malaria due to the following foreseeable reasons,

1. Operative
   - Hasty implementation of eradication program.
   - Poor surveillance.
   - Incomplete spray work.
   - Premature take off into consolidation and maintenance phase, without first eliminating malaria.
   - Undue reliance on untrained basic health workers.
   - Lack of community involvement in spray work.
2. Administrative
   - Poor supply of drugs and insecticides
   - Shortage of staff
   - Low budget allowance
   - Lack of national commitment.
3. Technical
   - Drug and insecticide resistance
   - Rapid industrialization, migration from neighboring countries.

Urban malaria scheme, 1971

During its resurgence, malaria began to invade in cities due to aggregation of labor, unplanned development, inadequate drainage and ongoing construction work. *Anopheles stephensi* was the predominant vector in urban regions. Strategies for control were

1. Antilarval spray (malathion, fenthion, abate, mineral oil) in any water collection.
2. Antiadult—Space spray with pyrethrum in peripheral belt of town (0.5–1 mile).
3. Filling and underground drainage.
4. Biological control of larva.
5. Mass education.
6. Drug treatment.

Modified plan of operation (MPO), 1977

In view of the above mentioned failure a new scheme was launched which was supposed to be *flexible* with changing epidemiological situations, stressed upon drug treatment and decentralized laboratory services.

1. Stratification of rural areas (based on API) and differential vector control measures
2. Active (house to house search by MPHWs) and passive (cases that visits hospitals and private practitioners) surveillance
3. Presumptive and radical treatment.

Table 9.19. Differential interventions in areas

Areas with API > 2	2 round DDT/ 3 round malathion/ 2 rounds synthetic pyrethroid spray Surveillance and treatment Entomological assessment
API < 2	*Focal* spray around houses with falciparum infection Surveillance and treatment Epidemiological assessment Follow up blood smears monthly upto 12 months

To ensure accessibility of services,

1. **Drug distribution centers** were set up, which gave drugs to key persons (AWC, teachers)
2. **Fever treatment centers** were set up, which took blood slides and provided presumptive treatment only

In addition, research for chloroquine resistant falciparum malaria and IEC on malaria is an important part of **MPO**. The current scheme of **surveillance** of malaria was introduced in MPO. Surveillance must cover entire population all through the year. In its basics, it consists of

1. **active** case detection by home visits every fortnight (the time to develop secondary cases) by MPHW (male); 1 MPHW caters a population of 10000; he visits each house, asks for a fever case which may have developed since his last visit, collects thick and thin smears and gives presumptive treatment. He sends the slides to PHC for examination and if +ve, returns to the house for radical treatment. He is bossed by the health assistant (male) from PHC.
2. **passive** case detection—Suspected malaria cases presenting to local health institutions, individuals, to be managed in the same manner.
3. mass blood exam (during outbreaks).
4. epidemiologic investigations to find out extent and trend of population and evaluate antimalaria program (API, ABER, SFR, SPR).

The MPO controlled deaths from malaria but the resurgence of malaria continued.

Malaria action program (MAP), 1995

The MAP stratified the country in **high risk areas** (i.e. areas with priority spray operating and dense treatment) and **low risk areas**.

Rural high risk area	Urban high risk area
SPR doubled in last 3 years (SPR at least 4%) Reported falciparum death Average SPR in last 3 years > 5% Focus of falciparum found Chloroquine resistant falciparum malaria present Falciparum proportion > 30% with SPR > 3% Aggregation of labor/new settlements	Population > 50000 and SPR > 5% Clinical malaria: fever = 1:3 SPR > 10% in last 3 years

Antimalaria program, 1999

It is a combination of MPO and MAP. It incorporates the classification of MAP (i.e. the concept of high risk and low risk areas), but retains the strategies of MPO. In 2003–04, it was merged with National Vector Borne Disease Control Program—Along with introduction of Integrated Vector Control in rural areas.

1. Indoor residual spray in selected high risk area (DDT, malathion, pyrethroids).
2. Distribution of insecticide treated nets – free/subsidized.
3. Larvivorous fish.
4. Environmental/minor engineering methods.

It also modified the strategies for vector control in *urban* areas,

1. Antilarval—No indoor spray, emphasis on drainage of water and insecticide spray in drain only.
2. Personal protection.

Roll back malaria program, WHO, 1998 (Fig. 9.30)

Figure 9.30. Roll back malaria

The Roll Back Malaria Partnership (RBM) is the global framework for coordinated action against malaria. The RBM Partnership was launched in 1998 by WHO, UNICEF, UNDP and the World Bank. In 2006, the RBM Partnership was redesigned in a process known as the 'Change Initiative'. Roll Back Malaria will seek to:

- strengthen health systems to ensure better delivery of health care, especially at district and community levels.
- ensure the proper and expanded use of insecticide-treated mosquito nets.
- ensure adequate access to basic healthcare and training of healthcare workers.

- encourage the development of simpler and more effective means of administering medicines; such as training of village health workers, mothers and drug peddlers on early and appropriate treatment of malaria, especially for children.
- encourage the development of more effective and new antimalaria drugs and vaccines.
- alleviate poverty, which is the breeding ground of malaria.

Dengue

Dengue, an usually nonsevere albeit debilitating viral fever ("breakbone fever"), is the most prevalent mosquito-borne viral disease in people. Dengue is endemic in most tropical and subtropical countries, many of which are heavily populated as well as a popular destination for tourists.

Problem

Some 2.5 billion people are estimated to currently be at risk of dengue in over 100 countries across the globe. It is estimated that between 50–100 million cases of dengue fever, 500 000 cases of dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS) and more than 20000 deaths from DHF/DSS occur each year. Dengue has become one of the most important emerging disease problems among international *travelers* and the *second most frequent cause of hospitalization after malaria* among travelers returning from the tropics. The past 25 years have seen the emergence and reemergence of epidemic dengue, with more frequent and larger epidemics associated with more severe diseases , probably related to population growth, massive unplanned urbanization, modern transportations and the lack of effective mosquito control.[324]

Dengue is spreading in India

1. Certain states have long history of dengue and regularly reporting cases with cyclical peaks.
2. New states are being affected (West Bengal).
3. Some states have been reporting dengue only for last 5–6 years (Bihar, Andhra, Rajasthan, Gujrat).
4. Dengue is endemic in almost half the country; there was an epidemic of dengue in 2005 in West Bengal.

Agent

Dengue virus (ssRNA); four serotypes DEN 1,2,3,4, which actually are four viruses almost as genetically different from each other as JE, West Nile and SLE viruses are from one another. Infection with *two* serotypes one after another increases chances of hemorrhagic fever (especially infection with DEN2).

Host

Manifestations are mild in children than adults. **Lifelong immunity** is provided after infection, but only serotype specific.

Environment

↑ popuation density, inadequate sanitation and drainage system, postmonsoon.

Chain of transmission

Reservoir/ source

Human cases, mosquitoes; the dengue viruses are the only known arboviruses that have fully adapted to humans and do not need an animal reservoir. Communicable period is from the day before onset of fever ↔ 5 days after.

Mode

Vector

- *Aedes aegypti* mosquitoes which are bery much adapted to humans, likes being around people (i.e. ↑ anthropophilicity), usually domestic, urban, rests indoor behind racks, under sofa and such other places; it lays its eggs in artificial water containers like flower vases and old tyres and water tanks in bathrooms. It is more closely associated with human habitation than any other species of mosquitoe and uses indoor breeding sites.
- *Aedes albopictus* and *Aedes polynensis* which are *peri* domestic, rural, rests outdoors feed on birds, reptiles, cows and humans; whereas *Aedes aegypti* is confined within the tropics and sub-tropics, *Aedes albopictus* also occurs in **temperate and even cold temperate regions**. In recent decades *Aedes albopictus* has **spread from Asia to become established in areas of Africa, Europe and the Americas**.The species *Aedes albopictus* thrives in a *wider range of water-filled breeding sites* than *Aedes aegypti*, including coconut husks, cocoa pods, bamboo stumps, tree holes and rock pools, in addition to artificial containers such as vehicle tires and saucers beneath plant pots. This diversity of habitats explains the abundance of *Ae. albopictus* in rural as well as periurban areas and shady city parks.

All these mosquitoes breed in artificial water collections, survive in prolonged dryness, bite at daytime, often several persons in sequence, and prefer human blood (all the factors which make them good vectors); *vector density* fluctuates with rainfall and improvement of water drainage systems; **extrinsic incubation period** = 8–10 days.

An infected mosquito can transmit the virus *directly* to her children (trans-ovarian transmission). *Eggs* can survive as long as a year without water.

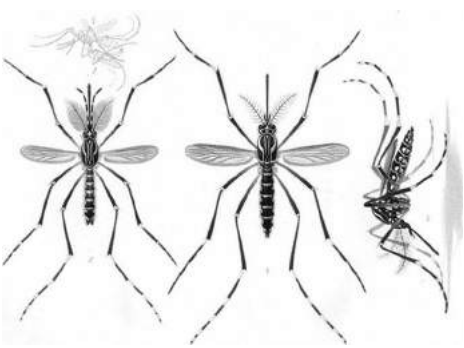

Figure 9.31. Aedes aegypti—Male (left), female (right); note the stripes[325]

Clinical course

Undifferentiated fever

Fever and rash like any other viral exanthem.

Dengue fever

Acute fever of 2–7 days with two or more of the following: headache, retrobulbar pain, myalgia, arthralgia, bleeding, rash, leukopenia. The rash typically appears during remission of fever or the 2nd febrile phase. The illness lasts for 5–7 days after which usually complete recovery occurs.

Dengue hemorrhagic fever

Dengue fever +

1. Hemorrage—Tourniquet test +ve (see 'Bleeding disorders' in a Textbook of medicine), petechiae, ecchymoses, purpura, bleeding from GIT or injection site, hematemesis, melena
2. Thrombocytopenia
3. Evidence of plasma leakage—Rise in hematocrit (for age and sex) > 20%; drop in hematocrit < 20% on rehydration, pleural effusion, ascites.

Table 9.20. Grading of DHF

I	Fever, constitutional symptoms, + ve tourniquet test
II	Spontaneous bleeding
III	Rapid weak pulse/ narrow pulse, hypotension, cold clammy skin, restlessness
IV	Undetectable pulse and BP

Dengue shock syndrome

DHF + SHOCK (rapid and weak pulse narrowing of pulse pressure ≤ 20 mm Hg)

Labs

1. Virus isolation from serum, plasma, autopsy, infected mosquito.
2. Viral antigen/ RNA in serum/autopsy.
3. Serology (IgM antibody is found after 5 days of exposure; *fourfold rise*/of IgG in paired samples taken at 10–14 day intervals is diagnostic of dengue).

Danger signs in dengue

- Minute skin bleeding
- Nose/gum bleeding
- Abdominal pain and melena
- Refusal of food and drink
- Abnormal behavior/drowsiness
- Dyspnea
- Oliguria
- Cold extremities.

Surveillance

***Surveillance* is essential for**

1. Monitoring dengue
2. Forecasting an epidemic
3. Timely control of epidemic.

Epidemiologic surveillance

1. Early detection of cases with standard case definition
2. Routine, sentinel and outbreak surveillance
3. Early warning signals detection
4. Effective and efficient response.

Entomologic surveillance

In endemic areas, it is essential to keep an eye on *Aedes* mosquitoes so that a dengue epidemic could be stopped.

1. **House density** = percentage of houses where Aedes larva is found.
2. **Container index** = percentage of stray containers (vases, pitchers, old tyres) where Aedes larva is found.
3. **Breteau index** = number of stray containers containing Aedes larva/total houses searched × 100.

Lab surveillance

Although time consuming and expensive, an ongoing serological surveillance of at risk population give early signal of asyptomatic/atypical dengue infections.

Treatment

Dengue fever

Bed rest, paracetamol (aspirin is contraindicated due to bleeding tendencies in dengue), mild sedatives, sponging, oral fluid; monitor till the patient is afebrile, platelet count and hematocrit are normal.

Dengue hemorrhagic fever (DHF)

Paracetamol, oral fluids/IV fluids over 2–3 hours; serial hematocrit levels and frequent assessment of urine output.

Criteria for hospitalization

- Rise in hematocrit > 20%
- Platelets < 50000
- Spontaneous hemorrhage
- Shock, oliguria
- Circumoral cyanosis.

Dengue shock syndrome

Immediate IV fluids (5% dextrose with either Ringer's lactate or normal saline) to expand plasma volume; check electrolytes and blood gases periodically; moist oxygen; blood/ FFP/ platlet transfusion as required.

Criteria for discharge in DHF/DSS

- Afebrile for 24 hours without antipyretics
- Improvement in clinical picture
- Stable hematocrit
- Adequate urine output
- Platelets > 50000
- No dyspnea.

Control

Controlling the reservoir

Early detection and standard case management.

Breaking the chain

Vector control, i.e.

- **Personal prophylaxis**—Mosquitoe repellants, wearing full sleeve shirts, pants and socks in daytime, use of insecticide treated bed nets even in daytime.
- **Biological**—Bacillus thuringensis (see the section on entomology).
- **Chemical**—Abate (larvicide) aersol spray at daytime.
- **Source reduction**—Detection and elimination of breeding places (roof tops, porticos, sunshades, tyres, coconut shells), proper covering of stored water, discourage to store water (reliable water supply), weekly dry day (empty all stored water once a week).
- **Community participation**—Sensitizing the community to find breeding places and eliminate them.

Protecting the host

Bad news. No vaccines yet.

Lymphatic filariasis

Lymphatic Filariasis, known as Elephantiasis, puts at risk more than a billion people in more than 80 countries. Over 120 million have already been affected by it, over 40 million of them are seriously incapacitated and disfigured by the disease. Imported from Africa with the slave trade, filariasis was long called "Barbados leg". It can disappear spontaneously — as it did in Barbados — when countries prosper and the poor are able to afford window screens and governments cover the sewers.

One-third of the people infected with the disease live in *India*,[326] and another third in Africa. In India LF is endemic UP, Bihar, Jharkhand, Andhra Pradesh, Orissa, Tamilnadu, Kerala, Gujrat. The north eastern states, J and K, Punjab and Rajasthan are *free* of the disease (Fig. 9.32).

Agent

Wuchereria bancrofti, *Brugia malayi*, *Brugia timori* (absent in India). The adult worms reside in lympahtics and release larva (microfilariae) in blood periodically,

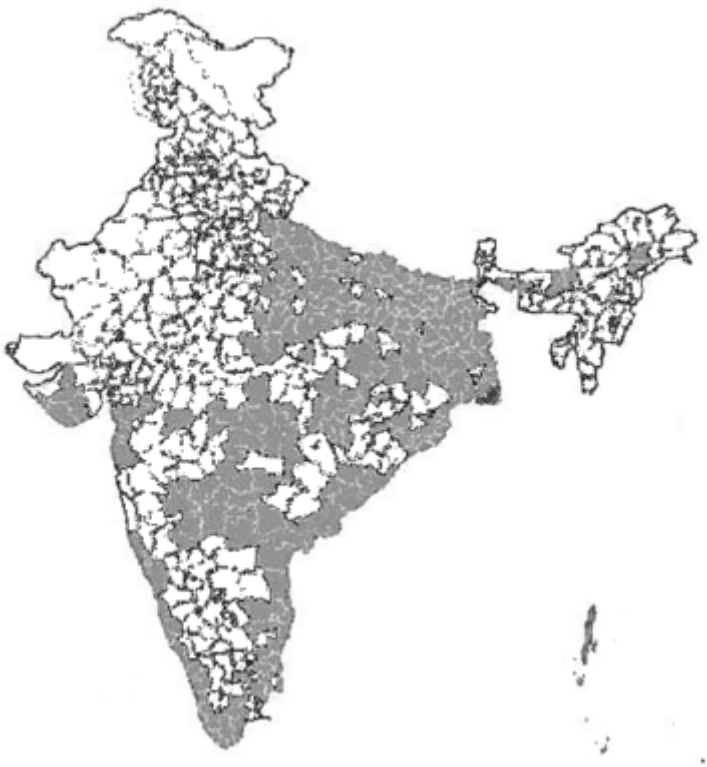

Figure 9.32. Filaria endemic districts in India [NVBDCP: Filaria]

at some particular time of day;[327] at other times the microfilaria take residence in pulmonary capillaries.

1. **Nocturnally periodic**—It is found in mainland of India, maximum density of microfilaremia occurs between 11pm–2am at night.
2. **Diurnally sub periodic**—Increase in microfilaremia in the afternoon, found in Nicobar.
3. **Nocturnally subperiodic**—Microfilaria are found in blood all through the day, but more in night.[328]

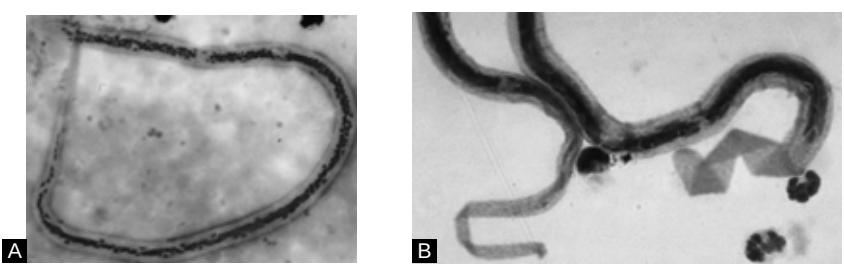

Figure 9.33. The two parasites: Microfilaria of *Wuchereria bancrofti* (left) and *Brugia malayi* (right). Note that Wuchereria has a graceful curve, uniformly tapered tail, discrete nuclei, and no nuclei of the end of its tail; now compare it to Brugia [CDC Public Health Image Library]

Host

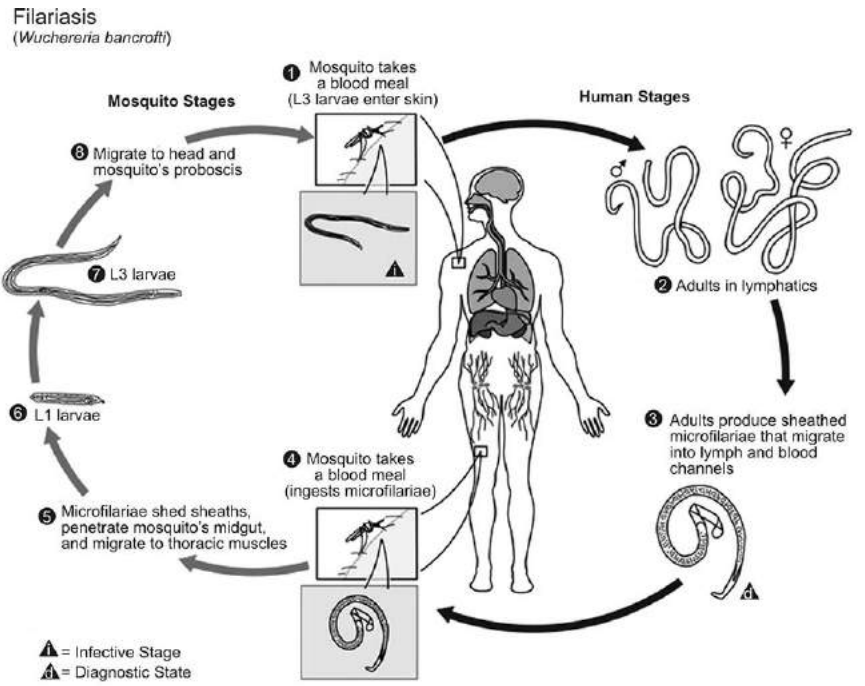

Figure 9.34. Life cycle of filaria parasite[329]

1. **Definitive host**—Man; adults are frequently and more heavily infected than children; also, microfilaria rate is greater in men than women; many subclinical cases persist
2. **Intermediate host**—Mosquito; the mosquito acquire microfilaria from human blood who develop into L1 → L2 → **L3 larva** (infective form) with no multiplication in number (only *cyclic* development, not *cyclopropagative*). **Extrinsic incubation period** = **10–14 days** (time to develop L3 larva).

Vector

Bancroftian filariasis (nocturnal)	Culex quinquefasciatus breeds in polluted water. Common breeding sites are wet pit latrines, septic tanks, cesspools, drains, disused wells, paddy fields. They are anthropophilic and night biters.
Malayan filariasis	*Mansonoides annulifera* *Mansonoides uniformis* *Mansonoides Indiana* All breed in water bodies with aquatic plants
Bancroftian filariasis (diurnally subperiodic)	*Ochlerotatus niveus*

Environment

Unplanned urbanization, industrialization, migration of rural population into cities, 22–38°C and a relative humidity of about 70% with bad water drainage facilities favors the growth of mosquitoes (and the disease).

Chain of transmission (Fig. 9.35)

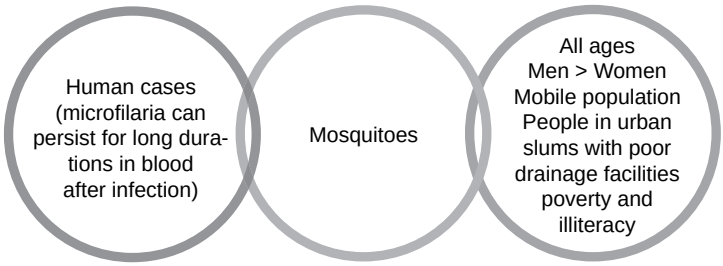

Figure 9.35. Chain of transmission of filariasis

Clinical course

Prepatent period = bite to first appearance of microfilaria in blood = 12–18 months. Clinical incubation period = 8–16 months.

Asymptomatic infection

Though the infection is generally acquired early in childhood, the disease may take years to manifest itself. Indeed, many infections are *asymptomatic*. However, such subjects may have hidden lymphatic pathology and kidney damage as well. Microfilaria can or cannot be present in blood.

Carriers

Long term microfilaremia (the adults produce microfilaria for 5–6 years before they die out).

Acute filariasis

Acute episodes of local inflammation involving skin, lymph nodes and lymphatic vessels often accompany. Some of these are caused by the body's immune response, but most are the result of bacterial infection of skin where normal defences have been partially lost due to underlying lymphatic damage (**entry lesions** like injury, candidiasis, pyoderma, paronychia, fissure foot, eczema, insect bites).

Figure 9.36. Elephantiasis [US Center for Disease Control—Public Health Image Library]

Chronic filariasis (Fig. 9.36)

The worst symptoms of the chronic disease generally appear in adults, and in men more often than in women. In endemic

communities, some **10–50% of men** suffer from hydrocele and elephantiasis of the penis and scrotum. Elephantiasis of the entire leg, the entire arm, the vulva, or the breast swelling upto several times normal size—It can affect upto 10% of men and women in these communities. It is important to note that Malayan filariasis does not involve genitals.

Although severely disfigured, patients of chronic filariasis no longer bear microfilaria in blood and do not transmit the disease.

Lymphatic filariasis is a social evil

The instantly recognizable symptom of lymphatic filariasis is elephantiasis of legs. But 10 times as common is the symptom that is almost never spoken of the hydrocele. In endemic communities, more than a quarter of the men are tormented by the condition. The task of eliminating LF is made more daunting by the stigma and secret shame that the affliction causes, particularly in men. It never kills but sears the spirit of the affected. Often, the patient cannot work, leading to economic losses and starvation. Sometimes, they cannot bear it anymore and try to operate it themselves or by quacks (they are too shameful to show a hydrocele to a doctor), leading to infection, bleeding and possibly death.

For those with swollen legs, all they can do is to wash their legs and feet daily to forestall infections as the skin breaks down. The limbs cannot be surgically drained because the damage is permanant. There is no localized fluid collection to drain. Every nook and corner of soft tissue is swollen up.

The combined effect can be devastating

For people with elephantiasis, big pants will not hide their affliction. Funguses that erupt between toes stink and draw flies. Children can be mocking. Lovers can be cruel.

—Donald G Mcneil Jr, The New York Times, April 9, 2006

Why doesn't filaria cause epidemics?

- The larva do not multiply in the mosquitoe
- The larva do not multiply in the human host
- The life cycle of the parasite is 15 years or more
- A single bite seldom transmits filaria (unlike malaria).

Labs

Detection of microfilaria in blood

1. Blood drawn between 8:30 pm–12 midnight and made into a thick film (containing 20 mm^3 of blood) → examined by Romanowski stains; Wuchereria has no terminal nuclei, while Brugia has two and is also unsheathed.
2. Membrane filtration culture method—To concentrate the microfilaria in a single film.
3. Diethylcarbamazine provocation test—To bring out the microfilaria in blood during daytime.

Antigen detection

Immunochromatography to detetct parasite antigens in blood is rapid, field friendly, sensitive, specific and can be done during day or night.

Treatment

Treatment of acute filariasis (acute dermatolymphangioadenitis)

Uncomplicated episodes can be treated by a peripheral health worker

1. Analgesic (paracetamol).
2. Oral antimicrobials (i.e. amoxycillin 500 mg × 3 × 8 days).
3. Antiseptic cleaning of limbs; check for entry lesions, apply antimicrobial and antifungal ointment if necessary.
4. No antifilarial drugs.
5. Plenty of water, rest, elevation of limb, active movements, cooling the limb.
6. Advice on prevention of chronic filariasis.
7. If this does not improve in 2 days, refer patient to PHC.

Complicated episodes must be treated in a PHC with benzylpenicillin 5 million IU IV × 3 until fever subsides, and then switch to oral phenoxymethylpenicillin 750 mg - 1 g × 3 times daily for a total course of 8 days. In case of penicillin allergy use erythromycin.

Chronic filariasis

Chronic filariasis is incurable, the parasite cannot be removed from the body, and the tissue changes are irreversible. *Hydrocele* could be corrected operatively, but not elephantiasis. The more important part is limb hygiene and prevention of entry lesions.

Indices of filaria status of community

Parasitological indices

1. **Microfilaria rate** = number of persons showing presence of microfilaria in blood/ total number of people examined × 100; it is 0.63% for whole of India
2. **Average microfilaria density** = total number of microfilaria in all +ve smears/ total volume of blood (in mm^3) collected from all persons × 20 (20 mm^3 of blood is regarded as *unit* of blood collection)
3. **Disease rate** = prevalence of filariasis
4. **Filarial endemicity rate** = percentage of persons found with microfilaria in blood OR clinical disease or both
5. **Average infestation rate** = total number of microfilaria in all +ve slides / total number of +ve slides × 100.

Entomological indices

1. **10 man hour density** = number of male and female *Culex quinquefasciatus* collected by one person/hours spent in collection × 10
2. **Vector infection rate** = percentage of female mosquitoes carrying L1/L2/L3 larva
3. **Vector infectivity rate** = percentage of female vectors carrying infective (L3) larva.

Control

Controlling the reservoir

The primary goal of treating the affected community is to eliminate microfilariae from the blood of infected individuals so that transmission of the infection by the

mosquito can be interrupted.The WHO mentions that "the use of **single doses of 2 drugs administered concurrently (optimally albendazole with DEC or ivermectin) is 99% effective** in removing microfilariae from the blood for a full year after treatment".

Diethylcarbamazine

DEC is an oral drug that is both safe and effective in clearing microfilaria. It can cause, however, severe side effects in the occassional case (nausea, dizziness, focal inflammation, lymphedema, orchitis and hydrocele). Earlier, it used to be given in 12 day courses (DEC 6 mg/kg × 12 days), nowadays a single dose has proven to be effective. In highly endemic areas (such as Lakshwadeep), *salt* has been fortified with DEC.

Breaking the chain

Vector control

Antilarval measures. Reduction of mosquitoe breeding places, mineral oil, removal of pistia plants from water (where *Mansonia* mosquitoes breed), regular drainage of stagnant water (see the chapter on entomology)

Antiadults. Most *Culex* mosquitoes are now sensitive only to pyrethrum sprays.

Protecting the host

Using mosquito nets, window screens, repellants.

National Filaria Control Program, 1955

Objectives

1. Delimit the problem
2. Implement control programs
3. Training health care staff to handle filariasis.

Control measures

1. Mass drug administration (MDA) with DEC 4 mg/kg × 5 doses
2. Antilarval measures in urban areas
3. Indoor residual spray in rural areas.

In 1997, MDA with *single* annual dose of DEC/ DEC + albendazole in selected areas were taken up. In 2003, LF was incorporated in NVBDCP and a target was set up to eliminate LF from India by 2015. Nation wide MDA was launched in 2004.

Global elimination of LF (WHO), 1997

A reduction of incidence close to zero as a result of deliberate efforts requiring continuous and coordinated activities.

Strategies

Interruption of transmission. Community-wide mass treatment programs for entire at-risk population.

- Once-yearly administration of single doses of two drugs given together: Albendazole plus either diethylcarbamazine (DEC) or ivermectin, the latter in areas where either onchocerciasis or loiasis may also be endemic; this yearly, single-dose treatment must be carried out for 4–6 years.
- An alternative is the use of DEC fortified salt for 1 year.

Morbidity management. Community education programs to raise awareness in affected patients about local hygiene and the possible improvement.

Revised LF control under National Vector Bourne Diseases Control Program

Following to the WHO recommendations, the filaria control policy has been changed in India since 1997.

Single day mass drug administration with DEC 6 mg/kg

Interruption of transmission by single annual MDA (DEC) for 5 years or more to the entire at risk population except children < 2, pregnant and lactating women, seriously ill-patient.

2–5 years	100 mg
6–14 years	200 mg
> 15	300 mg

Combination MDA

1. Areas where onchocerciasis/loiasis are coendemic - albendazole + ivermectin
2. Where onchocerciasis/loiasis are nonendemic - albendazole + DEC

Advantage of mass drug administration

1. As effective as 12 day therapy for public health
2. Avoids cost of mass blood examination and false negatives
3. Nobody feels left out of the program because microfilaria was not found in his blood
4. Lesser side effects
5. Better compliance than a 12 day course
6. Less delivery cost
7. Does not require complex infrastructure
8. Can be integrated with existing health care systems.

Treatment of acute and chronic filariasis

See 'treatment' earlier. The NVBDCP emphasises home based care of elephantiasis and up-scaling of hydrocele operations in selected CHCs / District hospitals/ medical colleges.

Continuing existing antivector measures

See 'breaking the chain' earlier.

Education to inculcate individual/community based protective and preventive habits

1. Behavior change communication (do not forget mosquitoes nets, do not keep stored water open).
2. Human resource development through capacity building.
3. Public private partnership.
4. Research.
5. Monitoring and evaluation.

Yellow fever

Yellow fever is a disease of the equatorial region over two continents around the Atlantic – South America and Africa. It does not occur in Asia. The **agent** is Flavivirus fibricus (a group B arbovirus belonging to family Togaviridae). All ages and sex are susceptible, especially rural young adults and those who work in forests. One attack gives lifelong immunity. The virus relishes in temperature > 24°C, humidity > 60%; unplanned extension of cities brings unexposed people closer to jungles, where the virus resides.

Chain of transmission

Monkey, mosquitoes, human cases (clinical and subclinical) → *Haemagogus* and *Aedes africanus* mosquitoes (in jungle), *Aedes aegypti* mosquitoes (in towns and cities) → human host.

Control

Controlling the reservoir

Quarantine of international travelers (who have no vaccination certificate) for 6 days (under International Health Regulations).

Breaking the chain

1. *Aedes aegypti* index to be kept < 1% around 400 m of airports and seaports
2. Aerosols spraying on ships and aircrafts with Pyrethrum and DDT
3. General vector control measures.

Protecting the host

The **17D vaccine** is a live freeze dried vaccine (diluted in sterile, cold, physiological saline), grown in chick embryo; must be stored at subzero temperatures, It is given 0.5 ml SC over deltoid single dose, provides immunity from 6 days after vaccination upto 35 years.

Travelers who have been given the vaccine are given a vaccination certificate so that they can enter a country where there is no yellow fever. It is valid from 10 days of vaccination > 10 years.

Protecting India from yellow fever

When you think of it, the Indian population has never been exposed to the yellow fever virus, and the virus will spread like wildfire once it enters India. This is because Aedes mosquitoes are present here in plethoric amounts, and the environmental conditions (warm and moist weather, inadequate water drainage systems) are all suitable for the disease to spread. The only thing missing in the chain of transmission is the virus itself. Thus we must stay on guard to stop the virus at every portal to India. The Government has adopted three strategies for doing so

1. Check vaccination certificates for all travelers from an endemic country (including transit passengers and infants); without a certificate, they are quarantined for 6 days
2. Spraying all containers of incoming ships and aircrafts with pyrethrum and DDT to kill any infected mosquitoes that might have come along.

3. Keeping Aedes mosquitoes out of ship and airports (*Aedes aegypti* index < 1 around 400 meters) so that they cannot bite any passenger who has the virus.

Kyasanur forest disease

It is a viral infection localized in Shimoga, Kannada and Chikmagalur districts of Karnataka.

Agent

The KFD is a Flavivirus belonging to the family Togaviridae.

Host

1. Squirrels, rats, birds and bats
2. Monkeys (amplifying host—The virus multiplies in monkeys and rapidly kills them)
3. Man—Incidental, dead end host, from where the virus has no route of escape; usually those who have visited forests with their cattle are affected first.

Environment

It occurs within January – June, the period when pasture greens dry up and the cattle have to visit the forest to get food. They provide blood meal for ticks, the vectors of the disease.

Chain of transmission

Small mammals and monkeys → hard and soft ticks → human

Clinical course

Incubation period = 3–8 days; headache, fever, hemorrhage, meningo encephalitis; a *relapse* often occurs after 7–8 days of primary illness.

Labs

Virus isolation from blood, serology.

Control

Breaking the chain

Tick control. Spraying carbaryl/fenthion from aircraft on a 'hot spots' (where monkeys are seen to be dying suddenly) in forests and 50 km around.

Protecting the host

1. Killed vaccine for woodcutters, surveyors, forest guards
2. Personal protection (mosquito repellant lotions while working in forest).

Chikungunya fever

Chikungunya (in the makonde language "that which bends up") virus is an insect-borne virus, of the genus Alphavirus, that is transmitted to humans by virus-

carrying Aedes mosquitoes. The disease shares some clinical signs with dengue, and can be misdiagnosed in areas where dengue is common. Chikungunya has only recently (2006–2007) flared up in some spots in India, after 41 years of absence.

Chain of transmission

Human cases → *Aedes egypti* and *Aedes albopictus* > mosquitoes → human host

Clinical course

Abrupt onset of fever with joint pain, muscle pain, headache, nausea, fatigue and a rash. Most patients recover fully, but in some cases joint pain may persist for several months, or even years.[330] Occasional cases of eye (conjunctivitis and photophobia), neurological and heart complications have been reported. Subclinical infections are common, and the disease can go unrecognized, especially in regions where dengue is endemic.

Labs

- **Virus isolation** provides the most definitive diagnosis but takes 1–2 weeks for completion and must be carried out in biosafety level 3 laboratories.[331]
- Reverse transcriptase PCR is faster (1–2 days).
- Serology—ELISA to measure Chikungunya-specific IgM levels. Results require 2–3 days and false positives can occur with infection via other related viruses such as O'nyong'nyong virus and Semliki Forest Virus.

Control

Same as dengue.

Japanese encephalitis

Japanese 'B' encephalitis (JE) is a disease of pigs and birds, and only occassionally infects humans. It continues to be endemic in West Bengal, Andhra Pradesh, Tamil Nadu, Karnataka, North Eastern states and some parts of UP (Fig. 9.37).

JE is important from a community perspective because of

1. Epidemic potential
2. High case fatality
3. Lifelong sequelae of encephalitis.

Figure 9.37. JE endemic districts in India [NVBDCP: Japanese Encephalitis]

Agent

The JE virus is a flavivirus with a preference towards neural tissue.

Host

It is a zoonosis of herons and egrets. *Pigs* serve as amplifier hosts and do not show signs of the disease. *Cattle* bear the virus and are the major attractants for mosquitoes. *Man* is the accidental, dead end host.

Environment

It is a disease of rural areas where there are adequate breeding places for Culex mosquitoes (i.e. paddy fields); the disease flares up in monsoon and postmonsoon.

Chain of transmission (Fig. 9.38)

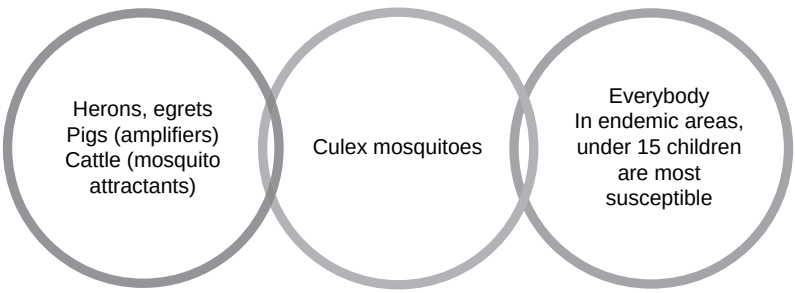

Figure 9.38. Chain of transmission of Japanese encephalitis

Vector

Culex tritaeniorhynchus (South India), *Culex vishnui* (West Bengal), *Culex pseudovishnui*. The culex is a rural mosquito which breeds in irrigated rice fields and shallow ditches. It prefers animals than human, and the night more than daytime. It rests outdoors, occasionally indoors in summer time. The average lifespan is 21 days and it is a strong flier (can fly 1–3 km at a stretch). **Extrinsic incubation period** = 9–12 days; once infected with JE, *remains* infected for its life.

Clinical course

The disease is characterized by an clinical: asymptomatic ratio of 1:250 to 1: 1000; however, both kinds of infection gives immunity. **Incubation period** is about 5–15 days.

Prodrome

Acute fever with chills, headache, malaise.

Acute encephalitis

High fever, neck rigidity, photophobia, nausea, vomiting, altered sensorium, seizures, variable neurologic signs.

Sequelae

Residual neurodeficit (paralysis, mental retardation), 25% case fatality.

Table 9.21. Diagnosis of Japanse Encephalitis[332]

Suspect case	Fever of variable severity + altered sensorium for > 6 hours + NO skin rash EXCLUDE—Other known encephalitis viruses
Probable case	Suspect case + presumptive laboratory results (detection of acute phase elevated and stable JE antibody by ELISA/ hemagglutination inhibition/viral neutralization, etc.)
Confirmed case	Detection of JE antigen/virus/genome in tissue/blood/other body fluids by immunofluorescence and PCR Or JE IgM in CSF Or Fourfold or greater rise of antibody in paired sera (acute and convalescent phase)

Treatment

No specific treatment; maintenance of fluid and electrolytes, lowering of intracranial pressure, antiseizure drugs, maintenance of airway.

Control

Factors that make JE difficult to control

- Outdoor habit of the vector (thus indoor residual sprays are of no use).
- Scattered distribution of cases spread over relatively large areas (because Culex mosquitoes can fly over long distances).
- Presence of different reservoir hosts.
- Specific vectors for different geographical and ecological areas.
- Immune status of various population groups is not known making it difficult to delineate vulnerable population groups.

Controlling the reservoir

Isolation of pigs and cattle away from general population and protecting them from mosquito bites.

Breaking the chain

Vector control

1. Source reduction—Intermittent irrigation and neem in rice fields.
2. Anti larval—Larvivorous fish and biocides.
3. Anti adult—Fogging with malathion and ultra low volume spray with fenthion during outbreaks; the affected village and villages within 2–3 km radius should be sprayed, along with local cattle shelters and pig styes.

Protecting the host

1. Behavior change communication—Use of insecticide treated nets and repellants especially during night, cover as much area of body possible with clothing.
2. **Vaccination** of 1–15 years children in endemic areas (and travelers to endemic areas.

Killed vaccine (from virus cultured in mouse brain tissue)	1 ml (0.5 ml under 3 years) SC × 2 at 7–14 days interval SC A booster within 1 month - 1 year of primary doses Another booster after 3 years
Live vaccine (SA-14-14-2 strain); freeze dried, diluted with distilled water	0.5 ml SC single dose, for children of 1–15 years

Vaccination is not recommended as an outbreak control measure as it takes at least one month after second dose to develop antibodies at protective levels and the outbreaks are usually short lived.

Others

Capacity building (through training workshops for health workers), research, monitoring of JE and evaluation of services (now conducted under NVBDCP).

Leishmaniasis

The members of the genus Leishmania cause a number of infections (visceral, cutaneous and mucocutaneous leishmaniasis) borne by the sandfly, P*hlebotomus*. Visceral leishmaniasis or kala-azar is a slow progressive disease. In India, *Leishmania donovani* is the only parasite causing this disease.

Problem

Kala-azar is endemic in eastern states of India namely Bihar, Jharkhand, Uttar Pradesh and West Bengal. It is a disease of ancient times, but was beginning to show decline in India around 1955–60 because of,

1. Successful drug treatment[333] and reduction of parasite load
2. Reduced vector population (collateral benefit of DDT spray as part of National Malaria Eradication Program)
3. Better immunity of the community

It started *reappearing* in 1971. The reasons for this surge was

1. Nonimmune population born after 1950
2. Nonfamiliarity of new age doctors with kala-azar
3. Inadequate supply of drugs
4. Incomplete treatment of cases.

Targets for 10th five year plan

1. Annual reduction of kala-azar deaths by 25% till 2004
2. Zero incidence of kala-azar by 2007 with at least 20% annual reduction from 2001 onwards
3. Elimination by 2010.

Agent (Fig. 9.39)

Visceral leishmaniasis	*Leishmania donovani*, intracellular parasite, life cycle consists of a mosquito phase (promastigote) and a human phase (amastigote)
Cutaneous leishmaniasis (oriental sore)	*Leishmania tropica*
Mucocutaneous leishmaniasis	*Leishmania braziliensis*

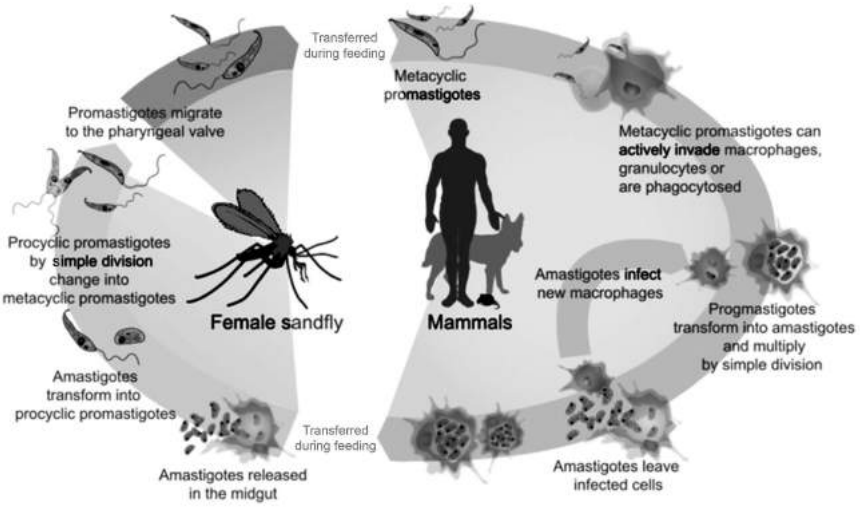

Figure 9.39. Life cycle of *Leishmania donovani* [334]

Host

All ages and sexes are susceptible, but it peaks in 5–9 years and males are twice as affected. People in mining, farming and forestry are easy victims. Population movement from endemic to nonendemic areas also spreads the disease. *Recovery* gives lasting immunity.

Environment

1. Altitude—Confined to planes (< 2000 ft from sea level)
2. Season—Two peaks: Postmonsoon (November) and spring (March and April)
3. Predominantly rural.

Developmental projects like forest cleaning, cultivation, setting up railway tracks, etc. expose the workers sandflies.

Chain of transmission (Fig. 9.40)

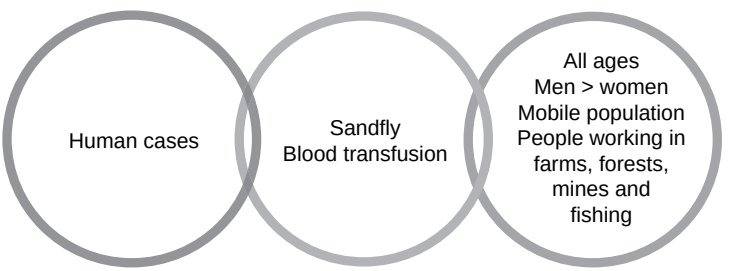

Figure 9.40. Chain of transmission of kala-azar

Vector

Female *Phlebotomus argentipes* (kala-azar), Phlebotomus papatasi and *Phlebotomus sergenti* (cutaneous leishmaniasis); **extrinsic incubation period** = 6–9 days.

Clinical course

Kala-azar

Incubation period = 10 days – 2 years (commonly 1–4 months)

1. **Fever**—Initially there is continuous, nocturnal fever which later becomes intermittent type. In 20% cases the fever shows a *double rise* in 24 hours.
2. **Anemia**.
3. **Soft nontender splenomegaly**—The spleen often engulfs the whole abdomen causing a pot belly.
4. Liver is only slightly enlarged and smooth in feel.
5. Clean moist tongue (unlike enteric fever).
6. The **skin** is dry, rough, and harsh and is often pigmented (thus the name 'kala-azar' which means 'black fever'); *hair* tends to be brittle and falls out.

Because the monocytes and macrophages are primary targets of the parasite—The patient will be highly susceptible to secondary bacterial infections. Death occurs if left untreated—Mostly due to other infections (pneumococcus, tuberculosis, entameba) or GI bleeding.

Cutaneous leishmaniasis

It bears three forms—Anthrophilic (ACL), Zophilic (ZCL) and Diffuse (DCL). It causes painful ulcers at the site of sandfly bite.

Mucocutaneous leishmaniasis

Ulcer similar to CL, around margins of mouth and nose. The disease causes mutilation of face, causing the patient to be socially ostracized and unacceptable by public.

Postkala-azar dermal leishmanoids

This is a type of nonulcerative cutaneous lesion prevalent in endemic areas of India, develops in about 10% kala-azar patients generally 1–2 years after treatment of original disease (however, it can present without a history of kala-azar too).

The manifestations of PKDL may be of three types.

1. Depigmented macules—Earlier lesions occurring in extremities
2. Erythematous patches—Appears on nose, cheek, skin (‘butterfly’ rashes); lesions are very photo sensitive.
3. Pinkish yellow nodules—Replace the earlier lesions – found all over skin, tongue, over the eyes, extensor surfaces; they are nonulcerated and painless, but disfigure the face of the patient.

