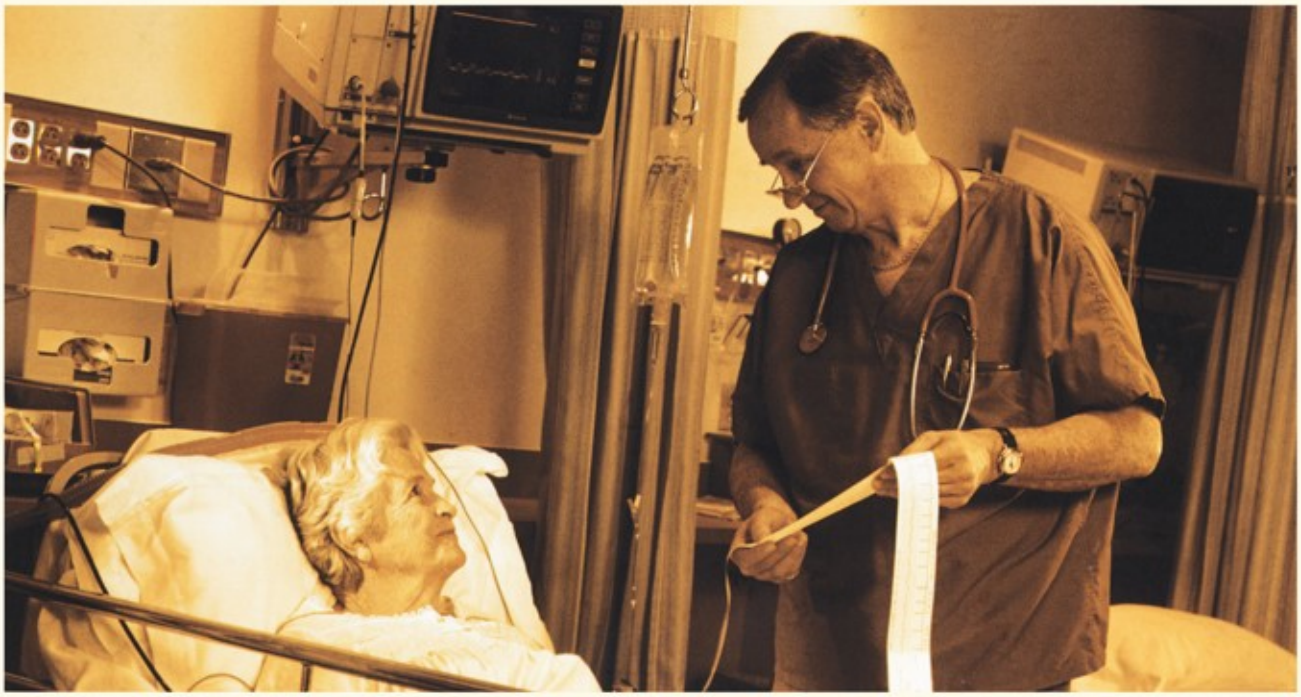
Classic Papers in Geriatric Medicine

with Current Commentaries



Edited by

Robert J. Pignolo, MD, PhD

Monica K. Crane, MD

Mary Ann Forciea, MD

Humana Press

Classic Papers in Geriatric Medicine
with Current Commentaries

Aging Medicine

Robert J. Pignolo, MD, PhD; Mary Ann Forciea, MD; Jerry C. Johnson, MD,
Series Editors

Age-Related Changes of the Human Eye, edited by Carlo A. Cavallotti and Luciano Cerrulli, 2008

Classic Papers in Geriatric Medicine with Current Commentaries, edited by Robert J. Pignolo, Monica K. Crane, and Mary Ann Forciea, 2008

Eldercare Technology for Clinical Practitioners, edited by Robin Felder and Majd Alwan, 2008

Handbook of Pain Relief in Older Adults: *An Evidence Based Approach*, edited by Michael F. Gloth, 2004

Classic Papers in Geriatric Medicine with Current Commentaries

Edited by

Robert J. Pignolo, MD, PhD
Division of Geriatric Medicine
University of Pennsylvania School of Medicine
Philadelphia, Pennsylvania, USA

Monica K. Crane, MD
Geriatric Medicine, Memory Health Clinic
Carolina Health Care System
Charlotte, North Carolina, USA

Mary Ann Forciea, MD
Division of Geriatric Medicine
University of Pennsylvania Health System
Philadelphia, Pennsylvania, USA

Humana Press

Editors
Robert J. Pignolo, MD, PhD
Assistant Professor of Medicine
Division of Geriatric Medicine
University of Pennsylvania
School of Medicine
Philadelphia, PA
USA

Monica K. Crane, MD
Medical Director
Geriatric Medicine
Memory Health Clinic
Attending Senior Health Connection
Carolina Health Care System
Charlotte, NC
USA

Mary Ann Forciea, MD
Clinical Associate Professor of Medicine
Division of Geriatric Medicine
University of Pennsylvania Health System
Philadelphia, PA
USA

Series Editors
Robert J. Pignolo, MD, PhD
Assistant Professor of Medicine
Division of Geriatric Medicine
University of Pennsylvania
School of Medicine
Philadelphia, PA
USA

Mary Ann Forciea, MD
Clinical Associate Professor of Medicine
Division of Geriatric Medicine
University of Pennsylvania Health
System
Philadelphia, PA
USA

Jerry C. Johnson, MD
Professor and Chief, Geriatric Medicine
Division
University of Pennsylvania School of
Medicine
Philadelphia, PA
USA

ISBN: 978-1-58829-998-7 e-ISBN: 978-1-59745-428-5
DOI: 10.1007/978-1-59745-428-5

Library of Congress Control Number: 2007941270

Printed on acid-free paper

9 8 7 6 5 4 3 2 1

springer.com

Preface: What Makes a Classic? Classic Papers in Geriatric Medicine with Current Commentaries

"Who said so?" "Why do we do that?" Just as in life, an awareness of our "ancestry" in the form of landmark papers and observations that guide our practice and thinking gives us a foundation and a base for further inquiry. The field of geriatrics is a relative newcomer as an organized specialty, but its foundations are rooted in the classic papers of early descriptions of aging and age-related diseases, as well as in more recent studies on the physiologic mechanisms and possible clinical interventions in these often-chronic processes. Easy access to those landmark papers can help us in our own reflections on our clinical practices, in achieving a more thorough understanding of the background of research, and in teaching the richness of our intellectual history for our trainees and students. In this book, the editors focus on the clinical discipline of geriatric medicine and some of the classic papers that have changed the way we think about and practice the care of elderly people.

In choosing the "classic" papers, we have used a combination of expert opinion and objective assessment. In most cases, these criteria were in agreement, but in some cases they were not, nor were they always expected to be. For example, it would be difficult to assign an impact factor to the original description of an age-related disease, but certainly there can be no doubt of its historic importance. Similarly, papers that have shown a clinical impact on patient care are surely at least as well read, if not more, by practitioners as researchers who continue to work in the field; and yet there is little objective means to quantitate this effect. The 15 areas chosen for inclusion in this volume represent the beginnings of practice and thoughts about the best ways to care for older patients. Many other contributions fell victim to space limitations.

We have often chosen early or representative examples of papers that have substantially contributed to care of the aged. We have selected papers in disease-specific areas (dementia), health systems (home care), and education. In general, despite the plethora of review articles in geriatric medicine, and also accepting that some of these papers represent clear and compelling thought in the field, we have chosen to exclude review articles, preferring primary source material wherever possible. The exceptions are those papers that have contributed sentinel ideas and hypotheses on which current work is so closely based; for example, Finucane's summary of evidence of the utility of tube feeding in patients with dementia.

Each paper is introduced by a commentary. These commentaries describe the singular contributions of the chosen paper, give a short list of other important early papers in that area, and, in many cases, reflect on progress in that particular field. The commentaries are personal statements by the authors

about the influence of the papers chosen; the commentaries are not meant to be exhaustive reviews of the clinical area in question.

An accepted measure of the importance of a paper is its ability to retain its impact over time. While many of the classic papers in this collection have stood the test of time, others have gained classic status by virtue of their profound influence in relatively quick measure or because of timeliness in filling a void in knowledge.

Clinical impact, citation frequency, historical value, timeliness or timelessness—these are the attributes that we assign these landmark papers in geriatrics. We hope that their rediscovery or first-time reading will surprise and motivate the reader to continue the clinical and research endeavors undoubtedly induced by these classics in their original readers.

Robert J. Pignolo, MD, PhD
Monica K. Crane, MD
Mary Ann Forciea, MD

Acknowledgments

We thank Yamina Hayman-Pratt for her technical help in putting the manuscript for this book together. We appreciate the support for this project given to us from Richard Lansing of Humana Press. We especially thank our families for their encouragement throughout the writing and editing process.