These patients are consistent human reservoirs.

Labs

Hematology

Peripheral blood shows *neutropenia*, *relative* lymphocytosis and monocytosis, severe anemia, thrombocytopenia, hypergammaglobulinemia (↑ γ globulins).

Aldehyde (Napier) test

It is positive only after 3 months of kala-azar. 40% formalin (1–2 ml) + 1–2 drop serum → formation of milky white opacity. The test is *very* nonspecific and found

to be positive in schistosomiasis, trypanosomiasis, liver cirrhosis and multiple myeloma. But the simplicity of this test, and that it can be performed by the lowest level of health workers, has kept it alive till now.

Visualization

Demonstration of the organism in aspirate. The sample to be taken is bone marrow /blood/ spleen aspirate. *Spleen* samples are 90% sensitive but spleen aspiration will cause hemorrhage which can sometimes turn uncontrollable. Leishmania parasites are present in small numbers in *blood*, especially within WBCs. On the very first occasion you draw the smear, make a *straight* leukocytic edge, not tongue shaped. This zone will show lot of organisms.

Serology

ELISA, indirect hemagglutination (IHA), indirect immunofluorescence (IIF), direct agglutination and rk39 dipstick tests. The ELISA method is the simplest and has most potential for field surveys.

Leishmanin (Montenegro) test

Washed promastigotes (10^6/ml) in 0.5% phenol saline or merthiolate are injected intradermally (0.1 ml) on flexor surface of forearm → after 48–72 hours, the induration is marked. The test is +ve if its induration > 5mm. It is not a diagnostic test for kala-azar but shows immunity to the parasite only. The test is negative during attack of kala-azar, becomes positive within a 1 year of recovery.

Treatment

Drug policy under kala-azar elimination program as per recommendations of expert committee (2000).

First line drugs

Short term

- Areas with sodium stibogluconate (SSG) sensitivity > 90%—SSG IM or IV 20 mg/kg × 30 days; SSG is contraindicated in severe liver, kidney or heart disease.
- Areas with SSG sensitivity < 90% - Amphotericin B 1mg/kg IV infusion daily or alternate day for 15–20 infusions. Dose can be increased in patients with incomplete response with 30 injections.

Long Term

- Areas with high level of SSG resistance (> 20%)—Miltefosine 100 mg daily × 4 weeks.
- Areas with SSG sensitivity > 80%—SSG IM or IV 20 mg/kg/day × 30 days OR Miltefosine 100 mg daily × 4 weeks.

Second line drugs

Because of emerging SSG resistance, we have to resort to second line drugs when

1. No response after 20 days with SSG
2. Partial response after 30 days
3. Partial response after 2 courses of treatment in a fresh case.

For failure with SSG only. Amphotericin B 1 mg/kg IV infusion daily or alternate day for 15–20 infusions. Dose can be increased in patients with incomplete response with 30 injections.
For failure with both SSG and Miltefosine. Liposomal Amphotericin B

Treatment of PKDL

SSG in usual dosages for kala-azar could be given upto 120 days. Repeated (3–4) courses of Amphotericin B can be given in patients failing SSG treatment.

Control

The Kala-azar Control Program was launched in Bihar and West Bengal in 1990–91, *intensified* in 1992. It brought the case rate down 75% by 2002. It was then merged with NVBDCP.

Strategies

1. Vector control—Residual insecticide spray in endemic areas twice annually
2. Early diagnosis and prompt treatment of cases
3. Monitoring, supervision and evaluation of services
4. Education on kala-azar and community participation
5. Capacity building.

Spraying

Twice yearly spraying of DDT indoor residual spray in endemic areas (once in May-July and again in Aug-Oct); to spray at least 1g/m^2 of DDT on all indoor surfaces which are *6 ft high from ground level* (sandfly rests only above 6 ft of gronud).

Elimination of sandfly breeding places

Fill up cracks in walls, rodent burrows, removal of wood, bricks and rubbish that lie around the house, place cattle sheds and poultry away from the house.

Early diagnosis

In endemic areas, the combination of fever > 3 weeks, palpable spleen, aldehyde test +ve and malarial parasites –ve is presumed to be kala-azar. If the aldehyde test is –ve, the blood sample is sent to secondary level care (district hospitals) for demonstration of parasites. The rk39 rapid test kit is now being promoted for confirmatory diagnosis at the primary level.

Treatment

See earlier

National Vector Borne Disease Control Program

Vision

Well informed, self sustained healthy India free of vector borne diseases with equitable access to health care.

Mission

Integrated and accelerated action towards

1. Reducing mortality of malaria, JE, dengue by half by 2010
2. Elimination of kala-azar by 2010
3. Elimination of lymphatic filariasis by 2015.

Strategies

Parasite elimination and disease management

1. Early diagnosis and prompt treatment
2. Strengthening of referral
3. Epidemic prepredness and rapid response.

Integrated vector control

(See the chapter on vectors).

Supportive interventions

1. Behavior change communication
2. Public private partnership
3. Human resource development
4. Monitoring and evaluation through periodic reviews and field visits and web based information system.

All the individual programs against malaria, kala-azar, filariasis, dengue, Japanese encephalitis and Chikungunya are now integrated into NVBDCP.

Plague

Plague has swept the planet in three pandemics in the middle ages, of which the most devastating second pandemic or the 'black death' (1347–1351), reduced the population of the world by one-thirds. So much was its repercussion that The Black Death contributed to the destruction of the feudal system in medieval time. In its aftermath, the black death may also have favored the use of more advanced farming tools as a smaller workforce was available (most laborers had died) and plots grew larger as a result of the population loss. The reputation of plague is so well entrenched into popular psyche that 'plague' has become a *verb* in the english dictionary (Fig. 9.41).

A revolution is interesting insofar as it avoids like the plague the plague it promised to heal.

—Daniel Berrigan

Figure 9.41. Der Doktor Schnabel von Rom" (English: "Doctor Beak of Rome") engraving by Paul Fürst (after J Columbina). The beak is a primitive gas mask worn by physicians, stuffed with substances (such as spices and herbs) thought to ward off the plague[335]

Problem

One of the oldest identifiable diseases known to man, plague remains endemic in many natural foci around the world. It is widely distributed in the tropics and subtropics and in warmer areas of temperate countries. Untreated, mortality particularly from pneumonic plague may reach 30–60%. When diagnosed and promptly treated, plague may be successfully managed with antibiotics such as streptomycin and tetracycline, reducing mortality from 60% to less than 15%. However, the recent appearance in Madagascar of a strain of *Yersinia pestis* showing *multiresistance* to antibiotics is a matter of much concern.[336]

India

Plague was a major public health problem until 1940s until widespread application of DDT (for mosquitoes) killed, as a parallel benefit, the rat fleas too. It *reappeared* in Beed, Maharashtra, September 1994, followed by a pneumonic plague epidemic in **Surat** that resulted in 53 deaths and in a large internal migration of about 300,000 residents, who fled fearing quarantine.[337] A combination of heavy monsoon rain and clogged sewers led to massive flooding which resulted in unhygienic conditions and a number of uncleared animal carcasses. It is believed that this situation precipitated the epidemic[338] More recently, there has been outbreak in Himachal Pradesh in 2002.

Agent

Yersinia pestis is a gram –ve bacillus which stains bipolarly in special stains (Giemsa, Wayson, methylene blue). It can survive and multiply inside burrows of rodents.

Host

All ages and both sexes are susceptible. Human activity like hunting, cultivation, construction and outdoor recreation, war spreads the disease.

Vectors

Contrary to popular belief, rats did not directly start the spread of the bubonic plague. It is mainly a disease in the fleas (*Xenopsylla cheopis*) that infested the rats, making the rats themselves the first victims of the plague.

Entomologic indices

1. **Total flea index** = number of all fleas/number of all rodents in a locality
2. **Specific flea index** = flea/rodent according to each species of fleas; if *Xenopsylla cheopis* index > 1, it indicates potential explosiveness of plague. In an epidemic, within 48 hours of spraying insecticides, *Xenopsylla cheopis* index should fall to zero.
3. **Burrow index** = free living flea of each species/number of rodent burrows.

Environment

1. **Season**—September–May is 'plague' season in North India; after this period, wild rats aestivate in burrows. In South India, it occurs all through the year.

2. Temeprature—20–25°C, 60% humidity.
3. Heavy rain floods the burrows and kills the rats, which keeps plague away from these areas; however, in urban areas, heavy rainfall can cause clogging of sewers and insanitary conditions, leading to increase rat activity, a factor which has been ascribed in the Surat breakout.
4. Poor housing conditions, abundance of rats and fleas in close proximity with humans.

Chain of transmission (Fig. 9.42)

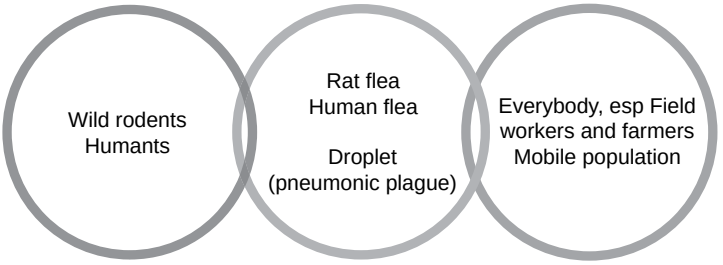

Figure 9.42. Chain of transmission of plague

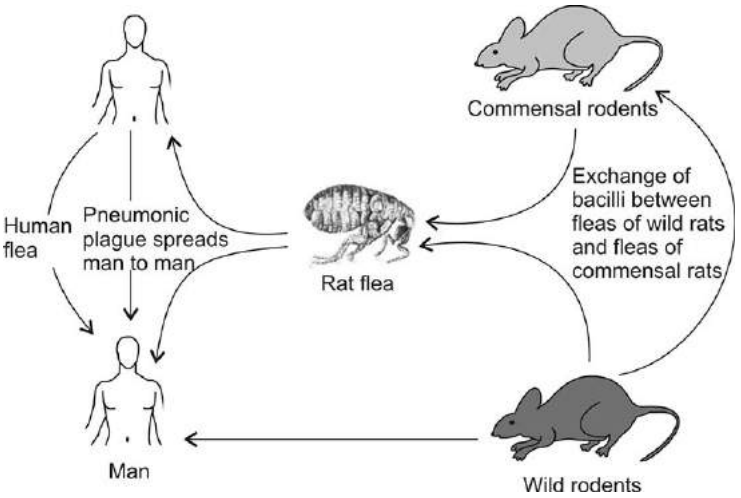

Figure 9.43. Cycles of plague observed in nature

Reservoir (Fig. 9.43)

Wild rodents (*Tatera indica* in India); the disease is maintained by rodents who have become immune to plague and effectively become carriers of the bacilli.

Source

Domestic rats (*Rattus rattus*) and peridomestic rats (*Rattus norvegicus*), in total the commensal rats, who have acquired the infection, human cases of *pneumonic* (bubonic plague cofines the bacilli to the lymph node and thus does not spread from man to man).

Mode

Vector borne

The plague bacteria multiply inside the flea, sticking together to form a plug that blocks its stomach and causes it to starve. The flea then bites a host and continues to feed, even though it cannot quell its hunger, and consequently the flea vomits blood tainted with the bacteria back into the bite wound. The bubonic plague bacterium then infects a new victim, and the flea eventually dies from starvation. A **partially blocked** flea can at least have some food and lives on, making it even more dangerous than a completely blocked flea.

Other than biting, fleas can also transmit the virus by defecating on the bite wound, and mechanically.

Direct contact with rodents

Man can acquire the infection directly from contact with wild rodents.

Man to man transmission

- Droplets from a case of pneumatic plague can spread from person to person
- The bacilli can also be transmitted from man to man by a vector, the human fles (Pulex irritans).

Clinical course

Incubation period = 2–7 days (except pneumonic plague, which shows up in 1–3 days).

- **Bubonic plague** is characterized by regional lymphadenopathy resulting from bite of a rat flea. The location of the primary bubo suggests the source of infection. *Inguinal buboes* in adults and older children indicate that infection was transmitted by flea bite on lower limb. *Axillary buboes* suggest upper extremity inoculation through handling of infected animal tissues, including cuts incurred while skinning an animal.
- Primary **septicemic plague** is an overwhelming plague bacteremia usually following a cutaneous exposure; it is rare.
- Primary **pneumonic plague** follows inhalation of aerosolized droplets from another human case of pneumonic plague; it is also much rare. **Secondary, pneumonic plague** results from hematogenous spread from bubonic plagues, usually a severe and fatal complication if untreated.

If the case is treated with bacteriostatic antimicrobials who do not cross the blood brain-barrier (i.e. tetracycline), the bacilli may cause meningitis.

Diagnostics

Samples

Lymph node aspirate, sputum, blood—Depending upon the type of case.

Visualization (Fig. 9.44)

The bacilli take bipolar stain with Giemsa, Wright or Wayson stain.

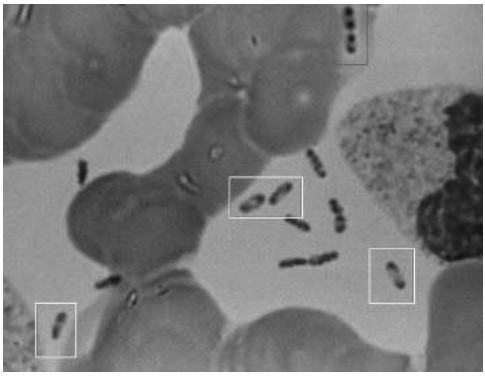

Figure 9.44. Dark stained bipolar ends ('safety pin' shaped) of Yersinia pestis can clearly be seen in this Wright's stain of blood from a plague victim [CDC Public Health Image Library, phil.cdc.gov]

Culture

Blood of all patients should be cultured, if possible on the very spot of collection. If this is not possible, the blood can be carried in the Carey Blair media to the laboratory.

Serology

Serum taken during the early and late stages of infection can be examined to confirm infection. Rapid dipstick tests have been validated for field use to quickly screen for *Y. pestis* antigen in patients.

Table 9.22. WHO case definitions of plague

Suspect plague	Compatible clinical and epidemiological features AND Suspicious organisms seen or isolated from clinical specimens.
Presumptive plague	*Yersinia pestis* F1 antigen detected in clinical materials by direct fluorescent antibody testing OR Isolate from a clinical specimen demonstrates biochemical reactions consistent with Yersinia pestis or PCR positivity OR A single serum specimen is found positive for diagnostic levels of antibodies to *Yersinia pestis* F1 antigen, not explainable on the basis of prior infection or immunization.
Confirmed plague	Isolate identified as *Yersinia pestis* by phage lysis of cultures OR A significant (4 fold) change in antibody titer to the F1 antigen in paired serum specimens.

Control

Controlling the reservoir

1. Early diagnosis of cases, notification to WHO, isolation of pneumonic plague cases, treatment (Streptomycin 30 mg/kg IM in two divided doses × 7–10 days, or gentamicin in patients who are pregnant), disinfection of fomites and corpse; destruction of carcasses of rats.
2. Education on prompt reporting of cases and sudden increase in death of rats (rat fall).

Breaking the chain

1. **Flea control**—Insecticide spraying (10% DDT, 2% Carbaryl or 5% malathion depending on sensitivity of fleas); amount of insecticide should be 2–3 g/m^2 of surface. Spraying should be done in all walls inside houses (from 3 ft and above), all crevices, doors, clothing, bedding and household pets. Rat burrows should be insufflated with insecticide powder. A radius of 5 miles of infected area should be sprayed within 48 hours of outbreak, to achieve a zero flea index.
2. **Rodent control**—The rodent population can be kept in check only by proper community sanitation in hygiene; it is important to clear the *fleas first* by insecticide and then kill the rats during an epidemic (or else, without rats to pry on, fleas will infest humans, and *that* will be very difficult to clear).

Protecting the host

Vaccination

Plague vaccines at one time were widely used but have not proven to be an approach that could prevent plague effectively. Vaccines are not recommended for protection during outbreaks, but only as prophylactic measure for high-risk groups (e.g. laboratory personnel, health workers).

The plague vaccine is a formol killed vaccine that is given in two primary doses 7–14 days apart: Males → 1 ml + 1.5 ml SC, females → 0.75 ml + 1 ml SC; a booster is required after 6 month (male → 1 ml, female → 0.75 ml). The vaccine gives immunity from 5 days after vaccination for 6 months. Doses needs to be reduced depending on age of the subject, and infants below 6 months are not vaccinated.

Chemoprophylaxis

Tetracycline 500 mg × 4 × 5 days should be given to all plague contacts and health care workers

Epidemiological surveillance

Plague pandemics of past centuries illustrate how quickly plague can spread through human population. Although no one expects to see those pandemics again, plague continues to live in some foci of the world. Effective plague prevention requires up-to-date information on the incidence and distribution of the disease. A **plague surveillance program** should identify cases and epizootics (plague outbreaks in rats) as quickly as possible so that steps can be taken to control disease spread.

Uses[339]

1. Predict areas where future human cases and rodent epizootics may occur.
2. Identify the most common zoonotic sources of human infection.
3. Identify the most important rodent and flea species maintaining a given focus of *Y. pestis.*
4. Indicate the hosts and flea species that should be targets for control measures.
5. Assess the effectiveness of plague prevention and control measures.

6. Identify local ecological factors or human activities that may result in increased plague exposure risks for humans.
7. Detect trends in the epidemiology and epizoology of plague in a given region.

Activities

Human surveillance. Reporting and notification of human cases, increasing plague awareness in the community, active surveillance for plague cases following a case report, collection of information on each case (including what kind of case it is, according to WHO case definitions), isolation of bacilli from cases is possible, treatment of cases, complete history of case including search for possible rodent exposure; pneumonic plague cases should be followed up with periodic sputum examination to detect infectivity; study of the local environment, land usage,farming and crop storage practices (rats frequent in stored grain); special attention should be given on surveillance of sea and airports, from where rodents (or a case in incubation) can be exported into another country. To identify carriers, serological surveys may be carried out in population at risk.

Rodent surveillance. Rodent sampling (collection and examination of dead rodents or trapping live rodents), diagnosis of Yersinia pestis from dead animals by direct immunofluorescence, serological survey of carnivore animals that eat rodents.

Flea surveillance. Collection of fleas from rodents and their burrows, calculation of flea indices.

Rabies

Rabies is a zoonotic disease of domestic and wild animals, and is spread through contact with infected saliva (via bites or scratches). Most human deaths occur in Asia and Africa (more than 95%). Once symptoms of the disease develop, rabies is 100% fatal. Rabies is enzootic and epizootic all over the world. The barrier of water has protected Australia, Japan, Taiwan, Ireland, UK, NZ, Lakshwadeep, Andaman and Nicobar and Maldives from rabies.

Agent

The rabies virus is a Type I Lyssavirus (Type II, III, IV are antigenically different, cause a rabies like disease). It is killed with soap, oxidizing agent, detergents, UV ray and temperatures over 60°C. The virus found in animals is the wild or 'street' virus, which turns into a 'fixed' virus over serial subculture in neural tissue.

Street virus	Fixed virus
Pathogenic to all mammals on bite anywhere in the body	Pathogenic only if injected in brain
Forms negri bodies	-
Incubation period = 2 weeks – 2 months	Incubation period = 4–6 days
Can multiply in extraneural tissue	Cannot

Host

Dogs, jackal, hyenas,foxes, bats are reservoirs for the virus. Man is an accidental, dead end host.

Risk group

1. Laboratory workers who deal with the virus
2. Veterinary doctors
3. Dog handlers
4. Hunters
5. Field naturalists
6. *Children* often play with animals and are less likely to report bites or scratches.

Chain of transmission (Fig. 9.45)

Rabies is maintained in three distinct cycles in nature. Among them, the urban cycle causes disease in humans.

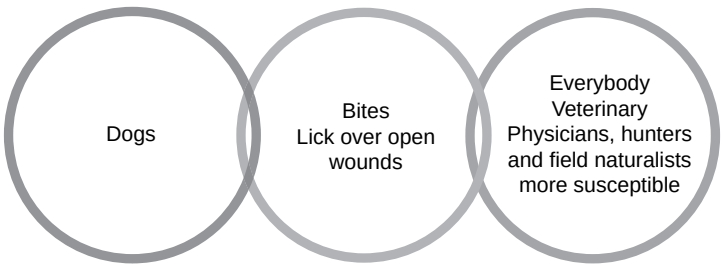

Figure 9.45. Chain of transmission of urban rabies

Why are dogs an ideal reservoir for rabies?

1. Long incubation period
2. Abundance of street dogs
3. Saliva is infective > 2 days before illness, so that infective dogs may not be symptomatic
4. Dogs are not vaccinated on a regular basis
5. Indian and Ethiopian dogs may be *chronic* carriers of the virus
6. Dogs become furious with disease and thus tend to bite.

Other cycles of rabies

The virus is maintained in the *wild* in the jackal, hyena and fox. *Bats* act as long term reservoirs of the virus and as a source of infection of wild animals. Notably, they can spread the virus through bites *and* aerosol sprays.

While the dog is the predominant cause of rabies in developing countries, in developed countries rabies continues mainly in wild animals. In the past few years, **bat rabies** has emerged as a public health problem in the Americas and Europe.

Can rabies spread from humans to human?

Not unless the patient bites a person; however, cornea/organ transplants from a rabies patient may infect the recipient.

Clinical course

The rabies virus travels to the brain by following the peripheral nerves. The **incubation period** of the disease is usually a few months in humans, depending

on the distance the virus must travel to reach the central nervous system.[340] The incubation period depends on,

1. Proximity of site of bite to brain
2. Number of wounds
3. Infective dose
4. The biting animal species
5. Clothing over the site
6. Treatment given.

Stages of rabies

Prodrome	Pain at site of bite, numbness and tingling, fever, malaise, headache, sore throat, priapism
Excitation	Restlessness, tremor, pharyngeal/laryngeal spasms, 'Hydrophobia' terror and excitement Respiratory/cardiac arrhythmias Convulsions on exposure to light, sound or air current Intense thirst and dehydration Change of voice
Paralysis	Restlessness, convulsions Flaccid and limp muscles, unconsciousness Respiratory arrest, coma, death

Differentials

1. Excitation phase—Lock jaw, encephalitis, hysteria, tetanus
2. Paralytic phase—Acute polyneuritis (Guillain-Barré syndrome), poliomyelitis, atropine poisoning, delirium tremens
3. Rabies postvaccinal encephalomyelitis.

Labs

The reference method for diagnosing rabies is by performing PCR or viral culture on brain samples taken after death, or from skin samples taken before death.[341] Inclusion bodies called **Negri bodies** are 100% diagnostic for rabies infection, not wholly sensitive.[342] If possible, the animal from which the bite was received should also be examined for rabies.

Serology

After 8 days of infection, neutralizing antibodies will appear in serum/CSF.

Control of urban rabies

Controlling the reservoir

1. Swift mass immunization of dogs—All dogs (at least 80–90%) should be vaccinated at 3–4 months of age. Live recombinant oral vaccines (see later) have given success in eliminating rabies in dogs.
2. Registration and licensing of dogs.
3. Restraint of dogs in public places.
4. Immediate destruction of rabid dogs.

5. Quarantine of imported dogs for 6 months.
6. Education on rabies and why to vaccinate your pet dog.

Breaking the chain

Beware of dogs.

Protecting the host

Neural (killed) vaccines

Suspension of infected nervous tissue of animals containing the 'fixed' virus. Because these are crude products which cause severe and sometimes fatal reactions, their production has stopped in India since 2004.

Egg vaccines (Killed)

1. Vaccine grown in **duck egg** (less antigenic than neural vaccines)
2. Vaccine grown in **chicken egg**.

Cell culture vaccines (killed)

These are much more potent and safer than neural vaccines.

1. Virus grown in human diploid fibroblast cells (HDC vaccine)
2. Virus grown in chick embryo fibroblast cells (Primary Chick Embryo Cell or PCEC vaccine), dog kidney cells or hamster kidney cells
3. Vaccine grown in vero cells[343] (Purified Vero Cell or PVC vaccine).

Schedule of immunization[344]

A cell cuture vaccine is recommended for all vaccination purposes by WHO. In India, the duck embryo vaccine, HDC, PCEC and PVC vaccine are available.

Intramuscular schedules. Causes less pain of injection, but each dose

1. Standard WHO regimen—0.5 ml of PVC vaccine or 1 ml HDC/PCEC vaccine injected IM into deltoid at 0, 3, 7, 14 and 28 days (0 = day of exposure) (Fig. 9.46).

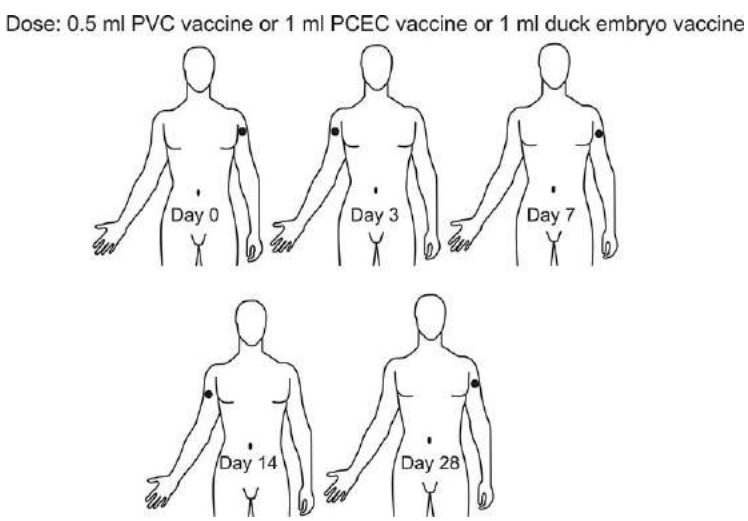

Figure 9.46. The standard intramuscular regimen

2. Abbreviated multisite regimen—0.5 or 1 ml (depending on the vaccine) IM 2 doses in two arms on day 0 → followed by 0.5 or 1 ml IM 1 dose on deltoid on day 7 and day **21** (Fig. 9.47).

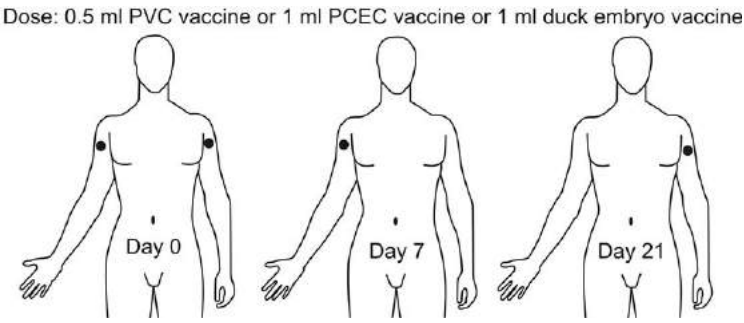

Figure 9.47. Abbreviated multisite regimen

Intradermal schedules. Intradermal dose is 1/5th of IM dose (thus intradermal dosing should be used when vaccines are in short supply), but causes more pain of injection.

1. **2 site schedule**—For use with PVC and PCEC vaccine; 1 dose of 0.1 ml PVC vaccine given at each of two sites on days **0**, **3**, **7** and at one site on days **28 and 90**; amount of each dose changes in other kinds of vaccines (Fig. 9.48).

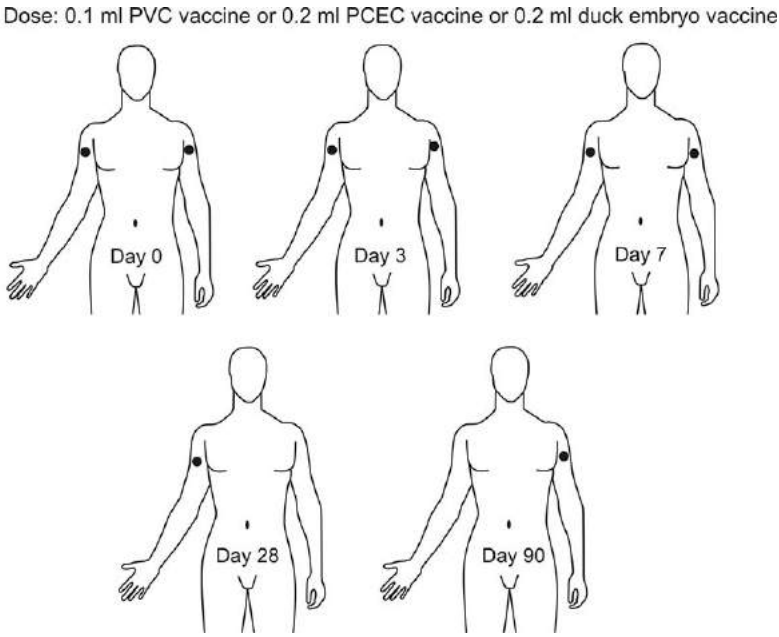

Figure 9.48. 2 site intradermal regimen

2. **8 site schedule**—For use with HDC and PCEC vaccine; 0.1 ml is given at 8 sites of two sides of the body – suprascapular, deltoid, lower abdomen and

lateral thighs on day 0. Next on day 7, the same dose is given at 4 sites of both deltoid and lateral thighs. On days 28 and 90, it is given at one deltoid only (Fig. 9.49).

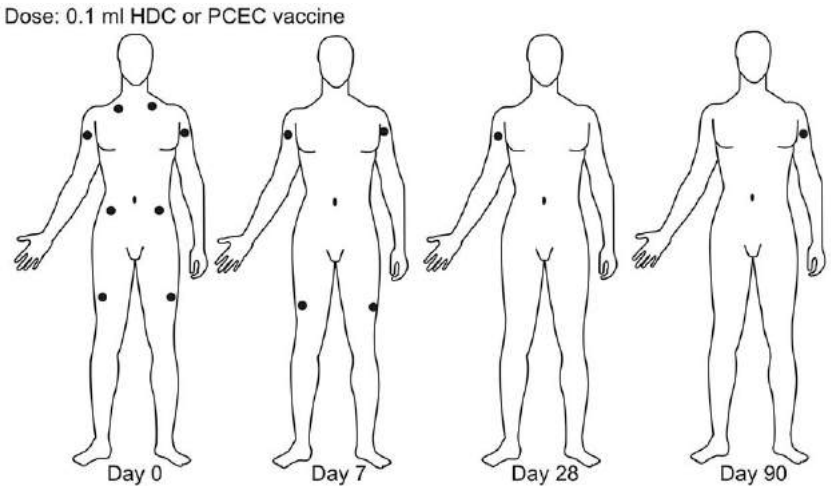

Figure 9.49. 8 site intradermal regimen

For adults, the rabies vaccine should always be administered in the deltoid area of the arm; for children aged < 2 years, the anterolateral area of the thigh is recommended. **Rabies vaccine should not be administered in the gluteal area, where the induction of an adequate immune response may be less reliable**.

Passive immunization

1. **Equine anti rabies serum**—Cheap but anaphylactic; after an exposure to virus, the '0' dose of 40 IU/kg (subject to maximum 3000 IU) is given; half of this dose is infiltrated around bite wound and other half is given IM. Because it is a foreign protein, it should be given only after sensitivity testing.
2. **Human rabies Ig (HRIg)**—Nowadays, HRIg is being recommended rather than crude preparations from horse serum because of less allergic reactions; dosage HRIg 20 IU/kg half by infiltration around wound and half IM.

Preexposure prophylaxis of rabies

Preexposure prophylaxis is recommended for everyone occupationally at risk of rabies. A cell culture vaccine is given in IM dose of 1 ml or 0.5 ml, depending on the vaccine type, or intradermal dose of 0.1 ml on days 0, 7 and 28 (day 28 preferable, but administration may be advanced towards day 21 if time is limited). **A booster dose** should be given at intervals ideally dictated by regular testing for antirabies antibodies (or every 5 years if such testing is not available. Antirabies neutralizing antibody ≥ **0.5 IU/ml** indicate protection.

Postexposure prophylaxis

Local treatment. Cleansing with soap water or detergent and copious amounts of water. *Suturing* of the wound should NOT be done before 24–48 hours.

Systemic management. Antimicrobials (to prevent secondary infection), tetanus toxoid

Animal surveillance. Observe the dog for 10 days; if it dies within these 10 days, examine its brain for rabies. Postexposure prophylaxis could be discontinued if

- The brain of the dead dog shows no rabies
- It *lives* for more than 10 days (it did not have rabies in the first place).

Rabies vaccination.

Table 9.23. The WHO guidelines for postexposure treatment of rabies

Category	Type of contact	Recommendation
I	Touching, feeding, licking on intact skin	None
II	Nibbling of uncovered skin Minor scratches or abrasions without bleeding Licks on broken skin	Vaccinate immediately STOP treatment if 1. animal is humanely killed and the brain found to have no rabies by fluorescent antibody test 2. if animal is healthy for 10 days
III	Single/multiple transdermal bites/scratches Contamination of mucosa with saliva	HRIg + vaccine STOP vaccination in above mentioned conditions

Indications of continuing rabies vaccination for full course

1. The dog shows signs of rabies or dies within 10 days
2. The dog cannot be traced
3. Unprovoked bite from an otherwise quite dog
4. The brain of the dead dog shows rabies
5. All bites of wild animals.

Postexposure treatment of who have been vaccinated earlier. HDC vaccine 1 ml im on days 0, 3, 7. If antibody titer in the serum of the person is known to be > 0.5 IU/ml, or if the bite is not severe, only two doses are needed.

Controlling wild rabies with a live oral vaccine

The V-RG vaccine is actually a vaccinia virus with the glycoprotein gene from rabies virus.[345] V-RG has been successfully used in the field in Belgium, France, Germany and United States to prevent outbreaks of rabies in wildlife. Because it is *oral*, mass vaccination of wildlife is possible by putting it in baits. A similar strategy of vaccinating "neighborhood dogs" has been used in Jaipur.[346]

Tetanus

Tetanus is a bacterial disease of high mortality (40–80%). Among the diseases of extended immunization program, neonatal tetanus is the next frequent killer after measles. It is also very under reported.

Tetanus has been eliminated from many developed countries and five South Asian countries (Bhutan, Korea, Maldives, Sri Lanka, Thailand). The World

Health Assembly resolved to eliminate tetanus (to reduce incidence < 1/1000 live births) by 1995, but in 1996, the date was extended to 2000.

Tetanus is endemic in India; **neonatal tetanus**, once a major problem, is on the decline due to improved hygiene during delivery.

Agent

Clostridium tetani which liberates a toxin (lethal dose 0.1 mg for a 70 kg man) that acts over motor end plate, spinal cord, brain and sympathetic system.

Host

Active age group (5–40); agricultural workers are most susceptible. Neonatal tetanus affects both sexes, but is more reported in male children. An incidence of tetanus gives no immunity for future, and *herd immunity* is impossible. This is also the reason that there is no maternal antibody to protect against neonatal tetanus.

Environment

Tetanus is an environmental disease; the bacteria primarily reside in the soil, and in the intestine of animals (thus excreted in their feces into soil).

Chain of transmission

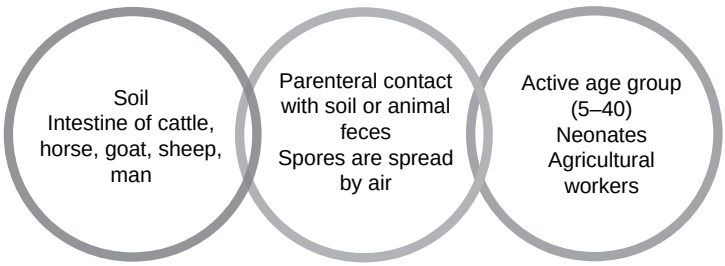

Figure 9.50. Chain of transmission of tetanus

Clinical course

Incubation period = 6–10 days; longer incubation period may be caused by dormant spores or treatment with ATS. Tetanus is characterized by muscle spasms, initially in the jaw muscles. As the disease progresses, mild stimuli may trigger generalized tetanic seizure-like activity, which contributes to serious complications and eventually death unless supportive treatment is given.

Types

1. Traumatic—Due to entry of bacteria through a wound; the wound may be so trivial so as to go unnoticed (a pinprick), or a larger one (accidents, burn, animal bite) or even iatrogenic (injections); because the bacilli is found in human intestine too, bowel surgery could leak the bacilli into blood.
2. Maternal (puerperal)—Specially after an abortion.
3. Otogenic—Through an infected middle ear; often the vehicle for the bacteria is a foreign body (pencils, beads and matches).

4. Neonatal tetanus (8th day disease).
5. Idiopathic—Due to inhalation(?) of tetanus spores or unrecognized *micro*trauma).

Prevention

Controlling the reservoir

Virtually impossible (you cannot disinfect soil).

Breaking the chain

- Adequate protection of agricultural workers (footwear)
- To stop applying soil/cowdung (!) over the cord stump of the newborn.[347]

Protecting the host

Active immunization

Because tetanus toxoid ('toxin like') is an effective vaccine, the aim is to vaccinate entire population as early as possible, so that antitoxin level 0.01 IU/ml in serum is maintained throughout life. The usual course of immunization is tetanus toxoid 0.5 ml IM in the upper arm, two doses at 1–2 months interval. A booster dose can be given after 1 year and another 5 years. The vaccines produced today are highly refined and adsorbed in aluminium phosphate, and reactions to vaccine are very uncommon.

Passive immunization

1. **Human hyperimmunoglobulin** in a dose 250–500 IU protects for at least 30 days
2. **Equine antitetanus serum** (ATS, derived from horse serum) 15000 IU sc (after sensitivity testing) protects for 7–10 days; because it is a foreign protein, it is more prone to cause reactions and loses efficacy over repeated dosing (the person develops antibodies *against* ATS).

Active + passive immunization

For previously unimmunized people as postexposure prophylaxis; the toxoid is given in one arm and the antitoxin in the other; the *rationale* is that the toxoid will give long term protection and antitoxin will cover this particular exposure.

Antimicrobials

After an exposure, an early (< 6 hours) single dose of 1.2 million unit IM benzathine penicillin/500 mg erythromycin 6 hourly can be given; antimicrobials only kill the bacilli but not their spores.

Postexposure prophylaxis

Table 9.24. Postexposure prophylaxis of tetanus

All wounds should receive surgical toilet; next, *categorize* the patient in one of four groups A = completed course of toxoid/ booster within last 5 years B = completed course of toxoid/ booster within last 5–10 years

Contd...

Contd...

C = completed course of toxoid/booster > 10 years ago D = unimmunized/unknown		
Wounds < 6 hours old, clean, non-penetrating, less tissue injury	A	No intervention
	B	Single dose of tetanus toxoid (TT1)
	C	TT1
	D	TT1 + TT2 after one month
Larger, more dirty wounds	A	No intervention required
	B	TT1
	C	TT1 + human Ig
	D	TT1 + human Ig + TT2 after one month

Elimination of neonatal tetanus program

Neonatal tetanus can be prevented by immunizing women of childbearing age with tetanus toxoid, either during pregnancy or outside of pregnancy. This protects the mother and through a transfer of tetanus antibodies to the fetus also her baby. Additionally, clean practices when a mother is delivering a child are also important to prevent neonatal and maternal tetanus. Worldwide, all countries are committed to "elimination" of maternal and neonatal tetanus , i.e. a reduction of neonatal tetanus incidence to below one case per 1000 live births per year in every district. As of December 2007, 47 countries remain that have not eliminated neonatal tetanus.[348]

Goals

Reduce incidence < 1/1000 live births in each district (number of neonatal tetanus cases is presumed to be 2 × reported *male* neonatal tetanus cases).

Strategies

1. Increase and sustain high tetanus toxoid (2 doses 1 month apart) coverage in pregnancy; no woman should be denied at least one dose of TT, whenever during her pregnancy, she may come to the health facility. If she has been immunized earlier, a booster dose is sufficient.
2. Distribution of disposable delivery kits to mothers.
3. Delivery by trained personnel.
4. Intensify dai training; develop community level skilled birth attendant.
5. Essential newborn care.
6. Surveillance.
7. Follow up where cases are reported.
8. Education on case reporting, clean deliveries, deliveries by trained personnel.
9. Monthly reporting to CMOH on incidence of neonatal tetanus.

Classification of districts

High risk	> 1/1000 live births or TT2 in pregnancy coverage < 70% or attended deliveries < 50%
Controlled	< 1/1000 live births and TT2 in pregnancy coverage > 70% and Attended deliveries > 50%
Eliminated[a]	< 0.1/1000 live births and TT2 in pregnancy coverage > 90% and Attended deliveries > 75%
[a] WHO and the Indian Government differ on what should be called 'elimination' of neonatal tetanus; WHO states that incidence < 1/1000 live births is elimination, while India has set the benchmark at < 0.1 / 1000 live births	

RESPIRATORY INFECTIONS

Smallpox

The last case of smallpox in India was Saiban Bibi, a Bangladeshi immigrant found in Karimganj railway station on 24th May, 1975. In April 1977, India was declared smallpox free. The last case in the world occurred in Somalia, 1978.

The WHO, on 8th may 1980, declared the earth 'Smallpox free'—An achievement no lesser than excursion to interplanetary systems. The virus is maintained in two laboratories of Moscow and Atlanta (to make vaccines, if needed, in future).

Why was it so easy?

Smallpox offers some distinct advantages:

1. No reservoir except humans.
2. No long term carriers.
3. No vector/vehicles.
4. Infection gives lifelong immunity.
5. Diagnosis is simple (characteristic rashes occuring over visible parts of body).
6. Subclinical infections are noninfective (because the only mode of spread is airborne infection from skin lesions).

Figure 9.51. A poster from the pre-eradication days [CDC Public Health Image Library]

7. Vaccine is highly effective
8. All the nations cooperated (finally, for once).

However …

1. Monkeypox, tanapox, yaba pox, molluscum contagiosum virus still remains, which may cause breakouts
2. There may be a laboratory accident any day
3. Terrorists may bomb any country any day with the viruses.

So…

Smallpox surveillance is still going on. Any atypical cases of chickenpox should be immediately reported.

Chickenpox (Varicella)

Agent

The Varicella Zoster Virus (VZV) or Human Herpes Virus 3 causes an acute infection (chickenpox/varicella) and a latent, recurrent dermatomial infection (zoster).

Host

Chickenpox usually occurs in children <10 years in the mild form. Infection in adults is more complicated. One attack gives lasting immunity. However, any waning of immunity may cause reactivation of latent foci of virus (zoster). The virus is also transmitted vertically (from mother to fetus).

Environment

Chickenpox occurs in its full spree within 1st six months of the year (the spring – summer disease). Overcrowding (as in hostels) helps its rapid dissemination.

Chain of transmission (Fig. 9.52)

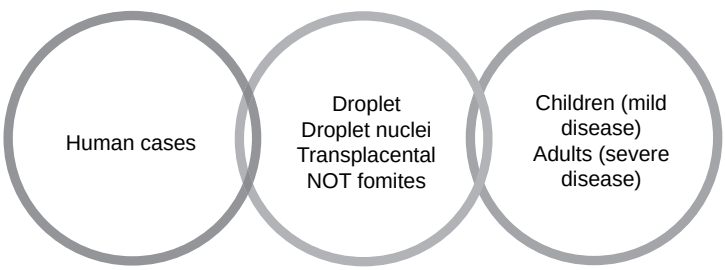

Figure 9.52. Chain of transmission of chickenpox

Communicable period extends from 1–2 days before ↔ 4–5 days after rashes appear. However, the healed scabs are noninfective, and usually herald the time the patient may be brought back to contact with his peers. Chickenpox is very much communicable (SAR 70–90%) but not a very severe disease.

Clinical course

The **incubation period** varies from 7–21 days, following which the disease evolves in these stages.

Preeruptive stage

It is the prodrome lasting about 24 hours with fever, back pain, shivering and malaise.

Eruptive stage

The rashes appear on the *same day* as fever. The rashes are pleimorphic, appear first on trunk and spreads centrifugally. The rashes evolve very rapidly. The fever starts afresh with each new crops of rashing but does not rise to a very high degree.

Table 9.25. Smallpox and chickenpox

	Smallpox	**Chickenpox**
Incubation period	12 days	7–21 days
Prodrome	Severe	Mild
Rashes start at Axilla Progression Depth Vesicles Stages seen at a time Evolution	Palm, sole, face Free Centripetal Deep Multilocular, umbilicated Monomorphic Slow	Trunk Affected Centrifugal Superficial Unilocular, dew drop like Pleiomorphic Fast
Scabs form after	10–14 days	4–7 days
Fever	Subsides with rashing but may rise again in pustular stage	Rises with each new crop of rash, subsides in 5 days

Complications

1. Pneumonia
2. Encephalitis/meningitis/cerebellar ataxia
3. Reye syndrome (encephalopathy + fatty degeneration of viscera).

Teratogenicity

Maternal varicella may cause fetal wasting, cutaneous scars, atrophic limbs, microcephaly and low birth weight.

Labs

Culture from lesional fluid, serology (immunofluorescence, fluorescent antibody to membrane antigen, ELISA, etc). Clinical diagnosis is recommended.

Table 9.26. Differences between varicella and zoster

	Varicella	**Zoster**
Occurrence	Epidemic	Sporadic
Season	Spring	None

Contd...

Contd...

SAR	90%	None[a]
Age	Children	> 50 yrs
Lasting immunity	Present	Not present
Site	Whole body	Dermatomes
Triggers	Unknown (in children) Immunosuppression in adults	Immunosuppression
Prognosis	Cure is usual	Cure/destruction of ganglia

[a] But cases where chickenpox has spread from a skin lesion of zoster are not unknown

Prevention

Controlling the reservoir

Early diagnosis of cases and isolation for 6 days after rashing.

Protecting the host

Passive immunization

Varicella Zoster Ig (VZIG) is given with 72 hours of exposure (i.e. contact to a case), 1.25–5 ml IM.

Indications. (1) Immunocompromised (2) Susceptible teens (i.e. who still have not had the disease) (3) Pregnant woman (4) Newborn whose mother has chickenpox within 5 days before and 2 days after delivery (5) Premature infants (6) Continuous household contact of patient (7) Hospital contact (8) Playmate of a patient for more than one hour.

Active immunization

A live vaccine of chickenpox is available (Oka strain), which *may* produce latent infection and zoster in later years. It is indicated before any surgery leading to immunocompromise (i.e. splenectomy) and diseases that may be aggravated by chickenpox.

Cons to chickenpox vaccination

1. Chickenpox is a very mild disease in children
2. One attack will give lasting immunity
3. LAV may cause zoster.

It is not rational policy to include chickenpox vaccine in the National Immunization Schedule. This is because if the chickenpox vaccine were to be added to the list of childhood vaccinations, it is feared that there would be a greater number of cases of zoster in adults, until the vaccination was given to the entire population. This is because adults who have had chickenpox as a child are less likely to have zoster in later life if they have been exposed occasionally to the chickenpox virus (for example by their children). The exposure itself acts as a booster vaccine. In this case, the disease in the child vaccinates the parent against zoster.[349]

Diphtheria

Diphtheria (Greek "pair of leather scrolls", referring to the pseudomembranes over the two tonsils) is an ancient disease. Historically quite common, diphtheria has largely been eradicated in industrialized nations through widespread vaccination.

Because diphtheria was once as endemic in developed countries as tuberculosis today is in developing countries, the history of diphtheria is well documented, and associated with many big names in medicine. In the 1920s, diphtheria was a menace in the US, killing a large number of under five children. One of the most famous outbreaks of diphtheria was in Nome, Alaska; the 1925 'serum run' to Nome to deliver diphtheria antitoxin is now celebrated by the "Great Race of Mercy".

The diphtheria bacilli were first observed by Klebs (of Klebsiella fame) in 1883 and cultivated by **Loeffler**, (1884). **Roux and Yersin** of the Pasteur institute, discovered the pathogenic mechanism, the diphtheria exotoxin, in 1988. The first effective treatments for diphtheria, endotracheal tubes that prevented the tonsils from blocking the airways, were implemented in the 1880s by US physician Joseph O'Dwyer. In the 1890s, the German physician **Emil von Behring** developed an antitoxin for diphtheria from animal serum, for which he was awarded the **first Nobel Prize in Medicine**. The first successful *vaccine* for diphtheria was also developed in 1913 by Behring.

The **Schick test** (see later) was invented by **Béla Schick** in 1911, a Hungarian-born American pediatrician. A massive five-year campaign was coordinated by Dr. Schick. As a part of the campaign, 85 million pieces of literature were distributed by the Metropolitan Life Insurance Company with an appeal to parents to "Save your child from diphtheria."

The palpable effect of vaccination on diphtheria can be exemplified by no other incidence than the breakup of Soviet Russia. After the breakup of the former Soviet Union in the late 1980s, vaccination rates in its constituent countries fell so low that there was an explosion of diphtheria cases. In 1991 there were 2,000 cases of diphtheria in the USSR. By 1998, according to Red Cross estimates, there were as many as 200,000 cases in the Commonwealth of Independent States, with 5,000 deaths. This was so great an increase that diphtheria was cited in the Guinness Book of World Records as "most resurgent disease".[350]

Agent

Corynebacterium diphtheriae is a gram +ve bacillus with three strains – *gravis*, *intermedius* and *mitis*. Some strains are toxinogenic while others not. Diphtheria toxin is only produced by Corynebacteria when it is infected with a bacteriophage. The bacteriophage integrates a gene into the bacteria that causes the toxin to be produced.

Resistance

It is killed by heat (100°C for 1 minute or 58°C for 10 minutes) and formaldehyde very easily, but can survive in the environment (i.e. fomites) for long time.

Host

Everybody can get affected, but the disease is most severe children under 5 years and people over 40, when it can cause a 20% case fatality.[351]

Cases

Cases may be clinical or remain subclinical. Also, the clinical stage may remain *mild* (occasional sore throat), thus hard to detect.

Carriers

Both temporary and chronic carriers (i.e. bearing depots of bacteria in their pharynx or nose) do occur. Route of exit is droplets in nasal or throat secretions.

Environment

Like all respiratory infections, diphtheria is spread by overcrowding and poor hygiene.

Chain of transmission (Fig. 9.53)

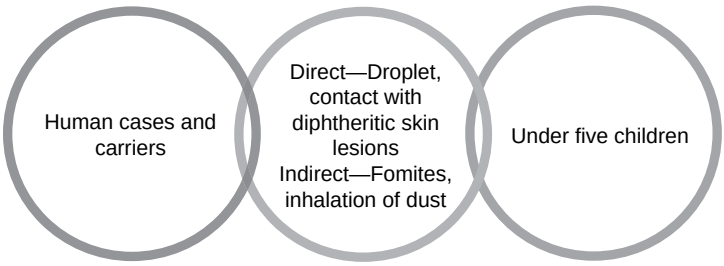

Figure 9.53. Chain of transmission of diphtheria

The disease is **communicable** for 14–21 days after onset of symptoms. If two thorat swabs taken 24–48 hours apart are found negative, then the case has ceased to be infective.

Clinical course

Incubation period is 2–6 days. The disease comes in two distinct types.

Disease in respiratory tract

1. Nasal (mildest)—Only bloody discharge from nose, very infective
2. Faucial/pharyngotonsillar—Thick, bluish to gray, adherent pseudomembrane that bleeds if picked
3. Laryngotracheal (severe disease)—Cough and hoarseness of voice, usually preceded by faucial diphtheria.

Nonrespiratory disease

In addition to symptoms at the throat, the patient may experience more generalized symptoms (listlessness, pallor, tachycardia, low blood pressure). Longer-term effects of the diphtheria toxin include cardiomyopathy[352] and peripheral neuropathy[353] (causing postdiphthetic paralysis). Diphtheria may also cause *skin lesions*, conjunctivitis and ear infections.

Labs

1. Culture and visualization of the bacilli from throat swab
2. Toxigenicity testing
3. Schick test—This is an intradermal hypersensitivity test which tests (a) the immune status and (b) hypersensitivity to diphtheria toxin. 0.2 ml toxin is introduced in one arm and the same amount of inactivated toxin in other arm (control).

Test arm	Control arm	Result
No reaction	No reaction	Immune
Circumscribed red flush 10–50 mm diameter which attains maximum size between 4th – 7th day	No reaction	Positive (susceptible)
Red flush which disappears on 4th day	Red flush which disappears on 4th day	Pseudopositive (hypersensitive) reaction
Red flush that attains maximum size in 4–7 days	Red flush which disappears on 4th day	Combined (patient is susceptible)

Treatment

Cases

The disease is manageable medically until respiration is obstructed, when emergency tracheostomy or intubation is required. Diphtheria can also cause paralysis in the eye, neck, throat, or respiratory muscles, cardiac arrhythmias and heart failure, and should be put in an ICU.

Antitoxin. The decision to infuse antitoxin should not await laboratory results, as the antitoxin is life saving. Dosage—20000–100000 IU of antitoxin given IM/IV (after sensitivity testing), depending on severity of disease.

Antimicrobials. The CDC recommends

- Erythromycin 40 mg/kg oral or IV (maxiumum 2 g per day) × 14 days
- Procaine penicillin G (300000 IU for patients weighing < 10 kg or 600000 IU for those weighing > 10 kg) IM × 14 days. Patients with allergy to penicillin G or erythromycin can use rifampin or clindamycin.

Carriers

Erythromycin 250 mg oral × 4 × 10 clears the infection.

Control

Controlling the reservoir

Early detection of cases and carriers (by active search within the contacts of a case), isolate them for the period of communicability (14 days) and appropriate treatment.

Breaking the chain

Disinfect clothes, fomites and sputum of cases and carriers.

Protecting the host

Vaccination

The diphtheria vaccine is a filtrate of toxigenic strain treated with 0.3% formalin and incubated at 37°C until toxicity has disappeared (it becomes a 'formol toxoid'). Now, it is adsorbed in aluminium hydroxide or phosphate.

The diphtheria toxoid is available combined with tetanus toxoid as pediatric diphtheria-tetanus toxoid (DT) or adult tetanus-diphtheria (dT) combination (the

adult preparation contains 10 times less diphtheria toxoid). **Children younger than 12 years** of age should receive either DPT (if under 5 years of age) or pediatric DT (if more than 5 years of age). **Persons 12 years of age or older** should receive the adult formulation (adult dT), even if they have not completed a series of DPT or pediatric DT. Both DT and dT require *two* doses only, while the inclusion of pertussis in DPT causes a minimum of 3 doses to achieve seroconversion.

Dosage. 3 IM doses in at least 1 months intervals (or 2 doses if no pertussis vaccine is given); booster doses may be given at 18 months later and another after 5–6 years. The diphtheria toxoid is combined as DPT, DT or dT and integrated in National Immunization schedule (refer to the beginning of this chapter).

Table 9.27. Specific protection for contacts of a case

Immunized within 2 years	No action
Immunized before 2 years	Booster dose of toxoid
Nonimmunized	Erythromycin + Antitoxin 1000–2000 IU + Diphtheria toxoid full course

Pertussis

Although childhood vaccination has dramatically reduced reported pertussis cases, the incidence of the disease has increased over the past 20 years in developed countries, most notably in previously immunized adolescents and adults.[354] In India, however, pertussis is still on the decline.

Agent

Bordetella pertussis and *Bordetella parapertussis* are gram –ve coccobacilli that liberate both endotoxins and exotoxins (pertussis toxin, adenylate cyclase like toxin and tracheal toxin). They are easily killed by heat and ordinary disinfectants and do not survive in external environment for long.

Host

Under five children are most susceptible, and the disease is most severe below 6 months of age. Females are prone have a more severe disease. There are only *clinical cases* and no subclinical cases/ carriers.

Environment

Overcrowding and low socioeconomic status.

Chain of transmission (Fig. 9.54)

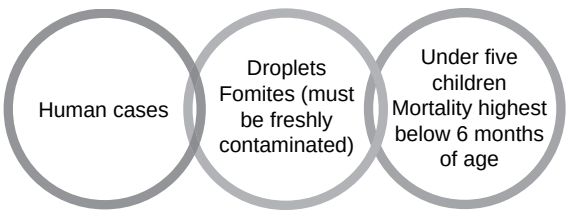

Figure 9.54. Chain of transmission of pertussis

The disease is very infective is 1st stage (SAR = 90%). **Communicable period** lasts for 1–3 weeks after paroxysmal cough.

Clinical course

Incubation period = 7–14 days
Stage 1—Catarrh (**2 weeks**). Insidious onset of cold, running nose, watering of eyes, mild fever, irritating cough that worsens at night
Stage 2–Paroxysm (**2–4 weeks**). Intense bouts of cough in quick succession > apnea > deep inspiration with a characteristic 'whoop' like sound.

Complications

1. Pressure effects—Subconjunctival hemorrhage, hematemesis, epistaxis
2. Dehydration and exertion
3. Bronchitis, emphysema, bronchiectasis, bronchopneumonia
4. Convulsion and coma (encephalitis).

It is one of the most lethal diseases in children who have not been immunized, or are malnourished.

Labs

The best sample for diagnosis is a **saline nasal wash**. Droplets can also be collected directly by holding the culture media (Bordet gengue media) in front of the patient while he coughs. Organisms are best visualised by fluorescent antibody testing.

Treatment

Linctuses and soothing agents, maintenance of hydration, good nursing care, continued feeding, Erythromycin 30–50 mg/kg oral daily (in 4 divided doses) × 10 days.

Control

Controlling the reservoir

1. Early detection of cases (the best chances of detection of bactera in nasal wash is at the first stage of disease, i.e. 2 weeks after onset of symptoms)
2. **Isolation** until clinically considered noninfective by culture (at least 3 weeks in untreated patients or 5 days after erythromycin treatment)
3. Treatment.

Breaking the chain

Disinfection of fomites of all cases.

Protecting the host

Pertussis vaccine

There are two kinds of vaccines

- **Whole cell killed vaccine (wP)**—Which is usually used in our country (it is cheaper), and can cause the same neurologic complications as the pertussis bacillus.

- **Acellular vaccine (aP)**—A combination of pertussis toxin + Hemagglutinin / 69kD Bordetella protein/Fimbrin II and III.

Pertussis vaccines are highly effective, strongly recommended, and save many infant lives every year. Though the protection they offer lasts *only a few years*, those few years include the preschool age, the time of greatest risk from pertussis.[355] Because the vaccine is effective only for short-term, and pertussis is increasingly occurring in adolescents (who may transmit the bacteria to infants), some countries have adopted a booster dose. Others **refrain from giving any pertussis vaccine (primary or booster) to anybody more than 5 years of age**, from concerns that neurologic side effects of wP increase with age. However, the aP can be used for vaccination of teens and adults.

Management of contacts of a case

Close contacts are defined as anyone coming into contact with the respiratory secretions of an infected person in the 21 days before or after the infected person's cough began. For all contacts of a case, implement

1. Separation
2. Prophylactic erythromycin
3. Booster dose of DPT (or only DT if the contact is > 5 years old)
4. Ring immunization (protecting a child by vaccinating his playmates, so that he is surrounded by immune individuals).

In general, for the community, the pertussis killed vaccine is included in DPT.

DPT

DPT has been combined into one because of logistic advantages (the child gets 3 vaccines at one go) and that pertussis killed vaccine increases immunogenicity of other two. The choice of vaccine is now the adsorbed vaccine (in aluminium hydroxide or phosphate) which increases both immunogenicity and shelf life of the vaccine.

The optimum temperature for DPT is 2–8°C, but not *freezing*. It is one of the most heat stable, and freeze sensitive vaccines.

1. Primary doses—Optimally at 6, 10, 14 weeks of age (must be completed before 1 year)
2. Booster doses—At 1½ years and 5–6 years (DT *only* after 5 years, as pertussis is unlikely to be a severe disease after 5 years of age and the vaccine also runs a high chance of cerebral complications after 5 years); recently, due to resurgence of pertussis in developed countries amoung adults, the CDC recommends daPT (adult diphtheria vaccine + acellular pertussis vaccine + tetanus toxoid).[356]

Depending on the pertussis component, there are two kinds of DPT vaccines—The DTaP and DTwP (see the section on pertussis vaccines).

Efficacy. After a primary series of three properly spaced diphtheria toxoid doses in adults or four doses in infants, a protective level of antitoxin (defined as greater than 0.1 IU of antitoxin/ml) is reached in more than 95% children.[357]

Dose. 0.5 ml deep IM in left anterolateral thigh/deltoid

Adverse reactions. Fever (within 24 hours and short lasting), soreness, convulsions and encephalitis. **Neural complications** like convulsion,

encephalopathy or Reye's syndrome are due to the pertussis component. If a child shows such reactions during 1st dose, drop pertussis from 2nd dose onwards.

Because of so many adverse reactions, drop out rate from immunization schedule is *maximum* after DPT vaccination, and the DPT vaccine has faced many lawsuits in countries where people prefer litigation rather than defaulting.[358]

Contraindications. Severe allergic reaction to a previous DPT injection. If the child is having convulsions and coma, drop pertussis and continue with DT.

Influenza

Influenza[359] is characterized by three important features

1. Propensity to cause periodic pandemics (Fig. 9.56)
2. High mortality during pandemics
3. Great antigenic variation that lead to these pandemics.

Influenza rapidly spreads around the world in frequent seasonal epidemics, affecting 5–15% of the population. Deaths mainly occur in **high-risk groups** (elderly, chronically ill).[360] However, sudden antigenic changes causes **pandemics** which are distinct from seasonal epidemics. *Three* influenza pandemics occurred in the 20th century (H1N1 Spanish flu in 1918, H2N2 Asian flu in 1957, H3N2 Hong Kong flu in 1968), each of caused by the appearance of a new strain of the virus. Often, these new strains appear

- When an existing flu virus spreads to humans from other animals
- When an existing human strain picks up new genes from a virus that infects birds or pigs.

An avian strain named H5N1 raised the concern of a new influenza pandemic, after it emerged in Asia in the 1990s, but it has not evolved to a form that spreads easily between people.[361] There was speculations that it was being transmitted directly from birds to humans.

The unique features of an influenza pandemic are

1. Suddenness
2. Short incubation period
3. Large number of subclinical cases
4. Short duration of immunity
5. Absence of cross immunity.

Agent (Fig. 9.55)

Influenza virus belongs to the famliy Orthomyxoviridae with three distinct types A, B, C of which type A causes pandemics and type C causes a milder disease. Among its many antigens, two surface antigens (hemagglutinin and neuraminidase) are responsible for its antigenic variation. Among 15 possible different hemagglutinins, only H1–H5, and two neuraminidases, N1 and N2, infect humans.

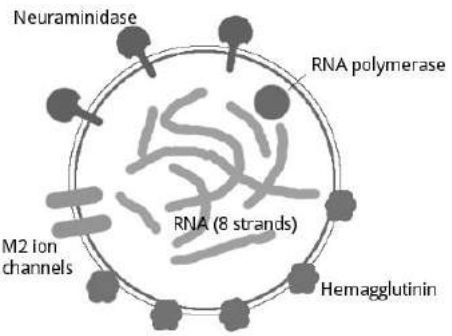

Figure 9.55. The influenza virus; note the eight strands of RNA inside the virus, which mix and match in any combination between themselves [CDC - public health image library]

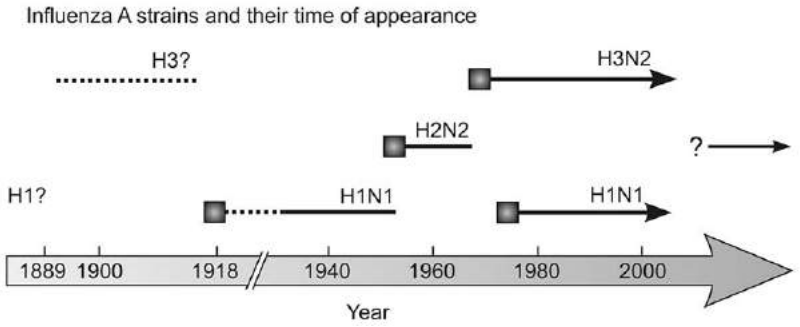

Figure 9.56. Influenza A strains presently circulating in humans

Influenza is harbored by aquatic birds who discharge the virus in feces into water. Terrestrial birds acquire the infection from water and release droplets of virus in their feces. The final host is pig which acquires the virus from feces of birds and humans. The human and avian viruses exchange genes to become a 'new' virus inside the pig, which are the 'mixing vessels'.

Antigenic shift and drift

Shift is a change of both hemagglutinin and neuraminidase (occurs only in influenza A virus due to *genetic reassortment* of the 8 RNA fragments contained inside the virus). Shift is a major change in antigenic structure and responsible for pandemics. However, much more frequent are small antigenic variations that occur continuously, known as *drifts*, due to point mutations in the RNA. Because of drifts there are seasonal epidemics, and influenza vaccine has to be *reformulated* annually.

Host

Everybody is affected, but the disease is most severe over 65 years of age, or in the immunocompromised, but in general children get more infected.

Environment

Most epidemics of influenza occur in the winter. Like all respiratory infections, overcrowding propagates the virus.

Chain of transmission (Fig. 9.57)

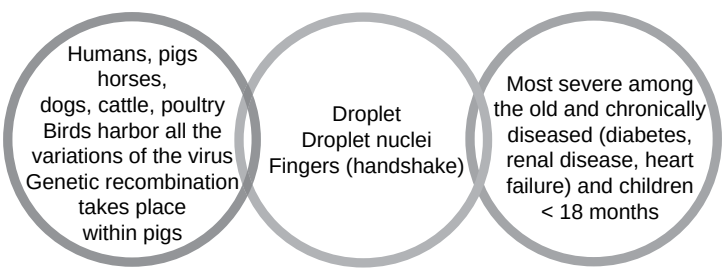

Figure 9.57. Chain of transmission of influenza

Communicable period = 1–2 days before onset of fever ↔ 1–2 days after onset of fever

Clinical course

Incubation period = 18–72 hours. The virus attacks mainly the upper respiratory tract – the nose, throat and bronchi and rarely also the lungs. The infection usually lasts for about a week. It is characterized by sudden onset of high fever, myalgia, headache and severe malaise, nonproductive cough, sore throat, and rhinitis. Most people recover within one to two weeks without requiring any medical treatment. However, these mild infections act as the source of the virus.

In the very young (< 18 months), the elderly and people suffering from medical conditions such as lung diseases, *diabetes*, *cancer*, *kidney* or *heart problems*, influenza poses *a serious risk*.

Labs

- Virus isolation from nasopharyngeal secretion (indirect fluorescent antibody technique)
- Fourfold rise in antibody titers in paired sera, one taken within 5 days of symptoms, another after 10–14 days.

Control

Controlling the reservoir

We can't 'screen' chickens regularly for flu. However, during an epidemic, everybody having even mild symptoms should stay at home and cover their mouths and noses. **Antiviral drugs** like amantadine and rimantadine can be taken : Amantadine 100 mg × 2 × 5 days. These drugs are effective against influenza A if given early in the infection but not against influenza B (which does not have the M2 protein which is the site of action of these drugs).

Breaking the chain

Use of handkerchiefs while coughing, talking or sneezing; proper *hand washing*

Protecting the host

Vaccination is recommended for high-risk groups.

Killed vaccine. Virus grown in chicken eggs and inactivated by detergents or β-propiolactone; it is given in a dose 0.5 ml 2 doses SC at 3–4 weeks intervals in previously unimmunized adults; during epidemics, a single dose is given which protects for 3–6 months.

Side effects. Fever, allergy (due to egg proteins), Guillain-Barré syndrome.
Indications. Children with chronic asthma, people above 65, people with cardio-pulmonary disease, immunosuppressed people (including HIV), 2nd trimester of pregnancy.
Contraindications. Children < 6 months, people allergic to egg.

Subunit vaccine. Specific viral components (the H or N antigens) can be isolated to be used as vaccines, or the antigens of a virulent strain may be transferred to a not so virulent strain to produce a vaccine virus.

Live attenuated vaccines

Produced by

1. introduction of hemagglutinin gene to vaccinia virus.
2. successive culture in low temperatures to produce a 'cold adapted strain' which may be given as nasal spray.

Due to the high mutation rate of the virus, a particular influenza vaccine usually confers protection for no more than a few years. Every year, the World Health Organization predicts which strains of the virus are most likely to be circulating in the next year, allowing pharmaceutical companies to develop vaccines that will provide the best immunity against these strains;[362] even then, it cannot possibly include *all* the strains and there can be vaccine failure.

The vaccine is not useful to control epidemics because to be effective, it must be given at least 2 weeks before exposure; and within 2 weeks, a new strain may develop which goes on to cause the epidemic. During epidemics, it is given only to high risk groups and people working in emergency sectors (traffic, police, health care, news, fire service) so that they do not abstain from duty for the fear of catching the flu.

The 2009 'swine flu' pandemic

In April 2009 a novel flu strain emerged in Mexico that *combined* genes from human, pig, and bird flu, initially dubbed **swine flu** and also known as influenza A H1N1.

Differences of the 2009 'swine flu' virus from the seasonal influenza virus

1. Genetically, the virus is *unrelated* to the seasonal H1N1 viruses that have been in general circulation among people since 1977; it is essentially a *new* virus and most people had no or little immunity to it when it emerged.
2. Unlike typical seasonal flu patterns, the new virus caused *high levels of summer infections* in the northern hemisphere.
3. The new virus causes patterns of death and illness not normally seen in influenza. Most of the deaths have occurred among *younger people*, including those who were otherwise healthy. Pregnant women, younger children and people with chronic lung or other medical conditions appear to be at higher risk.
4. The virus causes severe disease in many *healthy* individuals, which is not in lieu with seasonal influenza; the speed of deterioration is also much quicker, as well as increased need for ICU care.
5. Many of the severe cases have been due to *viral pneumonia*, which is harder to treat than bacterial pneumonias (usually associated with the 'conventional' seasonal influenza).

The World Health Organization officially declared the outbreak to be a pandemic on June 11, 2009. However, this declaration was an indication of *spread*, not severity, the strain *actually* having a lower mortality rate than common flu

outbreaks.[363] There is no evidence that it is endemic to pigs (i.e. actually a swine flu) or of transmission from pigs to people, instead the virus is spreading from person to person.[364] Contrary to popular belief, it is not spread by eating pork.

Manifestations

While most people will experience only mild symptoms, those at risk of a more severe infection include asthmatics, diabetics,[365] obese, heart disease, immunocompromised, children with neurodevelopmental conditions (cerebral palsy, muscular dystrophy, developmental delay),[366] and pregnant women.

Groups at Higher Risk for Severe Illness from 2009 Influenza A (H1N1) Infection (CDC)

1. Children younger than 5 years old.
2. Persons aged 65 years or older.
3. Children and adolescents (younger than 18 years) who are receiving long-term aspirin therapy and who might be at risk for experiencing Reye syndrome after influenza virus infection.
4. Pregnant women.
5. Adults and children who have asthma, chronic pulmonary, cardiovascular, hepatic, hematological, neurologic, neuromuscular, or metabolic disorders such as diabetes.
6. Adults and children who have immunosuppression (including immunosuppression caused by medications or by HIV).
7. Residents of nursing homes and other chronic-care facilities.

Warning signs[367]

Adults. Difficulty breathing or shortness of breath, pain or pressure in the chest or abdomen, sudden dizziness, confusion, severe or persistent vomiting, low temperature.

In children. Fast breathing or working hard to breathe, bluish skin color, not drinking enough fluids, not waking up or not interacting, being so irritable that the child does not want to be held, flu like symptoms that improve but then return with fever and worse cough, fever with a rash, being unable to eat, having no tears when crying (!).

Labs

The confirmatory diagnosis is **real time reverse transcriptase PCR** on nasal, nasopharyngeal or oropharyngeal swab.[368] However, confirmatory diagnosis usually does not alter the treatment but only serves as epidemiological data. The CDC recommends testing only for people who are hospitalized with suspected flu, pregnant women, and immunocompromised people.

Situation

World. As of 28 February 2010, worldwide more than 213 countries and have reported confirmed cases of influenza H1N1, including at least 16455 deaths.[369] Transmission of virus persists in some areas of Europe and Asia but influenza activity is declining at the time of writing and at low level in the most areas. The

most active areas of transmission are currently observed in parts of Southeast Asia and East and South-eastern Europe. Recently, influenza type B is increasingly reported in Asia.

India. India has been affected a lot less than many other countries, and if the drop in number of deaths as well as new infections is anything to go by, swine flu or influenza A (H1N1) seems to be receding in the country. Upto 5th March 2010 the overall death toll in the country now stands at 1387.

Treatment

Supportive care (rest, drinking plenty of fluids, a nonaspirin analgesic) should be enough in most cases. See a doctor if any of the warning signs develop.

People in at-risk groups or those with rapidly worsening symptoms should be treated with **antivirals** (oseltamivir or zanamivir); the CDC recommended oseltamivir treatment primarily for people hospitalized with pandemic flu.

Oseltamivir is approved for use in persons age 1 and over. The usual adult *theraputic* dosage is Oseltamivir 75 mg oral × 2 × 5 days, beginning within 2 days of the appearance of symptoms; dose must be reduced for children and patients with renal impairment. Oseltamivir may be given as prophylactic (in an epidemic or following close contact with a case). Standard *prophylactic* dosage is 75 mg once daily (for patients aged 13 and older) × upto 6 weeks.

The CDC recommends oseltamivir specifically to

- People hospitalized with more severe illness
- Children younger than 2 years old
- Adults over 65
- Pregnant women
- People with chronic medical or immunosuppressive conditions
- Adults under 19 on long-term aspirin therapy.

However, children and adults presenting with suspected flu that have symptoms of lower respiratory tract illness or clinical deterioration should also receive prompt empiric antiviral therapy.[370]

Control

Controlling the reservoir

Early detection of cases. During the epidemic, anybody having mild respiratory symptoms should isolate himself or herself. The CDC advises that school student/ office workers stay home sick for seven days after getting the flu, or 24 hours after symptoms end, whichever is longer. In the airports, thermal screens are used to detect anybody with a fever.

Breaking the chain

1. 2 weeks quarantine of international travelers
2. Use of air filters and cabin cleaning inside aeroplanes
3. Proper hand washing
4. The CDC recommends face mask or respirators only for.[371]

- health care professionals.
- A high risk individual (see earlier) taking care of a flu patient.
- People who are ill with the virus themselves (so that they don't spread the disease to others).

Facemasks do not seal tightly to the face, while most *respirators* (e.g. N95) are designed to seal tightly and filter out very small particles. For both facemasks and respirators, however, limited data is available on their effectiveness. Also, these devices may give people a false sense of security so that they forget other precautionary measured. The 2009 epidemic had created a mad rush for N95 masks, skyrocketing their prices in India,[372] which goes to show how people love to run after a hype.

Vaccination (Fig. 9.58)

Both the CDC of USA and NHS of UK have recommended vaccinating high risk people (who are over 6 months of age), pregnant women and health care workers. Single dose of the killed vaccine has been recommended as sufficient for the present pandemic for people above 10 years who are not immunocompromised.[373] This vaccine does not protect against seasonal influenza.

Figure 9.58. The live vaccine for influenza H1N1 given as nasal spray [CDC Public Health Image Library]

Mumps

Mumps is a global disease of low mortality.

Agent

The mumps virus is one of the family Paramyxoviridae, with only one serotype. The virus invades and is found in all body fluids, but has special predilection for glandular epithelium and neural tissue.

Host

Attacks children (5–9 years) preferentially than adults. One attack (clinical or subclinical) gives lasting immunity. Infants below 6 months are immune due to maternal antibodies.

Environment

Mumps is endemic, but incidence peaks in winter and spring; overcrowding will occasionally result in epidemics.

Chain of transmission

Cases (clinical and subclinical) → droplet, saliva (especially by kissing or sharing food), urine, blood transfusion, transplacental → human host, especially children between 5–9 years. **Communicable period** is 1 week before onset of symptoms ↔ 1 week after; it is maximally communicable *just* after parotid swelling has occurred. SAR is 86%.

Clinical course

Incubation period = 2–3 weeks after which there is acute tender parotid enlargement which subsides in another 2–3 weeks. There may be pain on opening of mouth before swelling is evident. About 30–40% infections are asymptomatic.

Complications

Orchitis (usually unilateral, self limiting, and *rarely* causes sterility[374]), ovaritis, pancreatitis, encephalitis, 'aseptic' meningitis, transverse myelitis, deafness, polyneuropathy, cerebellar ataxia and facial palsy (i.e. involves every kind of glands and neurons in the body). The risk of orchitis is increased if the infection is contacted during adolescence. The virus may cause abortion if contacted during first trimester of pregnancy, but not teratogenicity.

Control

Controlling the reservoir

It is difficult as the disease spreads during incubation period. However, isolate the all the cases until parotid swelling subsides.

Breaking the chain

Disinfect fomites of the case, use of handkerchiefs and face masks by the case.

Protecting the host

Vaccination

Live vaccines have been devised from many strains (i.e. the Jeryl Lynn strain), which is a freeze dried vaccine given single dose 0.5 ml SC at upper midthigh/ arm at 12–15 months of age, and another before entering school. It is stored at 2–8°C.

The WHO (World Health Organization) recommends the use of mumps vaccines in all countries with well-functioning childhood vaccination programs. Although not in National Immunization in schedule, MMR vaccine is recommended after 1 year of age by Indian Academy of Pediatrics.

Side effects. Cranial nerve palsy, Guillain-Barré syndrome, Encephalitis

Contraindications. Acute febrile illness, immunodeficiency and malignancy, within three weeks of another viral vaccine/BCG, egg/chicken allergy or allergy to antimicrobials (which are added in vaccine), infants, pregnancy and 3 months before conception, within 3 months of giving any normal human Ig.

Mumps elimination

Many developed countries have now targeted mumps for elimination. The strategies for elimination are:

- high coverage of first dose MMR vaccination.
- ensuring a second opportunity for vaccination.
- conducting 'catch up' immunization for susceptible children, who have been left out from being immunized.

Case definitions for surveillance (WHO)

Clinical mumps. Acute onset tender, self limited, unilateral or bilateral parotid (or other salivary swelling) lasting ≥ 2 days without other causes.

Confirmed mumps. Clinical mumps + mumps specific IgM antibody (who have been not immunized within 6 weeks with the mumps vaccine) or 4 fold rise of mumps IgG in paired sera or isolation of mumps virus from saliva, urine, CSF

Epidemiologically confirmed mumps. A patient with clinical mumps who is contact of a laboratory confirmed case. (i.e. his history of contact makes him equivalent to a laboratory confirmed case).

Rubella

Rubella ('little red', referring to the child with congenital rubella syndrome) has been known since the nineteenth century and was long thought to be a trivial disease, a type of either scarlet fever or measles. (only German doctors disagreed, who persisted that rubella was a disease in its own right—which is why it is sometimes called 'German measles'). Then, in 1941, the Australian ophthalmologist Norman Gregg reported a worrying trend—78 cases of severe cataracts among newborns, all of which could be traced back to rubella infection among the mothers in early pregnancy. Later, other problems such as heart defects, deafness, and mental retardation were noted in such babies.

Agent

The Rubella virus is an RNA virus which belongs to the Togaviridae family. It naturally infects only humans.

Host

Rubella in prevalent in children of developing countries, and in adults of developed countries. Natural infection and vaccine both give lifelong immunity.

Environment

Rubella is a disease of spring and winter, and cyclic trends occur in 4–9 years.

Chain of transmission

Cases (clinical and subclinical) → Droplets, droplet nuclei, transplacental → Susceptible host.

The **communicable period** is 1 week before rash ↔ 1 week after. It is less communicable than measles (because does not cause cough, so the child does not spread the virus with every bout of cough, as with measles).

Clinical course

Incubation period = 2–3 weeks; about 50% infections are subclinical, and among the clinical, most are mild infections.

Prodrome. Mild coryza, sore throat, low grade fever, postaural and post cervical lymphadenopathy

Rash. The rash appears 24 hours after fever, spreads from face towards trunk and limbs. They are maculopapular and discrete from each other.

Complications

1. Arthralgia
2. Encephalitis
3. Thrombocytopenia and bleeding manifestations.

Rubella in pregnancy

Rubella produces congenital defects in 1/3rd of infections. Usually damages occur only if the virus is acquired within 3–4 gestational months (rate of transmission to fetus in 1st trimester in 90%). Diagnosis is confirmed by high IgM in neonate (the mother gives IgG to the neonate, not IgM, thus any IgM found in the blood of a neonate is his own) and persistent high antirubella IgG after 6 months.

Within 1st month	Fetal death
1st month – 1st trimester	Cardiac defects, cataract, deafness
Later	Communication and developmental defects (autism, mental retardation)

Investigations

Virus isolation, and serology (RIA/ELISA), hemagglutination inhibition tests

Control of congenital rubella

For countries where rubella is endemic but vaccination is not widespread, vaccination can be introduced in three steps, one by one.

First, vaccinate women of pubertal period and child bearing age

↓

Then, interrupt transmission by vaccinating all 1–14 years children

↓

Finally, when the above two are done, start routine immunization of all children

Immunization

The rubella vaccine is a live attenuated freeze dried vaccine (RA 27/3 strain cultured in human diploid fibroblasts), 0.5 ml single dose SC; it can be given in any age, but not under 1 year (due to interference by maternal antibodies). It gives long term immunity and second doses are not required. In immunization schedules, it is given at 12–18 months of age in the MMR combination.

Side effects. Mild lymphadenopathy, fever, rash, sore throat, arthralgia, ITP/TTP, peripheral neuropathy

Contraindications. Infants under 1 year, pregnancy, immunocompromised.

Severe acute respiratory syndrome (SARS)

SARS emerged in Southern China in late 2002 and spread in the spring of 2003 to some 30 countries within Asia, Europe and North America. The epidemic finally

came to a stop in July 2003 through strict implementation of quarantine and isolation procedures and international collaboration. At that date, 8,096 cases had been identified worldwide and 774 patients had died, a **9.6% case fatality rate**.

Agent (Fig. 9.59)

The coronavirus reponsible for SARS (SARS-CoV) belongs to a newly identified group in the family Coronaviridae, which are enveloped RNA viruses whose envelope is characterized by crown-like proteinic spikes, and whose genome is an exceptionally long 29727 nucleotides single-stranded positive RNA molecule that encodes 23 different proteins. The virus can survive for 24 hours on a plastic surface at room temperature (and thus it could easily spread via aircraft from one country to other).

Clinical course

Incubation period = 3–5 days. It is a systemic infection accompanied by fever, myalgia, headache, cough and dyspnea, usually with a history of contact (casual/ sexual) with SARS, or travel to a country affected with SARS. The cases rapidly progress to respiratory failure and need ventilatory support.

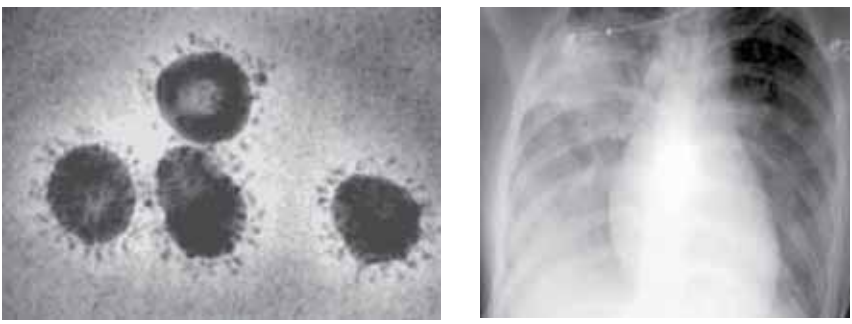

Figure 9.59. The SARS coronavirus (corona means a 'crown'—Can you see the crown around the virus?) and a chest X-ray of a SARS case
[CDC SARS resource: http://www.cdc.gov/niosh/topics/SARS/]

Case definitions

Suspect case	Any of the symptoms developing after 1st November 2002, including a fever of 38 °C (100.4 °F) or higher, and either a history of: • contact (sexual or casual) with someone with a diagnosis of SARS within the last 10 days • travel to any SARS transmissible regions (as of 10 May 2003–Parts of China, Hong Kong, Singapore and the province of Ontario, Canada) or a person who has *died* with undiagnosed acute respiratory illness with similar history
Probable case	Suspect case + positive chest X-ray findings of atypical pneumonia or respiratory distress syndrome/ autopsy findings of respiratory distress syndrome
Laboratory confirmed SARS	Positive laboratory diagnosis of SARS based on one of the approved tests (ELISA, immunofluorescence or PCR)

Chain of transmission (Fig. 9.60)

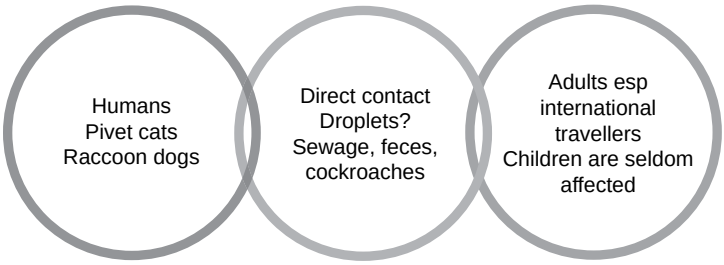

Figure 9.60. Chain of transmission of SARS

Maximum communicability occurs at **10 days after onset of symptoms**.

Reservoirs

Although there is evidence that SARS-CoV emerged from a nonhuman source, no animal reservoir has yet been identified with certainty. Masked palm civet cats and raccoon dogs have been found to be carriers of the virus.[375]

Treatment

Nothing specific. There was initially anecdotal support for steroids and the antiviral drug ribavirin, but no published evidence has supported this therapy.

Control

1. Identification and isolation of patients in negative air pressure (so that no airborne particle gets out of that room) with barrier nursing
2. Protecting health personnel – strict infection control of hospitals, universal precautions
3. Exit screening of international travelers for respiratory illnesses
4. Timely reporting of an outbreak to health authorities and to WHO[376]
5. Half a dozen candidate vaccines for SARS already were in development within 2004. Most of these efforts have however been put on hold, in view of the current elimination of the disease.

Measles

Problem[377]

Measles remains one of the leading causes of death among young children globally, despite the availability of a safe and effective vaccine. An estimated 164 000 people died from measles in 2008 – Mostly children under the age of five. The overwhelming majority (more than 95%) of measles deaths occur in countries with low per capita incomes and weak health infrastructures.

Measles is vaccine preventable. From 2000 to 2008 nearly 700 million children aged 9 months to 14 years who live in high risk countries were vaccinated against the disease. Global measles deaths decreased by 78% during this period.

The WHO/UNICEF global plan focuses on 47 priority countries that account for approximately 98% of global measles deaths. These countries, characterized

by weak health systems and chronically low immunization coverage, are among the world's poorest, and includes India.[378] However, after the introduction of routine vaccination, measles is gradually declining in India.

Agent

The measles virus is a member of geneus Morbillivirus, family Paramyxoviridae. Morbilliviruses, like other paramyxoviruses, are enveloped, single-stranded, negative-sense RNA viruses. The only source is a human case of measles (both clinical and subclinical); *carriers* do not occur.

Host

Measles is chiefly a disease of under five children, peak of incidence occurs between 6 months–3 years (the child is protected by maternal antibodies until 6 months). But as immunization proceeds, nonimmunized adults are now becoming more susceptible to measles (age shifting). The disease is much more severe and often fatal in malnourished children. Interestingly, a previous history of chickenpox reduces the severity of measles, and vice versa.

Environment

The disease flares up in winter and spring.

Chain of transmission (Fig. 9.61)

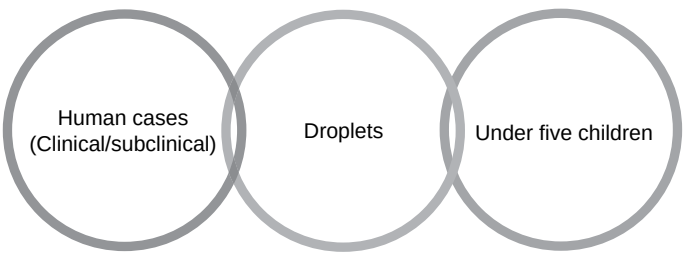

Figure 9.61. Chain of transmission of measles

Communicable period is 4 days before rashing RASH ↔ 5 days after. The virus remains active and contagious in the air or on infected surfaces for upto *two hours*, usually not enough time to infect another host. Thus, it does not spread via droplet nuclei or fomites.

Clinical course

Incubation period is 10 days.

Prodrome

High fever, malaise, running nose, headache, red eyes; **Koplik's spots** seen inside the mouth (small blush white spots opposite the lower first and second molars) are pathognomonic for measles but are not often seen, because they are transient and may disappear within a day of arising.

Eruption

Rashes appear 4 days after fever. They appear first on face (behind ear, forehead, along hairline) and spread downwards. The rashes are generalized, maculopapular, reddish (due to microhemorrhages), The rash is said to "stain", changing color from red to dark brown, before disappearing. It disappears by next 4–5 days.

Complications

1. Pneumonia—Primary (due to the measles virus itself) or secondary infection (due to bacteria); fatality is high in secondary bacterial pneumonia.
2. Otitis media—The measles virus causes secretory (allergic) rather than infective otitis media.
3. Encephalitis—Often resulting in long term neurologic sequelae.
4. Diarrhea.
5. Subacute sclerosing pan encephalitis—A severe debilitating complication of Measles resulting in paralysis, seizures, mental retardation and death within 1–3 years; it is best diagnosed by EEG and antimeasles antibodies in CSF.
6. Malnutrition (specially vitamin A deficiency)—Measles and malnutrition perpetuate each other.

Most measles-related deaths are caused by complications associated with the disease. Complications are more common in children under the age of five, or adults over the age of 20.

Treatment

1. Nutrition—Exclusive breast feeding, additional nutrition during convalescence, plenty of fluids, good complementary feeding
2. Vitamin A supplementation—All children in developing countries diagnosed with measles should receive two doses of vitamin A supplements, given 24 hours apart. This can help prevent eye damage and blindness. Vitamin A supplements have been shown to reduce the number of deaths from measles by 50%.
3. Antimicrobials for secondary infections.

Control

Controlling the reservoir

Case based surveillance—Identify, isolate the case for 7 days after reaching and follow up a case until recovery.

Protecting the host

The target is to develop herd immunity by vaccinating 95% children of each birth cohort and elimination of measles.

Measles vaccine

In 1954, the measles virus was isolated from an 11-years old boy from the United States, David Edmonston, and adapted and propagated on chick embryo tissue culture.[379] This culture provided the live vaccine of measles, known as the Edmonston Zagreb Strain. Nowadays, this strain is cultivated on human diploid cells (HDC) only and not on chicken eggs. The vaccine is a freeze dried preparation

reconstituted in *double distilled pyrogen free water*. **Shelf life** after opening vial is 4 hours, and after reconstitution, 1 hour (any delay may contaminate the vaccine with *Staphylococcus aureus*, a bacteria which shows special preponderance for this vaccine). The reconstituted vaccine must be kep in ice pack while administering (Fig. 9.62).

Figure 9.62. The OPV (left) and measles vaccine (right) kept in ice pack during an immunization session

Dosage. 0.5 ml SC over left anterolateral thigh at 9 months. Another dose may be given during an *epidemic* to all school going children (outbreak response). We stick to 9 months as a trade-off between

1. Maternal antibodies that dwindle in 6–12 months (an earlier than 9 months and the vaccine will probably be nullified by maternal antibodies)
2. High incidence of measles before 12 months of age (any later than 9 months could be too late).

However, the age can be lowered upto 6 months if there is a measles outbreak in the community, or delayed upto 15 months in communities where measles is on the verge of elimination.

Contraindication. Pregnancy, immunocompromised state

Side effects. Fever and rash after 5–10 days after vaccination; if contaminated by *Staphylococcus aureus*, the vaccine may introduce staphylococci in body which may result in upto **Toxic Shock Syndrome**. TSS typically occurs if the same vial has been used for two days. The symptoms are typical, severe watery diarrhea, vomiting and high fever within a few hours of vaccination. Because many infants are vaccinated from the same vial, TSS usually presents as a *point source epidemic*.

The measles vaccine is safe, effective and inexpensive. It costs *less than one US dollar* to immunize a child against measles.

Vaccination schedules

For all children. Routine immunization at 9 months (or any period after 6 months during an epidemic of measles); catch up immunization anytime between 6 months to 14 years.

For contacts of a case. The incubation period of the measles virus is 10 days, and time for inducing immunity by the vaccine virus (i.e. its 'incubation period') is 7 days. Thus, if a person is exposed to a case of measles today, he will have to be vaccinated within 10 – 7 = 3 days, otherwise the wild virus will win the race ahead of the vaccine virus. However, in endemic countries, under five children are not vaccinated if they come in contact of a case (they should

have been vaccinated already by keep up and catch up immunization campaigns). Prophylaxis for contacts is given only in children between 9–12 years. Although **immunoglobulins** are available, they are not much used due to the efficacy of the vaccine. They should be given if there is contraindications to vaccination, in a dose 0.25 ml/kg within 3–4 days of exposure.