Robert J. Pignolo, MD, PhD
Monica K. Crane, MD
Mary Ann Forciea, MD

Contents

Contributors

Vivian S. Argento, MD
Chief, Section of Geriatrics, Bridgeport Hospital Center for Geriatric Medicine, Yale New Haven Health Systems, New Haven, CT, USA

Lesley S. Carson, MD
Clinical Associate Professor of Medicine, Division of Geriatric Medicine, University of Pennsylvania Health System, Philadelphia, PA, USA

Monica K. Crane, MD
Memory Health Clinic, Medical Director, Geriatric Medicine, Attending Senior Health Connection, Carolina Health Care System, Charlotte, NC, USA

Kathy L. Egan, PhD
Director, Geriatric Education, Division of Geriatric Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA, USA

Mary Ann Forciea, MD
Clinical Associate Professor of Medicine, Division of Geriatric Medicine, University of Pennsylvania Health System, Philadelphia, PA, USA

Jerry C. Johnson, MD
Professor of Medicine, Chief, Geriatric Medicine Division, University of Pennsylvania School of Medicine, Philadelphia, PA, USA

Jennifer Kapo, MD
Assistant Professor of Clinical Medicine, Department of Medicine, Division of Geriatric Medicine, University of Pennsylvania Health System, Philadelphia, PA, USA

Jason H. Karlawish, MD
Associate Professor of Medicine, Division of Geriatric Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA, USA

Robert J. Pignolo, MD, PhD
Assistant Professor of Medicine, Division of Geriatric Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA, USA

Edna P. Schwab, MD
Assistant Professor of Clinical Medicine, Hospital of the University of Pennsylvania, Chief, Division of Geriatrics and Extended Care, Philadelphia Veteran Affairs Medical Center, Philadelphia, PA, USA

Joan Weinryb, MD, CMD
Clinical Assistant Professor of Medicine, Division of Geriatric Medicine, University of Pennsylvania Health System, Philadelphia, PA, USA

Jean Yudin, MSN, RN, CS
Director, Schnabel in Home Primary Care Program, Division of Geriatric Medicine, University of Pennsylvania Health System, Philadelphia, PA, USA

1
Perspectives on General Aging

Robert J. Pignolo, MD, PhD

Commentary

In February 1905, in his farewell address to The Johns Hopkins University School of Medicine, William Osler offered two "fixed" ideas regarding the uselessness of old age (1). The first idea was that men more than 40 years of age were comparatively useless:

> Take the sum of human achievement in action, in science, in art, in literature—subtract the work of the men above 40, and, while we should miss great treasures, even priceless treasures, we would practically be where we are today.

The second idea was "the uselessness of men above 60 years of age," and he suggested that "as a matter of course, men stopped work at this age." With reference to a satirical novel, Osler continued:

> In that charming novel, the *Fixed Period*, Anthony Trollope discusses . . . the admirable scheme of a college into which at 60 men retired for a year of contemplation before a peaceful departure by chloroform. That incalculable benefits might follow such a scheme is apparent to any one who, like myself, is nearing the limit, and who has made a careful study of the calamities which may befall men during the seventh and eighth decades!

Although these suggestions were likely made in jest, the press tried to attribute subsequent suicides to these remarks.

Since the mid-1900s, modern geriatrics has offered clinical descriptions of general aging and the expectations of chronic illnesses near the end of life. In 1951, Robert Perlman put forth the concept that aging was the manifestation of the decreased ability to respond adaptively to stressors (2). In his formulation of the so-called "aging syndrome," he considered the multifactorial nature of the process to include internal factors (resistance), as well as external factors (stressors, both environmental and socioeconomic). He described aging as pathologic and cumulative, but thought that normal aging could be distinguished from chronic degenerative conditions. Our current usage of the term "geriatric syndrome" probably has its roots in Perlman's aging syndrome; where he attempted to describe the multiple etiologies that come together to produce general aging, we consider the diverse causes that contribute to a specific geriatric syndrome. Finally, Perlman offered, in broad terms, levels of interventions for aged individuals of varying debilitated state, which depending on particular circumstances and goals could be passive, palliative, therapeutic, prophylactic and/or rehabilitative.

In 1980, a hypothesis was proposed that if the onset of chronic illness could be postponed and if that delay was greater than the increases in life expectancy, then the lifetime burden of illness could be reduced. This idea, known as the "compression of morbidity" theory, was originally proposed by Fries and is reproduced here. At the time of the original proposal, there was little demographic data on trends in morbidity, and so other scenarios for future morbidity and longevity were possible besides compression of morbidity. The least desirable scenario would be to have increased longevity and no delay in the onset of chronic illness, thus extending the period of poor health before death. Alternatively, chronic illnesses might be postponed, but only in equal extent to increases in life expectancy. Each scenario assumes some increase in life expectancy.

National health surveys since 1982 suggest that disability trends have declined at about 2% per year in the United States, while mortality rates have declined at about 1% per year, thus providing evidence for compression of morbidity in the United States (3). This implies that the burden of chronic illness may be offset by a delay in the onset of infirmity.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

References

1. Osler W. Valedictory address at Johns Hopkins University. JAMA 1905;44:705–710.
2. Perlman RM. The Aging Syndrome. JAGS 1951;2:123–129.
3. Fries JF. Measuring and monitoring success in compressing morbidity. Ann. Intern. Med. 2003;139:455–459.

130 THE NEW ENGLAND JOURNAL OF MEDICINE July 17, 1980

SPECIAL ARTICLE

AGING, NATURAL DEATH, AND THE COMPRESSION OF MORBIDITY

JAMES F. FRIES, M.D.

Abstract The average length of life has risen from 47 to 73 years in this century, but the maximum life span has not increased. Therefore, survival curves have assumed an ever more rectangular form. Eighty per cent of the years of life lost to nontraumatic, premature death have been eliminated, and most premature deaths are now due to the chronic diseases of the later years. Present data allow calculation of the ideal average life span, approximately 85 years. Chronic illness may presumably be postponed by changes in life style, and it has been shown that the physiologic and psychologic markers of aging may be modified. Thus, the average age at first infirmity can be raised, thereby making the morbidity curve more rectangular. Extension of adult vigor far into a fixed life span compresses the period of senescence near the end of life. Health-research strategies to improve the quality of life require careful study of the variability of the phenomena of aging and how they may be modified. (N Engl J Med. 1980; 303:130-5.)

THIS article discusses a set of predictions that contradict the conventional anticipation of an ever older, ever more feeble, and ever more expensive-to-care-for populace. These predictions suggest that the number of very old persons will not increase, that the average period of diminished physical vigor will decrease, that chronic disease will occupy a smaller proportion of the typical life span, and that the need for medical care in later life will decrease.

In forecasting health, the interaction between two sets of observations has gone unnoticed. The first set demonstrates that the length of the human life is fixed — that man is mortal and that natural death may occur without disease. The second set indicates that chronic disease can be postponed and that many of the "markers" of aging may be modified. If these two premises are granted, it follows that the time between birth and first permanent infirmity must increase and that the average period of infirmity must decrease.

THE LENGTH OF LIFE IS FIXED

Speculation about immortality is rooted in antiquity and in human hope. The bioscientific, medical model of disease, our prevalent model, assumes that death is always the result of a disease process; if there were no disease, there would be no death. This view is hard to defend.

If relative immortality were possible, one would expect to find some persons who anticipated the future and acted accordingly. Thus, a person genetically favored and fortunate enough to avoid disease might live much longer than actuarially predicted. Data fail to confirm the existence of such events. For example, adequate data on the number of centenarians have been available in England since 1837; over this time, despite a great change in average life expectancy, there has been no detectable change in the number of people living longer than 100 years or in the maximum age of persons dying in a given year.[1]

The *Guinness Book of World Records* notes that the correlation between the claimed density of centenarians in a country and its regional illiteracy rate is 0.83. In Sweden, where careful investigations of centenarians are carried out, not one has yet exceeded 110 years of age. The greatest authenticated age in the world was recorded in Japan — 114 years.[2] Approximately one in 10,000 persons in developed countries lives beyond the age of 100. Moreover, inspection of the "tail" of the human survival curve demonstrates the falloff expected from a normal distribution, rather than the emergence of a few persons with notably long life spans.[3] There has been no satisfactory documentation of any society with exceptional longevity.[4]

Several theoretical explanations of the finite life span have been presented. At the cellular level, Hayflick and others have argued extensively for a finite number of cell doublings in the life span of a species. The number of doublings of human fibroblasts is approximately 50 (ref. 5); before reaching this point, subcultivation of cells proceeds in an active and youthful way. However, over a short period after the 50th subcultivation, the cells first fail to grow and then die, although there has been no change in the nutrients or other conditions of the culture medium. The number of doublings is species specific, and long-lived species have more doublings than do short-lived species.[6]

At the level of the organism, life may be defined as internal homeostasis. The internal milieu is adjusted within strict limits by compensating mechanisms in many organs, including heart, lungs, kidneys, and liver. In young adult life, the functional capacity of human organs is four to 10 times that required to sustain life. The existence of "organ reserve" enables the stressed organism to restore homeostasis when it is deranged by external threat. Measurement of organ reserve over time shows an almost linear decline beginning at about the age of 30.[7] As organ reserve

From the Department of Medicine (S102B), Stanford University Medical Center, Stanford, CA 94305, where reprint requests should be addressed to Dr. Fries.

This work was performed while the author was a Kaiser Fellow at the Center for Advanced Study in the Behavioral Sciences, and was delivered in part at the 2d Annual Nova Behavioral Conference on Aging, Fort Lauderdale, Fla., January 25, 1980.

(Continued)

decreases, so does the ability to restore homeostasis, and eventually even the smallest perturbation prevents homeostasis from being restored. The inevitable result is natural death, even without disease. Although a disease process may appear to be the cause of death, the actual cause is loss of the organism's ability to maintain homeostasis. Any small perturbation, without coexistent organ reserve, would have the same fatal result. Observations since those of Gompertz demonstrate an exponential increase in mortality rate after the age of 30; the rate doubles every eight years.[8] The best mathematical models[9] relate the linear decline in organ function to the exponential mortality rate. Obviously, an exponentially increasing mortality rate ensures a finite life span.

The Average Length of Life Is Increasing

The average length of life in the United States has increased from approximately 47 years at the turn of the century to 73 years today, an increase of 26 years (Fig. 1). Life expectancy for white women is now 77 years and for white men 70 years. A steady rise in life expectancy in the early years of this century changed to a relative plateau after 1950, but the increase has resumed in recent years.[10] Such data form the basis for predictions that more people will live beyond the age of 65 and for projections of medical facilities likely to be required in the future.