Measles elimination

The WHO has targeted measles for 'elimination', and gas defined elimination as **absence of endemic measles in a community for a 12 months period in presence of adequate surveillance**. The World Health Assembly, 2005, has aimed for a 90% reduction in measles mortality from 2000 to 2010.

The Measles Initiative (2001)

Recognizing the potential of measles vaccination to reduce child mortality, and given that measles vaccination coverage can be considered a marker of access to child health services, *routine measles vaccination coverage* has been selected as an indicator of progress towards achieving millenium development goal 4 (reduction of under five mortality).

The measles initiative is a collaborative effort of WHO, UNICEF, the American Red Cross, the CDC, and the United Nations Foundation. The strategy for measles elimination was endorsed at the World Health Assembly 2003.

Strategies

1. **Strong routine immunization** for children by their first birthday—The 'keep up' campaign aims at vaccinating more than 95% of all children of each birth cohort within 1 year of life
2. A '**second opportunity**' for measles immunization through mass vaccination campaigns, to ensure that all children receive at least one dose; usually this is being implemented as '**catch up**' immunization campaigns. Every child between 9 months to 14 years is to be vaccinated regardless of his previous vaccination status. After this initial 'catch up' campaign, a 'follow up' campaign is done every 2–4 years to vaccinate all children born after the catch up campaign.
3. Effective surveillance in all countries to quickly recognize and respond to measles outbreaks.
4. Better treatment of measles cases, to include vitamin A supplements, antibiotics.

The MMR vaccine

Dosage. 2 doses of MMR vaccine, th first at 12–15 months of age and another at 4–6 years of age. However, the chidren can get the second dose at any age which is 28 days later from the first dose.

Contraindications. Allergy to gelatin or neomycin (which are used to make the vaccine), pregnant women, immunocompromised seriously ill children.

Side effects. Fever, rash, swelling of glands, joint pain, seizures, temporarily low platelet count, allergic reactions.

The chickenpox (varicella) vaccine has been combined with MMR and MMRV.

Acute respiratory infections

Acute respiratory infections (ARIs) continue to be the leading cause of acute illnesses worldwide (among all age groups) and the most important cause of infant and child mortality, accounting for about *two million* deaths each year and ranking first among causes of DALYs lost in developing countries. While upper RTIs are a source of mere irritation, lower respiratory tract infection like bronchiolitis and pneumonia are frequently fatal in children. Pneumonia, with a global burden of 5000 childhood deaths everyday, is a tangible threat that is **responsible for about 21% of all deaths in under five children**, leading to estimate that of *every 1000 children born alive, 12–20 die from pneumonia before their fifth birthday*.[380]

However, the most prevalent form of ARI is the common cold, which pertinently holds on to its reputation of being an incurable disease, placing it in the same rank with AIDS, and has enshrined itself in many urban legends. Here's what a patient felt when he was told by his doctor that he had simply caught the 'common' cold.

Go hang yourself, you old MD !
You shall not sneer at me.
...
I contemplate a joy exquisite
I'm not paying you for your visit.
I did not call you to be told
My malady is a common cold.
...
By racking snuffle, snort, and sniff;
By handkerchief after handkerchief;
This cold you wave away as naught
Is the damnedest cold man ever caught!
Give ear, you scientific fossil!
Here is the genuine Cold Colossal;
The Cold of which researchers dream,
The Perfect Cold, the Cold Supreme.
This honored system humbly holds
The Super-cold to end all colds;
The Cold Crusading for Democracy;
The Führer of the Streptococcracy.
...

— Ogden Nash, "Common cold"

Agent

The main etiological agents responsible for ARI in children are *Streptococcus pneumoniae*, *Haemophilus influenzae type B (Hib)*, *Staphylococcus aureus*, respiratory syncytial virus (RSV), measles virus, human parainfluenza viruses Type I, II, and III (PIV-1, PIV-2 and PIV-3), influenza virus, adenovirus, enteroviruses (especially Coxsackievirus) and varicella virus. Bacterial infections tend to be more severe and fatal than viral infections.

Host

Infants, adults in 3rd decade of life (who get infected by their own children) and elderly (>60) are most susceptible. Risk factors in children are—

1. low birth weight
2. inadequate breastfeeding
3. no immunization
4. vitamin A deficiency
5. malnutrition.

Upper respiratory tract infections are much more frequent in children of school age and adults than in infants.

Environment

Overcrowding, air pollution, under ventilated housing, passive smoking, indoor smoke pollution, rapid industrialization.

Chain of transmission (Fig. 9.63)

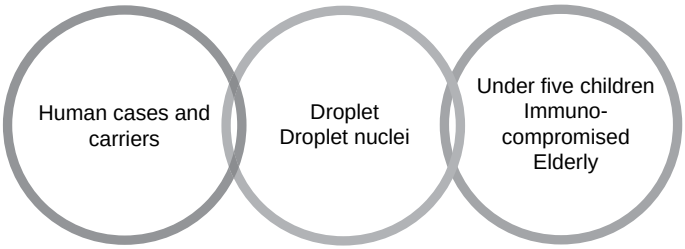

Figure 9.63. Chain of transmission of ARI

Clinical course

Bacterial infections

Streptococcus pneumoniae (pneumococcus) was identified in 30–50% of bacterial pneumonia cases in developing countries in the 1990s, followed by Hib, then *Staphylococcus aureus* and Klebsiella pneumoniae. Other organisms, such as Mycoplasma pneumoniae, *Chlamydia psittaci*, *Pseudomonas aeruginosa* and *Escherichia coli* also can cause pneumonia.

Pneumococcal (community acquired) pneumonia. Virtually every child in the world is colonized with one or more strains of pneumococcus and becomes a nasopharyngeal carrier during his first few years of its life. Many children will go on to develop otitis media, and a few will eventually develop invasive pneumonia or meningitis. The introduction of the **conjugate pneumococcal vaccine** in routine infant immunization should have a major impact on pneumonia.

Mycoplasma (atypical) **pneumonia**. Usually presents as acute bronchitis, pharyngitis and otitis, but 10% of infected children develop pneumonia.

Haemophilus influenzae B. Naturally acquired disease caused by *H. influenzae* seems to occur in humans only. In infants and young children, *H. influenzae* type b (Hib) causes bacteremia, pneumonia, and acute bacterial meningitis. Occasionally,

it causes cellulitis, osteomyelitis, epiglottitis, and joint infections. Unencapsulated *H. influenzae* (non B type) causes ear (otitis media) and eye (conjunctivitis) infections and sinusitis in children, and is associated with pneumonia. Around the world, 2–5% of under five children are nasopharyngeal carriers of the bacteria. The introduction of Hib conjugate vaccines has resulted in a remarkable decline in Hib disease. However, the vaccine is not yet routinely made available to a majority of children worldwide.

Viral infections

Measles (See last section)

Respiratory syncytial virus (RSV). The leading cause of serious respiratory illness in young children is RSV, the agent of **infantile bronchiolitis**.

Parainfluenza viruses. PIV-1, PIV-2 (which cause severe upper respiratory infection and stridor in under five children) and especially PIV-3 (which causes bronchiolitis in infants), are second in incidence immediately after RSV. All children by the age of 2 years have had at least one episode of PIV and/or RSV illness (as per data available in developed countries).

Both RSV and PIV can cause severe disease in the elderly, especially in patients with a chronic respiratory or cardiac condition.

Metapneumoviruses. Human metapneumovirus, a member of the Paramyxoviridae family, is a recognized cause of a large fraction of severe ARIs in infant, elderly and immunocompromised population.[381]

Coronaviruses. Recently discovered coronaviruses HCoV-HKU1 and HCoV-NL63 are significant pathogens that contribute to the hospitalization of children for ARI.[382] Other members of the family are HCoV-229E and HCoV-OC43, agents of the common cold, and the SARS-CoV, which caused SARS (see the section on SARS).

Dual infections with viral and bacterial may be quite common because, as seen in the developed world, epidemics of **RSV** and/or influenza coincide with epidemics of *S pneumoniae* year after year,[383] that administration of antimicrobials often improves a supposedly 'viral' infection, and the conjugate pneumococcal vaccine reduces mortality by 31% in pneumonias caused by virus.

Nosocomial pneumonia

It should be emphasized that nosocomial or hospital acquired pneumonia is a major public health problem: pneumonia is the second most common type of all nosocomial infections, with an associated case fatality rate of 20–50%.

Assessment of a child with ARI

This is the IMCI guideline for MPHWs so they can manage ARI cases with the simplest of algorithms. but this is handy for you as well.

Consciousness

Deterioration of level of consciousness and inability to take food or liquids is indicative of very severe in any age group.

Respiratory rate

Count breathing for full one minute when the child is calm. Expose chest and abdomen and count movements of both abdomen and lower chest wall. The normal respiratory rate is very high in infants, which gradually diminishes with age. Tachypnea is always pathognomonic of lung infection (pneumonia); in the absence of tachypnea we can conclude that the child only has upper respiratory infection (cough and cold).

Age	Margin for tachypnea
< 2 months	≥ 60/min
2–11 months	≥ 50/min
12 months – 5 years	≥ 40/min

Chest in drawing

If the child has chest in drawing, the *lower* chest wall goes *inwards* during inspiration. It is *physiological* in infants < 2m; but if it is seen in any child > 2 months of age, it is a marker of increased respiratory *effort* due to severe pneumonia. The child with such a severe pneumonia might NOT have tachypnea (if the child is exhausted and sinking slowly), and chest in drawing is then the only sign of severe pneumonia.

Stridor

Harsh sounds due to narrowing of larynx and trachea during inspiration. Stridor is often caused by acute epiglottitis (*Haemophilus infleunzae* B) or Parainfluenzavirus 1 and 2.

Wheeze

Fine, almost musical sounds during expiration due to narrowing of bronchioles. Recurrent wheeze or wheeze all over chest is probably not due to ARI but asthma.

Malnutrition

Malnutrition may mask all the above signs. If the child is malnourished, he needs a more thorough examination by a physician.

Cyanosis

A definitive sign of hypoxia, cyanosis can have many causes, including congenital heart disease and poisoning with some dyes, other than ARI.

Treatment

Table 9.28. Treatment chart for acute respiratory infections in children < 2 months

Any of these signs	Means	Treatment
Child stopped feeding well Convulsion Abnormally sleepy child Stridor in a calm child Wheeze Fever or low body temperature	Very severe disease	Refer urgently to a hospital Keep the child warm Give 1st dose of antimicrobial (Cotrimoxazole) Treat fever

Contd...

Contd...

Severe chest in drawing Tachypnea	Severe Pneumonia	Refer to hospital Keep the child warm Give 1st dose of antimicrobial (Cotrimoxazole) Treat fever
Mild chest in drawing (physiological in children of this age group) No tachypnea	No Pneumonia—cough and cold	Home care Breast feeding Clear the nose if secretions obstructs feeding Return quickly if child develops tachypnea, dyspnea, fever or gets sicker.

Table 9.29. Treatment of acute respiratory infections in a child between 2 months–5 years

Any of these signs	Means	Treatment
Not able to drink Convulsion Abnormally sleepy child Stridor in a calm child Malnutrition	Very severe disease	Refer to hospital Give 1st dose of antimicrobial (cotrimoxazole) Treat fever and wheeze If cerebral malaria is suspected, give antimalarial
Chest in drawing	Severe Pneumonia	Refer to hospital 1st dose AMA (cotrimoxazole) Treat fever and wheeze
No chest in drawing and Presence of tachypnea	Pneumonia	Home care Antimicrobial (cotrimoxazole) Treat fever and wheeze Return for reassessment within 2 days ↓ • if child gets Worse (not able to drink, chest in drawing)–Refer to hospital • remains Same–Consider change of antimicrobials and referral • feels Better (slower breathing, drop of fever, eats better)–Finish 5 day course of cotrimoxazole
No chest in drawing and no tachypnea	No Pneumonia—cough and cold	If the cough lasts for > 30 days → refer the child to hospital to exclude tuberculosis Assess ENT infections and treat accordingly

Contd...

Contd...

		Treat fever and wheeze Home care (feed child frequently, greater amount of fluids, soothe throat and relieve cough) Return if • breathing is faster/difficult • feeding problem • child gets sicker

Treatment regimen for children below two months

Very severe disease or severe pneumonia

After the 1st dose cotrimoxazole has been given by the health worker, the child must be hospitalized and treated with a combination of penicillins (Benzylpenicillin or Ampicillin) and gentamicin (50000 IU/kg/dose) or ampicillin (50 mg/kg/dose) and gentamicin (2.5 mg/kg/dose).

Drug	Amount in each dose	Frequency of dosing in infants < 7 days old	Frequency of dosing in infants > 7 days old
Benzylpenicillin	50000 IU/kg IM	12 hourly	6 hourly
Ampicillin	50 mg/kg IV/IM	12 hourly	8 hourly
Gentamicin	2.5 mg/kg IV	12 hourly	8 hourly

The WHO-UNICEF IMCI guidelines recommends health workers to give the 1st dose of ampicillin and gentamicin intramuscularly if they have identified a case to be pneumonia or very severe disease.

No pneumonia, cough and cold

These infants can be treated at home *without* antimicrobials, because most of tense cases are viral infections. The child must be given adequate breastmilk, and the nose of the child needs to be cleared periodically so that it does not obstruct breathing. The mother should be taught to identify dangers signs and report immediately.

Dangers signs in infants

1. Abnormally sleepy child (may caused by hypoxia resulting from pneumonia, or from meningitis)
2. Convulsions
3. Child stopped feeding well
4. Wheezing
5. Hypothermia is as grave in infants as fever.

Treatent for children between 2 months–5 years

Pneumonia

Cotrimoxazole (5:1 combination of sulfamethoxazole and trimethoprim) is the drug of choice because it is safe, effective and can be used safely by health workers at peripheral levels.

Age	Dosage of Cotrimoxazole (100 : 20) oral	Dosage of cotrimoxazole (200: 40) syrup (spoon size = 5 ml)
< 2 months	1 tablet × 2 × 5 days	½ spoon × 2 × 5 days
2–12 months	2 tablet × 2 × 5 days	1 spoon × 2 × 5 days
1–5 years	3 tablet × 2 × 5 days	1½ spoon × 2 × 5 days

After 2 days of therapy with cotrimoxazole, the child must be reassessed (see the chart earier).

Severe pneumonia

The child must be treated indoor with intramuscular antimicrobials (Benzylpenicillin 50000 IU/kg, Ampicillin 50 mg/kg, Chloramphenicol 25 mg/kg; Cloxacillin and Gentamicin in refractory cases). Give the drug 6 hourly for at least 48 hour and then assess the child.

- Improves—Continue same drug, consider switching to oral therapy
- No improvement—Switch to cloxacillin 25 mg/kg divided in 4 doses + gentamicin 2.5 mg/kg divided in 3 doses.

Therapy should continue 3 days after clinical recovery.

Very severe disease

Treat indoor. If some specific diagnosis can be done, go for specific treatment. Otherwise try Chloramphenicol 25 mg/kg 6 hourly IM for at least 48 hours. If the child does not improve, switch to cloxacillin and gentamicin.

Control of ARI

Controlling the reservoir

Standard diagnosis of cases and their treatment at all levels of health care. It is important to educate mothers about correct home care (frequent small feeds, greater fluids including breast milk, clearing nose, soothe throat, relieve cough).

Breaking the chain

Because the viruses and bacteria causing so common in the environment, there is no effective method to shield children from exposure.

Protecting the host

Vaccines

Many of the organisms causing ARI are vaccine preventable (measles, *Streptococcus pneumoniae*, *Hemophilus influenzae B*, influenza, chickenpox, RSV and PIV).

RSV vaccine

A vaccine trial in 1960s using a formalin-inactivated vaccine (FI-RSV), increased disease severity in children who had been vaccinated.[384] At present no vaccine exists, but subunit and live vaccines are in clinical trial.

However, palivizumab, a moderately effective prophylactic drug is available for infants at high risk. Palivizumab is a monoclonal antibody directed against

RSV surface fusion protein. It is given by **monthly injections** for five months. RSV prophylaxis is indicated for infants that are premature or have either cardiac or lung disease, but the cost of limits its use.

Parainfluenza vaccines

Bovine vaccine. Intranasally administered bovine PIV3 (bPIV3) vaccine is moderately effective against **both PIV3 and RSV**.

Cold adapted vaccine. The cold-adapted PIV3 vaccine has been extensively evaluated and is safe and immunogenic in seronegative children with a seroconversion rate of 79%.[385]

Pneumococcal vaccine

Polyvalent polysaccaride vaccine containing 23 capsular types of the bacteria (PPV23). The PPV23 contains 25 micrograms per 0.5 ml, of purified capsular polysaccharide from each of the 23 capsular types of *S. pneumoniae* that together account for most cases. Relatively good responses are elicited in most healthy adults during the two to three weeks following a single dose (PPV23 single dose 0.5 ml IM/SC. The immune response is *unreliable* in children < 2 years, and the immunocompromised. It is recommended for

- Elderly people (> 65), particularly those living in institutions
- Chronic organ failure
- Diabetes or certain immunodeficiencies
- Splenectomized patients
- Those immunocompromised due to organ transplant
- Sickle-cell disease.

The vaccine should not be mixed in the same syringe with other vaccines, but can be given on the same day with other vaccines. Following the vaccination of **pregnant women**, antibodies are transferred both via the placenta and in the breast milk. However, it is not yet documented that maternal vaccination actually protects newborn infants against pneumococcal disease. **Adverse reactions** include some soreness at the site of injection and, more rarely, low-grade fever. *Revaccination* is recommended for those with nephrotic syndrome or asplenia (both are immunosuppressed states).[386]

Pneumococcal conjugate vaccine containing seven selected polysaccharide capsules (PCV7). Recently, 7 selected capsular polysaccharides are bound to a protein carrier, such as a bacterial toxoid. The protein carriers induce a T-cell dependent immune response to the polysaccharides, leading to immunological memory and boosting upon repeated injection. It is **more effective in infants and the immunodeficient**. The vaccines not only to protects against invasive disease, but also to **suppress nasopharyngeal carriage** of the pathogen, i.e. they have a value in community prophylaxis and developing 'herd immunity'. Dosage: PCV7 IM at 6, 10 and 14 weeks + booster between 12–15 months. When first introduced in a community, a 'catch up' dose may be given to all children of 12–24 months and children between 2–5 years who are at high risk of the disease. There are only two disadvantages to the vaccine

- If a child is exposed to a serotype of pneumococcus that is not contained in the vaccine, he/she is not afforded any protection.

- Due to widespread vaccination, the strains covered in the vaccine will cease to cause infection and give way to other serotypes that are *not* covered.

Research is under way to develop of vaccine which does not contain the capsular polysaccharide (which varies between serotypes) but some other antigen which is common to all pneumococci.

Hemophilus influenzae B vaccine

Before 1985, Haemophilus influenzae type b (Hib) was the most common cause of bacterial meningitis in under 5 children, and a frequent cause of ARI. The cumulative risk for Hib invasive disease before the age of 5 was one in 200 children, similar to the risk for poliomyelitis during the 1950s.

In 1985, the first Hib polysaccharide vaccines were licensed for use in the United States. These vaccines contained purified polyribosylribitol phosphate (PRP) capsular material from the type b bacteria. However, it was ineffective in children less than 18 months of age (because it is only a polysaccharide, not protein, the immunity is entirely humoral and there is no T-cell involvement and no immune memory). *Conjugation* of the PRP polysaccharide with protein carriers confers T-cell-dependent characteristics to the vaccine and enhances the efficacy so that it could be used in infants. In 1989, the first Hib conjugate vaccines were licensed for use among children 15 months of age or older. The incidence of Hib is on the decline since in developed countries.

Dosage. Hib conjugate vaccine IM at 6, 10, 14 weeks + a booster at 12–15 months. It is combined with DPT vaccine for administrative convenience.

More than 90% of infants obtain long term immunity with 2–3 doses of the vaccine. The vaccine is not recommended for children ≥ 2 years of age.

Other measures

Vitamin A prophylaxis, breast feeding, surveillance.

Tuberculosis

Tuberculosis, the 'white plague' of human civilization, the harbinger of death to many (but none other more famous than John Keats) is caused by mycobacteria, usually Mycobacterium tuberculosis in humans. Most infections in humans occur in childhood result in an asymptomatic, latent infection in lungs (Ghon's focus), some of which progresses to clinical disease (Fig. 9.64).

Problem

Epidemiological indices

1. **Prevalence of infection** is assumed to be the prevalence of tuberculin test positivity; however, it is error prone, because many other factors could cause tuberculin positivity (BCG vaccination, other species of mycobacteria)
2. **Prevalence of infectious cases** = prevalence of sputum +ve tuberculosis in community
3. **Incidence of infection** = incidence of new tuberculin +ve (Tuberculin conversion index) per 100000 population; this is the indicator for 'attacking force' of tuberculosis in a community, i.e. it is the *absolute risk* of being infected

Figure 9.64. This 1919 poster from the Red Cross shows the protector of the family pushing the dreaded visitor out the door. The shrouded image of tuberculosis is comparable to the depiction of white plague [US National Library of Medicine]

by tuberculosis if you live in that community; each 1% rise in incidence of infection equals a risk of 50 new sputum +ve cases per 100000 population

4. **Incidence of infectious cases** = incidence of new sputum +ve tuberculosis in community
5. **Tuberculosis specific mortality rate** = (number of death due to tuberculosis in a year/mid year population) × 1000
6. **Proportional mortality due to tuberculosis** = number of deaths due to tuberculosis/total deaths in a year.

World[387]

A third of the world population is infected with tuberculosis.[388] The highest incidence of tuberculosis occurs in Sub Saharan Africa, followed by India and other countries of South-east Asia.

- Prevalence of disease = 13.7 million (2007); prevalence is slightly on the decline (due to rising population, not reduction of cases).
- Annual incidence of disease = 9.27 million (2007), i.e. 139/10000 population
- Annual incidence of infectious (sputum +ve) disease = 4.1 million (2007) or 66/10000 population.
- Annual deaths = 1.32 million HIV-negative people with tuberculosis (19.7 per 100000 population) and 456000 HIV-positive people with tuberculosis died in 2007.
- 4.9% of all new cases and relapse/defaulters in 2007 were *multi drug resistant*, and most cases of MDR tuberculosis were reported from India.

India[389]

Tuberculosis is the Largest Public Health Problem in India, accounting for one fifth of the global incidence.

- Prevalence of infection = 40% of Indian population
- Incidence of disease = 1.9 million new cases per year (5000 people per day develop some form of clinical tuberculosis)
- Incidence of infectious (sputum +ve) disease = 0.8 million per year
- Incidence of infection (annual risk of tuberculosis infection–ARTI) = **1.5%**, and once infected there is a 10% chance of developing disease. Because about half of diseased cases are sputum +ve pulmonary tuberculosis, an ARTI = 1.5 / 100 population translates to 1.5 × 100000/100 × 10% × 50% = 75 new smear positive pulmonary TB cases are *expected* per 100,000 population annually.
- Tuberculosis specific mortality = 2 deaths every 3 minutes, 5000 deaths per day and 28 deaths/100000 population per year; proportional mortality from tuberculosis exceeds the combined mortality from all other infectious disease, and tuberculosis is the leading cause of death in women, more than the combined mortality of all causes of maternal death
- 3% of new cases and 12% of retreatment cases have **multidrug resistant (MDR)** tuberculosis
- There have been isolated reports of tuberculosis resistant to most second line drugs (**extensively drug resistant or XDR**)
- Socioeconomic impact—Because tuberculosis predominantly affects working age men, it causes on an average, 3–4 months of loss of workdays; more than 300000 children have to leave school to earn money because their parents have tuberculosis, and > 100000 women are rejected by family because she has tuberculosis. An estimated $3 billion is lost to society indirectly and $300 billion directly because of tuberculosis.[390]

TB + HIV

In 2007, 5% of all tuberculosis patients in the world were HIV positive, and 14.8% of new tuberculosis cases in 2007 were also HIV +ve. Tuberculosis contributes 23% of proportional mortality in HIV positive people.

Agent

Except *Mycobacterium tuberculosis* a number of 'atypical' baceria like Mycobacterium marinum, *Mycobacterium kansasii*, *Mycobacterium avium-intracellulare* complex (MAC) have been seen to cause tuberculosis.

All the mycobacteria are acid fast, killed by heating in

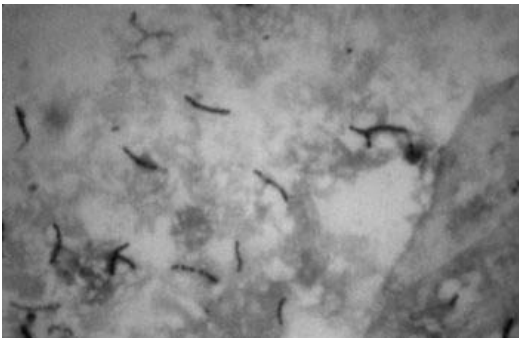

Figure 9.65. *Mycobacterium tuberculosis* (the red rods) in Ziehl-Neelsen stain of sputum

60°C for 15–20 mins, survive 24–48 hours in sputum, *6–8 hours in droplets* (thus they are transmitted through droplet nuclei).

Host

All age groups are affected, especially 15–44 years; men are more affected than women (probably due to increased exposure rather than susceptibility). Tuberculosis is a very common infection among medical students (in their clinical years) and health care professionals. Neither BCG nor an episode of disease gives protection. The BCG vaccine cannot itself inhibit a clinical attack but only reduces complications and severity.

Environment

Overcrowding, large families, lack of education and early marriage are consistently associated with spread of the disease. But tuberculosis is not confined to lower socioeconomic groups.

Chain of transmission (Fig. 9.66)

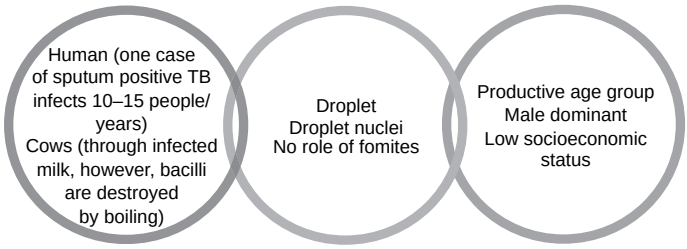

Figure 9.66. Chain of transmission of tuberculosis

The disease is *communicable* as long as the patient is discharging bacilli in sputum.

Clinical course

Incubation period = variable (according to host immunity). In the pulmonary form of the disease, there is persistent cough with sputum > 3 weeks, chest pain, evening rise of temperature, anorexia and weight loss, associated hemoptysis. In other forms, there may be involvement of lymph nodes, joints, intestine, skin and meninges.

Control

Tuberculosis is said to be controlled if prevalence of natural infection in 0–14 year age group is 1%.

Controlling the reservoir

Early diagnosis and treatment of cases (see RNTCP for details); isolation of cases; disinfection of sputum, slides, cups, broomsticks (used for making slides) by 5% NaOCl/ phenol.

Breaking the chain

Improving room ventilation, personal hygiene and healthful habits (covering nose while coughing or sneezing); health care workers should maintain universal precautions while dealing with tuberculosis patients.

Protecting the host

Vaccination

Since tuberculosis has no extra human reservoirs, eradication is theoretically possible, but is being hindered only due to the want of an effective vaccine.[391]

BCG

BCG is a live attenuated bovine tuberculosis bacteria, 'Danish 1331' strain. This was the first vaccine for tuberculosis and developed at the Pasteur Institute in France between 1905 and 1921.[392] However, mass vaccination with BCG did not start until after World War II.

Efficacy. The BCG does not give effective protection against pulmonary tuberculosis (efficacy varies between 0–80%, and protection lasts for 15–20 years), but gives 80% protection against complicated forms of tuberculosis (i.e. meningitis).[393]

Stability. Freeze dried vaccine is stable for 1 year under 2–8°C; it should be protected from light.

Dosage. BCG 0.1 ml intradermally at birth (or at 6 weeks, if not given at birth) just above the insertion of left deltoid, reconstituted in normal saline, *within 4 hours*/of reconstitution. A satisfactory injection should produce a wheal of 5 mm diameter (Fig. 9.67).

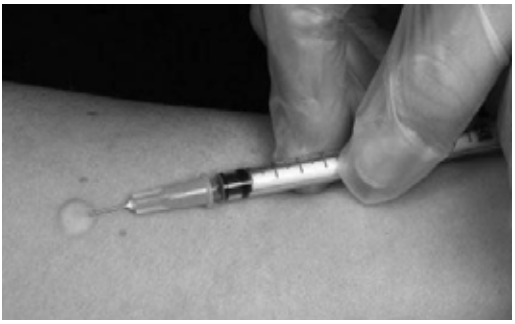

Figure 9.67. Note the tense, pale wheal which is the sign of successful intradermal injection; the syringe is at 5–15° angle with the skin [CDC Public Health Image Library]

Normal reactions. A papule at the site of vaccination → crusted ulcer which heals in 6–12 weeks leaving a permanent scar

>Mantoux positivity. Achieved after 8 weeks of vaccination

Side effects. Ulcer, suppurative lymphadenitis, osteomyelitis, disseminated BCG infection; *local abscesses* are best drained and treated with INH powder

Contraindications. Eczema, infective dermatosis, any kind of immunosuppression (including HIV infection); in areas where mycobacteria are less prevalent (i.e.

developed countries), BCG is not given to the entire population but only to high risk groups.

Newer vaccines

1. A recombinant tuberculosis vaccine rBCG30, entered clinical trials in the United States in 2004[394]
2. The MVA85A vaccine, a genetically modified vaccinia virus, is currently in phase II trials in South Africa by a group led by Oxford University.[395]

Chemoprophylaxis

Children < 6 years in contact with a smear +ve case are given isoniazid 5 mg/kg × 3 months → Tuberculin test → If –ve, stop isoniazid and give BCG vaccine; if +ve continue isoniazid for 3 more months.

Because of cost and variable efficacy, chemoprophylaxis is not recommended for tuberculosis control.

WHO Stop tuberculosis initiative, 2006

Vision. A world free of tuberculosis
Goal. To dramatically reduce the global burden of tuberculosis by 2015 in line with millennium development goals.

Targets

1. By 2005—At least 70% of people with sputum smear-positive TB will be diagnosed (i.e. under the DOTS strategy), and at least 85% successfully treated. The targets of a case detection rate of at least 70% and a treatment success rate of at least 85% were first set by the World Health Assembly of WHO in 1991.
2. By 2015—The global burden of TB (per capita prevalence and death rates) will be reduced by 50% relative to 1990 levels.
3. By 2050—The global incidence of active TB will be less than 1 case per million population per year.

Components

- **Pursuing high quality DOTS expansion and enhancement** by improving case finding and cure.
- **Address TB + HIV, MDR-TB and other challenges** through DOTS plus (see later), interlinking tuberculosis and HIV control programs.
- Strengthen health systems.
- **Engaging all care providers**, including private health care, to improve case finding and cure.
- **Empowering patients and communities** so that they understand tuberculosis and demand care for themselves.
- Enabling and promoting research for development of new drugs, vaccines and diagnoses.

Revised National Tuberculosis Control Program, 1997

The National Tuberculosis Program (NTP), which was set up in 1962, was *reviewed* in 1992 because of:

- Managerial weakness
- Low priority to tuberculosis
- Inadequate funding
- Over reliance on X-ray as diagnostic test
- Nonstandard drug regimens
- Low percentage of treatment completion
- Lack of systematic monitoring.

Full fledged RNTCP started in 1997, after pilot programs from 1993. In 2006, RNTCP has covered the entire country.

Goals

To Control tuberculosis – i.e. cut transmission of disease until it ceases to be a public health problem (i.e. reduce mortality and morbidity)

Objectives

1. To *achieve and maintain* a **cure rate of 85% in new sputum +ve cases** (not only does cure makes the patient happy, but it also makes him noninfective)
2. To achieve and maintain **detection of 70% of new sputum +ve cases** in population.

Structure of RNTCP

NATIONAL
Ministry of health, central tuberculosis division
Deputy Director General (Tuberculosis) – director of RNTCP
National level laboratories National Tuberculosis Institute (Bangalore), Tuberculosis Research Center (Chennai) and Lala Ram Swarup Institute of Tuberculosis (New Delhi)
Among these, the Tuberculosis Reserach Center has been designated as a 'supra national' reference laboratory and WHO collaborative center for research and training

↓

STATE
State Tuberculosis cell, headed by State Tuberculosis Officer (STO)
State Tuberculosis Training and Demonstration Center, headed by a director
State Drug Store
Intermediate reference laboratories: Medical college/public health laboratories of the state

↓

DISTRICT
District Tuberculosis Center (the nodal pont for tuberculosis control), headed by District Tuberculosis Officer (DTO)

↓

SUBDISTRICT
One **Tuberculosis Unit** is allotted per 500000 population; it consists of
- One Medical Officer for Tuberculosis control
- One Senior Tuberculosis Treatment Supervisor (STS)
- One Senior Tuberculosis Laboratory Supervisor (STLS)
- Five Designated Microscopy Center, one per 100000 population

↓

PERIPHERAL
Peripheral health staff
DOT center and DOT agent (treatment provider)

Strategies

1. Political commitment to increase human and financial resources and make tuberculosis control a nation-wide activity.
2. Access to quality-assured tuberculosis sputum microscopy for case detection
3. Standardized short-course chemotherapy to all cases of tuberculosis under Directly Observed Treatment (DOT); the DOT is nothing but a strategy to increase patient compliance by making the patient take the drugs while a health worker/ supervisor watches him.
4. Uninterrupted supply of quality-assured drugs.
5. Recording and reporting system enabling outcome assessment of each and every patient and assessment of the overall program performance.

Political and administrative commitment

Tuberculosis is a developmental indicator, and governments need to encourage RNTCP to ensure funds, human resources and to implement the other four components.

Good quality diagnosis

The RNTCP does not go for searching cases but examines those patients who present in a health facility with **cough > 3 weeks**, continuous fever, chest pain or hemoptysis.

Sputum smear microscopy

3 sputum samples : SPOT (at the time of first reporting) → Morning After (the patient coughs out first sputum sample after waking up next morning and brings it to microscopy center) → SPOT (when he comes, a third sample of sputum is collected) are examined for acid fast bacilli. Note that sputum needs to be collected, not saliva; the patient has to inhale deeply and bring out the secretion from the trachea, not the pharynx.

Sputum examination is

1. Simple (no great training needed to stain and observe AFB).
2. Inexpensive.
3. Feasible at most peripheral health centers.
4. Minimum inter/intrareader variation (usually the shiny red AFBs are not missed over the bluish background of sputum).
5. Prioritizes efforts to detect sputum +ve (infectious) cases.
6. An objective method to follow up of a case; 'cure' of a sputum +ve case is only possible when he becomes sputum -ve.

A **designated microscopy center** (DMC) is set up for each 100000 population (and each 50000 population in hilly and tribal areas; compare this with the distribution of PHCs and subcenters, which are also much more frequent in hilly areas than plains). A DMC is usually installed in a separate room of existing PHCs. A DMC should have[396]

1. RNTCP trained laboratory technician.
2. Binocular microscope (not the compound microscopes in your microbiology laboratory to see a binocular microscope, visit the tuberculosis unit of your medical college).

3. Physical infrastructure in laboratory should meet RNTCP guidelines.
4. The outdoor of that particular health facility should be visited by at least 60-100 adults per day.
5. At least 3–5 sputum smears should be examined per day.

Sputum collection centers. To improve access to diagnostic services in areas such as the tribal, hilly, difficult to reach areas of the country *sputum collection centers* may be established. Private practitioners in urban and rural areas can also collect sputum samples and send to the nearest DMC.

X-ray

Only a supportive diagnosis, *sensitive but not specific* (no finding is absolutely pathognomonic of Tuberculosis); variation between and within observers is great.

Culture

Sensitive and specific for tuberculosis, but very expensive, requires a full fledged microbiology lab and gives results only after at least 6 weeks.

Tuberculin test

Tuberculin test is an intradermal hypersensitivity test, the *only test* for latent tuberculosis infection.

Tuberculin. Tuberculin is a purified protein derivative (PPD) of *Mycobacterium tuberculosis*. **One tuberculin unit** = 0.00002 mg of PPD, which is contained in 0.1 ml solution of PPD.

There are three kinds of tests that could be done with tuberculin.

Mantoux test. Inject 0.1 ml PPD (1 tuberculin unit) intradermally in flexor surface of forearm and observe the region after 72 hours.

Diameter of erythema	Inference
< 6 mm	Negative (susceptible)—The subject has never been exposed to tuberculosis bacilli or BCG
6–9 mm	Doubtful (repeat test)
10–15 mm	Positive (immune) has been exposed to tuberculosis bacilli or BCG A positibe test indicates *active disease* in children below 2 years
> 15 mm	Hyperreactive—The subject has or will develop active tuberculosis disease

The test is very nonspecific, and only of prognostic value in children

1. *False +ve* results may be produced by infection with atypical mycobacteria; *repeated tuberculin testing* may cause a 'booster effect' and turn and initially tuberculin –ve subject to become hyperreactive.
2. False –ve results may be caused by immunosuppression by any cause—Measles, chickenpox, malnutrition, Hodgkin's disease, steroids, HIV, severe bacterial infection (including tuberculosis itself).

Like the tuberculosis bacilli, BCG also causes tuberculin positivity. Thus, in countries where BCG vaccination is established, tuberculin test has lost its value in detecting tuberculosis infections.

Tuberculosis remains undiagnosed if:

1. Suspect cases are not identified
2. Sputum is not sent for examination or stored for too long before examination
3. Sputum microscopy is of bad quality
4. There is a clerical error in labeling sputum samples or recording results.

To ensure quality control in diagnosis:

1. Rightly collect, transport and examine sputum preferably in the same day of collection, at most within 7 days of collection (otherwise you may obtain a false negative result).
2. Accurately record and grade results; there is a grading system of smears depending on number of bacilli seen in high power field, however, the sight of a single bacilli makes the smear +ve for tuberculosis.

Table 9.30. Grading of sputum smears, observed in oil immersion field (1000x magnification)

Observation	Result	Grade	Number of fields to be examined
> 10 AFB per oil immersion field	Positive	3+	20
1–10 AFB per oil immersion field	Positive	2+	50
10–99 AFB per 100 oil immersion fields	Positive	1+	100
1–9 AFB per 100 oil immersion fields	Positive	Scanty*	100
No AFB in 100 oil immersion fields	Negative	Negative	100

3. **Internal quality assurance** includes all means by which the laboratory personnel control the processes including checking of instrument, new lots of staining solutions, smear preparation, grading, etc.
4. **External quality assurance**: The RNTCP has arranged for on-site evaluation in each microscopy center by senior tuberculosis laboratory superviser (STLS, a member of the tuberculosis unit) who will regularly visit the microscopy centers and perform *cross checking* of all +ve smears and 10% of –ve smears. A few random slides are chosen from each DMC and rechecked at the ditrict tuberculosis center, in a double blind manner by the STLS (i.e. the STLS does not know which slides have been reported +ve by the DMC and which –ve). Thus the RNTCP maintains high fidelity of diagnosis (Fig. 9.68).

Special cases

1. Extrapulmonary tuberculosis and contacts of sputum +ve cases should undergo sputum smear examination irrespective of their symptoms.
2. Pediatric tuberculosis is best diagnosed by symptoms, history of *contact* with a adult tuberculosis patient, tuberculin test and chest X-ray; sputum examination is not always possible because children swallow their sputum; even if done, they are usually sputum -ve. Children showing neurological symptoms like irritability, refusal to feed, headache, vomiting or altered sensorium may be suspected to have **tubercular meningitis**.

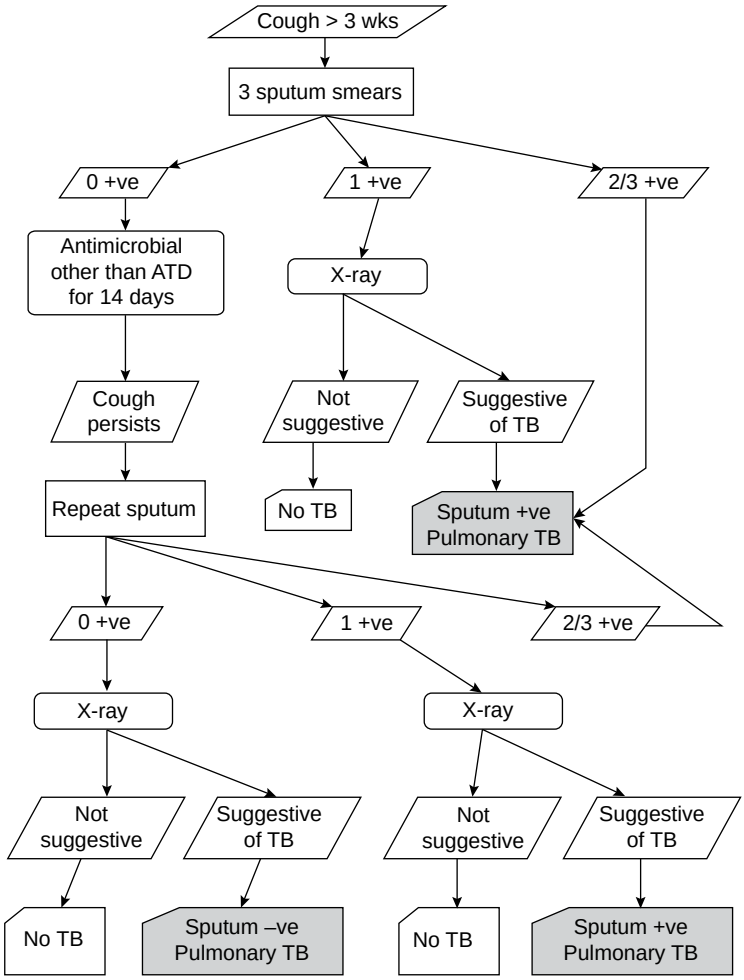

Figure 9.68. Diagnosis of tuberculosis

Case definitions

The major change in RNTCP has been treatment based on *case definitions*, with an aim to identify priority cases (sputum +ve cases), to make cost effective use of resources, and to minimize side effects of therapy.

Pulmonary tuberculosis, smear +ve

A patient with

- Two initial sputum smears +ve for AFB

  Or
- One initial sputum smear +ve for AFB and radiologic evidence of tuberculosis/culture positive for *Mycobacterium tuberculosis*

(See the diagnostic chart above).

Pulmonary tuberculosis, smear –ve

A patient with

- All three initial sputum smears –ve for AFB
- After 14 day course of antimicrobials (not antitubercular drugs) his sputum remains smear +ve, but
- Shows radiographic findings/culture that are consistent with tuberculosis.

Extrapulmonary tuberculosis

Tuberculosis in any organ other than lungs, diagnosed by culture, radiology or histologic examination. *Pleura* is considered separate from lungs and *pleural effusion* is extrapulmonary. A patient with both pulmonary and extrapulmonary involvement should be classified as *pulmonary* tuberculosis in the RNTCP.

Types of cases

New

A tuberculosis patient

- Who has never been treated for tuberculosis
  Or
- Has never taken ATD for more than a month.

Relapse

A tuberculosis patient who was declared *cured or treatment completed* (see below for definition of 'cured' and 'treatment completed'), but who *reports back* as a sputum +ve case

Transferred in

A tuberculosis patient transferred into a tuberculosis unit from another unit.

Treatment after default

A tuberculosis patient who

1. Received ATD for *one month or more* from any source
2. Has not taken ATD for consecutive 2 months or more
3. Now reports back to health facility
4. Is now sputum +ve.

Failure

A tuberculosis patient

- Who is sputum +ve after 5 months or more of starting treatment with ATD
  Or
- A patient who was started on category III (i.e. was intially sputum –ve), but has become sputum +ve anytime during course of treatment.

Chronic case

A tuberculosis patient who remains sputum +ve after completing a retreatment regimen (i.e. being treated with two completed courses of ATD).

Others

Some cases will have to be classified outside of these types. Mention reason of thus classifying.

Treatment outcomes

Cured

A patient who

- Initially sputum +ve
- Had completed treatment
- Undergone two sputum examinations, one of them at the end of treatment
- Sputum is –ve on both occassions.

'Cured' is the expected outcome of category I and II patients who are sputum +ve initially.

Treatment completed

There are three scenarios when a patient can only have an outcome 'treatment completed', not cured.

1. An initial sputum +ve patient → completed intensive phase of therapy → sputum –ve at end of intensive phase → completed full course of treatment → *no negative smears* at end of treatment (which means that either the patient was lost or the sputum could not be done for some reason).
2. An initial sputum –ve patient of pulmonary tuberculosis who does not become sputum +ve anytime during the course of treatment
3. A case of extrapulmonary tuberculosis who does not become sputum +ve anytime during the course of treatment.

Died

A patient who has died during course of treatment irrespective of cause of death (i.e. accidental deaths and deaths from other illnesses are also included in this category).

Failure

(See earlier in 'type of cases').

Defaulted

A patient who has

- Not taken ATD for 2 months or more
- After taking ATD for 1 month or more.

Transferred out

A patient transferred to another tuberculosis unit.

Chronic case

A patient who started on category II and remains +ve at the end of treatment (these patients high chance of becoming MDR).

Categories

Category I

1. New sputum +ve pulmonary tuberculosis
2. Sputum –ve but *seriously ill* pulmonary tuberculosis (miliary tuberculosis, extensive parenchymal infiltration, tuberculosis + HIV, cavitary lung lesions, all forms of pediatric tuberculosis except primary complex)
3. Extrapulmonary, *seriously ill* (meningitis, pericarditis, peritonitis, extensive bilateral pleural effusion, spinal, intestinal, urogenital tuberculosis, tuberculosis + HIV, all forms of pediatric extrapulmonary tuberculosis except lymph node tuberculosis and mild pleural effusion).

Category II

Relapse, failure, default, others (everybody who has to be treated a second time).

Category III

1. Sputum –ve pulmonary tuberculosis *not seriously ill*
2. Extrapulmonary tuberculosis *not seriously ill*.

Uninterrupted supply of good quality drugs

Drugs are supplied in patient-wise boxes (PWB) containing the full course of treatment, and packaged in blister packs. Giving all the drugs for the whole course of treatment ensures that patient will adhere to the regimen and complete the course, and will never have the excuse 'I ran out of drugs'. The PWB have a color code indicating the category of patient

- Red—category I
- Blue—category II
- Green—category III

Short course chemotherapy given in a program of Directly Observed Treatment (DOT).

Initiating treatment

The patient must be counseled and motivated to adhere to treatment as recommended. He also told about tuberculosis, its mode of transmission, precautions to be taken to prevent the spread, importance of directly observed treatment and its duration, and the need for *prompt evaluation of children under six years* or household contacts with cough of any duration.

Treatment is provided by direct supervision of a DOT provider, who can be a health worker, a family member, a cured person or any eminent person in the locality (i.e. teachers). DOT providers are employed under Rs 150 per month honorarium. The RNTCP has adopted the DOT principle for several reasons.

1. Convenient for patient
2. Bonds the patient and the DOT provider
3. Ensures compliance
4. Through good compliance, prevents emergence of drug resistance
5. Gives high cure rates.

Phases of therapy

1. **Intensive phase** with four or five drugs to make the patient sputum –ve as quickly as possible, so that he stops being infective.
2. **Continuation phase** with two or three drugs to clear disease from the patient.

Dosage

Table 9.31. Schedule for chemotherapy in tuberculosis: Note that 2 (HRZES)3 means "H (isoniazid), R (rifampicin), Z (pyrazinamide) and E (ethambutol) and S (streptomycin) 3 times weekly for 2 months"

Category I	$2\ (HRZE)_3 + 4\ (HR)3$
Category II	$2\ (HRZES)_3 + 1\ (HRZE)_3 + 5\ (HRE)_3$
Category III	$2\ (HRZ)_3 + 4\ (HR)_3$

Adults. For adults, drugs are given in recommended dose (Isoniazid 600 mg, rifampicin 450 mg, pyrazinamide 1500 mg, ethambutol 1200 mg, streptomycin 750 mg per dose) irrespective of body weight. However

- *For patients weighing more than 60 kilograms* an additional capsule of rifampicin 150 mg will be added to the treatment regimen
- Patients > than 50 years old receive streptomycin 500 mg only.

Children. For children and anybody weighing < 30 kg, the drugs will be given according to body weight (isoniazid 10–15 mg/kg, rifampicin 10 mg/kg, pyrazinamide 35 mg/kg, ethambutol 30 mg/kg, streptomycin 15 mg/kg). In RNTCP phase II, drugs for children will be supplied in prepackaged patientwise boxes (like adults); there will be one set of boxes for children weghing between 6–10 kg, and another for 11–17 kg.

Key principles

1. Protocol based treatment (eliminates the preference of individual physicians regarding treatment of tuberculosis and standardizes a common drug protocol, thus preventing emergence of drug resistance)
2. Intermittent dosing (thrice weekly)
3. Every dose of intensive phase and **first weekly dose** of continuation phase must be **directly observed** (the patient will bring empty packets at the beginning of next week).

Advantages of intermittent regimen over daily regimen

1. same efficacy
2. facilitates more regular observation
3. less side effects
4. reduction of total drug consumed
5. less expensive
6. patients are required to visit less frequently.

Adverse effects of drugs

Symptom	Drug	Action to be taken
Gastrointestinal upset	Any oral medication	Reassure patient; give drugs with less water and over a longer period of time, ever in empty stomach
Peripheral neuropathy (itching, tingling numbness)	Isoniazid (H)	Supplement pyridoxine 100 mg/day
Gout	Pyrazinamide (Z)	Evaluate cinically, reconsider pyrazinamide
Optic neuritis (diminution of vision)	Ethambutol	STOP ethambutol, refer patient for evaluation
Ototoxicity (tinnitus, deafness, vertigo, ataxia)	Streptomycin	STOP streptomycin, refer patient for evaluation
Jaundice	Isoniazid, rifampicin, pyrazinamide	STOP all drugs, refer patient for evaluation

NonDOTS treatment

In areas covered by RNTCP (that includes the whole country now) at least 95% of patients would be getting DOTS regimen. *Unobserved rifampicin should not be given under any circumstance*. However, RNTCP nonDOTS treatment[397] (self administered nonrifampicin containing regimen) may be needed in exceptionally few cases (e.g. adverse reaction to rifampicin and pyrazinamide). Every effort should be made to minimize NonDOTS.

NonDOTS regimen 1 (ND1)	Includes the same group of patients as Category I of DOTS	$2\,(HSE)_3 + 10\,(HE)_3$
NonDOTS regimen 2 (ND2)	Includes the same group of patients as Category III of DOTS	$12\,(HE)_3$

Special situations

1. **Hospitalization**—General policy of RNTCP is to treat patients at home. However, those admitted in hospitals are also to be treated with RNTCP regimens. On discharge, patients may be given a maximum of three doses (1 week drug supply) and told to visit their nearest DOT center.
2. **Pregnancy**—Streptomycin should not be given, other drugs used in RNTCP are safe; breastfeeding should continue regardless of the mother's tuberculosis status, but advise the mother to cover her mouth, if she is smear-positive, while breastfeeding. **Chemoprophylaxis** for the baby is recommended if mother is sputum smear-positive (see earlier for regimen of chemoprophylaxis).
3. **Liver disease**—Begin with ofloxacin, streptomycin and ethambutol; wiith careful monitoring of liver function, try to introduce rifampicin first, then isoniazid, but never pyrazinamide
4. **Renal failure**—Only streptomycin and ethambutol should be closely monitored with reduced dosage
5. **Oral contraceptive pills**—Rifampicin induces drug metabolizing enzymes and may cause OCP failure; switch to another contraceptive
6. In **tubercular meningitis**, the four drugs used during the intensive phase should be HRZS (instead of HRZE) due to poor penetration of blood brain-

barrier by ethambutol, and that ethambutol causes optic neuropathy which may be confused with that caused by tuberculosis itself; in meningitis and spinal tuberculosis with neurological complications *continuation phase of treatment should be given for 6–7 months*, thus extending the total duration of treatment to 8–9 months. In addition, steroids should be used initially in hospitalized cases of tubercular meningitis or pericarditis.

Systematic monitoring and accountability

Monitoring of patients is built into RNTCP. It has two components – follow up sputum examination and revised recording and reporting system.

Follow up sputum examination

The best method for diagnosis (sputum examination) is also the best method of follow up, as only sputum examination will indicate when a patient becomes non-infective.

New sputum +ve patients (Fig. 9.69)

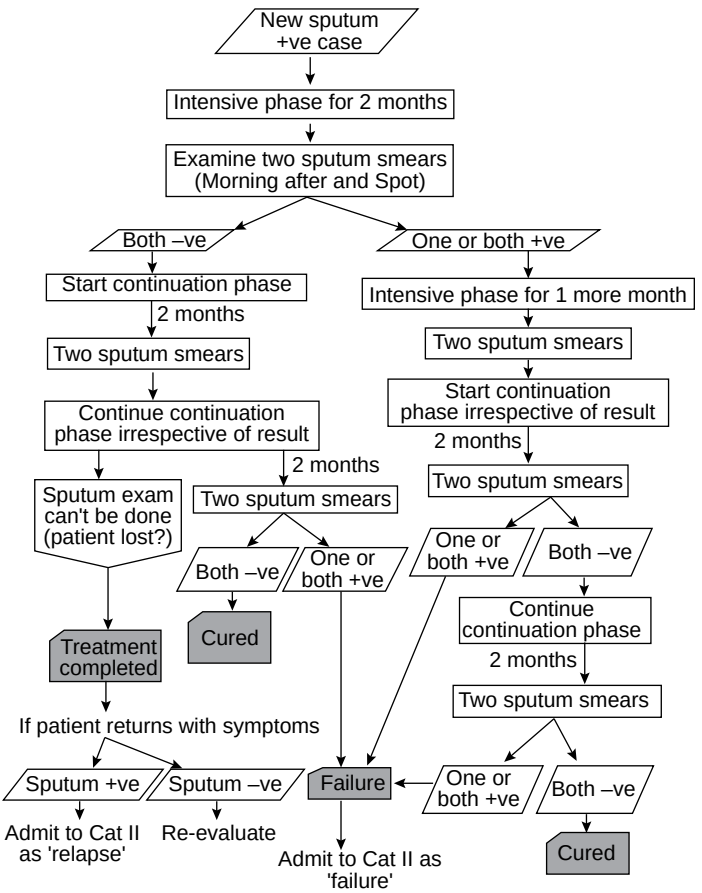

Figure 9.69. Follow up sputum examination of new sputum +ve patients

Sputum negative patients (Fig. 9.70)

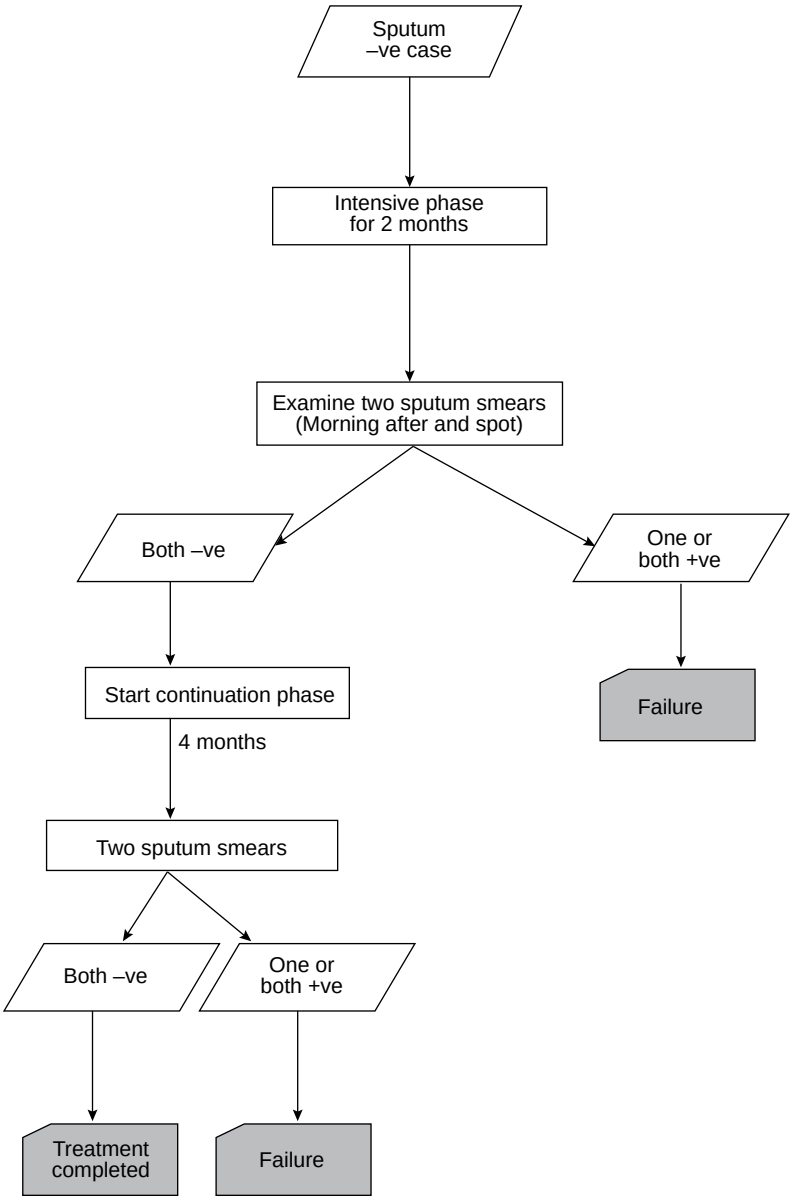

Figure 9.70. Follow up sputum examination of sputum -ve patients

The expected prognosis of category III is *treatment completed*, and any sputum positivity during the course of category III is failure.

Retreatment cases (category II) (Fig. 9.71)

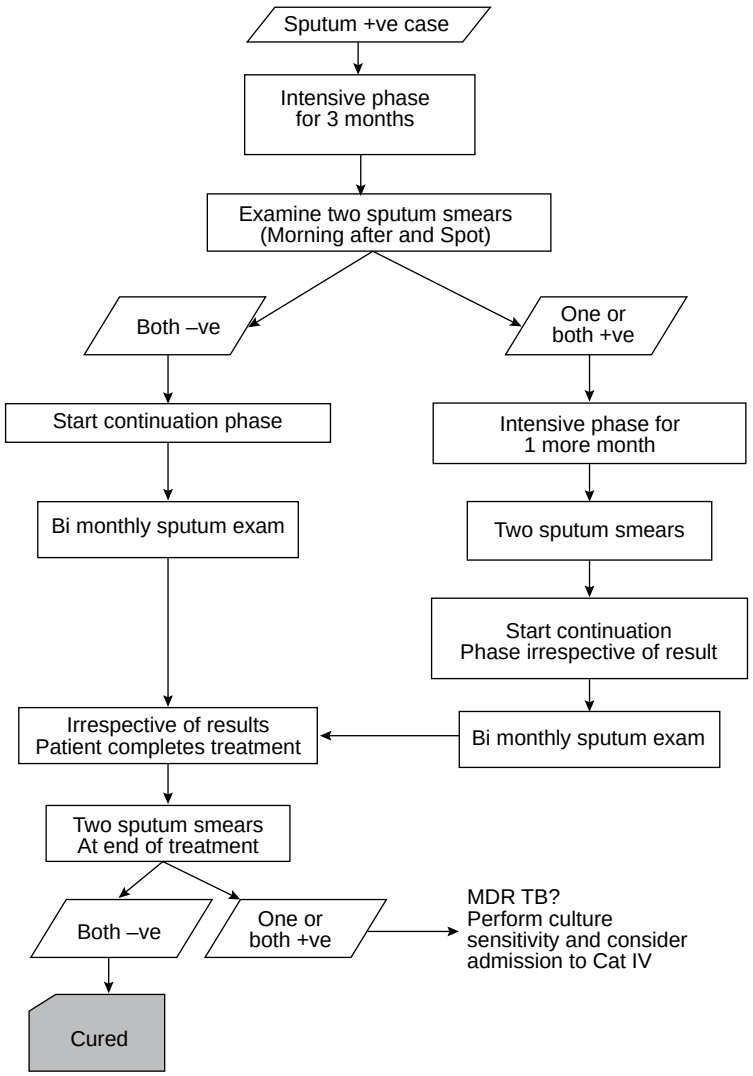

Figure 9.71. Follow up sputum examination of retreatment patients

Revised recording and reporting

Records and registers maintained by RNTCP

1. Laboratory form for sputum examination—To be filled by the medical officer who refers a case to RNTCP.
2. **Tuberculosis treatment card** is maintained at the PHC/subcenters; it contains all information regarding the patient and treatment regimens, duration of

treatment, follow up sputum results, outcome and also the information on DOT provider; a copy is also given to the DOT provider

3. **Tuberculosis ID card** (given to each patient) which includes his category, regimen and date of starting treatment, results of follow up sputum examination and date of visits.
4. **Referral form for treatment**—To be kept at all microscopy centers for referring patients to a health facility.
5. Transfer form is kept at all PHCs to transfer a patient from one tuberculosis unit to another
6. **Mycobacterial culture sensitivity form**—For a patient who does not respond to Category II treatment, the district tuberculosis officer must send his sputum with this form to a reference laboratory to know if the patient has MDR tuberculosis
7. **Tuberculosis register** is maintained by STS of Tuberculosis unit, and contains all information regarding every case (DOTS or NonDOTS) of that unit. It contains: tuberculosis number, date of registration, name , address, age/sex, name of PHC/SC where the patient has been referred from, date of starting treatment, regimen/category, disease classification, type of patient, details of sputum examinations, treatment outcome with date, remarks.

   The tuberculosis register should be updated by the STS during the supervisory visits to the health facilities. The **tuberculosis register** is the most important document in program monitoring; information from the TB register is used to compile the quarterly reports of RNTCP, and also in surveys regarding performance of RNTCP.
8. **Supervisory register**—It maintained at each health facility. All supervising officers should summarize their observations in this register after a visit to the facility.
9. **Referral for treatment register** should be maintained in all big hospitals and medical colleges where large numbers of cases are expected to be diagnosed and referred for treatment to other reporting unit.
10. **Stock register** is maintained at state/district/tuberculosis unit drug store for information of available drugs.
11. **Reconstitution register** is maintained at all the DTCs for recording the receipt of drugs of patients who have defaulted, died, failed treatment or transferred out. These drugs are reconstituted and reused.

Reporting. Each Tuberculosis unit must give **quarterly report** (4 monthly) on

1. **Diagnosis of new and retreatment cases**—At least *50% of pulmonary cases should be new sputum* +ve; at least *95% of all Tuberculosis cases must be treated under DOTS*.
2. **Sputum conversion**—At least 80% new sputum +ve cases should be –ve at 2 months of intensive phase, or 90% at 3 months of extended intensive phase.
3. **Outcome**—At least 85% of new sputum +ve cases should be *cured*; among new sputum +ve cases, *treatment completed* should be < 3% (treatment completed is not a good prognosis for new sputum +ve cases, but normal prognosis for sputum -ve cases), *failure* < 4% and *defaulters* <5%.

4. Program management—It indicates the status of involvement of health facilities, supervisory activities, referral activities, microscopy, treatment initiation, quality of DOTS, quality of microscopy, TB-HIV cross referrals, etc.

Management of tuberculosis + HIV

A lethal combination

1. In HIV infected people, the risk of progression from latent tuberculosis infection to disease is increased by 50% than normal people (in whom it is 10% only).
2. A *new* tuberculosis infection in an HIV patient can proceed quickly to clinical disease.
3. These patients are frequently extrapulmonary, or they have scanty amount of bacilli in sputum which are hard to find.
4. The tuberculin test does not work because HIV is a condition of immunosuppression.
5. Chest X-rays do not show cavitation which is typical of tuberculosis (cavitation is an immune response, which is absent in HIV infection).

In total, HIV makes more individuals have clinical disease, which is harmful from the point of view of both the individual and the community (more clinical cases means more infectivity).

Diagnosis of tuberculosis in HIV +ve patients

Irrespective of HIV or not, the RNTCP diagnostic algorithm should be followed for all tuberculosis suspects. Because HIV patients are often sputum –ve, sputum culture may occassionally be required.

Treatment

Antituberculosis treatment is the same for HIV-infected persons as it is for others.

- All *new* tuberculosis cases known to be HIV +ve are classified as **seriously ill** and treated in Category I
- The retreatment cases are to be treated with Category II

It is important to maintain confidentiality regarding HIV status of individuals.

Possible drug interactions

1. **Protease inhibitors** (saquinavir, amprenavir, lopinavir, ritonavir) and **nonnucleoside reverse transcriptase inhibitors** (nevirapine, delaviridine, efavirenz) may inhibit or induce cytochrome P-450 isoenzymes and thus alter the serum concentration of rifampicin
2. On the other hand, rifampicin induce cytochrome P-450 and substantially decreases blood levels of these antiretroviral drugs.
   Combinations of the above drugs should thus be avoided.

Scheme of treatment

All tuberculosis patients coinfected with HIV *should be treated* with a Rifampicin containing treatment regimen under DOTS. To avoid drug interactions, **treatment**

of tuberculosis should be completed prior to starting anti retroviral therapy, unless there is a high risk of HIV disease progression during antitubercular treatment (i.e. a CD4 count < 200/ μl or presence of disseminated tuberculosis).

In a patient who has been on treatment with rifampicin, **at least 2 weeks should have lapsed after the last dose of rifampicin before starting protease inhibitor or nonnucleoside reverse transcriptase inhibitors**, to avoid drug interaction.

Coordination between tuberculosis and AIDS control programs

RNTCP and the National AIDS Control Program (NACP) have devised a Joint Action Plan for TB-HIV coordination, whose objective is to reduce burden of tuberculosis in HIV patients through synergistic action of both programs. The areas of focus are

1. To sensitize the key policy makers about the importance of coordination
2. A joint training program for health workers
3. **VCTC RNTCP cross referral**—From the viewpoint of the RNTCP, a VCTC as is just another health facility (equivalent to a PHC) which refers tuberculosis suspects; and vice versa, tuberculosis patients who have other HIV-associated opportunistic infections, or report risk behavior for HIV, should be referred to a VCTC from the DOT center.
4. Universal precautions to prevent the spread of tuberculosis in HIV patients, and vice versa, the spread of HIV in tuberculosis patients in RNTCP (the last thing you want is transmission of HIV through unsafe injection practices by DOTS workers when giving streptomycin).
5. Establish a monitoring and evaluation system to assess the coordination.
6. To involve NGOs and private sectors (including private practitioners).

Drug resistant tuberculosis

Multi-drug resistant tuberculosis (MDR-TB) is caused by a bacilli which is resistant to at least (mind the language here) isoniazid and rifampicin. MDR-TB is a laboratory based diagnosis (from an **RNTCP quality assured culture and drug susceptibility testing laboratory**, usually the reference laboratories).

Cause of emergence of MDR TB

1. Provinding inadequate treatment regimens
2. Wrong categorization of patients (often the physician forgets to elicit previous antitubercular drug history, and classifies a 'relapse' case as a 'new' case)
3. Not 'directly observing' the treatment
4. Incompliance of patients due to lack of counseling.

Regular chemotherapy under DOTS strategy can prevent the emergence of MDR-TB.

Drug resistance surveillance

In order to monitor the level of drug resistance in the country, drug resistance surveillance (DRS) is being conducted among both new and previously treated cases in selected states. This will provide baseline data on MDR tuberculosis.

Diagnosis of MDR TB

Cases who continue to be **sputum +ve at the end of 4 months or later of Category II** treatment may be suspected of having MDR TB. *Only these patients*

should have a sputum sample collected and sent for culture sensitivity testing to an RNTCP quality assured laboratory.

Treatment

MDR-TB cases should be referred to and treated only at a specialized center. Treatment requires prolonged chemotherapy (24 months) with *at least 3 drugs that the patient has not consumed earlier* (*usually ofloxacin is included in regimen, alongwith Z, E and S*).

Table 9.32. A regimen for MDR tuberculosis[398]

Resistant or intolerance to H	6 $(RZE)_3$
Resistant or intolerance to H and R	12–18 $(ZEQ[a] + S[b])_3$
Resistant to all first line drugs	24 (1 injectable drug + any three of ethionamide, cycloserine, a fluoroquinolone or para-aminosalicylate)$_3$
[a] A fluroquinolone [b] Or another injectable drug—Amikacin, kanamycin, capreomycin; these must be discontinued after 6 months depending upon tolerance	

DOTS plus guidelines

In 1998, WHO the DOTS-Plus strategy, which aims to prevent emergence and manage MDR-TB. It is a *supplement* to the standard DOTS strategy, and NOT a replacement for DOTS. Also, it is not applicable universally (like DOTS), but only at selected sites. Components of DOTS plus are almost the same as RNTCP, with modifications for MDR TB.

1. Sustained political commitment
2. Diagnosis of MDR-TB through quality assured culture sensitivity testing
3. Appropriate treatment with second line drugs
4. Uninterrupted supply of quality assured second line drugs
5. A standardized DOTS Plus recording and reporting system.

RNTCP plans to form a network of DOTS plus sites around the country by 2010.

RNTCP Phase II

In the first phase of RNTCP (1998–2005), the program's focus was on ensuring expansion of DOTS to the entire country. From 2006, RNTCP has now entered its second phase, and it now aims to *consolidate* the gains made, to improve access of people to DOTTS, and to *sustain* the achievements for decades to come. All components of new **Stop TB Strategy** (see earlier) are incorporated in the second phase of RNTCP.

Leprosy

Leprosy is one of the oldest disease affecting mankind. The Bible tells of several lepers who were healed by Jesus Christ himself. Even before that, lepromatous leprosy was present in India by 2000 BC.[399] The interesting part is that even after 2000 years, we are yet to get rid of the stigma around leprosy. Although segregation of patients is unnecessary in places where adequate treatments are available, there are still more than 1,000 leper colonies in India,[400] and many people are afraid to self report a skin rash. It is a cliché in old Hindi films, the

villain does a really cruel thing (murders a man/rapes a woman) and years later, he is seen with a shrouded face, holding a walking stick in a bandaged hand, implying that he has been 'cursed' with leprosy for his sins. The disgust, the ghastly faces, the amputed limb wrapped in dirty bandage, the profound sense of nausea have given leprosy its reputation of being one of the filthiest of diseases, and not many, except a few really great ones, have come to alleviate the sufferings of the leper.

> Leprosy work is not merely medical relief; it is transforming frustration of life into joy of dedication, personal ambition into selfless service.
>
> *—Mohandas Karamchand aka "Mahatma" Gandhi*

In fact leprosy is neither sexually transmitted nor is it highly dangerous. 95% of people are *naturally immuneto* leprosy and cases are no longer infectious after as little as *3 weeks* of treatment with rifampicin. The only reason leprosy remains is that the bacilli die hard, and remain for years together within macrophages.

Leprosy has been eliminated from most of the world, including India (in 2005); prevalence of leprosy in India in 31/12/2005 was **0.95/10000 population**.[401] However, pockets of endemicity remain in many parts of the world, including India.[402]

WHO has defined leprosy a public health problem if prevalence > 1/10000. Leprosy is **eliminated** if prevalence comes below 1/10000.

Agent

Mycobacterium leprae; it is a very slow growing bacteria (generation time = 24 days) which has never been cultured in abiotic media; it can survived in dried droplets for 9 days and in moist soil for 46 days.

The bacilli were discovered in 1873 by the Norwegian physician Gerhard Armauer Hansen, who was searching for the bacteria in the skin nodules of patients with leprosy. It was the first bacterium to be identified as causing disease in humans.[403,404]

Host

Any age, especially between 10–20 years; ↑ incidence in infants indicates that the disease is active and spreading. Both clinical and subclinical infections provide cell-mediated immunity in against the bacilli; humoral immunity may also be afforded in lepromatous leprosy cases. Many tuberculoid and indeterminate types of disease get cured spontaneously.

Who gets what? (Fig. 9.72)

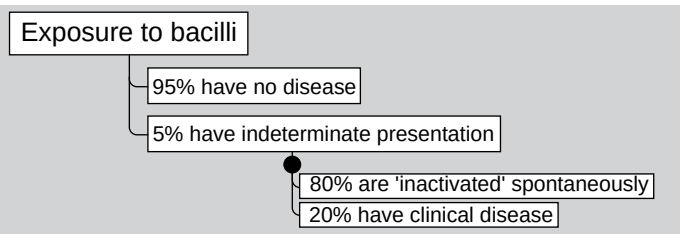

Figure 9.72. Leprosy—Who gets what?

Environment

Overcrowding, ↓ ventilation and ↑ humidity, low socioeconomic conditions

Social factors

1. False beliefs
2. Hiding early lesions due to stigma
3. Isolation.

Chain of transmission (Fig. 9.73)

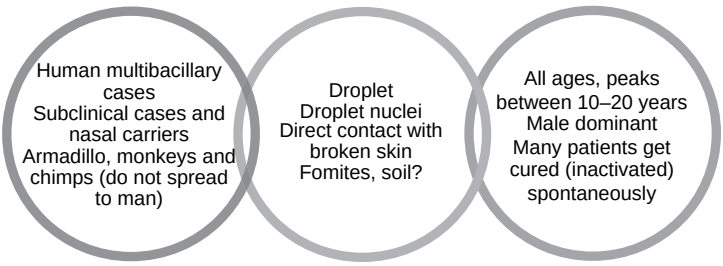

Figure 9.73. Chain of transmission of leprosy

Basically, there are two routes of exit, the skin and the nasal mucosa, although their relative importance is not clear. The main route of transmission is through droplets.[405] Whether the disease can be transmitted through direct contact with ulcerated skin is debated. It is true that lepromatous cases show large numbers of organisms deep down in the dermis; however, whether they reach the skin surface in sufficient numbers is doubtful. In a recent study, Job, et al. found fairly large numbers of *M leprae* in the superficial keratin layer of the skin of lepromatous leprosy patients, suggesting that the organism could exit along with the sebaceous secretions.[406]

Clinical course

Incubation period = 3–5 years or more (lepromatous leprosy), shorter in tuberculoid cases.

Skin lesions

Skin lesions occur in all cases of leprosy except pure neuritic types. Lesions range from a macule, papule, plaque, nodules to ulcers with anesthesia or hair loss and local nerve thickening. All sensory modalities are lost in leprosy, beginning with fine touch.

Nerve thickening

Low temperature, trauma, friction and movement favor nerve involvement. Thus, peripheral nerves like ulnar, median, great auricular, common peroneal and posterior tibial get involved easily. Examine sensory supply of nerves and power of muscles.

Questions to ask

Ask about treatment received in the past. A person who has completed a full course of MDT very rarely needs further treatment.[407] Also ask about family history of leprosy or contact with any case of leprosy.

Lab

Slit and scrape skin smears

Beause skin smears are very difficult to rightly perform and test, and treatment for leprosy should be based on clinical findings, not skin smears. Six sites are picked from, including both ear lobes. The sites should be marked to be examined again at follow up visits. Smears could also be taken from nose (patient blows his nose into a cellophane) or nasal scrappings (not usually used because it is painful). Smears are *fixed* immediately by heating over a spirit lamp amd transported to laboratory to be stained by Ziehl-Neelsen stain and examined in each smear under oil immersion view (Fig. 9.74).

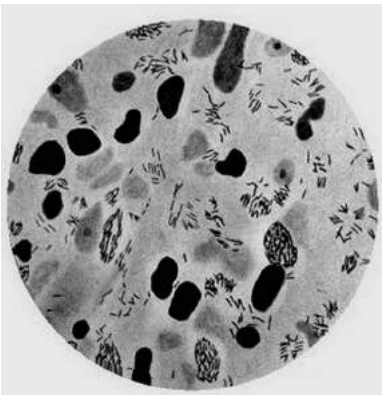

Figure 9.74. Photomicrograph of leprosy bacilli in skin smears; they frequently appear in clusters or globi[CDC public health image library]

Bacteriological index

The bacteriological index is the *grading of bacillary load* in skin from an average of 7 sites.

0	No bacilli in 100 fields
1+	1–10 bacilli/100 fields (i.e. 0.01–0.1 bacilli per field[a]
2+	1–10 bacilli/10 fields (i.e. 0.1–1 bacilli per field)
3+	1–10 bacilli in each field
4+	10–100 bacilli in each field
5+	100–1000 bacilli in each field
6+	> 1000 bacilli in each field

[a] This is only an aid to memory, to help remember the logarithmic nature of the scale; '0.1 bacilli' are not real

The average bacillary index of the subject is $\sum$ **scores from all sites / number of sites examined**. If the BI is 1–2+, at least 100 fields need to be examined. If BI is any more, you can give a score after examining only 25 fields. However, before declaring a smear negative, you have to examine 200 fields at least.

Morphological index. The morphological index is percentage of solid staining bacilli (i.e. which were yet alive during taking the smear). Criteria of solid staining bacilli are

1. Uniform staining
2. Parallel sides
3. Rounded ends
4. Length 5 times than width.

The MI is a more sensitive index of response to treatment than BI, because it counts only viable bacilli, not dead one.

Tests to detect cell-mediated immunity against leprosy

Lepromin test

Lepromin is a crude extract of lepromatous tissue, which contains the bacterial antigens (glycolipid 1).

0.1 ml lepromin injected in forearm
↓ 24–48 hours
Local erythema which subsides in 3–4 days (**Fernandez reaction**)
The early reaction is analogous to the mantoux test and indicates whether the person has been exposed to the bacilli previously or not
If the erythema > 10 mm, the test is considered positive
↓ 21 days after injection
A *nodule* develops at same spot which increases in size upto 4–5 weeks (**Mitsuda Reaction**)
If on 21st day, the size of nodule > 5 mm, the test is positive

The mitsuda reaction indicates **degree of cell-mediated immunity against leprosy**.

By lepromin test, we usually mean the Mitsuda reaction. It is found in TT and BT types of leprosy, but not in BL or LL types. The test is useful to classify leprosy based on CMI (see Ridley and Jopling classification).

Lymphocyte transformation test and leukocyte migration inhibition test

Although these provide good indication of CMI, they are expensive and not suitable for community application.

Tests to detect humoral immunity

The chief defence against leprosy is cell-mediated immunity (CMI), but humoral immunity is also active (although humoral immunity alone is futile against leprosy), and detection of antibodies have potential to identify subclinical infection and in follow up. The **fluorescent leprosy antibody absortion test** is now used for identification of subclinical cases.

Test to detect nerve damage

Histamine test. Histamine is injected into an hypoesthetic area. If the cutaneous nerve has been damaged, there is a wheal at the area but *not* the neurogenic flare

response (recall Lewis' Triple Response from your physiology book). The test is recommended for classifying indeterminate types of leprosy.

Definitive diagnosis

Skin and nerve biopsy

Culture

The bacilli can only be cultured in another living cell (i.e. foot pads of mice, armadillo) and drug susceptibility can be tested in such cutures. However, it is time consuming (6–9 months). Culture within macrophages gives more quick results (3–4 weeks).

Classification of leprosy

Indian classification

Table 9.33. Indian classification of leprosy

Type	Number of lesions	Type of lesions	Presence of bacteria in skin
Indeterminate	1–2	Vague hypopigmented hypoesthetic macule	–
Tuberculoid	1–2	Well-defined hypopigmented hypoesthetic macule/papule	–
Borderline	≥ 4	Well/ ill-defined hypopigmented hypoesthetic macule/ papule/plaque	+ / –
Lepromatous	Numerous	Poorly defined macule, papule, nodules which are shiny and smooth and distributed symmetrically	+
Pure neuritic	None	Only nerves involved	–

Ridley and joppling (Fig. 9.75)

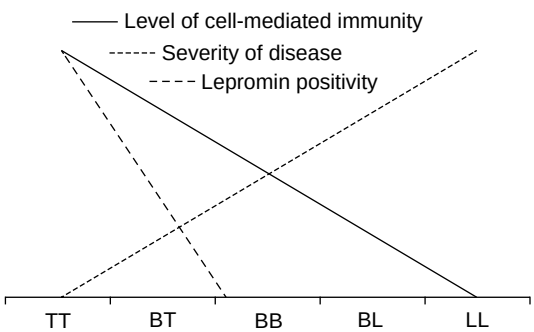

Figure 9.75. Ridley Jopling types of leprosy depending on cell-mediated immunity against leprosy

Table 9.34. Ridley and Jopling classification

WHO type	Ridley and Jopling type	Skin lesions	Nerve thickening	AFB in skin	Lepromin test
Paucibacillary	Tuberculoid (TT)	1–3 macules or plaques < 10 cm, hypopigmented or reddish, *hypo-esthetic*, with well-defined margins	0–1	–	+
	BT	More numerous macules or plaques, less well-defined, but still hypoesthetic; **satellite lesions** are present around a central lesion	> 1 Asymme-trical	–	
Multibacillary	BB	Most unstable (i.e. quickly becomes either a TT or an LL); inverted saucer shaped lesions		–/+	–
	BL	Lesions *tend* to be symmetrical; the macules and plaques are larger, and there is normal skin between lesions	*Tends* to symmetry	+	
	LL	Insidous onset, steady downhill course, multiple organs involved **Innumerable, ill-defined, symmetrical, hypopigmented macules** which are not anesthetic[a] Diffuse infiltration with many bacteria into skin causing **nodules** ('leonine' facies) Nasal stuffness and epistaxis lymphadenopathy, fever testicular involvement	Nerve damage slow to appear; symmetri-cal sensory loss from dorsal foream, leg, hands, feet	+	

[a] Nerve damage in leprosy is due to the host immune response (T-cell mediated), not due to the bacilli *per se*; lepromatous cases have little immunity against leprosy, so that they develop nerve damage very late or never. Thus, initially they have no anesthesia.

WHO classification

For the purpose of diagnosis and treatment by a peripheral health worker, both the Indian and Ridley Jopling classification fail spectacularly because of their complexity. Thus in 1981, WHO simplified things with an operational classification that has only two categories:

1. **Paucibacillary**—Any case whose bacteriological index (BI) is < 2 at All sites of skin; it includes indeterminate, TT and BT groups of Ridley Jopling classification.
2. **Multibacillary**—Any case whose BI is ≥ 2 at Any site of skin; it includes BB, BL and LL groups of Ridley Jopling classification.

Because examination of skin smears is not always possible in peripheral health facilities, the WHO classification could also be made clinically (Fig. 9.76).

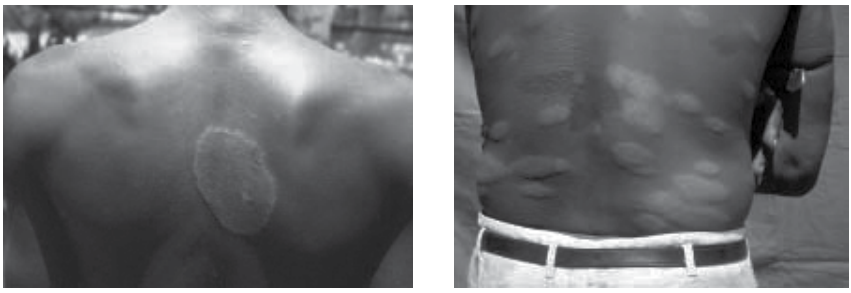

Figure 9.76. Classification of leprosy has become simple: Just count the lesions; paucibacillary (left), multibacillary (right)[408]

Table 9.35. WHO classification (1981)[407]

Paucibacillary	Multibacillary
1–5 skin lesions Or ≤ 1 nerve involved And Skin smear -ve at all sites	> 5 skin lesions Or > 1 nerve involvement Or Skin smear +ve at *any* site Or 5 skin lesions + 1 nerve involvement

Where there is any doubt, classify patient as *multibacillary*.

Treatment

Till the 1980s, leprosy was treated with only dapsone. Such **monotherapy** resulted in

1. Long duration of treatment.
2. ↑ side effects because of ↑ total amount of drug received.
3. Irregular compliance.
4. Emergence of drug resistance.
5. And consequently, loss of faith on treatment (this is the most dangerous of all; no public health program can afford to lose the *faith* of people).

6. Continued transmission of leprosy.
7. More incidence of relapse cases.

The WHO introduced multi drug therapy (MDT) in 1981.

Objectives of MDT

1. Interrupt transmission by sterilizing infectious cases with drugs
2. To ensure early diagnosis, prompt treatment and prevent deformities
3. Prevent resistance to drugs.

Prerequisites before starting treatment

Classify the patient according to WHO case definitions; inform patient about leprosy, how to prevent deformities, self care and that leprosy is curable.

Case definitions

Case

A *case* of leprosy is a person having ONE OR MORE of the following

1. Hypopigmented/reddish skin lesions(s) with definite hypoesthesia
2. Thickening of peripheral nerves
3. Skin smear shows acid fast bacilli (not necessary for diagnosis of leprosy)

who are yet to complete a course of MDT.

New case. A case who has never taken MDT.

Defaulter. A case who had started MDT but has not taken drugs for 12 consecutive months. A defaulter who returns to the health center should be given a new course of MDT if he has *new* skin lesion or nerve involvement, lepromatous nodules or signs of reactions to treatment (see later).

Relapse. A case who develops clinical features of leprosy (i.e. new skin lesions) after successfully completing adequate course of MDT (this is very rare). They must be retreated with MDT. The **relapse rate** is the best indicator of success of MDT.

MDT Regimens[409]

Table 9.36. MDT regimen

Paucibacillary (6 months)		
	Adults (> 14 years)	Children 10–14[a]
Supervised once monthly		
Dapsone Rifampicin	100 mg 50 mg	600 mg 450 mg
Unsupervised daily		
Dapsone	100 mg	50 mg
Multibacillary (12 months)		
Supervised once monthly		
Rifampicin Clofazimine[b] Dapsone	600 mg 300 mg 100 mg	450 mg 150 mg 50 mg

Contd...

Contd...

Unsupervised		
Clofazimine Dapsone	50 mg daily 100 mg daily	50 mg alternate day 50 mg daily

[a] Dosage needs to be proportionately reduced according to body weight in children < 10 years
[b] Clofazimine causes red coloration of skin, mucous membranes, urine and sweat; if this is unacceptable, it may be replaced with ethionamide or prothionamide (250–375 mg self administered dose)

Single skin lesion PB leprosy was earlier treated with single dose Rifampicin 600 mg, Ofloxacin 400 mg and Minocycline 100 mg.

With good compliance, lesions either heal or become inactive (do not progress and new lesions do not appear).

Accompanied MDT. To improve compliance, WHO has recommended to give patients a choice: they can collect their treatment at regular intervals from the health center or take the *entire course* with them when diagnosed. Patients should choose someone close to them to accompany them with their treatment.

Contraindications to MDT

1. Severe renal/hepatic dysfunction
2. Severe anemia
3. Allergy to sulfa drugs.

Neither pregnancy nor HIV infection should affect MDT.

Regularity of treatment

Patients who have taken combined drug therapy *for at least* **2/3rds of the time allotted**. For example, if within 6 months, the patient has taken combined therapy for at least 4 months, his treatment is considered to be regular.

Adequacy of treatment

Treatment is *adequate* if

- PB cases have completed all the drugs to be received in their 6 months period *within* 9 months (i.e. they have not been late for more than 3 months) MB cases who have completed all the drugs of their 12 months period within 18 months.

Posttreatment surveillance

PB cases should be clinically examined every year for at least 2 years and MB cases, 5 years.

Reactions

We know that *Mycobacterium leprae* does not cause disease *per se*, but leprosy is a state of chronic (delayed) hypersensitivity to the bacilli. A **reaction** is an *acute* inflammatory event, caused by sudden changes in immunologic activity of the patient, in the course of disease before, during or after treatment. Reactions are not a side effect of MDT. They are the body's response to leprosy and do not mean that the disease is becoming worse or that the treatment is not working.

There are two kinds of reactions

	Type I	**Type II (erythema nodosum leprosum)**
Occur in	BT and BL cases	BL and LL cases, usually within 2 years of starting treatment
Skin	Existing lesions show signs of acute inflammation (rubor, dolor, calor, tumor) New lesions appear Subsides leaving scales	Red painful, tender subcutaneous nodules on face, arms, legs; appear in groups and subsides in few days
Nerve involvement	Nerves close to skin (especially ulnar and common peroneal) become enlarged, tender, painful and may lose function overnight	Rare
Other organs	Not affected	Eye, joints, kidney, testes, bones may be involved
Systemic symptoms	None	Fever, joint pain, fatigue
Treatment	Prednisolone 40 mg once daily × 2 weeks 30 mg once daily × 2 weeks 20 mg once daily × 2 weeks 15 mg once daily × 2 weeks 10 mg once daily × 2 weeks 5 mg once daily × 2 weeks Rest, immobilization, analgesics, continue MDT	Prednisolone, same dose as in Type I Rest and immobilization Symptomatic treatment for headache and fever If symptoms are refractory to steroids, try Thalidomide 100–300 mg every night or Clofazimine 300 mg daily

If the Type I reaction occurs *before* starting treatment, it usually turns the patient more lepromatous, and such reactions are thus called *downgrading*. If the same reaction, however, happens after starting treatment, the case become more tuberculoid, and the phenomenon is known as *reversal*.

Relapse in leprosy

Patients whose therapy was terminated after adequate dose but who subsequently developed new signs and symptoms of disease either in surveillance period or thereafter. Because the reversal reaction presents in the same way as a relapse case, it is important to distinguish between the two

	Reversal	**Relapse**
Time interval	Occurs before or during treatment or 6 months after completion of treatment	Occurs > 6 months after treatment
Onset	Abrupt	Insidious
Existing lesions	Become acutely inflammed (*rubor*, *dolor*, *calor*, *tumor*)	May show rubor, but no other signs

Contd...

Contd...

Ulcers	Lesions may ulcerate	-
New lesions	Several	Not too many
Nerves	Multiple nerves become painful, tender and lose function	A single nerve is involved; usually no pain or tenderness
Fever	+	-
Response to steroid	Rapid	Nil

Disabilities

Primary disabilities (caused by the disease process itself)

Leprosy eats into the body, causing loss of eyebrows, hair, resorption of nose and finger and toes, perforation of the palate and nasal septum. In addition, the myriads of nodules over face gives the patient a 'leonine' (lion like) appearance. Involvement of nerves leads to several additional deformities.

Nerve affected	Result
Trigeminal	Corneal anesthesia
Facial	Lagophthalmos (inability to close eyes)
Ulnar	Claw hand; loss of sensation and sweat over medial palm
Median	Inability to abduct and oppose thumb; loss of sensation over lateral palm
Radial	Wrist drop; loss of sensation over dorsum of hand
Common peroneal	Foot drop; loss of sensation over legs and dorsum of foot
Posterior tibial	Claw toes; loss of sensation of sole

Secondary disabilities

Secondary deformities result from ignoring primary ones. For examples, in the eyes, the combination of corneal anesthesia + Lagophthalmos can lead to exposure keratitis and blindness. This could have been prevented by proper eye care (Fig. 9.77).

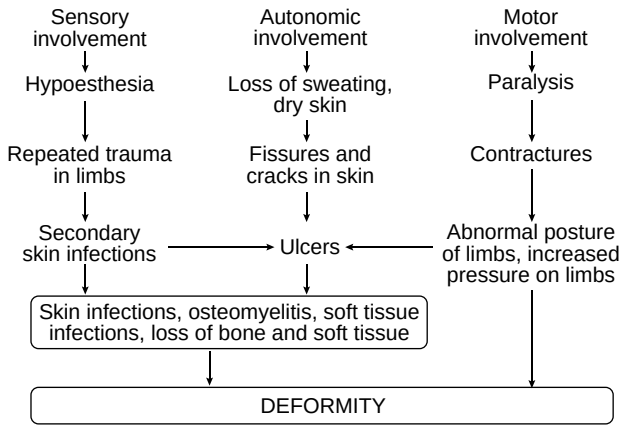

Figure 9.77. Disabilities in leprosy

WHO grading of deformities

Hands and feet	0	No anesthesia or visible deformity
	1	Anesthesia
	2	Visible deformity
Eyes	0	None
	1	Leprotic eye problem, but visual acuity > 6/60
	2	Visual acuity < 6/60

Disability limitation

1. Early diagnosis and treatment of cases and *reactions.*
2. Personal protection—Do not touch hot objects, do not walk long distances, do not walk barefoot, keep skin moist all the time, keep ulcers clean, dry and aseptic, use protective gloves, footwear and antiseptic creams.
3. Education on leprosy, importance of treatment and how to identify a reaction.
4. Provision of protective aids (splints, microcellular rubber footwear, eyeglasses, eyedrops, etc.).
5. Monitoring.
6. Reconstructive surgery.