A more critical look at these data, however, demonstrates that they reflect progress in the elimination of premature death, particularly neonatal mortality. For persons 40 years of age and older, life expectancy has increased relatively little; for those 75 years old the increase is barely perceptible. Figure 1 presents a largely unnoticed paradox: if these lines are extrapolated into the future, at some point in the 21st century the average life expectancy as projected at birth will exceed average age of death as projected at age 75.

A white woman aged 70 may now expect to live 14 years longer (on the average), and a white man of the same age 11 years. Present differences in life expectancy between sexes and races become much smaller as the age on which the analysis is based rises. Racial minorities and men are more subject to premature death.

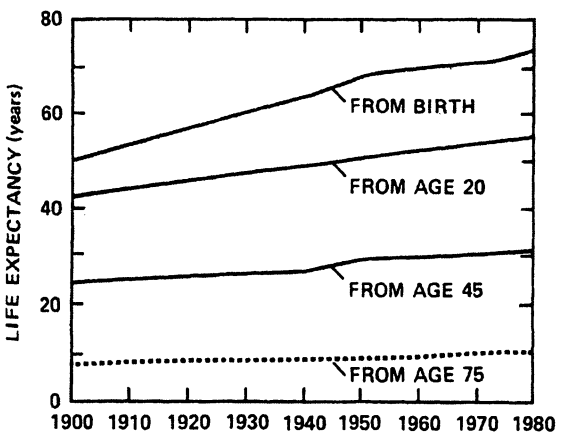


Figure 1. Life Expectancy Trends in the United States. Life expectancy at birth has increased by 26 years in this century, and expectancy at 75 (broken line) by only three years. The slope decreases as the life span is neared.

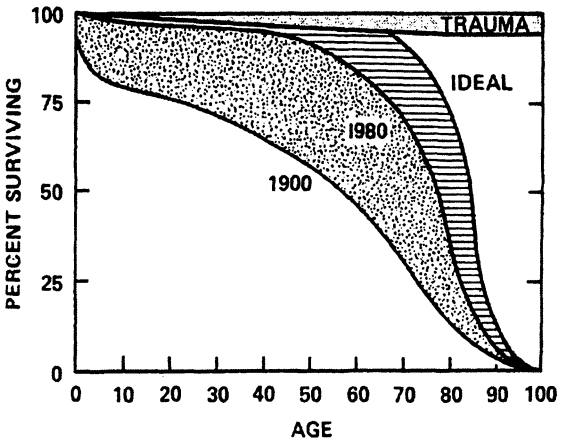


Figure 2. The Increasingly Rectangular Survival Curve. About 80 per cent (stippled area) of the difference between the 1900 curve and the ideal curve (stippled area plus hatched area) had been eliminated by 1980. Trauma is now the dominant cause of death in early life.

The shape of the survival curve provides additional insights. In antiquity, as in many species of animals now, death was almost a random event: an organism succumbs to an intercurrent problem before reaching the life span usual for members of the species. In 1900, the survival curve in the United States was not very different from this situation. However, sequential survival curves throughout this century show progressive "rectangularization" [1,8] as the elimination of premature death results in a sharp downslope to the natural life span (Fig. 2). The serial data allow calculation of the position and shape of a survival curve if all premature death were eliminated: an ideally "rectangular" survival curve. If we assume a normal biologic distribution, statistics suggest that under ideal societal conditions mean age at death is not far from 85 years.

The natural limit to the life span can be calculated in several ways. Perhaps the easiest, after study of the rate at which life expectancy at various ages is increasing, is to calculate the point at which the curves intersect (Fig. 3). For example, over the first eight decades of this century average life expectancy from birth increased at the rate of 0.33 year per year of the century, and life expectancy from age 65 has increased by 0.05 year per year. These curves intersect in the year 2009, at a mean age at death of 82.4 years. During the most recent decade, average life expectancy from birth has also increased 0.33 year per year, and life expectancy from age 65 has increased at 0.12 year per year. These curves intersect in the year 2018, at a mean age at death of 85.6 years.

Calculations based on other periods or from other ages converge at similar points. Figure 3 shows intersection at age 85 in the year 2045, a reasonable median projection. In actuality the curves will not be straight but will approach an asymptote; the limit will be approached more slowly, and the attainable average life expectancy will be less than the theoretical estimate. Predictions by the federal government (Fig. 3) make such nonlinear assumptions and suggest that the actual limit may be less than 85 years.

Mortality data describe a biologic distribution, which appears approximately normal in populations of laboratory animals. If the tail of the survival curve remains fixed and the biologic distribution is normal, an age of 100 years is about four standard deviations from the mean, and the standard deviation about four years. Thus, under ideal conditions, 66 per cent of natural deaths would occur in persons 81 to 89 years, and 95 per cent in persons aged 77 to 93 years. With a biologic distribution, the ideal survival curve will never be completely "rectangular," and, if the rate of violent and traumatic death (a category now accounting for more than half of deaths in persons under the age of 40 years[9]) remains roughly constant, there will always be some premature deaths.

Changes in survival curves in this century may be compared with the hypothetical ideal curve in Figure 2. Since 1900, Americans have covered most of the distance to that ideal, in terms of years of life saved: our progress has removed about 80 per cent of the area between the ideal curve and the 1900 curve (if the rate of violent death is disregarded). Moreover, the great change has occurred in the early years of life, with most remaining premature deaths concentrated in the years after age 60.

These changes are dramatic. In 1900 the average citizen died 38 years "prematurely" (short of the theoretical limit), in 1950 17 years, and in 1980 only 12 years. In 1980 white women will die on the average only seven years prematurely. Moreover, violent death accounts for three of the years by which we fall short of the limit. Clearly, the medical and social task of eliminating premature death is largely accomplished.

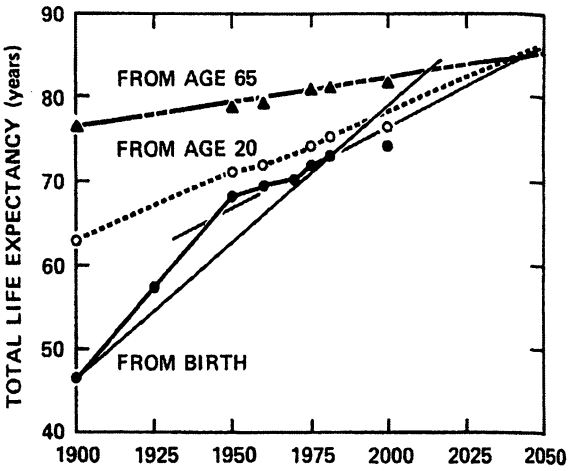


Figure 3. Trends, Limits, and Convergences in Life Expectancy.

Projection of total life expectancy data into the future shows convergence at the ideal average life span, 85 years, in 2045. Spans of 82.6 to 85.6 years can be calculated from projections from different ages (at birth, at age 20, and at age 65) and different years in this century. (Data are from the National Center for Health Statistics [1977][10]; values indicated by triangle and circles for the year 2000 are estimates from the Office of the Actuary.)

Chronic Disease Has Superseded Acute Disease

Acute illness has ceased to be the major medical problem in the United States. At the turn of the century, mortality patterns were dominated by acute, usually infectious disease. Tuberculosis, acute rheumatic fever, smallpox, diphtheria, tetanus, poliomyelitis, pneumococcal pneumonia in the young, and similar conditions constituted the principal threats to health.[10] Each of these now causes less than 2 per cent of the health problems that it caused in 1900.[11] Smallpox has been eradicated; polio, almost so. The decline in these diseases can be attributed to a number of factors, including improved nutrition, less crowded living arrangements, water sterilization, immunization, and specific antibiotics.[12] It is important to recognize that chronic diseases have replaced acute illness as major health threats.

Chronic illness now is responsible for more than 80 per cent of all deaths and for an even higher fraction of cases of total disability.[8] Arteriosclerosis (including coronary-artery disease and stroke), arthritis, adult-onset diabetes, chronic obstructive pulmonary disease (including emphysema), cancer, and cirrhosis represent the overwhelming majority of our health problems. They are widespread conditions that originate in early life and develop insidiously; the probability of their occurrence increases with age. They can be considered, broadly, as problems of accelerated loss of organ reserve. Generally, they develop slowly and asymptomatically below a clinical threshold, at which the process becomes clinically evident, progresses, and often culminates in death or disability.

Thus, the early arteriosclerotic plaque does not materially impede circulation, but gradually the probability of an acute thrombotic event or insidious vascular insufficiency increases. The osteoarthritic bone spur is evident on x-ray films for many years before pain or disability is noted in the affected joint. Glucose tolerance decreases gradually until sugar is excreted in the urine of the diabetic. The patient with emphysema has accelerated loss of pulmonary reserve. The probability of development of neoplasms increases with age.

Disability and lowered quality of life due to the most prevalent chronic diseases are thus inescapably linked with eventual mortality. These chronic diseases

(Continued)

are approached most effectively with a strategy of "postponement" rather than of cure. If the rate of progression is decreased, then the date of passage through the clinical threshold is postponed; if sufficiently postponed, the symptomatic threshold may not be crossed during a lifetime, and the disease is "prevented."

Some chronic illnesses definitely can be postponed; elimination of cigarette smoking greatly delays the date of onset of symptoms of emphysema and reduces the probability of lung cancer. Treatment of hypertension retards development of certain complications in the arteries. In other illnesses, circumstantial evidence of similar effects of postponement is strong but proof is difficult: that arteriosclerosis is retarded by weight reduction or exercise is suggested by associative data but has not yet been proved.