Rehabilitation

See the chapter on 'health and disease'.

Control of leprosy

Measuring the burden of leprosy

The 'burden' of leprosy can be looked at in three ways[410]

1. The **incidence—**Because incidence is difficult to measure directly, the '**Case Detection Rate**' is used as a proxy for incidence.It seems likely that some new cases never come for diagnosis. The global incidence of leprosy seems to be declining slowly but the decline is *faster* in some areas than in others; and in a few places the incidence rate seems to be *rising*. Incidence depends on many factors like BCG vaccination, socioeconomic status, MDT application, etc.
2. The **prevalence** of leprosy has decreased throughout the world over the last 20 years because of MDT. By reducing the duration of treatment to just one year or less, MDT has greatly reduced the numbers on treatment at anyone time. Prevalence is the key indicator to know when leprosy has been eliminated, but it is not an adequate indicator to reflect changes in the epidemiological trend of leprosy.
3. The third way in which the 'burden' of leprosy can be viewed through the eyes of affected people themselves. Leprosy can lead to **disability** of the hands and feet, blindness; but physical problems are often overshadowed by the social rejection and mental suffering caused by the stigma around leprosy. The true burden of leprosy is amplified many times by the amount of disability it causes.

Controlling the reservoir

Early diagnosis of leprosy by clinical methods and treatment by MDT is the best method of making the case noninfective.

Case finding methods

Prevalence of leprosy	Recommended method
< 1 / 1000	Search for cases within *contacts* of a case
< 10 / 1000	Search for cases within groups, i.e. preschool and school children, slum dwellers, military recruits, laborers; invite people with skin disease to 'skin camps' to screen leprosy
≥ 10 / 1000	Screen everybody in the community, visit each house for cases

All cases should be recorded in WHO specified manner.

Breaking the chain

Improved standard of living, ↓ overcrowding, ↑ ventilation, ↑ personal hygiene.

Protecting the host

The BCG vaccine affords some protection against clinial leprosy (the disease, not the infection) **Chemoprophylaxis** with dapsone and acedapsone in children are still in stages of evaluation.

National Leprosy Control Program, 1955

The National Leprosy Control Program was taken up in 1955 with objectives of

1. Early detection
2. Sustained and regular dapsone monotherapy.

The WHO recommended MDT instead of dapsone monotherapy in 1981. Thus NLCP was renamed in 1983 as National Leprosy *Eradication* program with the aim of reducing 'quantum' of infection (the exact language used) in population by reducing sources and breaking the chain of transmission. Many felt that an intermediate stage of elimination was required, before jumping from 'control' to 'eradication'.

In 1991, the WHO declared strategy for elimination of leprosy, as was expected, by reducing prevalence < 1/ 10000.

National Leprosy Eradication Program phase I, 1993–2000

There were two phases of NLEP (1993–2000 and 2001–2004) which were principally funded by World Bank. Today, the program in run entirely by Government funds.

Orginal goal

Reduce prevalence to < 1/ 10000 by the year 2005 (in accordance with National Health Policy)

Objectives since April 2005 onwards

- To continue efforts to eliminate leprosy
- To maintain the gains achieved
- To make quality leprosy services available.

Quality leprosy services (WHO)

Quality leprosy services

1. Are accessible to all who need them.
   - Coverage: MDT treatment can be provided at all health units.
   - No geographical, economic or gender barriers.

2. Are patient-centered and observe patients' rights, including the rights to timely and appropriate treatment and to privacy and confidentiality.
3. Address each aspect of case management, based on solid scientific evidence
   - Diagnosis is timely and accurate, with supportive counseling
   - Treatment with MDT is timely, free-of-charge and user-friendly
   - Prevention of disability interventions are carried out appropriately
   - Referral for complications and rehabilitation is done as needed
   - Maintain simple records and encourage review and evaluation.

Strategies[411]

1. *Decentralization* of NLEP to States and Districts and strengthening of services-to integrate leprosy into the three tier health care system (as RNTCP has been); quality leprosy services are being made available in all health facilities through outdoor on each working day. A referral system for difficult cases is also being set up.
2. Leprosy *training* of all health workers
3. Early diagnosis and prompt MDT, through routine and special efforts
4. *Education* on leprosy using local and mass media for reduction of stigma and discrimination.
5. Prevention of disability and medical rehabilitation—To promote reconstructive surgery and free distribution of protective footwear
6. Monitoring and periodic evaluation
7. Intersectoral collaboration.

Barriers to elimination

1. Long and variable **incubation period**.
2. Long **communicable period**.
3. Many **modes of transmission** (some of them unknown).
4. Great number of **subclinical cases**.
5. Complicated **spectrum of disease** (is this case BB or BL? or is it a histoid variant?).
6. Emergence of **antimicrobial resistance** in bacteria.
7. **No vaccines** available.
8. No artificial **culture** media (so we cannot perform experiments on the bacilli as we wish).
9. Extrahuman **reservoir** (armadillo) present.
10. Taboo and social **stigma**, which delays diagosis.

Impact

Due to the NLEP, the prevalence of leprosy reduced from 24/10,000 in 1992 before to 0.95/10000 on 31/12/2005 (eliminated).

Special action project for elimination of leprosy (SAPEL)

Aside the NLEP, between 2001–2004, many SAPELs were organized which addressed the 'cases of consequence', i.e. > 5 smear +ve skin lesions, in *underpriviledged* and *difficult* to access (i.e. tribal areas).

WHO 'final push' strategy on elimination of leprosy

1. **Expand** MDT all health facilities
2. Ensure that all existing and new cases are given **appropriate** MDT regimens
3. **Encourage** all patients take treatment regularly and completely
4. Promote **awareness** in the community on leprosy so that individuals with suspicious lesions will report voluntarily (without fear)
5. Set **targets and time table** for activities and make all efforts to achieve them
6. Keep good **records** of all activities in order to monitor the progress towards elimination.

SEXUALLY TRANSMITTED DISEASES

Reproductive tract infections

Reproductive tract infections are classified as

1. STDs
2. Overgrowth of commensal bacteria
3. Iatrogenic infections
4. Infections due to vaginal delivery/spontaneous abortion (sepsis).

STDs are a group of communicable diseases *predominantly* transmitted by sex.

Agent

Bacteria	*Neisseria gonorrheae* *Treponema pallidum* *Chlamydia trachomatis* *Hemophilus ducreyi* *calymmatobacterium granulomatis* *Mycoplasma hominis* *Ureaplasma urealyticum* *Shigella* *Campylobacter jejuni* *Streptococcus agalactiae*
Virus	*Herpes simplex* *Cytomegalovirus* *HIV* *Human papilloma virus* *Hepatitis B* *Molluscum contagiosum*
Protozoon	*Entamoeba histolytica* *Giardia lamblia* *Trichomonas vaginalis*
Fungi	*Candida albicans*
Ectoparasites	Phthirus pubis Sarcoptes scabiei

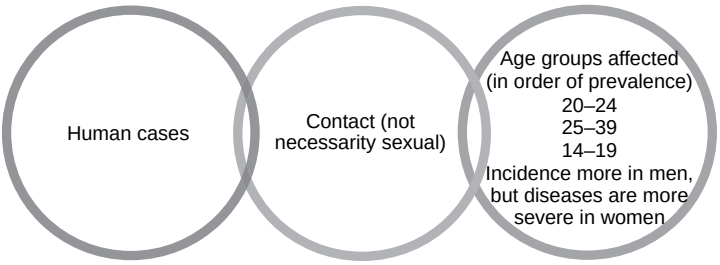

Figure 9.78. Chain of transmission of STDs

Sociodemographic factors

If sex is confined between two people only, STDs cannot escape the couple; the reason behind the 'explosion' of STDs in the middle of last century was the revolution of 'casual sex', and the second world war.

1. **Population explosion** with ↑ younger age groups have contributed to the spread of STD.
2. ↑ **urbanization**—Because people from different backgrounds and exposures gather in a city, STD spreads quickly in a city rather than the closed population of a village.
3. Broken families and promiscuity.
4. **Prostitution**—It is a known truth that women, especially prostitutes, are at the receiving end of STDs; usually STDs are brought into brothels not by a new girl but by a new customer.
5. **Unemployment** causes depression in young men, who search for sexual relief, often with prostitutes.
6. **Industrialization**—The power and value of women in an industrial society is much less than in an agricultural one; this is because in an industrialized corporate society, the men attend the outdoor jobs while women sit around the house. This is not so in agricultural societies where men and women take equal part in crop production. Thus, industrial societies lower the position of women, thus precipitating objectification of women and rise of casual sex.
7. **International travel**—In middle ages, *sailors* used to be the vectors for STDs; nowadays it can be any gentleman returning home from a trip.
8. Social unrest—**War** exposes too many young men to a new country; depraved of sexual contact for long periods, they turn to local women (or each other) for sex, often forcibly, and bring home an STD; **disasters and riots** cause exposes women to multitudes of sex starved men in the streets, and STDs have their way.

STDs are a public health problem

Magnitude is not known, because of

Under reporting and social stigma; people usually bear with STD until it hurts badly, and in the meantime, STD spreads to each sexual contact.

Health related consequences

Infertility, ectopic pregnancies, fetal wastage, cancer, death, ↑risk of HIV, development of AMA resistance

Need for a syndromic management

Current diagnostic strategies, which are laboratory based, are expensive, time consuming, unreliable, needs a lot of training and importantly, need a *second visit* from the patient, which he may not come back for. Thus the transmission continues and the patient may be lost to follow up. Neither an etiological diagnosis or a clinical guess about etiology seems to provide good results.

> Some clinicians feel that, after examining a patient, it is easy to make a clinical diagnosis, such as gonococcal urethritis or chlamydial urethritis. However, even specialists sometimes misdiagnose STD when relying on their own clinical experience. Why? In many instances it is not possible to differentiate clinically between the various infections and, in addition, it is common for mixed infections to occur. A patient who has multiple infections needs to be treated for each of them. Failure to treat one infection may result in the development of serious complications.
>
> *—STD case management workbook 2: Using flow charts for syndromic management: WHO 1995*

Again, its hard to refer patients of STDs as they want to stick to a PHC or private clinics, they do not want too much publicity about their disease.

Syndromic management

It comprises of

1. Diagnosis by a set of symptoms
2. Comprehensive therapy of all possible organisms that could cause such symptoms.

At *first* contact with health system.

Essential components

1. Syndromic diagnosis and treatment
2. Education on reduction of risk of acquiring STD, and condom provision
3. Counseling (one to one and confidential)
4. Management of the partner
5. Follow up.

Advantages of syndromic management

- Simple, cheap, rapid
- Can be implemented on large scale
- Requires minimum training
- Can be learnt by wide range of health workers
- Diagnosis and treatment in a *single* visit
- Prevents transmission and complication of disease
- Provides education regarding STDs.

Treatment regimens

Gonorrhea	Azithromycin 2g Or Ciprofloxacin 500 mg oral single dose Or Ceftriaxone 250 mg IM single dose
Chlamydia	Azithromycin 2g Or Doxycycline 100 mg × 2 × 7–14 days
Vaginitis	Metronidazole 400 mg × 2 × 7 oral + Topical nystatin 1 lakh IU × 1 × 14 days Or Metronidazole 2g oral + Fluconazole 150 mg oral
Cervicitis	Treat gonorrhea + chlamydia (Azithromycin 2g single dose clears both)
Herpes	Acyclovir 200 mg × 5 × 7 oral Recurrent form: Acyclovir 500 mg × 3 × 5 oral
Syphilis and Chancroid	Benzathine penicillin 2.4M units IM in 2 equally divided doses in each buttock, after sensitivity test + Azithromycin 1 g orally under supervision

The following flow charts are to be used for syndromic approach;[412] individual nations, including India, have developed their own flow charts based on these.

Urethral discharge (Fig. 9.79)

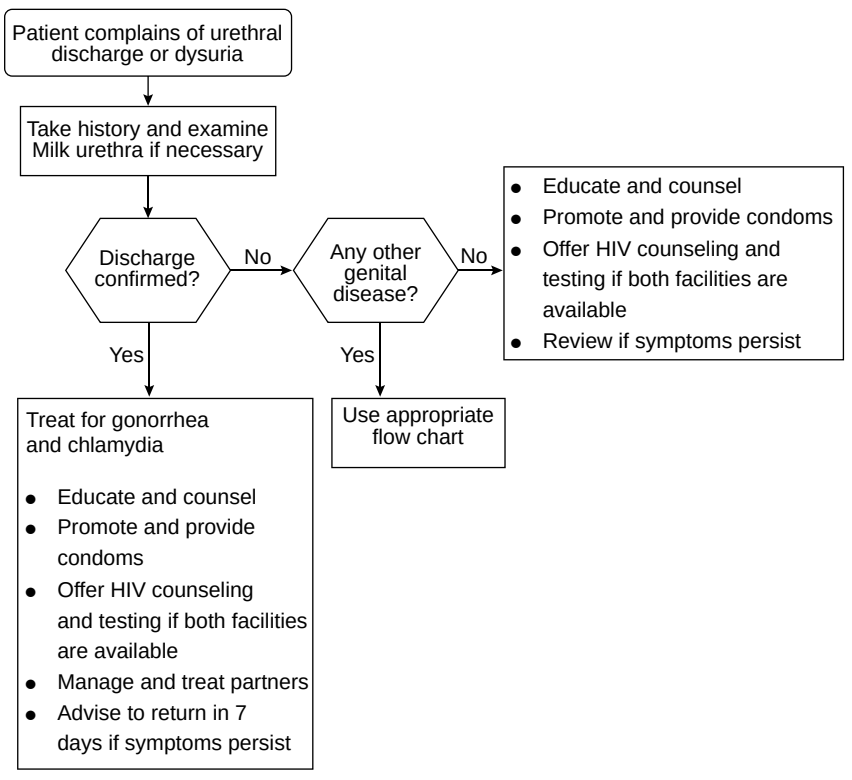

Figure 9.79. Urethral discharge

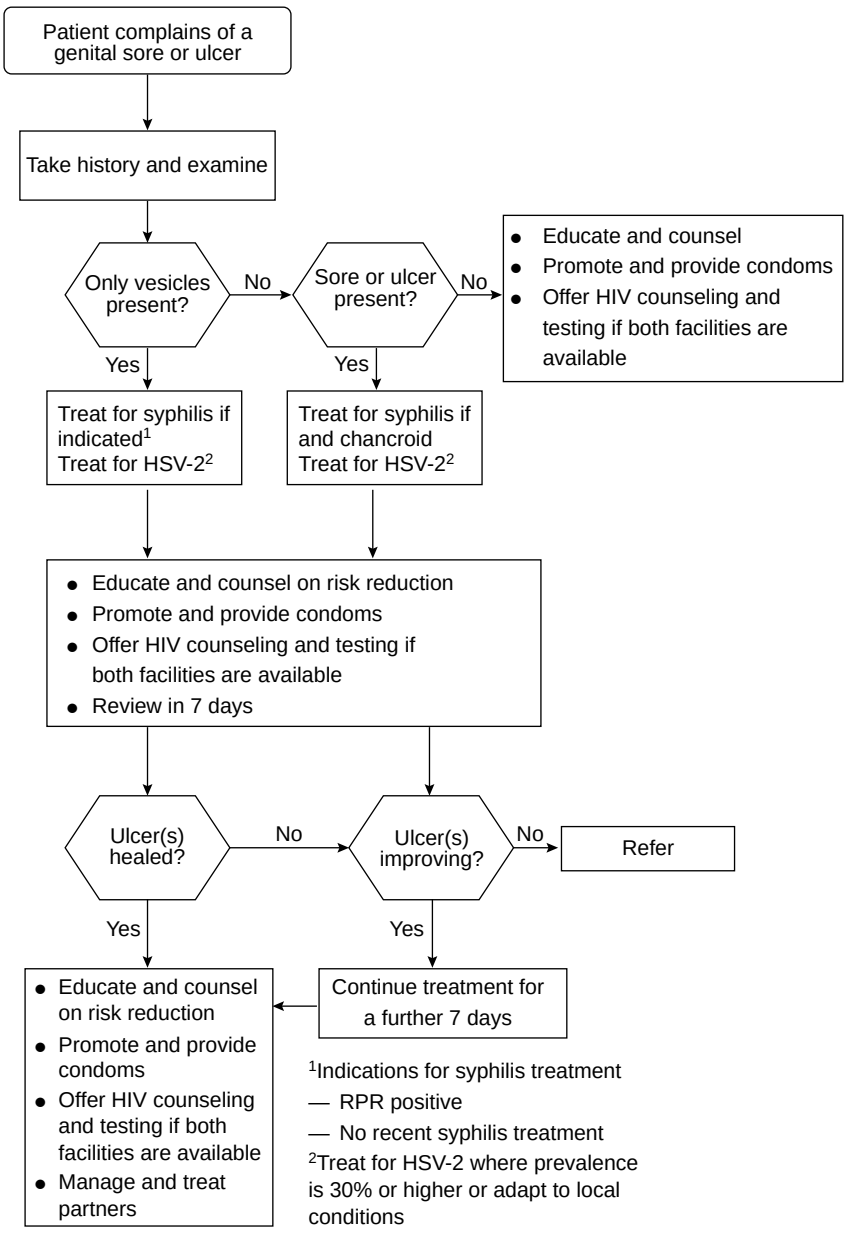

Figure 9.80. Genital ulcer

This flow chart should be replaced with a more relevant national version if either syphilis or chancroid are of low prevalence. Equally, if granuloma inguinale and/ or lymphogranuloma venereum are prevalent, include the relevant treatment. Genital herpes is the most frequent cause of genital ulcer in many parts of the world.

Vaginal discharge (Fig. 9.81)

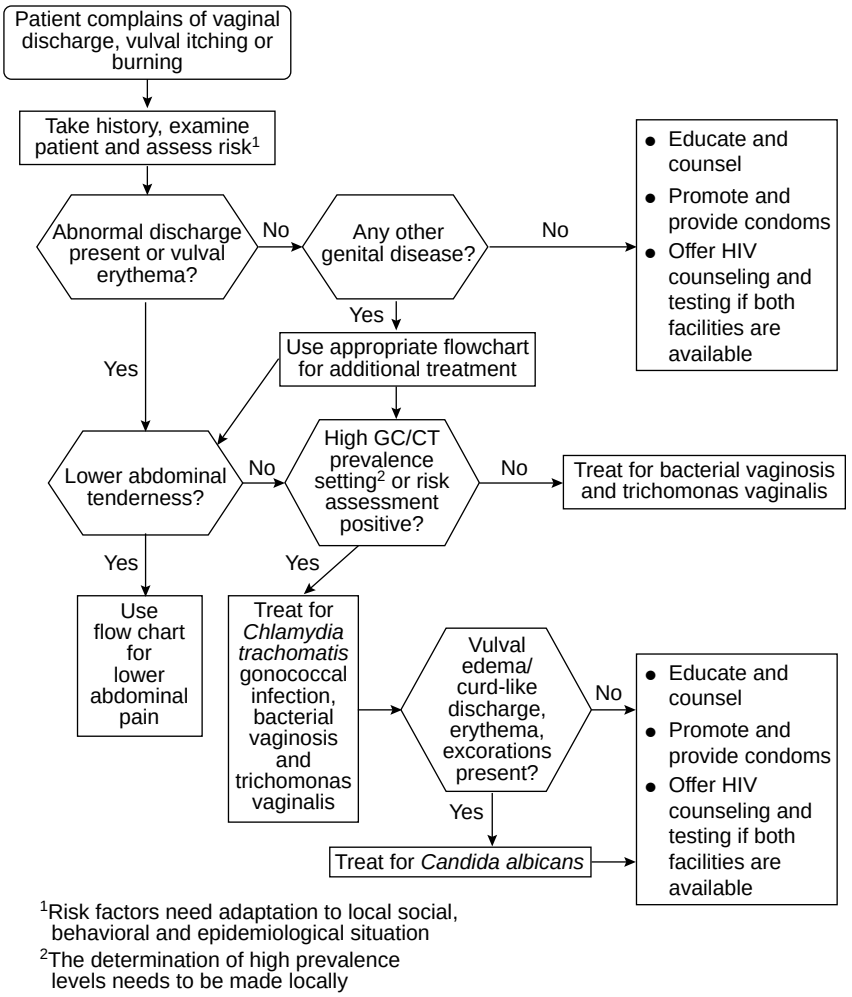

Figure 9.81. Vaginal discharge

While vaginal discharge is highly indicative of vaginal infection, it is poorly predictive of cervical infection with gonorrhea and/or chlamydia. The flow chart may become more predictive of cervical infection if a number of risk factors are included.

- Being under 21 years of age (or 25 in some settings)
- Being unmarried
- Having more than one sexual partner in the last three months
- Having a new partner in the last three months
- The current partner having a sexually transmitted infection.

The availability of a simple instrument like a vaginal speculum could make the diagnosis easily. On speculum examination, if discharge is seen to be coming from *within* cervix, the cervix is infected. This distinction is necessary because there are significant difference between vaginitis and cervicitis.

Vaginitis	**Cervicitis**
Caused by trichomoniasis, candidasis and bacterial vaginosis	Caused by gonorrhea and chlamydia
Most common cause of vaginal discharge	Less common cause of vaginal discharge
Easy to diagnose	Difficult to diagnose
No serious complications	Major complications
Treatment of partner unnecessary, except for trichomoniasis	Need to treat partner

Scrotal swelling (Fig. 9.82)

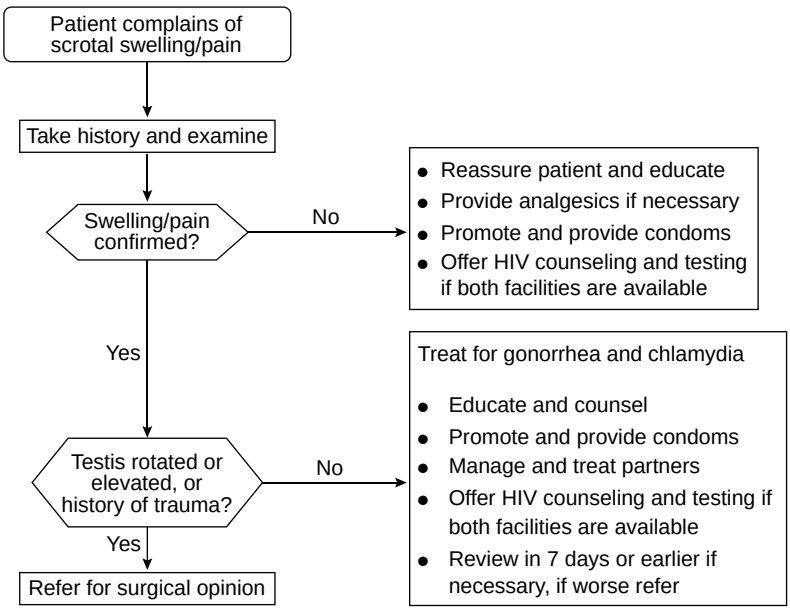

Figure 9.82. Scrotal swelling

Infection of the testis or epididymis is a serious complication of gonococcal urethritis and chlamydial urethritis. When infected, the testis becomes swollen, hot and very painful. If early and effective therapy is not given, the inflammation will heal with fibrous scarring and destruction of testicular tissue. This may lead to infertility.

It is important to consider possible noninfectious causes of scrotal swelling and pain, as well as nonsexually transmitted infections. Noninfectious causes include trauma, tumor and testicular torsion and all require referral.

Lower abdominal pain in female (Fig. 9.83)

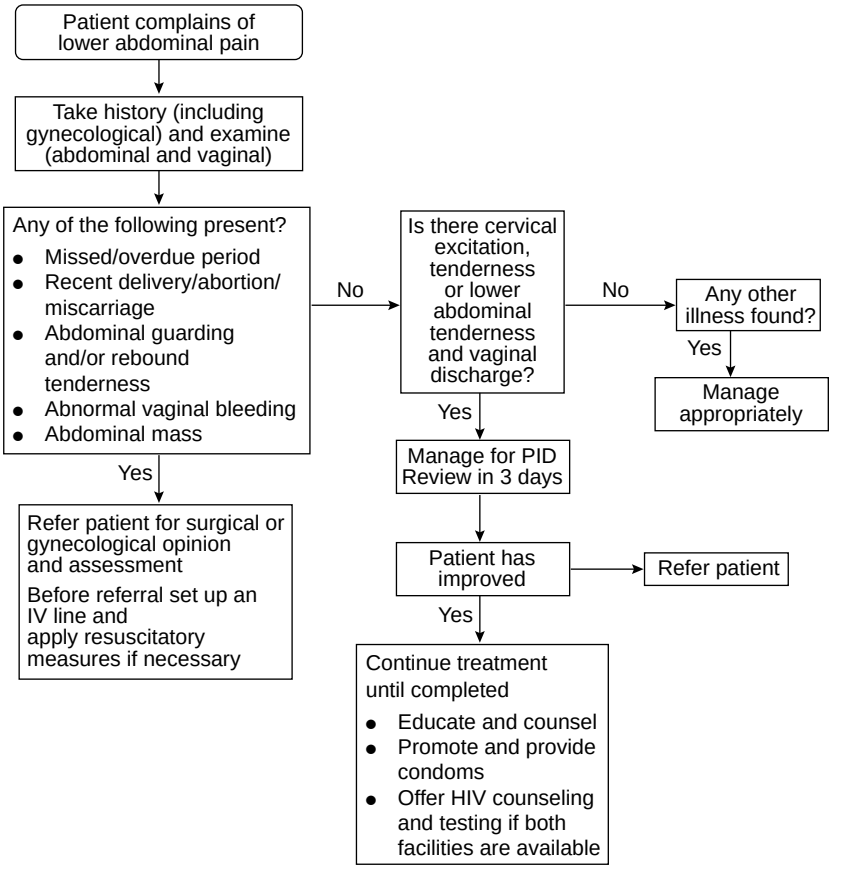

Figure 9.83. Lower abdominal pain in female

The term **pelvic inflammatory disease** (PID) refers to infections of the female upper genital tract: the uterus, fallopian tubes, ovaries or pelvic It can be caused by gonorrhea, chlamydia and some anaerobic bacteria. It can also lead to **generalized peritonitis**, a potentially fatal condition. **Salpingitis** may lead to a blocked fallopian tube, resulting in decreased fertility or total infertility if both tubes become infected. It may also lead to *partial* tubal obstruction, allowing *spermatozoa to pass through*, but not the relatively larger fertilized ovum. The result can be a tubal or **ectopic pregnancy**, which will eventually rupture, causing massive intra-abdominal hemorrhage and possibly death.

Women with PID usually have a history of

- Lower abdominal pain (may be absent) and vaginal discharge
- Pain during intercourse
- Abnormal uterine bleeding

- Painful urination
- Fever
- Menorrhagia.

Inguinal bubo (Fig. 9.84)

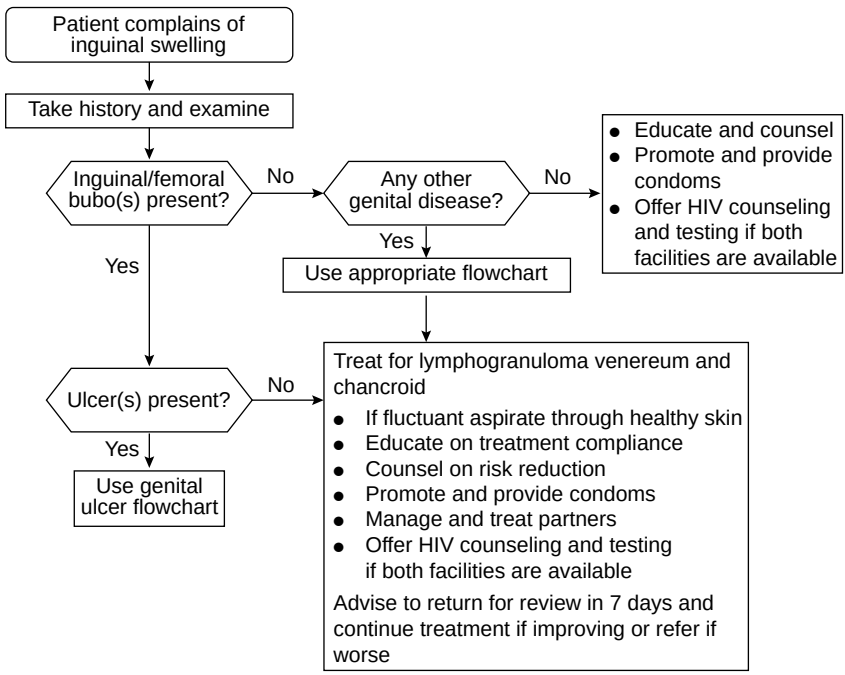

Figure 9.84. Inguinal bubo

This is a painful, often fluctuant, swelling of the lymph nodes in the inguinal region (groin). Buboes are usually caused by either chancroid or in many, but not all, cases of chancroid, a genital ulcer may be visible. If so, you should refer to the genital ulcer flowchart and treat the patient for that syndrome.

Neonatal conjunctivitis

Neonatal conjunctivitis (ophthalmia neonatorum) is defined as purulent conjunctivitis occurring in a baby less than one month of age. The most important causes of this potentially sight-threatening condition are gonorrhea and chlamydia. If caused by gonorrhea, blindness often follows.

Prevention

Controlling the reservoir

Case detection

1. **Screening** of at risk population (blood donors, prostitutes, industrial workers, army, navy, refugees, hotel workers, etc.)

2. Contact tracing—To find all sexual partners of a case and testing them for disease
3. Cluster testing—The case is asked for other people who have been exposed to the same risk as of the case; a man who regularly visits brothels could be asked to name other visitors.

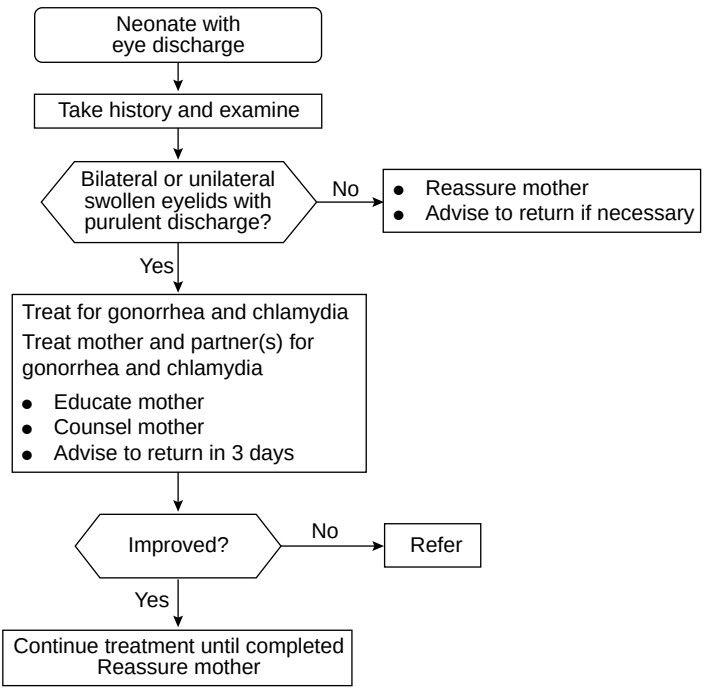

Figure 9.85. Neonatal conjunctivitis

Case management

It is best to use syndromic approach as it delivers comprehensive treatment at first visit.

Contact treatment

Treatment of all sexual contacts of the case, irrespective of whether they are diseased or not, while awaiting for test results.

Breaking the chain

Promotion of condoms; even with no condoms, basic hygiene should be maintained during sex.

Protecting the host

Vaccination

Except hepatitis B and human papillomavirus infection, no other STD is vaccine preventable.

HIV infection

Problem

World

Table 9.37. HIV/ AIDS epidemic: Global Summary, 2008,[413] figures in million

Indicator	Total	Adults	Children under 15
People living with of HIV/AIDS	33.4	31.3 (15.7 million women)	2.1
New cases of HIV/AIDS	2.7	2.3	0.43
Number of deaths from HIV/AIDS	2.0	1.7	0.28

After most developed countries have become HIV aware, most new HIV infections now occur in developing countries, and 48% of the newly infected were women. The epidemiological picture is not the same in the two worlds. In developing countries, homosexual men are predominantly affected. In developing however, the sex ratio is almost equal (see later for why women are vulnerable).

There are three kinds of HIV epidemics, classified on the basis of prevalence of HIV infection in high risk groups and in general population. Prevalence of HIV in pregnant women is taken as a surrogate indicator for prevalence of HIV in general population, as antenatal mothers are easily available to health facilities.

Epidemic	Prevalence of HIV in high risk group	Prevalence of HIV in pregnant women	Characteristics
Low level	≤ 5%	≤ 1%	Confined to a few individuals with high risk behavior
Concentrated	> 5%	≤ 1%	Confined to several people in high risk groups, but not yet spread to general population, because of lack of briding population (see later)
Generalized	> 5%	> 1%	Is maintained by the general population by sexual activity between themselves, and not dependent on virus imports from high risk groups

The bloodline: Fragments from the history of HIV

The earliest known case of HIV was from a blood sample collected in 1959 from a man in Kinshasha, Congo.[414] However, it did not get limelight until 1981 when rare type of pneumonia (due to *Pneumocystis jiroveci* and a rare cancer (Kaposi sarcoma) was reported in 5 gay men in Los Angeles[415] and New York. The disease did not yet have a name. The press called it Gay-related immune deficiency (GRID);

the CDC called it “the 4H disease,” as it seemed to infect Haitians, homosexuals, hemophiliacs, and heroin users. The public simply called it 'gay plague', implying that it was a some kind of punishment of God for being gay.

> "One thousand one hundred and sixty!" my friend said. "That's the median number of sexual partners the guys getting the disease have had. Can you believe that? One thousand one hundred and sixty!" ... "It affects homosexual men, drug users, Haitians and hemophiliacs," says a woman.
> "Thank goodness it hasn't spread to human beings yet."
>
> *—The Plague Goes Public, from The Plague Years: A Chronicle of AIDS, The Epidemic of Our Times, David Black (New York: Simon and Schuster, 1985)*

However, it was soon discovered straight men and women were not immune to the disease. In view of this, the term AIDS was introduced in July 1982, because "..it was reasonably descriptive without being pejorative". Thailand was the first Asian country to be infected, and Tamil Nadu the first Indian State (1986).

India

With 2.31 million infections, India has the third highest number of HIV infections, after South Africa and Nigeria. About 0.34% of people over 15 have the HIV infection at the end of 2007. The epidemic of India is yet *concentrated* (see the table earlier).[416]

Demographic impact

The gain in life expectancy which Africa had achieved over past 50 years by fighting with malaria, yellow fever, trypanosomiasis, etc. has been NULLIFIED by HIV. The HIV has eaten out the middle of African age pyramid and made a ‘waist’ around there. Many kids of HIV +ve parents now find themseles orphaned and carrying HIV (a double blow).

Economic impact

HIV and AIDS affects economic growth by reducing the availability of human capital[417] (it predominantly affects the working age group). In addition, the cost of therapy is a burden to many poor countries.

Socioeconomic factors

1. **Stigma**—The HIV has brought sex out in the open, literally. But this liberal air has come after decades of ignorance, prejudice and judgemental attitude towards HIV infected people. In many parts of our country, HIV destroys not only the body of the individual, but also his reputation of being a respectable person. The connotation of sex, especially the 'darker' types of sex (anal sex, gay sex, paid sex, etc.) still gives people the chills when dealing a person with HIV. And the fear of being outcast, of losing dignity, is what keeps people not coming to test for HIV, or denying it after they have been diagnosed of it (Fig. 9.86).
2. **Women** are the receiving end of HIV problem. In developing countries, women are in no position to ask their husbands to use a condom. And if the husband is promiscous, he could bring the virus from the brothel to his home (about 30 million men in India *buy* sex on a regular basis). Thus, women

account for about 39% of all infections in India despite the fact that more than 90% of them are in monogamous relationships, and have not had sex before marriage (except if they have been raped, in which case HIV finds yet another route into them).[418]

3. **Marginalized population** are at risk; street people, refugees, homeless people and everybody far from their homes (i.e. truck drivers) practice the kind of casual sex and aberrant behavior (i.e. IV drug abuse) that result in quick spread of HIV.
4. **Education** about life and sex in general seems to keep HIV rates down; but most Indian parents are afraid to raise the subject of sex in front of their children. This does not mean that they want their children to be hermits. They only blush away from the matter and hope that 'somebody' else will tell the kid all about it. This somebody often turns out to be a wrong person, i.e. a school senior who inculcates all the wrong ideas about sex (i.e. girls are always 'available', one night stands are fun, a steady boy/girlfirend is boring, etc). If that 'somebody' doesn't turn up, there is always the television.
5. **Rapid urbanization** which has increased the number of homosexuals, IV drug users, aberrant sexual practices; it seems people in villages are more prone to follow traditional ways of life. People in cities, however, because of their economic independence, are removed far from the ground realities; staying in close quarters of highrise buildings and clubbing in blazing music can take a toll on your mind.
6. **Separation from family**—Prisoners make a mess of themselves by turning homosexuals[419] and return home with HIV, again to infect his wife; **truck drivers**, removed from their homes (and wives) for months at a stretch, long for some sexual release, and will take any such opportunity that presents itself. They carry the virus over long distances.

Figure 9.86. This poster from 1989 uses a seventeenth-century painting of Saint Sebastian. The image shows Sebastian, who was condemned and persecuted by emperor Diocletian, being rescued by angels after he was fatally shot by Roman archers. The artist appropriates Sebastian the martyr as a symbol of suffering for people living with HIV/AIDS: by Charles Michael Helmken [US National Library of Medicine, nlm.nih.gov]

7. Problem families—Children who are bereft of parental care, and land up alone in too big a world, are easily dragged by adults into sexual acts, and catch the virus
8. **Customs**—Tattooing/body painting with unsterilized needles increase the risk of HIV infection; barbers who do not wash their razors between two customers are greatly risking a transmission. However, male circumcision seems to be protective against woman to man infection.[420]

Agent

The HIV belongs to family retroviridae, genus lentivirinae. Of the two varieties, HIV1 is found all over the world HIV2 is almost entirely confined to Africa. The receptor for viral surface glycoprotein (gp41) is CD4, and this defines the target cell population for HIV (CD4 lymphocytes and many antigen presenting cells).

The *structure* of the virus is simple enough. There are two surface glycoproteins **gp120 and gp41**, one matrix protein **p17**, one core protein **p24**, two molecules of **RNA**, two molecules of **reverse transcriptase** and several **proteases** for posttranslational modifications of viral proteins.

Host

The virus affects the sexually active age group (20–49). In most developing countries sex ratio is equal. Apart from being socially vulnerable (see earlier), women have a greater biological plausibility of infection, especially adolescents and postmenopausal women, whose cervical mucus is thinner.

High risk group (Fig. 9.87)

1. Male homosexuals (or as called by the NACO, 'men who have sex with men' or MSM)
2. Commercial sex providers ('prostitutes'—A term which has lost meaning in the new global economy) and their clients
3. IV drug abusers (IDU)
4. Frequent blood transfusion recipients.

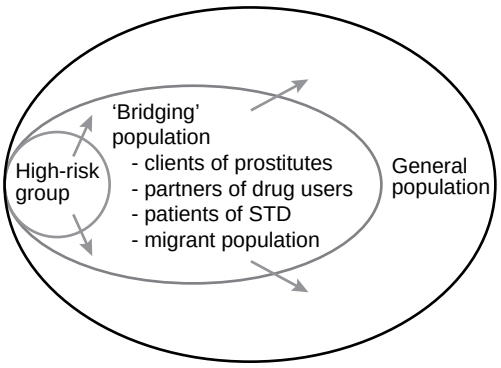

Figure 9.87. Bridging population: People who bring HIV into general population from high-risk groups

Chain of transmission (Fig. 9.88)

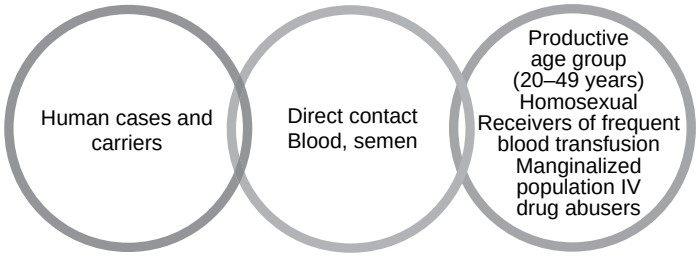

Figure 9.88. Chain of transmission of HIV

Mode

Sexual

Majority of HIV infections are acquire by unprotected sex. The transmission of the virus depends on

1. Strength and virulence of strain
2. **Concomitant STDs**, which causes mucosal breaks and rise in local macrophage population, so that HIV can gain entry easily and finds macrophages to infect
3. **Age and gender of receiver**—In high-income countries, the risk of female-to-male transmission is 0.04% per act and male-to-female transmission is 0.08% (i.e. women are *twice* as vulnerable from getting the infection from men than men from women). This is because semen contains a much higher concentration of virus than vaginal fluid, and women are exposed to the virus over a much greater surface area (the whole of their vagina, cervix and endometrium) than me (who contact vaginal fluid only at their glans). For various reasons, these rates are 4–10 times higher in low-income countries.[421] Adolescent girls and postmenopausal are more susceptible than those in reproductive age (see earlier).
4. **Type of sex**—Anal sex makes more mucosal breaks than vaginal or oral sex; however, if the women is menstruating, vaginal sex can also get very risky.
5. Stage of disease—The HIV infected people are most infectious in Window period and after they have developed AIDS (see later).

The correct use of latex condoms reduces risk of infection by 85%;[422] but spermicides may increase the risk.

Transplacental

HIV may pass on during delivery of an HIV +ve mother. Probability of infection is about one-fourth[423] (if the mother does not breastfeed) to two-thirds (if she does breastfeed). However, transmission rates can be lowered too 1% if the mother is given chemotherapy. Mothers who already have AIDS will almost always transmit the virus. Neonates born with HIV progress rapidly to AIDS.

Contaminated needles

Accidental needle pricks (such as in health workers) with a needle that has been contaminated with HIV +ve blood has only a 0.3% risk of HIV transmission.[424]

Needle sharing between intravenous drug users (IDU), often multiple times a day, increases the risk to 0.67%. Often such IDUs are also homosexuals.

Blood transfusion

Blood is the **most effective vehicle** for HIV transmission (95% efficacy[425]), and in this regard, whole blood, cells, and coagulation factors are all infective but NOT albumin, immunoglobulins or vaccines that are prepared from blood (i.e. HBsAg subunit vaccine).

HIV does NOT spread through

- Casual contact over intact skin (touching, hugging, handshake)
- Water or food
- Vectors
- Fomites.

Clinical course (Fig. 9.89)

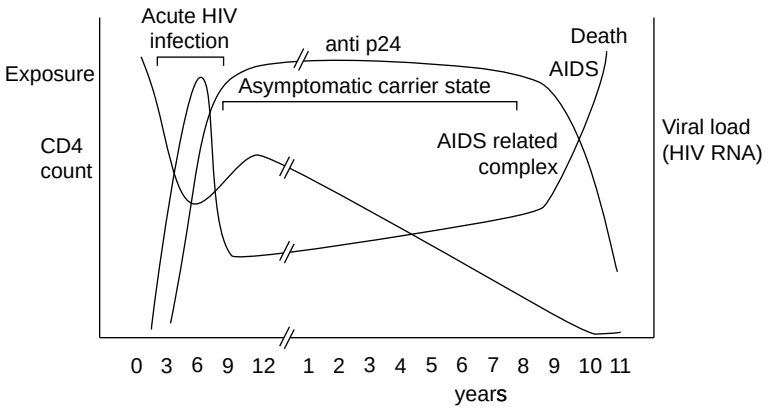

Figure 9.89. Natural history of HIV infection

Incubation period

The 2–4 weeks immediately following infection are silent both clinically and serologically.

Acute HIV infection/seroconversion illness

As the viral load rises, so as to be detectable in blood the patient becomes *infective*, but has not yet begun to produce antibodies, which will appear only after 2–12 weeks after infection. The period *before* antibody production is the **window period**, when the person is particularly infective because, but will test negative for HIV antibodies, which are the standard tests for HIV infection. Such patients can be diagnosed only by HIV RNA / p24 antigen testing in blood.

The appearance of antibodies (seroconversion) produces one or more of fever, arthralgia, myalgia, lymphadenopathy, sore throat, mucosal ulcers, headache and photophobia, meningitis, encephalitis, peripheral neuropathy or myelopathy in 50–70% of patients. The illness lasts between 6–9 weeks of infection and resolves

completely as antibody titers rise in blood. Viral RNA and p24 antigen, and occassionally antibodies are detectable in blood this stage.

Asymptomatic carrier state

On an average, the virus replicates for ~10 years while the person is asymptomatic. The viral load increases, and the CD4 counts ~50/ μl/year. Symptomatic disease appears only when CD4 count < 200.

AIDS related complex/lesser AIDS

This precursor to AIDS surfaces with diarrhea for > a month, fatigue, loss of weight, malaise, persistent generalized lymphadenopathy (> 1 cm enlargement of lymph nodes at two or more extrainguinal sites for more than 3 months in the absence of other causes) or splenomegaly. Severe opportunistic infections are yet absent at this stage.

AIDS

Barring some rare nonprogressors, most HIV infection will progress to AIDS. There are two kinds of manifestations of AIDS: those due to HIV itself (AIDS dementia complex, weight loss, chronic diarrhea) and those due to immunosuppression (opportunistic infections, cancers).

Opportunistic infections

These are infections in the immunodeficient state:

- Infections by obscure pathogens.
- Atypical presentations by known pathogens.
- Typically the inflammatory response is absent (i.e. no neck rigidity may be seen in meningitis).
- Best diagnosed only by visualization and culture (serology in unreliable).

Preventing opportunistic infections

1. Avoid exposure to pathogens—*Food hygiene* protects against *Salmonella* and *Cryptosporidium*; keeping away from your cat protects from Toxoplasma; having protection during sex keeps away herpes simplex, hepatitis B and papillomaviruses; *screening blood* for transfusion protects from hepatitis B and C.
2. Chemoprophylaxis—Primary prophylaxis is effective in reducing the risk of *Pneumocystis jiroveci*, *Toxoplasma gondii* and MAC infections. Also, antiretroviral therapy helps prevent opportunistic infections if it can keep CD4 count of at least 200.