Until recently, progress in health care could be conceived of as an exchange of acute medical problems for chronic ones: the person who survives an illness appearing abruptly early in life will have more lingering problems later. Since early death would cost relatively little in direct expenses as compared with the expenses of a later chronic problem, the exchange of acute illnesses for chronic ones has resulted in a massive need for additional medical services. The end of this era is nearing because there are now few acute illnesses to be "exchanged."

The most recent increases in average life expectancy are due principally to a decline in arteriosclerosis, particularly cerebrovascular disease. This decline is the first demonstration of a national decrease in mortality from a major chronic disease, and most observers attribute the change to changes in life style and to better treatment of hypertension.[13] The 26 per cent decline in per capita tobacco consumption over the past 15 years,[14] now accelerating, may effect at least a similar percentage of decrease in the incidence of chronic obstructive pulmonary disease and lung cancer, after a delay of a few years. Moreover, the preventive approach to chronic illness is still in its infancy. The long-term effects of increased exercise, lower weight, and growth in personal autonomy and personal responsibility for health are also likely to be positive.[15]

The Compression of Morbidity

The amount of disability can decrease as morbidity is compressed into the shorter span between the increasing age at onset of disability and the fixed occurrence of death. The end of the period of adult vigor will come later than it used to. Postponement of chronic illness thus results in rectangularization not only of the mortality curve but also of the morbidity curve.

The social consequences of this phenomenon will be profound. Death and disability, occurring later, become increasingly unavoidable. The incremental cost of marginal medical benefit inevitably rises. Intervention in the patient without organ reserve will be recognized as futile. The principles of fixed mortality and of natural death without disease carry profound implications.

Some caveats must be mentioned. War, depression, pestilence, or natural disaster could reverse recent trends. The human life span may not be fixed but may be slowly increasing, perhaps a month or so each century; the data are consistent with this hypothesis. The Hayflick phenomenon may have nothing to do with human aging. Medical progress may increase the number of cell doublings, learn to slow organ decay, or extend the maximum life span in some other way, notwithstanding its failure to do so to date. But it is highly unlikely that any such change will occur during our lifetime. The likelihood depends on whether the lowest curve of Figure 1 (life expectancy after age 75), after being relatively stable for many decades, will show a sudden upturn. Many of the chronic diseases, including arteriosclerosis, may be susceptible to "cure," and efforts directed at finding curative treatments must be continued. There will always be illness; theoretical curves may be approached but not reached. The surprising fact is that we are already approaching the limits.

By implication, the practical focus on health improvement over the next decades must be on chronic instead of acute disease, on morbidity not mortality, on quality of life rather than its duration, and on postponement rather than cure. The complex nature of the major diseases calls attention to multifactorial influences on outcome, in particular social and psychologic factors. Outcome is related to choice; assumption of personal responsibility, education for making decisions about personal health, and ability to encourage self-care are clearly essential to changing health behaviors.[16] Returning responsibility to the patient may cause anguish.

The Compression of Senescence

An important shift is occurring in the conceptualization of chronic disease and of aging. Premature organ dysfunction, whether of muscle, heart, lung, or joint, is beginning to be conceived as stemming from disuse of the faculty, not overuse. At the Stanford Arthritis Clinic I tell patients to exercise, and to "use it or lose it"; "Run, not rest" is the new advice of the cardiologist. The body, to an increasing degree, is now felt to rust out rather than to wear out. If loss of reserve function represents aging in some sense, then exercising an organ presents a strategy for modifying the aging process.

The links between the widespread chronic diseases and aging are the insidious loss of organ reserve common to both processes and the often identical factors that influence the rate of development. In preventive medicine these variables are seen as antecedents to disease, whereas in gerontology they are markers of age. Serum cholesterol, vital capacity, and systolic

blood pressure are examples of such variables. Exercise, weight control, and diet are some of the common modifying factors.

The modifiability, or "plasticity," of aging has been demonstrated in studies in which performance can be bettered despite age, within surprisingly broad limits. This important phenomenon has been largely unnoticed partly because of an emphasis on average rather than individual performance and partly because disparate disciplines are involved. Average declines in variables in aging can hide remarkable individual variation. The marathon runner is an example (Fig. 4). A runner in middle life who completes a marathon in 3½ hours is in the 99th percentile for this endeavor; yet not until age 73 would that time set an age-group record. These marathon data are important in that they show the maximum rather than the average performance, but here too there is a linear decline in performance between age 30 and 70. Still, the age-related decrement in maximal performance is only 1 per cent per year. Variation between healthy persons of the same age is far greater than the variation due to age; age is a relatively unimportant variable, and training in marathon running is clearly more important than age.

Similar observations on increased variation between individuals with age and on modifiability with training, even after age 70, have been made for intelligence testing,[17,18] social interaction,[19] health after exercise,[20] and memory.[21] Certain data indicate improvement with age, against the gradient of linear decline, for some persons. An inference is that personal choice is important — one can choose not to age rapidly in certain faculties, within broad biologic limits.

Such considerations suggest that research strategy toward aging be fundamentally shifted. Analysis of variation, not of the mean values, becomes crucial. Indeed, one can argue that the number of studies showing that the mean of every function declines steadily with age is already sufficient. Research now requires measurement of standard deviation between individuals, not of standard error between populations or between chronological ages.

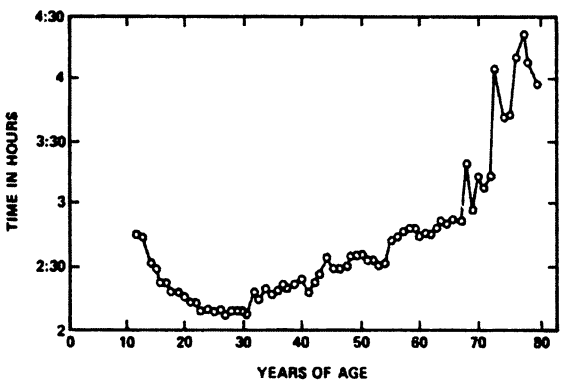


Figure 4. World Marathon Records for Men.
Note the slow but linear decline in maximum performance between the age of 30 and 70.

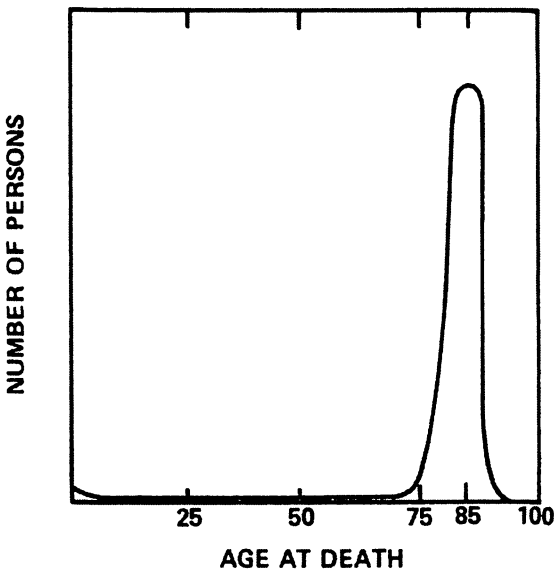


Figure 5. Mortality According to Age, in the Absence of Premature Death.
The morbidity curve is made rectangular, and the period of morbidity compressed between the point of the end of adult vigor and the point of natural death.

A new three-stage research strategy may be urged: measure the variability of a marker of aging (e.g., oxygen uptake, satisfaction as assessed by questionnaire, or intelligence as measured by IQ test) at a given chronological age; determine retrospectively the differences between the individual who has aged more rapidly and the one who has aged more slowly in that marker — hypothetically, such differences may be expected to correlate with the individual's practice in self-maintenance and to be confounded by self-selection; and design prospective intervention studies to explore causality.

At the top of the list of nationally important health-research subjects must be the ability to postpone chronic illness, to maintain vigor, and to slow social and psychologic involution. We must know for certain whether change is possible and how to accomplish it best. Personal autonomy has been emphasized above as a probable final common pathway to improved health. This emphasis is meant broadly, since clearly the collective efforts of individuals are required for removal of environmental hazards and the development of incentives to encourage rather than discourage the exercise of personal choice. We know relatively little about the specific relations between social changes and personal decisions, and much information in great depth is needed.

Summary

I have presented a model for national health that foresees continued decline in premature death and emergence of a pattern of natural death at the end

(Continued)

of a natural life span. Present approaches to social interaction, promotion of health, and personal autonomy may postpone many of the phenomena usually associated with aging. The rectangularization of the survival curve may be followed by rectangularization of the morbidity curve and by compression of morbidity (Fig. 5).

These considerations suggest a radically different view of the life span and of society, in which life is physically, emotionally, and intellectually vigorous until shortly before its close, when, like the marvelous one-hoss-shay,[22] everything comes apart at once and repair is impossible. Such a life approaches the intuitive ideal of many and confounds the dread of others for the opposite model, that of evermore lingering death. Paradoxically, predictability of death may prove soothing.

Since maintenance of organ capacity appears to require practice on the part of the individual, the implications for the societal role are as fundamentally different as are the two models. Indeed, the choice of societal postures toward the aged is likely to prove self-fulfilling. The older person requires opportunity for expression and experience and autonomy and accomplishment, not support and care and feeding and sympathy. High-level medical technology applied at the end of a natural life span epitomizes the absurd. The hospice becomes more attractive than the hospital. Human interaction, rather than respirators and dialysis and other mechanical support for failing organs, is indicated at the time of the "terminal drop." Anguish arising from the inescapability of personal choice and the inability to avoid personal consequences may become a problem for many. For others, exhilaration may come from recognition that the goal of a vigorous long life may be an attainable one.

I am indebted to Margret Baltes, Paul Baltes, John Bunker, Larry Crapo, Sarah Fries, Victor Fuchs, Halsted Holman, Elizabeth Loftus, Jack Riley, Matilda Riley, David Rogers, Martin Seligman, and George Valliant for their comments and criticisms.

References

1. Comfort A. The biology of senescence. 3d ed. New York: Elsevier Press, 1979:81-6.
2. McWhirter N. Guinness book of world records. 17th ed. New York: Bantam Books, 1980.
3. Greenwood M, Irwin JO. The biostatistics of senility. Hum Biol. 1939; 11:1-23.
4. Mazess RB, Forman SH. Longevity and age exaggeration in Vilcabamba, Ecuador. J Gerontol. 1979; 34:94-8.
5. Hayflick L. Aging under glass. Exp Gerontol. 1970; 5:291-303.
6. *Idem.* The cellular basis for biological aging. In: Finch LE, Hayflick L, eds. Handbook of the biology of aging. New York: Van Nostrand Reinhold, 1977:159-86.
7. Shock NW. Mortality and measurement of aging. In: Strehler BL, Ebert JD, Glass HB, Shock NW, eds. The biology of aging. Washington, D.C.: American Institute of Biological Sciences, 1960:14-29.
8. Upton AC. Pathology. In: Finch LE, Hayflick L, eds. Handbook of the biology of aging. New York: Van Nostrand Reinhold, 1977:513-35.
9. Strehler BL, Mildvan AS. General theory of mortality and aging. Science. 1960; 132:14-21.
10. National Center for Health Statistics. Health in the United States, 1978. Hyattsville, Md.: National Center for Health Statistics, 1978. (DHEW publication no. (PHS)78-1232).
11. Fries JF, Ehrlich GE, eds. Prognosis: contemporary outcomes of disease. Bowie, Md.: Charles Press, 1980. (in press).
12. McKeown T. The role of medicine: dream, mirage, or nemesis. 2d edition. Princeton, N.J.: Princeton University Press, 1979.
13. Stern MP. The recent decline in ischemic heart disease mortality Ann Intern Med. 1979; 91:630-40.
14. Walker WJ. Changing United States life-style and declining vascular mortality: cause or coincidence? N Engl J Med. 1977; 297:163-5.
15. Farquhar JW. The American way of life need not be hazardous to your health. New York: WW Norton, 1978.
16. Vickery DM, Fries JF. Take care of yourself: a consumer's guide to medical care. Reading, Mass.: Addison-Wesley, 1976.
17. Baltes PB, Schaie KW. On the plasticity of intelligence in adulthood and old age: where Horn and Donaldson fail. Am Psychol. 1976; 31:720-5.
18. Plemons JK, Willis SL, Baltes PB. Modifiability of fluid intelligence in aging: a short-term longitudinal training approach. J Gerontol. 1978; 33:224-31.
19. Rodin J, Langer EJ. Long-term effects of a control-relevant intervention with the institutionalized aged. J Pers Soc Psychol. 1977; 35:897-902.
20. Paffenbarger RS Jr, Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol. 1978; 108:161-75.
21. Langer EJ, Rodin J, Beck P, Weinman C, Spitzer L. Environmental determinants of memory improvement in late adulthood. J Pers Soc Psychol. 1979; 37:2003-13.
22. Holmes OW. The deacon's masterpiece: or the wonderful "one-hoss-shay." In: The autocrat of the breakfast table. Cambridge, Mass.: Houghton Mifflin, 1881.

2
History of Geriatric Medicine

Mary Ann Forciea, MD

Reproduced paper following commentary: Warren, Marjory. The Evolution of a Geriatric Unit. Geriatrics 1948;3:42–50. Reprinted with permission from the American Geriatric Society.

Commentary

From the years following the founding of the United States up to the period before World War II, the majority of ongoing care of the elderly in the United States and Great Britain was provided in three sites:

- homes of those older adults or the homes of family members
- grouped with the poor (poor homes or work farms)
- grouped with the mentally ill (asylums).

Placement within these sites was determined primarily by economics rather than by medical illness or functional status. Physicians made little distinction between the care of older patients and treatment of any other adult, other than to spend less time in diagnosis and therapy of older patients, especially if those patients were suffering from chronic diseases. The achievement of longevity was unusual; surviving to retirement slowly became more common throughout the twentieth century.

Along with increases in longevity during the twentieth century, several other developments contributed to changes in the care of older adults:

- increased professionalism in medicine and nursing
- standards in training of physicians and nurses
- increasing standards of research into clinical problems of older patients
- large numbers of WWI wounded veterans who needed chronic care were forced into existing institutions
- large numbers of conscientious objectors assigned to chronic care hospitals began to write about and lobby for improved care.

In the United States, the Flexner report had revolutionized the training of physicians away from "apprenticeships" toward formal curricula in accredited universities. Academic physician teachers and researchers began to look critically at the care of all patients, even occasionally at older patients. Similarly, nursing training began to move toward academic centers. At the same time, younger, mentally intact patients and their families who demanded more than custodial care challenged institutional facilities. The observations and activism of staff assigned to these facilities as alternatives to military service were positive forces for change.

In the 1940s in Great Britain, Marjory Warren began publishing papers describing her transformative work for elderly patients in a hospital for the chronically ill. She summarized her process of assessment of patients in that facility and the development of her "Geriatric Unit" in a paper in 1948 (1). She described the categories of patient that she felt belonged more appropriately together:

The chronic "up-patients" (ambulatory patients)
Chronic continent bed bound
Chronic incontinent bed bound
Senile, quietly restless (not noisy or annoying) and
Senile, demented (noisy and/or annoying).

She strongly advocated, for the first time in major medical journals, for careful assessment of patients on entry into institutions of care, for special training of medical students in the assessment and care of those patients, and for strong links to hospitals or teaching centers. She shared information on design, equipping, and staffing her units, and she provided data on admissions, mortality and discharge rates. The enduring quality of some of the challenges of care for these frail, older patients was captured in the passage:

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

In the beginning, the Geriatric Unit was regarded by most of the medical staff as a convenient unit to which to transfer all their unwanted patients, medical and surgical, old or young and usually without consultation. This old tradition has died slowly . . . (1)

Her advocacy for assessment of the older patients in her care greatly influenced the generation of British physicians whose members established Geriatric Medicine in the National Health Service after World War II. Geriatric evaluation of the older adult began to develop in the United States in the 1960s and 1970s. This protocol of initial evaluation, usually by a team of professionals and often linked to function status complaints and outcomes, spread to nursing homes (2), office practices (3), and housecalls (4). Williams and colleagues (5) published a protocol of evaluation for patients referred for long-term institutional care and documented unnecessary loss of autonomy without careful evaluation prior to placement. In the United States, the commitment of the Veterans' Administration to specialized care of older veterans allowed the development, testing, and training of specialized programs focused on older patients. The first controlled trial of a program of initial geriatric assessment and subsequent care appeared in 1984 (6). Rubenstein and colleagues demonstrated that specialized evaluation and care resulted in improved function that was maintained longer than usual care.

While techniques of assessment and care have been developing over the last thirty years of practice (see later chapters in this volume), education in the interdisciplinary team practice, which is the foundation of that assessment, has been evolving more slowly. The Hartford foundation has invested funds and support to develop instructional methodologies to promote team practice (7). Certainly the blossoming of the field of education in the skills involved in successful practice of medicine and nursing should aid efforts to prepare health professionals for the world of practice with older adults in the twenty-first century.

References

1. Warren MW. The Evolution of a Geriatric Unit. Geriatrics 1948;3:42–50.
2. Libow L. Geriatric Medicine and the Nursing Home: A Mechanism for Mutual Excellence. Gerontologist 1982;22:134–141.
3. Millman A, Forciea MD, Fogel D, Johnson JC. A model of interdisciplinary ambulatory geriatric care in a Veterans' Administration Medical Center. Gerontologist 1986;26(5):471–474.
4. Cherkasky M. The Montefiore Hospital Home Care Program. Am J Public Health 1939;39:163–166.
5. Rubenstein LZ, Josephson KR, Wieland GD et al. Effectiveness of a geriatric evaluation unit: A randomized clinical trial. New Engl J Med 1984;311:1664–1670.
6. Williams TF, Hill JG, Fairbank ME et al. Appropriate placement of the chronically ill and agend: A successful approach by evaluation. JAMA 1973;226:1332–1335.
7. Fulmer T, Hyer K, Flaherty E, et al. Geriatric Interdisciplinary Team Training Program: Evaluation Results. J Aging Health 2005;17:443–470.

THE EVOLUTION OF A GERIATRIC UNIT

Marjory W. Warren, M.D.

During the present century, as a result of greatly improved welfare of almost all sections of the community, the tremendous expansion of preventive medicine and research work in the realms of therapy, there has been a marked age shift in the population of Great Britain.

The following tables fully endorse this statement.

TABLE 1. GREAT BRITAIN—NUMBER AGED 60 OR OVER

Year	Men	Women	Total
1901	1,071,519	1,336,907	2,408,426
1939	2,511,200	3,197,400	5,708,600
1944	2,737,000	3,590,000	6,327,000
1946	2,828,000	3,759,000	6,587,000

TABLE 2. GREAT BRITAIN—TOTAL POPULATION

Year	Total
1901	37,000,000
1944	47,628,000

TABLE 3. GREAT BRITAIN—EXPECTATION OF LIFE

Year	Men	Women
1891–1900	44.1 years	47.8 years
1901–1910	48.5 years	52.4 years
1942	61.7 years	67.4 years

In addition to these facts are two other conditions which have seriously added to the problem of the overall care of the elderly sick, namely: the enormous loss of houses from aerial destruction during the World War II and the retardation of the building programme brought about by the war, and the post war economy; the diminishing tendency for the elderly to be cared for by their own family. This latter reason would form subject matter for an article on its own. Suffice it here to point out that various factors contribute to the condition, such as, the smaller size of the family, the frequent scattering of the members of the family to widely separated parts of Great Britain, or even overseas, and to the present day weakening of the sense of filial responsibility.