Nervous system

1. **AIDS dementia complex** is deterioration of cognitive functions at a late stage of the disease, caused by cortical atrophy.
2. Aseptic meningitis is usually seen *before* development of clinical AIDS; it presents with CSF findings of typical viral meningitis without a discernible cause.
3. The commonest cause of opportunistic meningitis is *Cryptococcus neoformans*; diagnosis is made by CT scan of brain and Indian ink staining of CSF (negative stain); start treatment with amphotericin B + flucytosine and introduce

fluconazole after 2 weeks, and continue until the patient has CD4 count of at least 200; meningitis may also be caused by *Coccidioides*, *Histoplasma* or occassionally *Acanthamoeba castellanii* or *Naegleria fowleri*.

4. **Toxoplasmosis** of the brain is declining after introduction of antiretroviral therapy.
5. CMV lumbosacral radiculopathy leads to paraparesis and sphincter disturbances; functional recovery may never occur.
6. JC virus, a polyomavirus which infects of oligodendrocytes leading to **progressive multifocal leukoencephalopathy**, which is a demyelinating disease; the features are of progressive intellectual impairment, aphasia and hemiparesis.
7. Myelopathy which may take the form of a pure sensory ataxia, subacute combined degeneration (B_{12} deficiency), or a vacuolar myelopathy as part of HIV encephalopathy; the cytomegalovirus can cause a combined myeloradiculopathy, usually late in the disease.
8. Peripheral neuropathy (especially a distal sensory polyneuropathy) is common in all stages of disease; myopathy may be caused by HIV itself (the wasting syndrome) or zidovudine.
9. Autonomic neuropathy presents as postural hypotension and diarrhea (zalcitabine and didanosine produce a similar neuropathy).

HAART has a strongly beneficial effect on HIV neurological disease, except PML.

Eye

1. Retinal 'cotton wool' spots (ischemic areas) are the most common ophthalmologic finding.
2. **Cytomegalovirus retinitis** is the most serious risk. The patient presents with painless diminution of vision with complain of 'floaters', and fundoscopy shows hemorrhage and exudates (so called 'pizza pie' appearance). Routine fundoscopy should be carried out in all HIV patients who have CD4 < 100. Treatment should start immediately with *ganciclovir + foscarnet* for 3 weeks, and maintenance continued till CD4 remains between 100–150 for 6 months; the major side effect of ganciclovir is myelotoxicity, and foscarnet is nephrotoxic.
3. Herpes simplex and the varicella zoster virus can cause acute retinal necrosis, which is painful and accompanied by uveitis/ keratitis.
4. Uveitis (especially when rifabutin has been used for treating AIDS + tuberculosis).

Skin and mucous membranes

1. Due to HIV itself—Seborrheic dermatitis, prurigo of HIV, intractable pruritus in infants, icthyosis, generalized dry and itchy skin, refractory psoriasis
2. **Eosinophilic folliculitis** is caused by a mite that presents as multiple, urticarial perifollicular papules that coalesce into plaque like lesions, and show an eosinophilic infiltrate of the hair follicle; treat with a topical antihelminthic drug.
3. **Herpes simplex** virus causes recurrent orolabial, genital and perianal warts that are red and exquisitely tender; treat with acyclovir.
4. Many infections, such as bacillary angiomatosis (*Bartonella quintana*), atypical mycobacterial infections, *Acanthamoeba castellanii* infection and Kaposi sarcoma present as erythematous nodules.

5. **Molluscum contagiosum** causes flesh colored umbilicated papules, and tend to regress with antiretroviral therapy.
6. Some rare fungi may infect the skin of HIV patients, like *Penicillium*
7. Scabies may turn very severe (Norwegian scabies) with hyperkeratotic psoriatsiform lesions.
8. Zoster may be the first sign of immunodeficiency, and it could be affecting more than one dermatone.
9. Every kind of **drug reaction** may occur in skin; while most of them are mild, severe ones such as **Stevens-Johnson syndrome** may be caused by cotrimoxazole, the nonnucleoside reverse transcriptase inhibitors and protease inhibitors.

Blood

1. Of course, lymphopenia is what HIV is all about.
2. **Anemia** may be caused by systemic fungal infections, drug induced myelosuppression, nutritional deficiency (specifically, B_{12} deficiency caused by achlorhydria and HIV enteropathy), and parvovirus B_{19} infection.
3. Isolated thrombocytopenia, may drop to dangerously low levels (< 10000)
4. **Pancytopenia**—It is caused by underlying cancers, MAC infections, disseminated cytomegalovirus infection, or due to side effects of drugs.
5. **Epstein Barr virus** can lead to some nonHodgkin's lymphoma, primary cerebral lymphoma.

Oral

- **Thrush** (due to *Candida albicans*) appears as a white, cheesy exudate, often on an erythematous mucosa in the oropharynx. The diagnosis is made by direct examination of a scraping for pseudohyphae.
- **Oral hairy leukoplakia** (due to Epstein Barr virus) is a whitish, frond like lesion along the lateral border of tongue, and often respond to podophyllin or anti herpes drugs.
- **Aphthous ulcers** have no known cause but respond to thalidomide.

Gastrointestinal

1. Esophagitis may be caused by *Candida*, cytomegalovirus (single large ulcer) or herpes simplex (multiple ulcers).
2. Achlorhydria, gastric lymphoma and Kaposi sarcoma of stomach.
3. **HIV enteropathy** = diarrhea + malabsorption (due to villus atrophy) + HIV infection of mucosa associated lymphoid tissue.
4. **Gastroenteritis** due to a large and diverse number of organisms.

Organism	Clinical course	Diagnosis	Treatment
Salmonella (the typhi, typhimurium or cholera suis species)	Fever, anorexia, fatigue, malaise and/or diarrhea	Culture of blood and stool	Long term ciprofloxacin
Shigella, particularly the flexneri species	Severe bloody diarrhea and/or bacteremia	Blood culture, stool examination	Ampicillin or Ciprofloxacin

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Organism	Clinical course	Diagnosis	Treatment
Campylobacter jejuni	Crampy pain, fever, bloody diarrhea, proctitis, bacteremia	Stool examination	Erythromycin
Mycobacterium tuberculosis and MAC	Abdominal pain and diarrhea	Peritoneal aspirate staining and culture, biopsy	Antitubercular treatment
Cryptosporidium parvum	Severe diarrhea in patients with CD4 < 300, cholecystitis	Acid fast cysts in stool	Nitazoxanide
Isospora belli	Diarrhea	Large acid fast cysts in stool	Cotrimoxazole
Cytomegalovirus	Colitis, diarrhea	Endoscopy and biopsy reveal intranuclear inclusion bodies	Ganciclovir or foscarnet; monitor for CMV retinitis

5. Perirectal ulcers and erosions due to herpes simplex virus, respond well to acyclovir.
6. **Hepatitis B** if often seen along with HIV infection (more so in IV drug abusers), and HIV patients are more prone to develop as chronic carriers of hepatitis B and less prone to develop immune mediated liver injury (i.e. fulminant hepatitis or cirrhosis); there is evidence that severity of hepatitis actually *increases* with antiretroviral therapy as the immune mechanism gets active; hepatitis C is also seen with increased severity in HIV patients.
7. Hepatitis due to other causes—MAC, Coccidioides immitis and Histoplama capsulatum, cytomegalovirus.
8. Many antiretrovirals (especially atazanavir and nevirapine) may cause fatal hepatic injury; didanosine causes acute pancreatitis.

Renal

1. **HIV nephropathy** shows early in the disease and causes nephrotic syndrome due to focal segmental glumerulosclerosis; after availability of antiretroviral therapy, it has become amenable to treatment with ACE inhibitors and/or prednisolone.
2. Drug-induced nephrotoxicity (foscarnet, amphotericin B, pentamidin, sulfadiazine, indinavir, adefovir, cidofovir, tenofovir).
3. **Urinary tract infections** are seen in increased frequency; **syphilis**, other than presenting in many atypical forms (such as a necrotising vasculitis in skin), also progresses to neurosyphilis (a once forgotten complication of syphilis) in 1% of HIV patients. In the HIV patient with syphilis, VDRL test in *unreliable* and dark field microscopy should be done.
4. **Vulvovaginal candidiasis** is common in HIV +ve women, as is *Trichomonas* infection.

Respiratory system

1. Acute bronchitis and sinusitis, due to the common organisms like *Streptococcus pneumoniae* or *Hemophilus influenzae*, or due to more uncommon ones, like mucors.
2. Lymphocytic *interstitial pneumonitis* is well documented in children but rare in adults; it is usually a self limiting condition, and rarely a clinical problem since introduction of antiretroviral therapy.
3. Bacterial (i.e. *Streptococcus pneumoniae* and *Hemophilus influenzae*) and fungal (*Aspergillus*, *Histoplasma*, *Coccidioides*, *Cryptococcus*) pneumonias; the pneumococcal vaccine is recommended for all HIV patients who still have a CD4 count < 200.
4. *Pneumocystis jiroveci* pneumonia is a marker of immunosuppression; the patient usually presents with vague chest symptoms, with no sputum, and in early cases X-ray may not show anything; only CT scan reveals a ground glass appearance. Definitive diagnosis requires demonstration of the organism in induced sputum, bronchoalveolar lavage or lung biopsy. Drug of choice, both for treatment and *prophylaxis*, is cotrimoxazole or IV pentamidine if patient cannot tolerate cotrimoxazole.

Chemoprophylaxis for Pneumocystis jiroveci infection

Indications. Any HIV-infected individual who has had a prior Pneumocystis jiroveci pneumonia, any patient with a CD4 count of < 200/μl or < 15% of all T-cells, patient with unexplained fever for > 2 weeks, or with a history of oropharyngeal candidiasis.
Regimen. Cotrimoxazole one double-strength tablet daily. This regimen also provides protection against toxoplasmosis and some bacterial respiratory pathogens. For patients who cannot tolerate TMP/SMX, alternatives are dapsone plus pyrimethamine plus leucovorin (tetrahydrofolic acid) or aerosolized pentamidine.

5. **Tuberculosis**, once thought to be extinct from the developed world, has been given a new lease by HIV; unlike atypical mycobacteria, *Mycobacterium tuberculosis* flares early in the course of HIV infection. Initially, when CD4 counts are moderately low (median 326/μl), tuberculosis presents in its pulmonary form. With progressive loss of CD4 cells, tuberculosis presents presents in more diffuse forms like miliary tuberculosis, mediastinal adenpathy, pleural effusion and extrapulmonary sites (lymph nodes, bone marrow, meninges, liver andd spleen)[426]; tuberculosis in HIV can't be cured , and in tuberculosis endemic country like ours, isoniazid prophylaxis is recommended for HIV patients.

Prophylaxis for tuberculosis in AIDS

Prophylaxis should be given to all HIV +ve patients with a tuberculin reaction > 5 mm (because of the immunosuppressive condition, we don't expect the 10 mm erythema, and 5 mm is taken for being a +ve reaction). Dosage: Isoniazid 300 mg daily × 9 months– 1 year

6. Atypical mycobacteria infections (especially those due to *Mycobacterium avium intracellulare* complex of organisms, or MAC) appears when patients

are profoundly immunosuppressed (CD4 < 50); clinical features are too vague and diagnosis by sputum smear microscopy is most sensitive. Treatment consists of a combination of clarithromycin + rifabutin + ethambutol, usually for life. However, if by anti retroviral therapy, the CD4 count can be raised to > 100 for more than 3–6 months, therapy for MAC can be discontinued.

Prophylaxis for MAC

Rifabutin may be used for prophylaxis in patients with CD4 count < 200.

Cardiovascular

1. **HIV cardiomyopathy** is a form of dilated cardiomyopathy, which presents as heart failure.
2. Antiretroviral drugs like nucleoside analogues can also cause cardiomyopathy, as well as opportunistic infections like cryptococcosis, toxoplasmosis and Chagas diseases, and Kaposi's sarcoma.
3. Hypercholesterolemia, atherosclerosis and coronary artery disease is seen to be associated with HIV infection, and more so with antiretroviral therapy.

Endocrine

1. **Lipodystrophy**, reminiscent of Cushing syndrome, may be seen anytime during HIV infection.
2. Advanced HIV disease may cause SIADH and hyponatremia.
3. Many factors in HIV contribute to **hypoadrenalism**, i.e. CMV, tuberculosis, cryptococcosis, histoplasmosis added to drug toxicity of antifungals (azoles).
4. Advanced HIV disease is associated with **hypogonadism** in ~50% of men, partly due to the disease itself and partly ganciclovir therapy.

Neoplasia

1. **Kaposi's sarcoma** is an cancer caused by human herpes virus 8 (HHV8), which is multicentric and pigmented, occurs almost exclusively in homosexual men and who have acquired HIV sexually. In addition to skin, it also affects lymph nodes and many internal organs (Fig. 9.90).

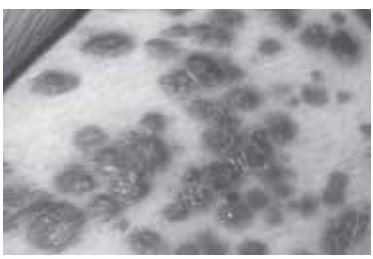

Figure 9.90. Kaposi sarcoma [US National Cancer Institute]

2. NonHodgkin's large B-cell *lymphomas* in lymph nodes, lung, GI tract and brain (primarily CNS lymphoma), strongly associated with Epstein Barr virus.
3. Squamous cell carcinomas, specially of cervix and anus (cased by papillomavirus).

WHO criteria for clinical diagnosis of HIV/ AIDS

In 1986 the WHO published the first set of criteria for clinical diagnosis of AIDS, known as the Bangui criteria.[427]

The Bangui Criteria, 1986

2 MAJOR SIGNS	+ 1 MINOR SIGN	=
weight loss ≥ 10% of body weight chronic diarrhea > 1 month fever > 1 month	cough > 1, generalized pruritic dermatitis zoster oropharyngeal candidiasis disseminated herpes simplex generalized lymphadenopathy	AIDS

Note that there are no investigations involved, that is these criteria apply whether an individual has tested HIV +ve or not. These criteria were specific, but not sensitive enough, that is many AIDS patients presented with neither of these signs but a different set of signs. For this reason, the criteria were modified in 1994 to publish the expanded case definition, which includes a labortaory test for HIV antibodies.

Expanded WHO definition for AIDS[428]

One or more in an HIV +ve person > 12 years of age

1. > 10% weight loss with diarrhea AND/ OR fever for at least a month
2. Cryptococcal meningitis
3. Pulmonary/extrapulmonary tuberculosis
4. Kaposi's sarcoma
5. Neurological impairment sufficient to impede daily activities
6. Esophageal candidiasis
7. Recurrent or life-threatening pneumonia
8. Invasive cervical cancer.

WHO clinical staging for patients with confirmed HIV infection[429]

	For adults and adolescents (> 15 years)	For children (< 15 years); only points that differ from adults are mentioned
Stage 1	• Asymptomatic • Persistent generalized lymphadenopathy	Same as adults
Stage 2	• **Moderate unexplained weight loss** (< 10% of body weight) • Recurrent upper respiratory tract infections • **Herpes zoster** • Angular cheilitis, oral ulcers • Papular pruritic eruptions • Seborrheic dermatitis • Fungal nail infections	• Recurrent or chronic **upper respiratory tract infections** (incuding otitis media • Unexplained persistent hepatosplenomegaly • Linear **gingival erythema** • Extensive **warts** • Extensive **molluscum contagiosum**

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		• Unexplained persistent **parotid enlargement** *Weight loss* is NOT a criteria for children; the rest of the criteria (oral ulcers, etc.) are same as adults
Stage 3	• **Unexplained severe weight loss** (> 10% of body weight) or **chronic diarrhea** (> 1 month) or **persistent fever** (above 37.6°C) for > 1 month • Persistent **oral candidiasis** or hairy leukoplakia • Pulmonary tuberculosis • Severe bacterial infections like osteomyelitis, pyomysitis, etc. • Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis • Unexplained anemia (< 8 g/dl), neutropenia (< 500 / µl) or chronic thrombocytopenia (< 50000)	• Unexplained **moderate malnutrition or wasting** or diarrhea (14 days or more) or **fever** (above 37.5°C) for > 1 month • Persistent oral **candidiasis** (*after first 6–8 weeks of life*) or hairy leukoplakia • **Lymph node tuberculosis** • Severe recurrent bacterial **pneumonia** • Symptomatic lymphoid **interstitial pneumonitis** • Chronic **HIV-associated lung disease** including bronchiectasis The rest same as adults (acute gingivitis, pulmonary tuberculosis, hematologic changes, etc).
Stage 4	• HIV wasting syndrome • Pneumocystis jiroveci pneumonia • Recurrent severe bacterial pneumonia • Chronic **herpes simplex** infection • **Candidiasis** of esophagus or respiratory tract • **Extrapulmonary** tuberculosis • **Kaposi's sarcoma** • Cytomegalovirus infection (especially retinitis) • Central nervous system **toxoplasmosis** • HIV encephalopathy • Extrapulmonary cryptococcosis including meningitis • Disseminated **nontuberculous mycobacterial** infection • **Progressive multifocal leukoencephalopathy** • Chronic **cryptosporidiosis** (with diarrhea) • Chronic **isosporiasis**	• Unexplained severe **wasting, stunting or severe malnutrition** • Recurrent **severe bacterial** infections *other than pneumonia* • **Cytomegalovirus** infection (specially retinitis) with onset at age older than one month • Central nervous system **toxoplasmosis** (*after one month of life*) • HIV encephalopathy The rest same as adults, *except* leishmaniasis, cervical carcinoma, nontyphoidal Salmonella are *not* included in criteria for children

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	• Disseminated mycosis (coccidiomycosis or histoplasmosis) • Recurrent **nontyphoidal Salmonella** bacteraemia • **Lymphoma** (cerebral or B-cell nonHodgkin) or other solid HIV-associated tumors • Invasive **cervical carcinoma** • Atypical disseminated **leishmaniasis** • Symptomatic **HIV-associated** nephropathy cardiomyopathy	

Labs

Detection of antibodies

As noted earlier, antibodies to p24 appear 2–12 weeks after exposure, which can be detected by an **ELISA** test (> 99% *sensitive* but not very specific). For screening purposes, ELISA can be used. Those positive on ELISA can be subjected to a more *specific* test, which is the **Western Blot**. The Western Blot is considered +ve is antibodies exist to two of the three antigens p24, gp41 and gp120/160. When combines with ELISA, the Western blot has a specificity > 99.99% in detecting HIV infection. However, it can produce results that are indeterminate (i.e. only one antibody found, or antibodies to some other viral component like p31 being found) in early HIV infection, presence of autoimmune disease, pregnancy and recent tetanus toxoid injection.

Detection of viral components

The viral RNA can be detected by RT-PCR, and is the usual indicator for viral load. The *p24 antigen* (marker for viral *replication*) itself can be detected by ELISA.

Prognostic marker

The CD4 + T-cell count is done by flow cytometric analysis. Normally, there are 950 CD4+T-cells per µl (thats a microliter, or mm^3). The CD4 counts gives the present risk of opportunistic infections and also measures the response to therapy. Different opportunistic infections show up at different CD4 counts. Some like MAC infections, primary CNS lymphoma (due to Epstein Barr virus) and CMV retinitis occur only when CD4 count is very low (< 50). Others like tuberculosis, herpes simplex, candidiasis, hairy leukoplakia and Kaposi sarcoma are very early features of immunosuppression (occur even when CD4 count is > 200).

The **percentage** of CD4+T-cells among all lymphocytes is considered more useful for predicting risk, and is normal if > 20%.

WHO case definition for HIV infection[430]

For purpose of HIV surveillance, WHO has published standard case definitions. However, there are for purpose of surveillance only, and not for clinicians, who might prefer many other methods for diagnosis.

Adults and children 18 months or older

- Positive HIV antibody testing (rapid testing kit or ELISA).
- Confirmed by a second HIV antibody test (rapid testing kit or ELISA) against a different antigen.
- AND/ OR positive virological test for HIV or its components (HIV-RNA or HIV-DNA or ultrasensitive HIV p24 antigen).

Children younger than 18 months

Positive virological test for HIV or its components (HIV-RNA or HIV-DNA or ultrasensitive HIV p24 antigen) taken more than four weeks after birth. HIV antibody testing is not necessary for children.

Strategy for testing in India

There are three levels of testing

I. Single rapid ELISA with highest sensitivity
II. If I is reactive, second ELISA with a different kit
III. If II reactive again, third ELISA with highest *specificity*.

Purpose	Prevalence of AIDS in the area	Testing to be done upto level
Screening of blood for transfusion	Any	I
Surveillance	> 10% ≤ 10%	I II
For diagnosis of HIV in symptomatic patient	> 30% ≤ 30%	I II
For diagnosis of HIV in asymptomatic	> 10% < 10%	II III

Treatment

There are several parts in treatment of HIV/AIDS

1. Antiretroviral therapy
2. Treating opportunistic infections, and chemoprophylaxis (i.e. for *Pneumocystis jiroveci* and mycobacteria)
3. Counseling the patient and raising his morale, knowing the disease can only be slowed down, not cured.

Potential problems in treating antiretroviral therapy

Adverse drug recations—Liver, kidney and bone marrow toxicity.
Immune reconstitution phenomenon (disease aggravates after antiretroviral therapy due to the immune system regaining power and causing immune mediated damage) specially with Mycobacterial illnesses.
Dementia which makes compliance difficult to achieve.
Drug interactions between antiretroviral drugs themselves and with other classes of drugs (i.e. antitubercular drugs).

Antiretroviral drugs

Nucleoside reverse transcriptase inhibitors (NRTI)

Zidovudine (azidothymidine or AZT), didanosine (ddl), lamivudine (3TC), stavudine (d4T), zalcitabine (ddC) and abacavir (ABC) are nucleoside look alikes who inhibit the viral reverse transcriptase enzyme.
Dosage. AZT 300 mg × 2 daily or 3TC 150 mg × 2 daily.
Adverse effects. All of these drugs cause bone marrow suppression, lactic acidosis, steatosis, headache, nausea, fatigue. In addition, both AZT and 3TC may cause pancreatitis, a tendancy most seen with ddl. d4T causes a sensory neuropathy and hepatotoxocity which may become severe.

Protease inhibitors (PI)

Amprenavir, saquinavir, ritonavir. indinavir, nelfinavir and lopinavir inhibit the protease that cleaves out viral proteins from a single polypeptide coded by the RNA. All of them cause hyperlipidemia.

Nonnucleotide reverse transcriptase inhibitors (NNRTI)

These include nevirapine (NVP), delavirdine, efavirenz (EFV). All of them causes skin rash. Nevirapine is hepatotoxic, allergic (may cause Stevens-Johnson syndrome) and efavirenz may cause neural tube defects.
Dosage. Nevirapine 200 mg oral daily × 2 weeks + 200 mg oral × 2 contd. and Efavirenz 600 mg oral daily.

WHO guidelines of antiretroviral treatment[431]

Principles

1. Do not harm
2. Ensure access and quality
3. Promote quality and efficiency
4. Ensure sustainability.

When to start anti retroviral treatment (ART)

The timing of therapy should be as early as to prevent progression of disease and death, but not so early that the individual suffers unnecessary reactions to drugs. Because WHO stages 1 and 2 have uncertain prognosis, these patients should be given ART only after CD4 count.

WHO clinical stage	CD4 count / µl	What to do
Adults and adolescents		
Any	≤ 350	Start treatment
1 and 2	≤ 350 Unavailable	Start treatment Do not start treatment
3 and 4	Any	Start treatment
Co-infections		
Any stage + co-infected with active tuberculosis	Any	Start treatment; anti-tubercular treatment first, followed by ART

Contd...

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WHO clinical stage	CD4 count / µl	What to do
Any stage + co-infected with hepatitis B	Any	Start treatment
In pregnancy		
Any	≤ 350	Start treatment
1 and 2	≤ 350	Start treatment
	Not available	Do not start treatment
3 and 4	Any	Start treatment
Infants and children (< 15 years)[a]		
4	Any	Start treatment
3[b]	Any	Start treatment
2	CD4 or WBC count less than normal	Start treatment
1	CD4 less than normal	Start treatment
[a] Antiretroviral therapy of HIV infection in infants and children: Towards universal access: Recommendations for a public health approach, WHO 2006 [b] For children over 12 months of age, treatment may be delayed in wait for CD4 count		

Table 9.38. Cut off values of CD4 cells in pediatric age group

	< 11 months	12–35 months	36–59 months	> 5 years
CD4 percentage	< 25%	< 20%	< 15%	< 15%
CD4 count	< 1500	< 750	< 350	< 200

What to start

One of the following regimens

- AZT + 3TC + (EFV or NVP)
- TDF + (3TC or FTC) + (EFV or NVP)

WHO has dealt away with stavudine (d4T) based regimens because of disfiguring and potentially life-threatening toxicity of the drug.

Co-infections.

- In patients *co-infected with tuberculosis*, use efavirenz as the NNRTI.
- In patinets *co-infected with hepatitis B*, use a TDF + (3TC or FTC) containing regimen.

Pregnancy. The regular regimens can be started, except that efavirenz should be avoided in the first trimester which may cause neural tube defects.

When to change ART?

Where available, test viral load every 6 months; a **persistent viral load > 5000 copies/ml** confirms *failure* of treatment.

Second line ART

Use of a boosted protease inhibitor (ATV or LPV) + 2 NRTIs.

What to use in children and infants?

Start with combination of (AZT or d4T or ABC) + 3TC + (NVP or EFV), that is a combination of 2 NRTI and one NNRTI. To reduce toxicity of therapy, and because NVP can not be given in patients co-infected with tuberculosis (it interacts with rifampiciin), a combination of 3 NRTI can also be used, i.e. (AZT + d4T) + 3TC + ABC.

Treatment of HIV infected pregnant women and prevention of transmission to newborn[432]

Which pregnant women need ART?

1. Women with HIV infection in need of ART (see the table earlier) need lifelong ART irrespective of pregnancy.
2. Any HIV infected pregnant woman with CD4 < 350 should receive ART irrespective of clinical stage.
3. Any HIV infected pregnant woman in clinical stage 3 or 4 should receive ART irrespective of CD4 count.

When to start and how long to continue

Pregnant women who need ART can start it anytime during pregnancy and continue lifelong. The drug regimens to be used are shown earlier.

What to do with the infant born with the HIV +ve mother who has been on ART? (Fig. 9.91)

If the mother is breastfeeding. Give the infant NVP daily until 6 *weeks of age*. In this case, because the mother has received full ART during treatment, NVP does not need to be given to the infant for more than 6 weeks. The risk of HIV transmission through breastmilk, though exists, is smaller than the risk of not breastfeeding at all, which could be disastrous for the heath of the child, especially in developing countries.

If the mother is not breastfeeding. Give the infant AZT or NVP daily for 6 weeks. For mother who have received *4 weeks or less* of ART during antenatal period, AZT is the drug of choice for the infant.

What if the mother herself does not need ART, but only needs prophylaxis for mother to child transmission

Ideally, antiretroviral prophylaxis (ARV) should be started at 14 weeks of pregnancy, or whenever the woman comes to a health facility. The ARV prophylaxis consists of

- Daily AZT
- NVP at onset of labor
- AZT + 3TC during labor and delivery
- AZT + 3TC for 7 days postpartum.

If the mother will breastfeed, the infant needs NVP daily until 1 week after completion of breastfeeding. If she will not breastfeed, however, the infants should be given AZT or NVP for 6 weeks.

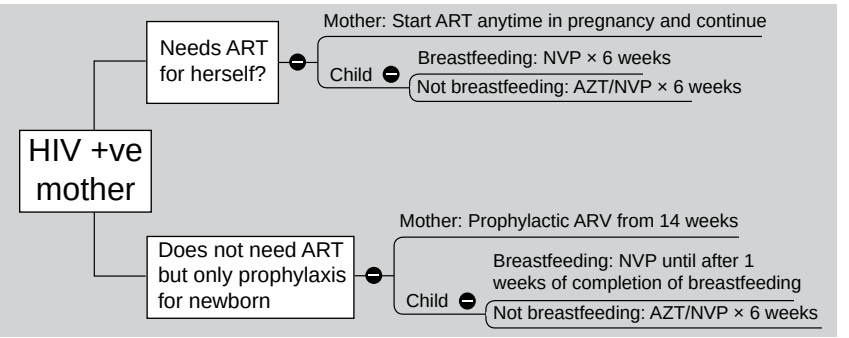

Figure 9.91. The HIV +ve mother

Monitoring therapy

1. Clinical improvement
2. WBC count
3. CD4 count
4. Viral load (HIV RNA copies in plasma).

Control

Controlling the reservoir

- Treatment of cases with antiretroviral drugs
- Preventing HIV +ve cases to donate blood, have sex or entering drug user communities.

Breaking the chain

- **Blood safety**—People in high risk groups should be urged to refrain from donating blood (or any other tissue, for that matter), at all. All blood should be screened for HIV1 and HIV2.
- Prevention of mother to child transmission (see later).
- **Universal precautions**—Strict sterilization and asepsis must be maintained in all health care facilities (see 'universal precaution' at the beginning of this chapter), injections kept to a minimum (when absolutely necessary) and preference of IV fluids rather than blood transfusion.

Protecting the host

Education. The awareness of HIV (and if necessary, the *fear* of it) must be impressed upon everybody; stress upon use of condoms during sex, to stick to one sexual partner (however boring he or she may be), refraining from sharing needles/ razors/ toothbrushes, using only sterilized instruments for piercing and tattooing. HIV +ve women should be taught not to get pregnant at all. Education campaigns should take advantage of all available media.

Vaccine. There is NO vaccine yet. However, several 'candidate' vaccines are in line, of which gp120 subunit vaccine is in trial. POSTEXPOSURE prophylaxis seems to be more important than preexposure.

Postexposure prophylaxis[433]

As interns and house physicians, chances are that you will prick yourselves with a needle sometime during patient care. Without panicking, this is what you need to do.

First aid. Immediately wash skin water and soap, and rinse. Do not scrub. Do *not* use antiseptics or skin washes (bleach, chlorine, alcohol, betadine). If there has been a splash on the eye, irrigate with water or normal saline. Do not panic. DO NOT PUT PRICKED FINGER IN MOUTH (you guessed that, right!)

Do you need PEP for HIV? A significant exposure from a HIV +ve person (or of unknown HIV status) needs to be considered for PEP within 72 hours of exposure. No laboratory testing is needed for initiation of PEP.

Drugs. Two NRTI (AZT + 3TC) for 28 days; add a protease inhibitor if drug resistance is likely.

HIV testing. Two tests, one just after exposure and another after 3–6 months. Also, check the patient's blood (the blood of the one you have been pricked with) for HIV and hepatitis B and C.

National AIDS control program

The AIDS task force was formed in 1985, even before the 1st case in India. NACP was lauched in 1987, and two separate wings NACO (central, 1992) and State Aids Control Organization (SACS) were launched later. After phase I and II, NACP III was lauched in 2007.

NACP III (2007–2012)

Goal

To halt and reverse the epidemic in India over the next 5 years.

Objectives

1. To reduce incidence by 60% in the first year in high prevalence state, to revert the epidemic
2. To reduce incidence by 40% in the vulnerable states, to stabilize the epidemic.

The major activities of NACP II are as follows.

Targeted intervention to high risk groups

1. Behavior change communication—To abstain from rash sexual acts and not to share needles
2. Condom promotion
3. Treatment of STDs.

After the high risk groups, the next priority of NACP are the *bridging population*.

Blood safety

The **National Blood Policy** (2002) recommends the following

1. Ban on 'professional' donors, who have great chance of being infected; such donation is banned since 1998, and only voluntary donation is allowed (i.e. you cannot sell your blood).

2. Mandatory HIV, hepatitis B and C, malaria and VDRL testing of blood which is for transfusion.
3. Only licensed blood banks are allowed to collect blood.
4. Better utilization of blood by component separation.

2177 licensed blood banks and 82 components separation units now exist in India.

Education to general population

Mass education is the only way to achieve a behavioral change, generating demand for services (i.e. condoms, VCTC), and removing myths and stigma about HIV. The NACO and SACS have occupied every kind of media (electronic, print and folk media) in India for many years now, and 'Buladi' from the West Bengal SACS) is one of the most successful educational campaigns. The **World AIDS day** is celebrated 1st December each year. A nationwide AIDS helpline has also been set up (call 1097 toll free!).

School AIDS education

Quiescent for many years, 'sex education' has finally surfaced in Indian schools in a training module called 'learning for life', which is to be implemented by the SACS. In colleges and universities, the 'Talk AIDS' project aims to open up voices on HIV/AIDS among the youth, who are often clueless about it even at the beginning of their sex life.

Control STDs

Not only does an STD increase chance of HIV infection from contact, STDs are linked with HIV epidemiologically and behaviorally. The **syndromic approach** should be integrated into primary health care to deal with STDs, and set up STD clinics in district/block hospitals and all medical colleges.

Condom promotion

Hetrosexual sex is commonest mode of HIV transmission in India, and never forget that condoms, in addition to protecting against STDs and HIV, are also contraceptives. The NACP is distributing WHO specified condoms by both *social marketing* (see the chapter on sociology) and free distribution (condoms are kept in open boxes outside every health facility to be picked up at your will). In high risk sites (i.e. colonies of commercial sex providers), condom dispensing machines are placed at phone booths.

Family heath awareness campaigns

FHAC targets to raise the awareness of HIV/AIDS in rural and slum areas, to promote use of only safe blood, to tell that HIV can spread from mother to her newborn, and about the services provided by NACP. 15 days campaigns are held by each state according to convenience.

Voluntary counseling and testing (VCT)

It is the most enduring way to change the behavior and remove stigma. A VCT center is the place to go if you ever suspect that you may have HIV, either because of your symptoms or your risky behavior. The VCTC screens for HIV infection.

Pretest counseling

1. Personal, sexual history and assessment of risk
2. Assessment of related factors and knowledge of HIV.

Posttest counseling

- For the HIV –ve:
  - the patient may have come in the window period (try a second test after sometime)
  - promote safe behavior anyway
- For HIV +ve:
  - Explain HIV, difference between HIV infection and AIDS
  - Counseling (how to live life with HIV)
  - Referral for antiretroviral treatment
  - Maintain confidentiality.

Integrated Counseling and Testing Centers have been set up in all medical colleges, district and subdistrict hospitals. In addition to functioning as VCTCs, they also provide single doses of NVP to HIV +ve mother at the time of labor, and to the newborn.

Antiretroviral treatment

The NACP provides free ART at Government hospitals of high prevalence states (TN, Andhra, Maharashtra, Karnataka, Manipur, Nagaland and in Delhi). The priority are under 15 children, pregnant women and AIDS cases.

Prevention of parent to child transmission (PPTCT) in HIV +ve mothers

The PPTCT program was started in the country in the year 2002.

1. Proper **counseling** regarding high risk behavior in antenatal clinic (IV drug use, smoking which causes early rupture of membrane)
2. Prevent **early rupture of membrane during labor**
3. Periodic cleansing of birth canal by iodine
4. Properly done Caesarean section is safe, but if there's not adequate prepartion, go for vaginal delivery if Caesarean is not absolutely necessary
5. Early cleansing of baby and **bath** (differs from routine neonatal care)
6. Use of breast milk substitutes, if possible
7. Chemoprophylaxis (see WHO guidelines earlier).

Surveillance

HIV Sentinel Surveillance

HIV sentinel surveillance means carrying out cross-sectional studies (also known as prevalence surveys) of HIV prevalence rates at regular intervals among selected groups in the population known as "Sentinel groups". HIV sentinel surveillance can be either community-based or clinic/health facility-based; the latter is much more convenient and hence is always preferred.

1. High-risk groups—Injectable drug users, truck drivers, STD clinics, men who have sex with men (250 patients).

2. General population—Antenatal clinics (250 patients). The results from antenatal clinics will not be fruitful if the HIV prevalence in high risk group of the state is < 2%. In such a case, *donated blood* may serve as a source for sentinel surveillance.

Each year the sentinel survey is carried out by NACO from 1st August to 31st October. 2 ELISA tests with better specificity is used for the purpose. The whole thing must be done in absolute anonymity and blind manner.

AIDS Case Surveillance

It is hospital/ health provider based, and depends on reporting of diagnosed AIDS cases.

STD Surveillance

Incidence of STD may give an indication of HIV infection because

1. STDs and HIV infect the same group of people
2. STD is itself a risk for HIV infection
3. STD clinics are an important sources to get HIV patients who have come for some other disease without knowing that they may have HIV
4. STD is easier monitored than HIV; it is a surrogate indicator.

Behavioral Surveillance

To know how much people know about HIV/AIDS, and whether they have modified their lifestyles accordingly, and to what extent.

Appendix

APPENDIX A: WORKOUT

AWW = Anganwadi worker, MPHW = Multipurpose health worker.

Six months old Priya is having loose motions for one day; she is of normal nutritional status. At present she is alert, thirsty, skin turgor is reduced and she is able to drink.

She has **some dehydration**. She should be kept under observation in a Diarrhea Treatment and Training Center.

Fluids

ORS 400–600 ml in 1st 4 hours and then recategorize.
1. Improves—ORS at maintenance dose (50–150 ml after each stool)
2. Same—Continue for 4 more hours
3. Deteriorates—IV fluids.

Food

Normal feeding, continue breastfeeding, ↑ during convalescence.

Drugs

None.

Education to mother

Signs of severe dehydration
ORS preparation and use
Follow up at 5 days.

Raju, a seven months old boy is brought to subcenter because he has passed stool 5 times since morning; he is of normal nutritional status, alert, normal skin turgor, normal thirst, eyes are not shrunken.

He is having no dehydration. He needs oral rehydration therapy (ORS + feeding).

Fluids

Plenty of home available fluid to prevent dehydration, frequently and also after each stool. Provide mother with ORS and advice on preparation (50–100 ml after each liquid stool).

Feeding

Breastfeeding, normal feeding, increase during convalescence.

Drugs

None.

Education to mother

Same as above.

Measures to prevent future occurrence of diarrhea

Measles vaccination
Personal hygiene
Use freshly prepared food
Supply of potable water
Proper excreta disposal.

Shyama (30) has come to subcenter for family planning advice; she has a daughter of 8 months. Her husband Kanai is an alcoholic and beats her regularly. Shyama has suffered from jaundice twice in last year. Her menstrual history is normal.

She needs an IUD (copper T). Ideal time for insertion is within 10 days of first day of last menstrual period. She must follow up regularly to check displacement/ bleeding and for reassurance.

Methods not suitable

Condoms (because of husband)
OCPs (jaundice)
Sterilization (she has only one child).

A couple has come to postpartum unit for family planning advice. the husband is 24-year-old and the wife is 20. Their only child is a 5 months old baby girl, who is exclusively breastfed. But on questioning, the wife gives history of ectopic pregnancy.

Condoms are suitable for this couple.

Methods not suitable

IUD (ectopic pregnancy)
OCP (the mother is lactating)
Sterilization (they have only one child).

How to improve efficacy of condom

Spermicidal jelly
Consistent use.

Disadvantages of condoms

Failure 2–20/100 woman year
Needs continuous motivation.

An eleven months old boy presented at subcenter with bilateral pearly white foamy triangular spots on bulbar conjunctiva.

Management

Vitamin A in oil 1 lakh IU stat + 2 lakh IU after 4 weeks (the boy will be 12 months old after a month, so 2 lakh IU has to be given then) + continue prophylactic schedule (doses at 6 months interval until 36 months).
Measles vaccination
Vitamin A rich food
Correct any coexisting malnutrition.

Six months old Mohan's mother gives history of cough for 2 days. Respiratory rate is 42/min; temperature is normal; no stridor/ wheeze/chest indrawing/malnutrition.

He is suffering from no pneumonia–cough and cold.

Drugs

None (probably viral infection).

Symptomatic treatment and care at home

Home remedies (honey, tulsi) to soothe the throat
Clear the nose by saline if nose is blocked (no artificial nose drops)
Continue breastfeeding and normal feeding and increase during convalescence.
Educate mother of danger signs.

Jennifer (10 months) has cough and dyspnea since 3 days and not eating well but drinking alright; respiratory rate is 54/min and temperature 39.4°C. She has no chest indrawing/ stridor/wheeze/ unconsciousness. Her nutritional status is normal.

She is suffering from pneumonia (only tachypnea present, no other signs).

Drugs

Cotrimoxazole (100:20) 2 tablets × 2 × 2 days and then reassess.

1. Deteriorates—Refer to FRU
2. Same—Continue for 2 more days, and then if it is worsening, refer
3. Improves—Continue 5 day course.

Paracetamol may be given for fever.

Symptomatic treatment and care at home/health facility

Same as above.

Advice to mother

Identify danger signs.

Satish is the son of Pradip and Dipali who have recently come to the village. Satish was born on July 2000 and his weight was as follows: July 3.25 kg, August 4 kg, September 4.5 kg.

Comments on the growth chart of Satish

Birth weight normal
Normal growth pattern (direction upwards, over 80% of standard)
Current weight 4.5 kg
Monitoring has been regular.

What the AWW must tell to the mother

Exclusive breastfeeding
Begin complementary feeding at 6 months, increase feeding during illness
To complete primary immunization in time
Family planning (cafeteria approach)
Continue regular growth monitoring.

Mozammel was born on June 2000 with 2.8 kg. His weight was recorded twice in August and October, and was 4.8 kg on both occasions.

Comments on the growth chart of Vishwanath

Normal birth weight
Growth was normal earlier, now static and entering grade I malnutrition (direction rising → horizontal)
Current weight is 4.8 kg
Monitoring has been irregular.

What the AWW must tell the mother

Exclusive breastfeeding
Observe and teach good breastfeeding practices
Start complementary feeding from 6 months, increase feeding during convalescence
Regularize growth monitoring
Immunization.

The frozen ice pack in your vaccine carrier has melted within 2 hours.

Causes

Repeated opening
Lid was not properly closed
Crack in the carrier
Kept in direct sun.

What to do

Train staff better
Discard vaccines.

Shibani (24), 2nd gravida, delivered a boy at home with an untrained dai, with no antenatal care. 5 days later, the boy stops sucking, develops trismus, convulsions and dies in 2 days.

He had neonatal tetanus (points in favor—no antenatal care, 5 day onset, untrained dai, home delivery, trismus, convulsion).

Immunization that should have been taken by mother

TT1 at first contact + TT2 4 weeks later, dosage completed before 4 weeks of expected date of delivery. If documented history of complete immunization within 3 years is present → 1 booster dose is required only.

Measures during delivery that should have been taken

5 cleans (hands, surface, blade, cord tie, cord stump)
Trained personnel (institution/home).

Bakul, a primigravida, registers to health center at 28 weeks with a weight of 50 kg, BP 120/80, Hb 9 g%.

Not an ideal time to register (beyond 16 weeks).
She has anemia.

Treatment

IFA (100: 500) × 2 × 100
Diet: Green leafy vegetables, animal flesh, jaggery, more lemon; do not take tea/coffee/milk before meals (which inhibit iron absorption).

A child ages 6 months (nonimmunized) reports to the sub center? what immunization schedule is recommended till the age of 15?

Because the child is still not immunized, OPV-1, BCG and DPT-1 may be given together on this visit. Subsequent doses follow at 4 weeks intervals and from 9 months, takes the course of National Immunization Schedule.

6 months (today)	OPV-1, BCG, DPT-1, Hepatitis -1
7 months	OPV-2, DPT-2, Hepatitis-2
8 months	OPV-3, DPT-3, Hepatitis-3
9 months	Measles
18 months	OPV-booster, DPT-booster
5–6 years	DT-booster
10 years	TT-booster
15 years	TT-booster

Aziz, a 6 years old boy has come to PHC with no history of previous immunization; what vaccines does he need now?

It is imperative that the child has already some tuberculosis exposure. It is best to do Mantoux test and then give BCG.

Vaccines to be given on the very day (remember that several live vaccines can be given on same day at different sites)	BCG if Mantoux negative OPV-1, DT-1, Hepatitis-1 Measles If acellular pertussis vaccine is available, DTaP may be given in three doses at 4 weeks interval
4 weeks later	OPV-2, DT-2, Hepatitis-2
4 weeks later	OPV-3, Hepatitis-3
10 years	TT-booster
15 years	TT-booster

Manisha, a 12 years old girl has come to PHC with no history of previous immunization; what vaccines does she need now?

BCG and OPV can be skipped because the child is assumed to be exposed to these organisms already

1. dT (adult type diphtheria, tetanus vaccine) 2 doses at 4 weekly intervals and a booster dose 6 months – 1 year later
2. Hepatitis B, 3 doses at 0, 1 and 6 months
3. Measles vaccine single dose; it will be useful to give.
   MMR if it is available (the rubella component because the child is a girl).

The vaccines are all started on the very day of reporting.

In November 2000, the dai told AWW that Rama (19) gave birth to her third girl named Neeta 2 days ago. Rama was married at 15 and made her second delivery in November 1999. Neeta now weighs 2.3 kg.

Causes of LBW

Teenage pregnancy
Too many pregnancies
Too frequent pregnancies.

What the AWW should discuss

Exclusive breastfeeding
Immunization
Keep the baby warm
Minimum handling of baby (to prevent infections)
Family planning.

Ganesh (40) has 2 hypopigmented patches for last 1 year. They are anesthetic, well-circumscribed. His left ulnar nerve is thickened.

He has PB leprosy.

Treatment

Dapsone 100 mg × 1 × 6 months

Supervized: Dapsone 100 mg + Rifampicin 600 mg once monthly for 6 months.

Allow his daily business

PB is noninfective
Isolation is not desirable.

ASSESS AND CLASSIFY THE SICK CHILD AGED 2 MONTHS UPTO 5 YEARS

ASSESS

ASK THE MOTHER WHAT THE CHILD'S PROBLEMS ARE

- Determine whether this is an initial or follow-up visit for this problem.
  - If follow-up visit, use the follow-up instructions on *TREAT THE CHILD* chart
  - If initial visit, assess the child as follows:

CHECK FOR GENERAL DANGER SIGNS

ASK:	*LOOK*:
• Is the child able to drink or breastfeed?	• See if the child is lethargic or unconscious.
• Does the child vomit everything?	• Is the child convulsing now?
• Has the child had convulsions?	

A child with any general danger sign needs *URGENT* attention; complete the assessment and any prereferral treatment immediately so that referral is not delayed.

THEN ASK ABOUT MAIN SYMPTOMS:

Does the child have cough or difficult breathing?

IF YES, ASK:	LOOK, LISTEN, FEEL:	
• For how long?	• Count the breaths in one minute. • Look for chest indrawing. • Look and listen for stridor. • Look and listen for wheezing.	CHILD MUST BE CALM

If wheezing and either fast breathing or chest indrawing: Give a trial of rapid acting inhaled bronchodilator for upto three times 15–20 minutes apart. Count the breaths and look for chest indrawing again, and then classify.

Classify COUGH or DIFFICULT BREATHING

If the child is:	Fast breathing is:
2 months upto 12 months	**50** breaths per minute or more
12 months upto 5 years	**40** breaths per minute or more

CLASSIFY

USE ALL BOXES THAT MATCH THE CHILD'S SYMPTOMS AND PROBLEMS TO CLASSIFY THE ILLNESS.

IDENTIFY TREATMENT

SIGNS	CLASSIFY AS	TREATMENT (Urgent pre-referral treatments are in bold print)
• Any general danger sign or • Chest indrawing or • Stridor in a calm child	**SEVERE PNEUMONIA OR VERY SEVERE DISEASE**	♦ **Give first dose of an appropriate antibiotic** ♦ **Refer URGENTLY to hospital***
• Fast breathing	**PNEUMONIA**	♦ **Give oral antibiotic for 3 days** ♦ If wheezing (even if it disappeared after rapidly acting bronchodilator) give an inhaled bronchodilator for 5 days** ♦ Soothe the throat and relieve the cough with a safe remedy ♦ If coughing for more than 3 weeks or if having recurrent wheezing, refer for assessment for TB or asthma ♦ Advise the mother when to return immediately ♦ Follow-up in 2 days
• No signs of pneumonia or very severe disease	**COUGH OR COLD**	♦ If wheezing (even if it disappeared after rapidly acting bronchodilator) give an inhaled bronchodilator for 5 days** ♦ Soothe the throat and relieve the cough with a safe remedy ♦ If coughing for more than 3 weeks or if having recurrent wheezing, refer for assessment for TB or asthma ♦ Advise the mother when to return immediately ♦ Follow-up in 5 days if not improving

* If referral is not possible , manage the child as described in ***Integrated Management of Childhood Illness***, Treat the Child, Annex: Where Referral is Not Possible, and WHO guidelines for inpatient care.

** In settings where inhaled bronchodilator is not available, oral salbutamol may be the second choice

Does the child have diarrhea?

IF YES, ASK:

- For how long?
- Is there blood in the stool?

LOOK AND FEEL:

- Look at the child's general condition, is the child
  - — lethargic or unconscious?
  - — restless and irritable?
- Look for sunken eyes.
- Offer the child fluid, is the child
  - — not able to drink or drinking poorly?
  - — drinking eagerly, thirsty?
- Pinch the skin of the abdomen. Does it go back
  - — very slowly (longer than 2 seconds)?
  - — slowly?

Classify DIARRHEA

for DEHYDRATION		
Two of the following signs: • Lethargic or unconscious • Sunken eyes • Not able to drink or drinking poorly • Skin pinch goes back very slowly	**SEVERE DEHYDRATION**	♦ If child has no other severe classification: — Give fluid for severe dehydration (Plan C) OR ***If child also has another severe classification:*** — ***Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way*** — ***Advise the mother to continue breastfeeding*** ♦ ***If child is 2 years or older and there is cholera in your area, give antibiotic for cholera***
Two of the following signs: • Restless, irritable • Sunken eyes • Drinks eagerly, thirsty • Skin pinch goes back slowly	**SOME DEHYDRATION**	♦ Give fluid, zinc supplements and food for some dehydration (Plan B) ♦ ***If child also has a severe classification:*** — ***Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way*** ***Advise the mother to continue breastfeeding*** ♦ Advise mother when to return immediately ♦ Follow-up in 5 days if not improving
Not enough signs to classify as some or severe dehydration	**NO DEHYDRATION**	♦ Give fluid, zinc supplements and food to treat diarrhea at home (Plan A) ♦ Advise mother when to return immediately ♦ Follow-up in 5 days if not improving
and if diarrhea for 14 days or more		
• Dehydration present	**SEVERE PERSISTENT DIARRHEA**	♦ Treat dehydration before referral unless the child has another severe classification ♦ Refer to hospital
• No dehydration	**PERSISTENT DIARRHEA**	♦ Advise the mother on feeding a child who has PERSISTENT DIARRHEA ♦ Give multivitamins and minerals (including zinc) for 14 days ♦ Follow up in 5 days
and if blood in stool		
• Blood in the stool	**DYSENTERY**	♦ ***Give ciprofloxacin for 3 days*** ♦ Follow-up in 2 days

Does the child have fever?

(by history or feels hot or temperature 37.5°C** or above)

IF YES:
Decide Malaria Risk: High or low

THEN ASK:	***LOOK AND FEEL:***
• For how long?	• Look or feel for stiff neck
• If more than 7 days, has fever been present everyday?	• Look for runny nose
• Has the child had measles within the last 3 months?	Look for signs of MEASLES • Generalized rash and • One of these: Cough, runny nose, or red eyes

If the child has measles now or within the last 3 months:
- Look for mouth ulcers. Are they deep and extensive?
- Look for pus draining from the eye
- Look for clouding of the cornea

High Malaria Risk / ***Low Malaria Risk*** / ***Classify FEVER*** / ***If MEASLES now or within last 3 months, Classify***

HIGH MALARIA RISK

• Any general danger sign or • Stiff neck	**VERY SEVERE FEBRILE DISEASE**	♦ ***Give quinine for severe malaria (first dose)*** ♦ ***Give first dose of an appropriate antibiotic*** ♦ ***Treat the child to prevent low blood sugar*** ♦ ***Give one dose of paracetamol in clinic for high fever (38.5°C or above)*** ♦ ***Refer URGENTLY to hospital***
• Fever (by history or feels hot or temperature 37.5°C** or above)	**MALARIA**	♦ ***Give oral co-artermether or other recommended antimalarial*** ♦ ***Give one dose of paracetamol in clinic for high fever (38.5°C or above)*** ♦ Advise mother when to return immediately ♦ Follow-up in 2 days if fever persists ♦ If fever is present everyday for more than 7 days, rever for assessment

LOW MALARIA RISK

• Any general danger sign or • Stiff neck	**VERY SEVERE FEBRILE DISEASE**	♦ ***Give quinine for severe malaria (first dose) unless on malaria risk*** ♦ ***Give first dose of an appropriate antibiotic*** ♦ ***Treat the child to prevent low blood sugar*** ♦ ***Give one dose of paracetamol in clinic for high fever (38.5°C or above)*** ♦ ***Refer URGENTLY to hospital***
• No runny nose and No measles and No other causes of fever	**MALARIA**	♦ ***Give oral co-artermether or other recommended antimalarial*** ♦ ***Give one dose of paracetamol in clinic for high fever (38.5°C or above)*** ♦ Advise mother when to return immediately ♦ Follow-up in 2 days if fever persists ♦ If fever is present everyday for more than 7 days, refer for assessment
• Runny nose PRESENT or • Measles PRESENT or • Other causes of fever PRESENT	**FEVER MALARIA UNLIKELY**	♦ ***Give one dose of paracetamol in clinic for high fever (38.5°C or above)*** ♦ Advise mother when to return immediately ♦ Follow-up in 2 days if fever persists ♦ If fever is present everyday for more than 7 days, refer for assessment
• Any general danger sign or • Clouding of cornea or • Deep or extensive mouth ulcers	**SEVERE COMPLICATED MEASLES*** **	♦ ***Give vitamin A treatment*** ♦ ***Give first dose of an appropriate antibiotic*** ♦ ***If clouding of the cornea or pus draining from the eye, apply tetracycline eye ointment*** ♦ ***Refer URGENTLY to hospital***
• Pus draining from the eye or • Mouth ulcers	**MEASLES WITH EYE OR MOUTH COMPLICATIONS*** **	♦ ***Give vitamin A treatment*** ♦ ***If pus draining from the eye, treat eye infection with tetracycline eye ointment*** ♦ If mouth ulcers, treat with gentian violet ♦ Follow-up in 2 days
• Measles now or within the last 3 months	**MEASLES**	♦ ***Give vitamin A treatment***

** *These temperatures are based on axillary temperature. Rectal temperature readings are approximately 0.5°C higher.*

*** *Other important complication of measles —Pneumonia, stridor, diarrhea, ear infection, and malnutrition are classified in other tables.*

Does the child have an ear problem?

IF YES, ASK:	*LOOK AND FEEL:*
• Is there ear pain?	• Look for pus draining from the ear.
• Is there ear discharge?	• Feel for tender swelling behind the ear.

Classify EAR PROBLEM

• Tender swelling behind the ear.	**MASTOIDITIS**	♦ ***Give first dose of an appropriate antibiotic.*** ♦ ***Give first dose of paracetamol for pain.*** ♦ ***Refer URGENTLY to hospital.***
• Pus is seen draining from the ear and discharge is reported for less than 14 days, or • Ear pain.	**ACUTE EAR INFECTION**	♦ ***Give an antibiotic for 5 days.*** ♦ Give paracetamol for pain. ♦ Dry the ear by wicking. ♦ Follow-up in 5 days.
• Pus is seen draining from the ear and discharge is reported for 14 days or more.	**CHRONIC EAR INFECTION**	♦ Dry the ear by wicking. ♦ Treat with topical quinolone eardrops for 2 weeks ♦ Follow-up in 5 days.
• No ear pain and No pus seen draining from the ear.	**NO EAR INFECTION**	♦ No treatment.

THEN CHECK FOR MALNUTRITION AND ANEMIA

CHECK FOR MALNUTRITION

LOOK AND FEEL:

- Look for visible severe wasting
- Look for edema of both feet

CLASSIFY NUTRITIONAL STATUS

• Visible severe wasting or • Edema of both feet	**SEVERE MALNUTRITION**	♦ ***Treat the child to prevent low sugar*** ♦ ***Refer URGENTLY to a hospital***
• Very low weight for age	**VERY LOW WEIGHT**	♦ Assess the child's feeding and counsel the mother on feeding according to the feeding recommendations ♦ Advise mother when to return immediately ♦ Follow-up in 30 days
• Not very low weight for age and no other signs of malnutrition	**NOT VERY LOW WEIGHT**	♦ If child is less than 2 years old, assess the child's feeding and counsel the mother on feeding according to the feeding recommendations — If feeding problem, follow-up in 5 days ♦ Advise mother when to return immediately

CHECK FOR ANEMIA

LOOK AND FEEL:

- Look for palmar pallor. Is it
  - severe palmar pallor?
  - some palmar pallor?

CLASSIFY ANEMIA

• Severe palmar pallor	**SEVERE ANEMIA**	♦ ***Refer URGENTLY to hospital***
• Some palmar pallor	**ANEMIA**	♦ Give iron ♦ Give oral antimalarial if high malaria risk ♦ Give mebendazole if child is 1 year or older and has not had a dose in the previous 6 months ♦ Advise mother when to return immediately ♦ Follow-up in 14 days
• No palmar pallor	NO ANEMIA	♦ If child is less than 2 years old, assess the child's feeding and counsel the mother on feeding according to the feeding recommendations – If feeding problem, follow-up in 5 days

ASSESS, CLASSIFY AND TREAT THE SICK YOUNG INFANT AGED UPTO 2 MONTHS

DO A RAPID APRAISAL OF ALL WAITING INFANTS

ASK THE MOTHER WHAT THE YOUNG INFANT'S PROBLEMS ARE

- Determine if this is an initial or follow-up visit for this problem.
  - If follow-up visit, use the follow-up instructions
  - If initial visit, assess the young infant as follows:

USE ALL BOXES THAT MATCH INFANT'S SYMPTOMS AND PROBLEMS TO CLASSIFY THE ILLNESS

CHECK FOR VERY SEVERE DISEASE AND LOCAL BACTERIAL INFECTION

ASK:

- Is the infant having difficulty in feeding?
- Has the infant had convulsions (fits)?

LOOK, LISTEN, FEEL:

- Count the breaths in one minute. Repeat the count if 60 or more breaths per minute.
- Look for severe chest indrawing.

YOUNG INFANT MUST BE CALM

- Measure axillary temperature.
- Look at the umbilicus. Is it red or draining pus?
- Look for skin pustules.
- Look at the young infant's movements. If infant is sleeping, ask the mother to wake him/her.
  - — Does the infant move on his/her own?

  If the infant is not moving, gently stimulate him/her.
  - — Does the infant move only when stimulated but then stops?
  - — Does the infant not move at all?

Classify ALL YOUNG INFANTS

SIGNS	CLASSIFY AS	TREATMENT (Urgent prereferral treatments are in bold print)
• **Any one of the following signs** • Not feeding well or • Convulsions or • Fast breathing (60 breaths per minute or more) or • Severe chest indrawing or • Fever (37.5ºC or above) or • Low body temperature (less than 35ºC*) or • Movement only when stimulated or no movement at all	**VERY SEVERE DISEASE**	♦ ***Give first dose of an intramuscular antibiotic.*** ♦ ***Treat to prevent low blood sugar.*** ♦ ***Refer URGENTLY to hospital.***** ♦ ***Advise mother how to keep the infant warm on the way to the hospital.***
• Umbilicus red or draining pus • Skin pustules	**LOCAL BACTERIAL INFECTION**	♦ ***Give an appropriate oral antibiotic***. ♦ Teach mother to treat local infections at home. ♦ Advise mother to give home care for the young infant. ♦ Follow-up in 2 days.
• None of the signs of very severe disease or local bacterial infection	**SEVERE DISEASE OR LOCAL INFECTION UNLIKELY**	♦ Advise mother to give home care for the young infant.

* The thresholds are based on axillary temperature. The thresholds for rectal temperature readings are approximately 0.5ºC higher.

** If referral is not possible, see **Integrated Management of Childhood Illness**, Management of the sick young infant module, Annex 2 "Where referral is not possible"

THEN CHECK FOR JAUNDICE

LOOK, LISTEN, FEEL:

If jaundice present, ***ASK:***

- When did jaundice first appear?
- Look for jaundice (yellow eyes or skin)
- Look at the young infant's palms and soles. Are they yellow?

Classify Jaundice

SIGNS	CLASSIFY AS	TREATMENT (Urgent prereferral treatments are in bold print)
• Any jaundice if age less than 24 hours or • Yellow palms and soles at any age	**SEVERE JAUNDICE**	♦ ***Treat to prevent low blood sugar.*** ♦ ***Refer URGENTLY to hospital.*** ♦ ***Advise mother how to keep the infant warm on the way to the hospital.***
• Jaundice appearing after 24 hours of age and • Palms and soles not yellow	**JAUNDICE**	♦ Advise the mother to give home care for the young infant. ♦ Advise mother to return immediately if palms and soles appear yellow. ♦ If the young infant is older than 3 weeks, refer to a hospital for assessment. ♦ Follow-up in 1 day.
• No jaundice	**NO JAUNDICE**	♦ Advise the mother to give home care for the young infant.

THEN ASK: Does the young infant have diarrhea*?

IF YES, LOOK AND FEEL:

- Look at the young infant's general condition:
  - Infant's movements
    - Does the infant move on his/her own?
    - Does the infant move only when stimulated but then stops?
    - Does the infant not move at all?
- Is the infant restless and irritable?
- Look for sunken eyes.
- Pinch the skin of the abdomen.
  Does it go back:
  - Very slowly (longer than 2 seconds)?
  - or slowly?

Classify DIARRHEA FOR DEHYDRATION

SIGNS	CLASSIFY AS	TREATMENT (Urgent prereferral treatments are in bold print)
Two of the following signs: • Movement only when stimulated or no movement at all • Sunken eyes • Skin pinch goes back very slowly.	**SEVERE DEHYDRATION**	♦ If infant has no other severe classification: – Give fluid for severe dehydration (Plan C) OR ***If infant also has another severe classification:*** ***— Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way*** ***— Advise the mother to continue breastfeeding.***
Two of the following signs: • Restless, irritable • Sunken eyes • Skin pinch goes back slowly.	**SOME DEHYDRATION**	♦ Give fluid and for some dehydration and continue breastfeeding (Plan B). ♦ ***If infant has any severe classification:*** ***— Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way.*** ***— Advise mother to continue breastfeeding.*** ♦ Advise mother when to return immediately ♦ Follow-up in 2 days if not improving.
• Not enough signs to classify as some or severe dehydration.	**NO DEHYDRATION**	♦ Give fluids to treat for diarrhea at home and continue breastfeeding (Plan A) ♦ Advise mother when to return immediately ♦ Follow-up in 2 days if not improving.

**** What is diarrhoea in a young infant?***

A young infant has diarrhoea if the stools have changed from usual pattern and are many and watery (more water than fecal matter).

THEN CHECK FOR FEEDING PROBLEM OR LOW WEIGHT FOR AGE

If an infant has no indications to refer urgently to hospital:

ASK:

- Is the infant breastfed? If yes, how many times in 24 hours?
- Does the infant usually receive and other foods or drinks? If yes, how often?
- If yes, what do you use to feed the infant?

LOOK, LISTEN, FEEL:

- Determine weight for age.
- Look for ulcers or white patches in the mouth (thrush).

ASSESS BREASTFEEDING:

- Has the infant breastfed in the previous hour?

If the infant has not fed in the previous hour, ask the mother to put her infant to the breast. Observe the breastfeed for 4 minutes.

(If the infant was fed during the last hour, ask the mother if she can wait and tell you when the infant is willing to feed again).

- Is the infant well-attached?

not well-attached *good attachment*

TO CHECK ATTACHMENT, LOOK FOR:
- *More areola seen above infant's top lip than below bottom lip*
- *Mouth wide open*
- *Lower lip turned outwards*
- *Chin touching breast*

(All of these signs should be present if the attachment is good).

- Is the infant suckling effectively (that is, slow deep sucks, sometimes pausing)?

not suckling effectively *suckling effectively*

Clear a blocked nose if it interferes with breastfeeding.

Classify FEEDING

SIGNS	CLASSIFY AS	TREATMENT (Urgent prereferral treatments are in bold print)
• Not well attached to breast or • Not suckling effectively, or	**FEEDING PROBLEM OR LOW WEIGHT FOR AGE**	♦ If not well-attached or not suckling effectively, teach correct positioning and attachment. • If not able to attach well immediately, teach the mother to express breast milk and feed by a cup.
• Less than 8 breastfeeds in 24 hours, or		♦ If breastfeeding less than 8 times in 24 hours, advise to increase frequency of feeding. Advise her to breastfeed as often and for as long as the infant wants, day and night.
• Receives other foods or drinks, or		♦ If receiving other foods or drinks, counsel mother about breastfeeding more, reducing other foods or drinks, and using a cup. • If not breastfeeding at all: — Refer for breastfeeding counseling and possible relactation. — Advise about correctly preparing breastmilk substitutes and using a cup.
• Low weight for age, or • Thrush (ulcers or white patches in mouth)		♦ Advise the mother how to feed and keep the low weight infant warm at home. ♦ If thrush, teach the mother to treat thrush at home. ♦ Advise mother to give home care for the young infant. ♦ Follow-up any feeding problem or thrush in 2 days. ♦ Follow-up low weight for age in 14 days.
• Not low weight for age and no other signs of inadequate feeding.	**NO FEEDING PROBLEM**	♦ Advise mother to give home care for the young infant. ♦ Praise the mother for feeding the infant well.

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his homeland, i.e. his movement is restricted. But a *patient* who already has an infective disease also has to be restricted, called **isolation.**

21. *Primordial prevention* refers to inhibition of appearance of a risk factor in a group in which it is yet to appear. Typical example is that children are generally drawn back from smoking.
22. World Health Organization's 2005 Bangkok Charter.
23. Werner Heisenberg, in particular (conceptor of the 'undertainity principle').
24. These things do not really need explaining, do they? But if you really want one, consider this: You carry out a screening for diabetes, and you select a fasting sugar cut off of 80. Of course you will detect all diabetics, but in addition, so many normal people who range over 80 mg/dl will be caught delinquent (loss of specificity). Think yourself what may happen if you select a cut off 160 mg/dl.
25. From xkcd (xkcd.com/539), under Creative Commons Attribution Noncommercial 2.5 license.
26. 'Student' is a pen-name taken by WA Gossett.
27. The Student's test is valied for less than 30 members in a sample.
28. British Medical Journal (bmj.com) tutorial on Epidemiology.
29. British Medical Journal (bmj.com) tutorial on Epidemiology.
30. The most famous of failed experiments is the Mitchelson Morley experiment, which showed that *light* has no relative velocity and its velocity is constant in *any* reference frame.
31. One of the problem faced by new researchers are which data to collect. Often, you begin a study with a protocol designed by you. Gradually, as you conduct the study, you realise the subjects are providing you some data which is not in your protocol, but seems to be important considering the number of subjects who have reported it. For example, while investigating the cause of low birth weight in an area, you ask mothers about the usual factors (maternal malnutrition, anemia), etc. But most of the women in the area go to work in a rice mill 6 kilometers away by foot, daily, even when they are pregnant. When you encounter this piece of information, you must modify your original study design, which is terribly difficult once you have begun your study. It is, thus useful to 'pretest' your protocol by conducting a **pilot study** in the population, to now whether extra variables emerge.
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133. Goldin, Claudia, and Lawrence Katz (2002). "The Power of the Pill: Oral Contraceptives and Women's Career and Marriage Decisions". J Political Economy 110 (4): 730–770.
134. Because the OCPs dissociate sex from reproduction very effectively (0.3% failure rate, to be precise), it contributed to the world-view of 'sex is fun' and one of the harbingers of 'casual sex' as it know it today. Thus it has faced opposition from many religious circles, including the Catholic Church.
135. "FDA Approves Seasonale Oral Contraceptivel". 2003-09-25. http://www.fda.gov/bbs/topics/ANSWERS/2003/ANS01251.html.
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143. http://www.csua.berkeley.edu/~monac/norplant.html.

144. How do failure rates relate to Pearl index? A failure rate 0.05% means if 100 women use Norplant implants for 1 year, only 0.05 will become pregnant, or if they use it for 4 years, only 2 will become pregnant.
145. Kippley, John; Sheila Kippley (1996). The Art of Natural Family Planning (4th ed.). Cincinnati.
146. Hristiansen C, Sandlow J (1 May 2003). "Testicular Pain Following Vasectomy: A Review of Postvasectomy Pain Syndrome". J of Andrology 24 (3): 293.
147. William R. Finger (Spring 1998). "Attracting Men to Vasectomy". Network 18 (3). http://www.fhi.org/en/rh/pubs/network/v18_3/nw182ch8.htm.
148. Ninaad S. Awsare, Jai Krishnan, Greg B. Boustead, Damian C. Hanbury, and Thomas A. McNicholas (2005). "Complications of vasectomy.". Ann R Coll Surg Engl 87 (6): 406–10.
149. "Essure System - P020014". Food and Drug Administration. 2003-04-28. http://web.archive.org/http://fda.gov/cdrh/pdf2/p020014.html.
150. WHO Factsheet (no 244) on emergency contraception, 2005.
151. WHO Factsheet (no 244) on emergency contraception, October 2005.
152. 'good birth'.
153. Some terms are not as big as they spell. *Decentralization* is simply distribution of decision-making power in all peripheral centers rather than just the Parliament.
154. Many Indian women spend hrs in queue for a pot of water in drought affected areas; it is surprising how we have ascribed the toughest jobs in the house to be 'womanly', so that the men in the house have an excuse of not doing it.
155. Comte, Auguste, A Dictionary of Sociology (3rd ed.), John Scott and Gordon Marshall (eds), Oxford University Press, 2005.
156. The same man who hypothesized that *omnis celluli e celluli* (all cells spring from some other cell), generally regarded to be a doctor, anthropologist, prehistorian, biologist and politician, and "the father of pathology". He has coined *fifteen* eponyms after his name in medical textbooks (Virchow's gland, Virchows's triad ...). His achievements in pathology, and later, his political thoughts about social and state medicine, have pervaded the medical thinking of modern times.
157. Lexicon of alcohol and drug terms published by the World Health Organization, http://www.who.int/substance_abuse/terminology/who_lexicon/en/
158. Census, 2001.
159. http://www.unicef.org/infobycountry/india_statistics.html.
160. UN Human Development Report, 2009.
161. http://www.who.int/reproductive-health/strategy.htm.
162. The association between reproduction and women is so close, that 'gynaecology' has, over years, been confined to the reproductive pathology of women, excluding all other organ systems, in spite of the fact that many other diseases behave differently in men and women. But 'andrology' has not yet emerged as a separate discipline, simply because men never need an abortion, they never go into labor and don't die while giving birth.
163. http://www.snopes.com/pregnant/medina.asp.
164. Govt of India sample Registration System, Statistical report 4 of 2007.
165. Govt of India sample Registration System, Statistical report 4 of 2007; according to CIA world factbook, it has dropped to 30.15 in 2009.

166. Sample Registration System, Maternal Mortality in India, 1997-2003.
167. Govt of India sample Registration System, Statistical report 4 of 2007.
168. UNICEF: State of World's children, 2009.
169. UN Millenium Development Goals: Maternal Mortality: http://www.endpoverty2015.org/goals/maternal-health.
170. This is a *ratio* as we have not included stillbirths/abortions in the denominator (i.e. the numerator is not a subset of the denominator).
171. http://www.who.int/features/qa/12/en/index.html.
172. Sample Registration System, statistical report 4 of 2007.
173. Remember, unlike internal medicine, we focus on only those things which are *simple* in community medicine.
174. Sample Registration System, report 4 of 2007.
175. CIA world factbook.
176. SRS 2003, cited in Singh B, "Infant Mortality Rate in India: Still a long way to go", Indian J of Pediatrics, Volume 74—May, 2007.
177. Probably they just expect anybody born among haystacks will turn out to be Jesus Christ.
178. Govt of India, Sample Registration System: Statistical report 4 of 2007.
179. UNICEF: State of the World's Children, 2009.
180. Cited in Pinheiro, Paulo Sérgio, World Report on Violence against Children, published by the United Nations Secretary-General's Study on Violence against Children, Geneva, 2006, p. 12.
181. "Child Labor as an Institution in India", Shruti Tripathi, Indira Gandhi Institute of Development Research (IGIDR), February 4, 2008, availible at http://papers.ssrn.com/sol3/papers.cfm?abstract_id=1090116.
182. It is surprising that how many 'unacceptable' things we have accepted.
183. Feeling exhausted with this sordid tale of children and their sufferings? Think how *living* the life of a poor, underfed, malnourished, laboring and sexually abused child feels like. Close this book, and ponder for a minute.
184. It is no major surprise that even in 2010, the major issues in the Indian Political Scenario is caste, religion, cricket, bollywood and *not* food, education or our children; we are Indians, the 'spiritual leaders' of the world; somehow it seems contrary to our 'spiritual' image to worry about mundane matters like price of food and education of girls - we find it more fashionable to be concerned with whether our cricket team stays at top of the world.
185. WHO-UNICEF: IMCI guidelines, 2008.
186. After Virginia Apgar.
187. Guidelines for Drinking-water Quality, WHO, Geneva 2008.
188. 2003 G8 Evian summit "Action Plan".
189. WHO, "Safer water for better health", 2008.
190. Allen Burton G, Robert Pitt. Stormwater Effects Handbook: A Toolbox for Watershed Managers, Scientists, and Engineers. New York: CRC/Lewis Publishers, 2001.
191. WHO: Safer water, better health, 2008.
192. WHO: Online QandA - How does safe water affect global health?
193. WHO: Safer water, better health, 2008.

194. WHO, "Safer water: better health", 2008.
195. Center for Affordable Water Sanitation and Treatment, "Household water treatment", 2006.
196. Center for affordable water and sanitation technology (Calgary, Canada): Household water treatment manual, 2008.
197. Center for affordable water and sanitation technology (Calgary, Canada): Household water treatment manual, 2008.
198. Guidelines for safe recreational waters". WHO, 2006.
199. Guidelines for Drinking Water Quality, 3rd ed, WHO.
200. As defined in Accelerated Rural Water Supply Program.
201. Toftum J. Thermal Comfort Indices. Handbook of Human Factors and Ergonomics Methods, CRC Press, 2005.
202. Havenith G. Heat balance when wearing protective clothing. The Annals of Occupational Hygiene: 1999;43(5), 289.
203. Wolkoff P, Kjaergaard SK. The dichotomy of relative humidity on indoor air quality. Environment International. 2007;33(6), 850.
204. European Environment Agency's 2005 Emission Inventory Guidebook.
205. Claude Monet (1840–1926). made several trips to London between 1899 and 1901, during which he painted views of the Thames and Houses of Parliament which show the sun struggling to shine through London's smog-laden atmosphere. Years later, the Great Smog of 1952 darkened the streets of London and killed approximately 4,000 people in the short-time of 4 days (a further 8,000 died from its effects in the following weeks and months).
206. AP 42 report by Environmental Protection Agency.
207. Estimated deaths and DALYs attributable to selected environmental risk factors, by WHO Member State, 2002.
208. Many people dismiss global warming as entirely statistical, and even doubt its existence. It is upto you what viewpoint you take, but the practices to stop the progression of a 'supposed' global warming are good for health and well-being of your in general; so that's there no harm in starting them.
209. "Summary for Policymakers"; Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change.
210. Copyright Dragon's flight under Creative Commons Attribution Share Alike 2.5 license.
211. Global Warming: Frequently Asked Questions - National Oceanic and Atmospheric Administration, National Climatic Data Center.
212. Global Warming: Frequently Asked Questions; National Oceanic and Atmospheric Administration, National Climatic Data Center.
213. Knutson, Thomas R. "Simulated reduction in Atlantic hurricane frequency under twenty-first century warming conditions". Nature Geoscience. 2008;1: 359.
214. King, Gary M; et al. (PDF). Global Environmental Change Microbial Contributions Microbial Solutions. American Society for Microbiology. pp. 7.
215. Parry ML, Canziani OF, Palutikof JP, et al. (Eds) "Chapter 8: Human Health". Climate Change 2007: Impacts, Adaptation and Vulnerability. Contribution of Working

Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press. 2007.

216. Shaffer G, Olsen SM, Pederson GOP. "Long-term ocean oxygen depletion in response to carbon dioxide emissions from fossil fuels". Nature Geoscience. 2009;2:105–9.
217. Raven, John A; et al. (2005–06–30) (ASP). Ocean acidification due to increasing atmospheric carbon dioxide. Royal Society.
218. WHO: Air quality guidelines for Europe. 1987.
219. Bansal RK, Saxena DM. Overcrowding and health. Indian J Med Sci [serial online] 2002 [cited 2010 Jan 2];56:177–9. Available from: http://www.indianjmedsci.org/text.asp?2002/56/4/177/11970.
220. Proposed by UN Seminar on Social Aspects of Housing, 1975.
221. Entropy is the amount of chaos in a system. The second law of thermodynamics states that the entropy of the universe is always increasing, i.e. things naturally place themselves in a most chaotic state. A cup of tea spills over very easily, but the reverse, water coming back into the cup of tea from the ground, never happens. The opposite of entropy, that is the amount of organization and harmony in a system, is called information.
222. Copyright Drstuey at the English Wikipedia under Creative Common Share Alike License 3.0.
223. The Hindu Business Line, Oct 28, 2009: "Hospital waste management should start at source, say entrepreneurs".
224. Nasrin Khan, public health specialist, Bangladesh.
225. UNICEF/WHO Joint Monitoring Program estimate for, 2004.
226. Harvey, Peter, Baghri, Sohrab and Reed, Bob. Emergency Sanitation: Assessment and program design. Water, Engineering and Development Center (WEDC), Loughborough University, UK. http://wedc.lboro.ac.uk/publications/pdfs/es/ES06CD.pdf. 2002.
227. Harvey et al. 2002 (see earlier notes).
228. Harvey et al. 2002 (see earlier notes).
229. Harvey et al. 2002 (see earlier notes).
230. Harvey et al. 2002 (see earlier notes).
231. The theme of traveling through, hiding, or even residing in sewers is a common cliché in fiction and comics. Famous examples of sewer dwelling or scenes are Teenage Mutant Ninja Turtles, Stephen King's It and Les Miserables.
232. Rosenhall U, Pedersen K, Svanborg A. "Presbycusis and noise-induced hearing loss". Ear Hear. 1990;11(4):257–63.
233. Rosen S, Olin. P, Hearing Loss and Coronary Heart Disease, Archives of Otolaryngology, 1965;82:236.
234. Field JM, Effect of personal and situational variables upon noise annoyance in residential areas, J of the Acoustical Society of America, 1993;93:2753–63.
235. Jesús Barreiro, Mercedes Sánchez, Montserrat Viladrich-Grau. "How much are people willing to pay for silence? A contingent valuation study", Applied Economics. 2005;37(11).
236. www.dosits.org/glossary/pop/lvr.htm.
237. Rosen S and Olin P, Hearing Loss and Coronary Heart Disease, Archives of Otolaryngology, 1995;82:236.

238. Generic Procedures for Assessment and Response during a Radiological Emergency, IAEA TECDOC Series number 1162, published in 2000.
239. Mayo Clinic Staff (May 9, 2008). Radiation sickness: Symptoms.
240. 'Welfare' are those services provided by any state or organization on a nonprofit basis, towards building a goodwill towards the community.
241. Osteoporosis of mandible among the girls working at a London match factory, a classic case of occupational intervention.
242. United States Environmental Protection Agency, "Pesticides and Food: What Integrated Pest Management Means".
243. A vector is *resistant* to a substance if it tolerate such doses of the chemical to which majority of the same species resusceptible.
244. The shell of insects.
245. Mosquito control through source reduction, University of Florida.
246. American Mosquito Control Association.
247. Stuart M Bennett, 2006; the-piedpiper.co.uk.
248. By Noodlesnacks (www.noodlesnacks.com) under Creative Common Attribution Share Alike 1.0 generic license.
249. Stuart M Bennett, 2006; the-piedpiper.co.uk.
250. A *nymph* resembles a mature tick (it has eight legs) without capacity of reproduction
251. André Karwath, under Creative Commons Attribution ShareAlike 2.5.
252. Luc Viatour (www.lucnix.be) under Creative Commons Attribution Share Alike 3.0 license.
253. Image by Kalumet (http://de.wikipedia.org/wiki/Benutzer:Kalumet) under Creative Commons Attribution Share Alike 3.0 license.
254. John Travis, "The Naked Truth", www.sciencenews.org.
255. Vincent S Smith, under Creative Commons Attribution 2.5 license, published in Public Library of Science J.
256. From the Archival Research Catalog of the National Archives and Records Administration (USA) under the ARC Identifier 514159.
257. Fellow of Royal Society who discovered 'cells'; his adventures with the microscope are documented in vivid detail in his book of pictures, Micrographia.
258. Fleas, HYG-2081-97 William F. Lyon, Ohio State University entomology page).
259. The term pesticides include insecticides + rodenticides + repellants + fungicides.
260. These are unicellular algae characterized by the silicified cell made by two halves. (Silicified meaning infiltration or replacement of organic tissues or of other minerals such as calcite by silica).
261. Of the three kind of accessibility mentioned here, *social* acceptability seems to be the challenge in India; many communities and cultures regard modern systems of medicine as incompetent, disrespectful of traditions or simply profane. Add to that the hostile behavior of health care workers across the country. It is no wonder that people prefer wizards, quacks and illegitimate practitioners, who are closer to them, than health services.
262. Sample Registration System, Statistical report 2006, Report 4 of 2007.
263. Census 2011: Provisional report.
264. UN Human Development Report, 2009.

265. UN Human Development Report, 2009.
266. NRHM Mission Document.
267. Ghulam Nabi Azad, Union Health Minister, 25th December, 2009.
268. Planning Commission Statement, 2007.
269. Which translates as introducing shiny new computers into every office without proper training of employees. The usual scenario is something get botched up, the computer is dumped within a year, and requisition for a new, shinier computer is presented. Meanwhile, 'e-governance' gets itself strangled in red tape.
270. Programs for the six diseases malaria, filariasis, dengue, chikungunya, Japanese encephalitis and kala-azar have been incorporated into National Vector Borne Disease Control Program.
271. Recommendations by Govt of India: Indian public health standards for primary health care (2007).
272. NRHM framework document, Govt of India.
273. http://www.who.int/topics/essential_medicines/en/
274. Current Index of Medical Specialities, Jan 2008.
275. 'Politics' (from Latin polis, a city or a society) as in "man is a political animal", not parliamentary politics; the introduction of low budget small cars in the market is a dream come true for many middle class people, but to the environmentalist, the sheer number of these cars in the street is a nightmare. The difference in political thought between the two is causing the difference in intelligence.
276. Census 2011: Provisional report.
277. WHO International Health Regulations, 2005.
278. Omran AR. The epidemiologic transition; A theory of the epidemiology of population change. Milbank Memorial Fund Quarterly 1971;49:509–38.
279. WHO Facsheet on Cardiovascular Disease: http://www.who.int/mediacentre/factsheets/fs317/en/index.html.
280. WHO: Atlas of Heart Disease and Stroke.
281. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. JAMA 2003; 289: 2560–72.
282. WHO: Atlas of Heart Disease and Stroke.
283. Health system: Improving performance. In: The World Health Report 2001. Geneva, World Health Organization, 2001:144–55.
284. Rheumatic fever and rheumatic heart disease: Report of a WHO Expert Consultation; Geneva, 29 October–1 November 2001.
285. WHO cancer factsheet, http://www.who.int/mediacentre/factsheets/fs297/en/index.html.
286. Danaei et al (2005) based on noted risk factors.
287. WHO: Cancer Prevention, http://www.who.int/cancer/prevention/en/index.html.
288. WHO: Screening and early detection of cancer, http://www.who.int/cancer/detection/en/index.html.
289. The new HPV vaccine: A Q and A sheet for girls and their parents on the HPV vaccination: NHS, www.nhs.uk/hpv.
290. Laurence D R, Bennett P N, Clinical Pharmacology, seventh edition 1992, ELBS/Churchill Livingstone.

291. NCCP Guidelines, Govt of India, Ministry of Health and FW, 2005.
292. http://www.who.int/mediacentre/factsheets/fs311/en/index.html.
293. American Diabetes Association, 2007, cited in Harrison's Principles of Internal Medicine, 17th Ed.
294. WHO Diabetes factsheet: http://www.who.int/mediacentre/factsheets/fs312/en/index.html.
295. WHO diabetes factsheet: http://www.who.int/mediacentre/factsheets/fs312/en/index.html.
296. WHO: Health Situation in the SEAR, 1998–2000.
297. NPCB Website, http://mohfw.nic.in/npcbnew/index.asp.
298. http://www.who.int/gpsc/clean_hands_protection/en/index.html
299. http://www.cdc.gov/OD/ohs/symp5/jyrtext.htm
300. From the point of view of the microorganism, this is the most successful type of parasitism. If an infection is rapidly fatal, the host dies soon and the organism has to now search for a new host. But if the organism stays quiet inside and goes on reproducing without causing as much distress so that the host will pop a drug or die, its purpose is served.
301. Droplets ranging from 0.5 to 5 μm, 40,000 droplets/sneeze are released at an average speed of 21 m/s, which can reach close to speed of sound if the sneeze is forceful enough.
302. CDC Q and A on HIV
303. Economically speaking, the 'price' that these mothers are paying for your services is a fully immunized child, which is an asset to the community.
304. Safe vaccine handling, cold chain and immunizations, WHO 1998.
305. Source: http://www.amicale-genealogie.org/Histoires_temps-passe/Epidemies/chol01.htm
306. New York Times. July 18, 1911.
307. "Is a vaccine available to prevent cholera?" CDC disease info: Cholera. http://www.cdc.gov/nczved/dfbmd/disease_listing/cholera_gi.html.
308. "Typhoid vaccines: WHO position paper". Wkly. Epidemiol. Rec. 83 (6): 49–59. February 2008.
309. Greek 'polios' = gray, 'myelos' = spinal cord, so poliomyelitis = inflammation of gray matter of spinal cord.
310. AFP Surveillance Bulletin - India: Report for week 07, ending 20th February 2010
311. WHO Polio Factsheet: http://www.who.int/mediacentre/factsheets/fs114/en/
312. Paul JR (1971). A History of Poliomyelitis. Yale studies in the history of science and medicine. New Haven, Conn: Yale University Press. pp. 16–18.
313. Kew O, Sutter R, de Gourville E, Dowdle W, Pallansch M (2005). "Vaccine-derived polioviruses and the endgame strategy for global polio eradication". Annu Rev Microbiol 59: 587–635.
314. Atkinson W, Hamborsky J, McIntyre L, Wolfe S (eds.) (2007). "Poliomyelitis" (PDF). Epidemiology and Prevention of Vaccine-Preventable Diseases (The Pink Book) (10th ed.). Washington DC: Public Health Foundation. pp. 101–14.
315. Spice B (April 4, 2005). "Tireless polio research effort bears fruit and indignation". The Salk vaccine: 50 years later/second of two parts (Pittsburgh Post-Gazette). http://www.post-gazette.com/pg/05094/482468.stm

316. The cold chain that has to be maintained for transport from the periphery to the center, i.e. the reverse of vaccines, which travel from center to periphery.
317. CDC FAQ on viral hepatitis.
318. WHO Malaria Factsheet: http://www.who.int/mediacentre/factsheets/fs094/en/index.html
319. WHO Global Malaria Report 2009.
320. WHO World Malaria Report 2009.
321. National Drug Policy on Malaria: 2007.
322. See the section on entomology.
323. DDT has a residual effect of 6 months, the same fact which is making it gradually unsuitable for ecological reasons. Malathion, fenitrothion and pyrethroids are being used more and more for residual spray, especially after emergence of DDT resistance.

    In an important policy shift, the World Health Organization (WHO) announced on 27/12/2009 that it is urging the use of the pesticide DDT to control the spread of malaria, as their is no cheaper and effective alternative. The notion has been challenged by many environmentalists.
324. WHO Degue Factsheet: http://www.who.int/vaccine_research/diseases/vector/en/index1.html
325. Drawing by EA Goeldi (1905) Os Mosquitos no Pará. Memorias do Museu Goeldi. Pará, Brazil; scanned by Wikipedia user Küchenkraut.
326. WHO Filaria factsheet, http://www.who.int/mediacentre/factsheets/fs102/en/
327. Take a deep breath and behold the wonders of evolution: the vector of filariasis is *Culex* mosquitoes, that predominantly bite at night. Thus the microfilaria *present themselves in blood during night time*, so that they have better chance of being picked up by a mosquitoe, and better chance of reproduction. It is downright amazing to see how mosquitoes and microfilaria have coevolved.
328. From US Center For Disease Control—Public Health Image Library.
329. From US Center for Disease Control (CDC)—Public Health Image Library.
330. Powers AM, Logue CH (2007). "Changing patterns of chikungunya virus: Reemergence of a zoonotic arbovirus" (PDF). J. Gen. Virol. 88 (Pt 9): 2363–77.
331. "WHO—Laboratory Diagnosis of Chikungunya Fevers".
332. NVBDCP: Japanese Encephalitis.
333. The first successful drug against kala-azar, urea stibamine, was discovered by an Indian, Upendranath Bramhachari in Campbell Medical School, Kolkata (now NRS medical college) in 1922.
334. Wikipedia user Lady of Hats.
335. Uploaded by Wikipedia use Paul Furst.
336. WHO Plague Manual: Epidemiology, Distribution, Surveillance and Control.
337. Pneumonic Plague Epidemic in Sural. Association of American Geographers. http://www.aag.org/hdgc/www/health/units/unit4/html/4bkground.html
338. Surat: A Victim of Its Open Sewers". New York Times. September 25, 1994.
339. WHO Plague Manual: Epidemiology, Distribution, Surveillance and Control.
340. Cotran RS, Kumar V, Fausto N, et al. (2005). Robbins and Cotran Pathologic Basis of Disease (7th ed.). St. Louis: Elsevier/Saunders. pp. 1375.

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342. Drew WL (2004). "Chapter 41: Rabies". in Ryan KJ, Ray CG (editors). Sherris Medical Microbiology (4th ed.). McGraw Hill. pp. 597–600.
343. A special lineages of cells used in cell cultures. The vero lineage was isolated from kidney epithelial cells extracted from an African green monkey
344. Rabies vaccines: WHO position paper: Weekly epidemiological record, 7/12/2007, No 49/50, 2007;82:425–36.
345. Wiktor TJ, Macfarlan RI, Reagan KJ, Dietzschold B, Curtis PJ, Wunner WH, Kieny MP, Lathe R, Lecocq JP, Mackett M (1984). "Protection from rabies by a vaccinia virus recombinant containing the rabies virus glycoprotein gene". Proc. Natl. Acad. Sci. USA 81 (22): 7194–8.
346. Reece JF, Chawla SK. (2006). "Control of rabies in Jaipur, India, by the sterilization and vaccination of neighborhood dogs.". Vet Rec 159: 379–83.
347. Once an established practice in India.
348. WHO Programs and Projects: Neonatal Tetanus, http://www.who.int/immunization/topics/tetanus/en/index.html
349. NHS "choices" website: Why isn't the chickenpox vaccine available in the UK?
350. Wikipedia: Diphtheria; http://en.wikipedia.org/wiki/Diphtheria
351. Atkinson W, Hamborsky J, McIntyre L, Wolfe S, eds. (2007). Diphtheria: In Epidemiology and Prevention of Vaccine-Preventable Diseases (The Pink Book) (10 ed.). Washington DC: Public Health Foundation. pp. 59–70. http://www.cdc.gov/vaccines/pubs/pinkbook/downloads/dip.pdf
352. Havaldar, PV, Sankpal MN, Doddannavar RP. (2000). "Diphtheritic myocarditis: Clinical and laboratory parameters of prognosis and fatal outcome.". Ann Trop Paediatr. 20 (3): 209–15.
353. Solders, G, Nennesmo I, Persson A. (1989). "Diphtheritic neuropathy, an analysis based on muscle and nerve biopsy and repeated neurophysiological and autonomic function tests." J Neurol Neurosurg Psychiatry 52 (7): 876–80.
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