The fascination of Medicine lies in its basic qualities—its wide social and humanitarian aspects, its progressive nature and its variety. Of all branches of Medicine, that of treatment of the chronic elderly sick has received, so far, less attention than others and consequently offers the widest scope for pioneer work and research. With the ageing of the population of Great Britain this subject has become not only one of academic interest, but one calling for urgent reform and practical solution.

Marjory W. Warren, M.D., is Deputy Medical Director, West Middlesex County Hospital, Isleworth, England; member of the Medical Society for the Care of the Elderly recently formed in England.

(Continued)

THE EVOLUTION OF A GERIATRIC UNIT 43

Although the term "geriatric," first used by Nascher in 1909 in America, has not yet been generally accepted in medical circles in Great Britain, there is a good deal to recommend its use. The word has the merit of defining patients in the upper age groups and separating them from the so-called "chronic" patients of the younger groups. At the present time, the majority of the elderly sick are referred to as "chronic" because the maladies and multiplicity of diseases from which they suffer call for treatment of long duration and almost always leave residual disabilities.

Of recent years, there has been a gradual awakening to the general inadequacy of treatment meted out to the majority of elderly sick and many sections of the community realise the urgent need for a much better service both for medical treatment and social care.

From the figures quoted previously and our knowledge that the morbidity rate rises with increasing age there can be no doubt that in the future provision must be made for much larger numbers of elderly sick than has been necessary in the past, and this provision must, moreover, be varied and suitable for all types. With a changed economic outlook, it must be presumed that many more classes of persons, whether sick for short or long term conditions, will need hospital accommodation. In the immediate future, if indeed ever, there is no likelihood that nursing and domestic personnel will be available for individual hire as in the past. Even if such help is obtainable, the greatly increased rates of pay, especially of domestic workers, will certainly make it impossible for the elderly and sick professional and middle class persons, on a small fixed pension, to remain cared for in their own homes. This side of the problem is manifest today, for already there are many such persons who have not the means to obtain the help that is essential and who find it almost impossible to gain admission to hospitals or institutions providing beds for long term illness of elderly sick. We also see pathetic cases of the well-to-do with ample means and large homes, yet who are unable to obtain the help that they need and are living in considerable discomfort and great loneliness.

All this at first suggests the need for a much larger number of hospital beds at a time when building is difficult, and the shortage of nurses acute. There are, however, one or two other factors which should be considered before plans are drawn up. Firstly, the treatment of many of the acute conditions amongst younger patients is shortened by modern therapy, such as sulpha drugs and penicillin, to mention but two, and so the turnover of such conditions should be possible in fewer beds, thus releasing some for the long stay cases. Some preventable diseases may never need hospitalization at all. Secondly, and this is not yet sufficiently widely appreciated, much can be done to rehabilitate elderly patients so that a considerable number may be discharged from the hospital. Some could return to their own homes and those unfit to return home could enter a small residential home, thus retain-

ing a modicum of liberty and using much less costly accommodation than would be provided in a hospital bed with full equipment and nursing staff.

In this connection it should be remembered that a number of the most crippling conditions from which the long-term chronic sick suffer are preventable. Many more conditions may be found preventable or treatable when more time and research has been devoted to such cases.

THE EVOLUTION OF THE GERIATRIC UNIT

During the past twenty years certain changes in administration affecting the treatment and care of large numbers of the long-term elderly sick patients have appeared. In 1929 large numbers of sick persons in hospitals, previously administered by Boards of Guardians, became the responsibility of the respective County Councils with their County Medical Officers. Later, certain additional institutions for the chronic sick and infirm, previously the responsibility of Public Assistance under the Poor Law, were appropriated by Public Health. In July 1935, one of these Poor Law Institutions, carrying about 700 chronic sick and infirm persons, became by change in legislation part of the general Public Health Hospital, which had itself been taken over by the County Council from the local Board of Guardians in 1929.

This change in responsibility called for immediate reform in order to raise the standard of the care and treatment of persons in the Institution to that of the Public Health hospital of which it had now become a part.

As there must still be many such institutions both in Great Britain and in other countries carrying large aggregate numbers of chronic sick and infirm persons, it is thought that it may be of interest to study the development and growth of one which has built up a Geriatric Unit from some of these wards, and to discuss the functions and future rôle of such a unit in the general hospital. It is the account of this pioneer unit still in the making which is the subject of this article.

CAUSES OF FAILURE IN THE PAST

There can be little doubt that in the past failure to obtain good results for the elderly sick and infirm, has been brought about by too little attention from medical and nursing staffs. It may be profitable here to consider the reasons for these failures.

1. Lack of continuity in the care of the elderly sick brought about mainly by lack of medical interest and consequent early transfer of the long stay patient away from senior medical staff to the hospital for "chronic" patients.
2. Failure to investigate and treat fully by modern methods all patients with whatever condition and of whatever age.
3. Failure to provide first class equipment for the chronic elderly sick comparable to that used for the younger acute patients.
4. Lack of appreciation on the part of medical persons as to how much can be done to rehabilitate and resettle elderly sick patients.

(Continued)

With these points in mind, and before any plans were made as to the ultimate development of a Geriatric Unit, every patient (about 700 in all) was fully examined, medically assessed and, where necessary, given appropriate treatment. In the majority of cases, relatives and friends were interviewed concerning the future. Almost immediately about 200 relatively able-bodied elderly persons were transferred to a Residential Home for old people close to the Hospital and run by Public Assistants, thus freeing accommodations for acute medical patients. Approximately 150 patients attached to the mental observation wards were left in the care of the psychiatric staff. This left about 350 really chronic sick patients to be cared for and treated.

The survey took in all about seven months, and on its completion certain facts emerged and certain preliminary recommendations were made. Facts revealed were:

1. That hitherto there had been no classification of patients.
2. That, in consequence, all wards contained so many different types of patients that the accommodation and equipment could not possibly be best for each, nor could any one staff cater really well for all.
3. That none of the modern ancillary services for patients were in use in the wards, nor had it been considered necessary that there should be such services. Indeed, physiotherapists and occupational therapists thought it unprofitable to work on such wards, and a fully trained nursing staff felt it a bore and somewhat beneath them to work with the chronic elderly sick.
4. That wards were large, overcrowded, dull and inadequately lighted.
5. That ward equipment was neither modern nor adequate.
6. That beds were all low, black and of an old-fashioned type and, therefore, difficult from a nursing point of view.

DEVELOPMENT OF A GERIATRIC UNIT

Preliminary plans for reorganization were drawn up and substantial changes were made in March 1936. Classification was started as follows:

1. Wards for investigation and treatment of geriatric patients, including new admissions and those transferred from other departments in the hospital.
2. A ward provided with a majority of cot beds for patients needing some restraint for physical or psychological reasons, but not bad enough to be segregated in the mental observation ward.
3. Ward for female patients whose main or only disability is incontinence of urine and/or faeces.
4. Ward for patients getting up and about and awaiting vacancies in Residential Homes or with friends.
5. Ward for patients no longer needing active medical treatment or rehabilitation, but still requiring a good deal of nursing and some medical supervision.

(These patients need not necessarily be kept in wards of the hospital but could be nursed in a Long Stay Annexe if such accommodation were available outside the hospital, *but* they *should remain the medical responsibility of the Geriatric Unit.*)

Although no major reconstruction was sanctioned, certain improvements and structural alterations of a minor character were undertaken between 1936-1938. These included removal of frame work obstructions interfering with free movement between one part of a ward and another, such as, the replacement of narrow doors by swing doors and a considerable improvement in lighting by installation of individual indirect lighting. The wards were repainted cream in place of the dark colours previously used and the low, black, old-fashioned beds were replaced by high nursing beds, easy-moving, with adjustable back rests and in light pastel shades. In the ward for up patients, low beds were used of a similar modern pattern, and in light colours. The total number of beds was reduced to conform to the standard spacing and each was furnished with a single modern locker, a bed table and a pair of ear phones conveniently hung at the head of the bed.

The wards were equipped with modern clinical aids, similar to those used in the acute wards, and gradually they fell into line with the general wards of the hospital. Additional equipment was added to these wards suitable to their special kinds of patient, for example, hand rails were fixed in parts of the ward devoid of beds so that patients could maintain independence in walking, even in the early stages. Additional arm chairs of varying types were obtained, as more of this type of furniture is needed when larger numbers of long-stay patients are under treatment. For the cot bed ward and the ward for incontinent patients larger stocks of bed linen were supplied.

Following this preliminary classification and segregation, with improved and additional equipment and in a new spirit for this type of patient, the unit set to work to deal with its large numbers of inherited patients, many of whom were bedridden without adequate medical cause, and to tackle its new admissions with an air of hopefulness. Under these new conditions the embryo of the Geriatric Unit was born.

GROWTH OF THE GERIATRIC UNIT

The early years were devoted to pioneer work in internal organization and administration, to improvement in staffing, to details of equipment and to the building up of teamwork, especially amongst nursing and ancillary staff, including physiotherapists and occupational therapists. Many improvements and changes have been made, and today, though far from perfect, an experimental centre is well-established.

In 1942, when the turn-over on these wards had increased considerably, it was found possible to gradually allocate about 90 beds for the Tuberculosis Service, and so the Geriatric Unit was reduced to approximately 200 beds.

(Continued)

New admissions to the Geriatric Unit are never made directly into the ward for incontinent patients, or into the ward for up patients, as it is thought that all patients admitted to these wards should be assessed first. On the male side, the numbers being much smaller (about 1:3) than on the female side, it has not been possible so far to arrange for the same degree of segregation. Incontinence amongst men is never such a serious problem, and a well-trained staff can deal with the majority of incontinent patients by simple nursing methods. At present men needing cot beds have to go to the observation ward attached to the mental block.

Progress at first was necessarily slow, the patients were difficult to re-educate, some were hopeless, and in the early days there was no really good teamwork. One ward which was entirely satisfactory from the start was the ward of 45 female patients set aside for incontinents. In this ward, first-class nursing has been done and a good deal of re-education satisfactorily completed. Many elderly patients have been "cured" of their incontinence and have been able to return home or enter a residential home on leaving this ward. It has never been regarded as a permanent home for patients admitted, although, of course, the turnover must inevitably be very slow, and a certain number of patients must finally be regarded as incurable.

In a different way, the cot bed ward has also done very good work, and the staff on this ward has also treated in an atmosphere of hopefulness and with the right psychological approach. The results have been very promising, considering the type of patient.

In the beginning, the Geriatric Unit was regarded by most of the medical staff as a convenient unit to which to transfer all their unwanted patients, medical and surgical, old or young and usually without consultation. This old tradition has died slowly, but recently there has been evidence that this custom is disappearing and now patients are usually referred in consultation first as to suitability. Also, it is more generally accepted that young chronic patients should not in principle be sent to geriatric wards.

The onset of the war temporarily relieved the hospital of numbers of infirm patients when relatives took their folk with them to be together in the greater danger which lay ahead. With air raids, however, new problems arose and numbers of elderly persons were rendered homeless, and sometimes in one night lost all the younger members of the family. Others were themselves the victims of air raids and suffered physical and psychological trauma. Such cases clearly needed full medical treatment and custodial care in whatever seemed the best accommodation.

The majority of the patients admitted were suffering from:

1. General debility including malnutrition, anaemia, etc.
2. Psychiatric conditions, including senile dementia.
3. Cardio-vascular degenerations, including cerebral arterio sclerosis, cerebral thrombosis, etc.

4. Arthritis—usually of the degenerative type.
5. Progressive nervous diseases.
6. Chest diseases.
7. Neoplasms.
8. Injuries—often referred from the orthopaedic department for rehabilitation.

Patients were unselected except on age (those over 60 years being preferred) and a number were primarily treated in the acute wards. When it became apparent that the prognosis was hopeless they were transferred to the Geriatric Unit, while others of advanced years who were expected from the outset to do well were admitted to the wards for acute patients.

For the aforementioned reasons, and because of the war and difficult housing conditions, the over-all picture could not be considered as quite normal. The results on statistical grounds are obviously less good than would have been obtained had the Unit admitted all new geriatric patients to its wards and if it had refused all geriatric patients who were transferred owing to poor prognosis.

In spite of all these abnormal conditions, however, in 1944 two wards in the unit, one male and one female, started keeping full records of all patients admitted or transferred to its wards, and some interesting figures have emerged. These figures are now published from this young and experimental unit for academic interest, as the writer knows of no previously published comparable figures from a Geriatric Unit of a general hospital, and hopes that other similar units may be encouraged to publish their figures for comparison.

TABLE 4. MALE WARD—CONTAINING 35 BEDS

Year	Admitted	Discharged Home	Discharged Resident Home	Died	Percentage Total Discharges	Percentage Deaths
1944	297	90	23	123	38	41.4
1945	292	86	25	139	38	47.6
1946	191	48	17	78	34	40

TABLE 5. FEMALE WARD—CONTAINING 45 COT BEDS

Year	Admitted	Discharged Home	Discharged Resident Home	Died	Percentage Total Discharges	Percentage Deaths
1944	155	35	15	65	32	43
1945	207	49	30	93	38	45
1946	222	41	27	91	30	41

These figures suggest that of all new admissions probably only about 25 per cent will form a residuum.

THE GERIATRIC UNIT TODAY

The department today carries almost 200 beds and accepts new patients admitted to the hospital (in the age group 60 years plus) and also patients referred to it from medical or surgical staff. These transfers are mainly patients from the medical side who are considered likely to need a very long stay in hospital or who are unlikely to improve much. Those transferred

(Continued)

from the surgical or orthopaedic departments fall into two categories: (a) those whose rehabilitation will require a very long period in hospital; (b) those whose primary condition, e.g., fracture, etc., is complicated by another disease.

The Unit does not accommodate *all* patients over 60 or even over 65, as a number of senile dements are admitted directly to the psychiatric wards attached to the mental observation wards and all surgical emergencies and some medical cases are admitted directly to the general medical and surgical wards.

Recently there has been a greater tendency for medical staffs from general wards to acknowledge that these so-called "chronic" elderly patients will probably do better in the atmosphere of the Geriatric Unit, and in the hands of those staffs who are most interested in such conditions. Such recognition of the value of this department is of course very encouraging.

There have been requests recently for the Unit to conduct Out-Patient sessions, which are being arranged for and will undoubtedly develop in the future as patients themselves learn of their existence. Already old patients are writing in and asking to be seen in the Geriatric Unit.

PHYSICAL MEDICINE

The great need for physiotherapy in these wards is fully recognised and this work is developing slowly but surely. Better results are being obtained as more staffs become available, and in addition to the services of the trained physiotherapists, some of the assistant nurses are doing excellent work under medical direction and supervision. Patients are, therefore, having combined treatment as individuals in the wards and also in small classes in the gymnasium. The gymnasium is a part of a vacated ward and has been very simply equipped for this sort of rehabilitation.

All the wards of the unit are now visited frequently and regularly by the occupational therapists who are beginning to take great interest in the results of their labours and are naturally greatly encouraged when they witness the increase in morale and the physical improvement of their patients. All the usual useful handicrafts are presented and a remarkably high standard of work is being done.

SOCIAL MEDICINE

All wards in the unit enjoy the services and invaluable help of the medico-social workers who work in very close co-operation with medical staff, often doing combined rounds with the medical officer and the ward sister.

FUTURE OF THE GERIATRIC UNIT

As the department gains experience, confidence and more skill, it should be able to prevent a great number of the conditions which are so prevalent and so crippling amongst elderly persons today. It should also be in a position to undertake still more treatment for both in-patients and out-patients

with a quicker turn-over, due to better teamwork and improved technique. It is essential that it should develop its teaching side both for medical and nursing students and post graduates, so that the future generations will be much better equipped by experienced personnel. The department will surely develop research as soon as its foundations are well-laid, for without this stimulus, progress must inevitably remain slow. Eventually, it should become the recognised department for advice concerning old age and conditions mainly dependent upon senescence. It should, in time, overcome the opposition from the more conservative sections of the medical and nursing professions, proving itself to be an invaluable and practical department in the hospital, enjoying the prestige and dignity which it has earned.

Medicine has responsibilities towards the elderly sick and infirm, equal to any other section of the community and must undertake these if it is to remain worthy of its high traditions.

3
Palliative Care of Older Patients

Jennifer Kapo, MD

Reproduced paper following commentary: Saunders C. Watch with me. Nurs Times 1965;61(48):1615–1617.

Commentary

In 1948, Cicely Saunders cared for a young man dying of a cancer in a hospital in Europe. The man was so touched by her attention to all aspects of his suffering—psychological, physical, and spiritual—that he left her a relatively small amount of money in his will to be a "window in your house" (1). At that time, Saunders was a nurse turned social worker. She had strong ideas about the best way to care for patients at the end of life, but no formal program to deliver this care and limited support from the doctors with whom she worked. To gain the respect of incredulous colleagues, she enrolled in medical school. With the training and experience of a self-contained intradisciplinary team, Dr. Saunders built the foundations of hospice care with the creation of St. Christopher's hospice in 1967.

Eighteen years before the Medicare Hospice Benefit was passed in the United States in 1983, Cicely Saunders articulated a vision of her life's work, the creation and nurturing of London's St. Christopher's Hospice in her article "Watch with Me" published in 1965 and reproduced here. In this classic paper, she outlines her goals for the hospice that have since evolved into the enduring principles of the modern hospice movement and the interconnected field of palliative medicine. These principles include respect for the patient and family, assessment of pain as well as other suffering (including psychological and spiritual), and the importance of research and education in forwarding the field.

In the United States, the hospice movement has grown substantially since the first hospice opened in Connecticut in 1974. According to data from 2005, there were approximately 4100 hospices in the United States that provided care for more than 1.2 million patients, or one-third of all patients who died that year (2). The growth in volume has been accompanied by a growth in the expertise of hospice workers to provide high quality end-of-life care. In "Watch with Me," Dame Saunders clearly describes the great need for research to improve this expertise. The field of hospice and palliative medicine research has grown substantially, cumulating in a National Institutes of Health State of the Science Report published in December 2004 that describes the many accomplishments of researchers in hospice and palliative medicine, but also highlights the need for further work (3). Of note, research suggests that hospice improves many outcomes at the end of life including improved pain assessment and treatment, improved communication with patients and families, and greater overall satisfaction with the dying process (4–7).

In "Watch with Me," Dame Saunders describes the need to train hospice care providers, as well as other healthcare providers who may encounter dying patients. Parallel to the growth of research programs in hospice and palliative care, education in hospice and palliative care has grown substantially. At present, the majority of U.S. academic medical centers offer required educational experiences in hospice and palliative medicine to all levels of trainees, including medical students and residents (8, 9). Some programs have palliative medicine fellowships to train specialists in hospice and palliative care. In the last year, the American Council of Graduate Medical Education (ACGME) formally recognized palliative medicine as a unique specialty.

This article has served as inspiration for the hundreds of hospice programs that were developed based on Dame Cicely Saunders' principles. These principles have guided hospices to provide comprehensive, compassionate care for dying persons. She died receiving this care, spending the last days of her life at St. Christopher's Hospice in London.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

References

1. Saunders C. The evolution of palliative care. J R Soc Med 2001;94:430–432.
2. National Hospice and Palliative Care Organization WD. National Hospice Trend Report. 2005.
3. Panel SotS. National Institutes of Health State of the Science Conference Statement on Improvement of End-of-Life Care. 2004.
4. Baer WM, Hanson LC. Families' perception of the added value of hospice in the nursing home [see comments]. J Am Geriatr Soc 2000;48(8):879–882.
5. Singer PA, Martin DK, Kelner M. Quality end-of-life care: patients' perspectives. JAMA 1999;281(2):163–168.
6. Teno JM, Clarridge BR, Casey V, Welch LC, Wetle T, Shield R. Mor V. Family perspectives on end-of-life care at the last place of care. JAMA 2004;291(1):88–93.
7. Casarett D, Hirschman K, Henry M. Does hospice have a role to play in end-of-life care in nursing homes? J Am Geriatr Soc 2001;49:1493–1498.
8. Billings JA, Block S. Palliative care in undergraduate medical education. Status report and future directions [see comments]. JAMA 1997;278(9):733–738.
9. Billings JA, Pantilat S. Survey of palliative care programs in United States teaching hospitals. J Palliat Med 2001;4: 309–314.

NURSING TIMES NOVEMBER 26 1965 1615

'Watch with Me'

Cicely Saunders, OBE, MA, MB, BS, SRN, AIMSW

Advent is the season before Christmas when, while awaiting the Feast of the Nativity, it is traditional to contemplate the Four Last Things

We can think about the foundations of St. Christopher's in various ways. We can say that they consist of all the interest and the money that has been given and promised and that have made the building and the laying of the foundation stone possible. We can think about them as all the work that has ever been done in this field in the past by people other than ourselves and on which we will build our own work. We can think about them as the people who have gradually joined in thinking, praying and working for St. Christopher's ever since the vision was first given more than 17 years ago. I think you all know that I like best of all to think of St. Christopher's as being founded on patients, those we have known and who are now safely through this part of their lives. One used to speak for all of them each time I told her of some meeting, as she said 'I'll be there'. Now I want to look at our foundations by taking one particular phrase which I believe expresses our ideals concerning St. Christopher's.

Ideals and Aims of St. Christopher's

I am sure the most important foundation stone we could have comes from the summing up of all the needs of the dying which was made for us in the Garden of Gethsemane in the simple words 'Watch with Me.' I think the one word 'Watch' says many things on many different levels, all of importance to us. In the first place it demands that all the work at St. Christopher's should stem from respect for the patient and very close attention to his distress. It means really looking at him, learning what this kind of pain is like, what these symptoms are like, and from this knowledge finding out how best to relieve them. It means continually gaining new skills, developing those already learnt from St. Luke's Hospital and the writings of its founder Dr. Howard Barrett, from all the work of St. Joseph's Hospice and from discussion with many other people both here and in the USA. I have not found any individual place concentrating on these problems alone but many have helped to shed light on different facets of them and all this we want to bring together and develop into new skills in an area that is very greatly neglected.

'Not only skill but compassion also'

We want to plan and carry out research in the relief of distress such as has not been done anywhere else, so far as I have been able to discover. It is often easier in a specialist setting to go on learning in this way and by building what we think is an ideal unit we hope to be able to help not only our own patients but to raise standards generally and also to stimulate others to think about these problems. A patient comes to my mind here, a young woman who said 'You seem to understand the pain from *both* sides.' Our aim in learning such understanding is to give the kind of relief described by another woman who said 'It was *all* pain but now it's gone and I am free.'

Seventeen years ago a young Pole died and left me £500 to be 'a window in your Home.' This was the very beginning of St. Christopher's. I also remember his saying 'I only want what is in your mind and in your heart.' This was echoed years later by another Pole who said to us 'Thank you. And not just for your pills but for your heart.' I think both of them showed that they wanted not only skill but compassion also. They needed warmth and friendship as well as good technical care. I think our understanding of what real watching means must include this. We have, indeed, to learn what this pain is like. Still more we have to learn what it feels like to be so ill, to be leaving life and its activity, to know that your faculties are failing, that you are parting from loves and responsibilities. We have to learn how to feel 'with' patients without feeling 'like' them if we are to give the kind of listening and steady support that they need to find their own way through.

Here again comes a key phrase I have often quoted—'I look for someone to look as if they are trying to understand me.' These patients are not looking for pity and indulgence but that we should look at them with respect and an expectation of courage, a heritage from seeing people like the woman who said to me 'You can tell them all that it was *all right*.' She was not going through a strange, dramatic or just unlucky experience, to be written up as such with sentimentality or sensationalism, but an all-too-common experience such as ordinary people have always faced and somehow managed to come through.

'I don't *want* to die'

We will be seeing patients who go along the path which leads from the honest but wistful plea—'I don't *want* to die, I don't *want* to die', to the quiet acceptance of 'I only want what is right.' We will not only see acceptance but also a very real joy, the true gaiety of someone who has gone through doubt, fear and unwillingness and come out the other side. I remember coming away from the bedside of a man who had come along that difficult path just about an hour before he died and saying to myself—'He looked *amused*'—and he really did. Certainly we are going to see hard things, but we are also going to see rewards and compensations and insight given to our patients here and now and we will see an extraordinary amount of real

Taken from a talk given to the annual general meeting of St. Christopher's Hospice.

(Continued)

happiness and even lightheartedness.

Planning an ideal unit is not enough to interpret all the meaning of the word 'watch' if teaching is not a vital part of what we do. We want St. Christopher's to be a place where all kinds of people can join us to learn from our experience and learn from our patients with us. This does not mean burdening the patients with the demands of continual bedside teaching. It does mean that you can give them an interest I know they enjoy if it is done in the right way. It can also reveal a new purpose in what is happening to them and what they are doing themselves. Certainly they are not all going to be saints. Some will be, indeed, and we will be very honoured and helped by their coming to us. Others will be splendidly maddening and I have no time to suggest the various crises with which we are going to have to cope. But who is to say who does the best—the person whose last weeks are the crown of a life of devotion, the young girl who makes the whole ward into a party for months on end and never shows you how much it costs her, or the old man who just manages to stop grumbling for his last 10 days or so? Certainly we will never fail to learn from them and some of the things that we will learn may surprise our future staff. Work here will not just be solemn. Rather I would just say it will be real and reality is gay and funny as well as serious. Above all, it will never be dull.

Being There

'Watch with me' means, still more than all our learning of skills, our attempts to understand mental suffering and loneliness and to pass on what we have learnt. It means also a great deal that cannot be understood. Those words did not mean 'understand what is happening' when they were first spoken. Still less did they mean 'explain' or 'take away'. However much we can ease distress, however much we can help the patients to find a new meaning in what is happening, there will always be the place where we will have to stop and know that we are really helpless. It would be very wrong indeed if, at that point, we tried to forget that this was so and to pass by. It would be wrong if we tried to cover it up, to deny it and to delude ourselves that we were always successful. Even when we feel that we can do absolutely nothing, we will still have to be prepared to stay.

'Watch with me' means, above all, just 'be there'. I remember the patient who said of the people who had really helped her, 'They never let you down. They just keep on coming.' I also remember she described the way God had met her by saying, 'He sends me people.' I am quite certain that St. Christopher's has to learn to be a place where people do not let you down but instead give the feeling of reassurance and safety that comes from faithfulness.

I think from this need especially stems the demand that we should grow into a real community. It is very important that we should be a group of people who have confidence in each other and that St. Christopher's should be the kind of family and home that can give the welcome and hospitality of a good home, where people are accepted as themselves and can relax in security. It must also be a place where everyone knows that the individual contributions matter and that there is no hierarchy of importance in what is done. Who will know what or who matters most to an individual patient as his manifold problems are dealt with by various members of such a group? There is a kind of compassionate matter-of-factness that develops in such a place and in this the hard-pressed worker is not overwhelmed by her own responsibilities.

St. Christopher's 'topping-out' ceremony takes place next week. See News of the Week, page 1604

The Community of all Men

Above all, I think it is here that we see the very great need for a religious foundation. We must remember that we belong to the much wider community of the whole Church, to the whole Communion of Saints and, indeed, to the whole community of all men. It is because of this that St. Christopher's is ecumenical and undenominational. We will welcome people of all sorts and kinds and be of all sorts and kinds ourselves. We are not emphasizing that there is just one way but rather that there is one Person coming in many ways.

The same words 'Watch with me' remind us also that we have not begun to see their meaning until we have some awareness of Christ's presence both in the patient and in the watcher. We will remember his oneness with all sufferers, for that is true for all time whether they recognize it here or not. As we watch them we know that he has been here, that he still is here and that his presence is redemptive.

Re-interpreting an old Truth

We do not help patients through this part of life by denying that it can sometimes be very hard. We do not see it truly if we just think somewhat vaguely of immortality and 'going on' rather than of death followed by resurrection. Dying followed by rebirth has been a dominant theme of man's religion from the very beginnings of belief. For

NURSING TIMES NOVEMBER 26 1965 1617

Christians this has once and for all been summed up and made truth in Christ himself. I believe that it is very important that this message should be shown at St. Christopher's in every possible way for it has hardly any meaning to a great majority of people in Great Britain today. This stands out with sad vividness in Geoffrey Gorer's survey, *Death, Grief and Mourning in Contemporary Britain.* It is a truth which needs to be re-interpreted in terms that are relevant to all those who will come to us, to the patients, to their relations and to all the visitors. Perhaps we may have a contribution to make to the 'new theology' as we learn about this very simply, seeing this truth, this Person, meeting people today.

Through Symbols and Sacraments

Christ will be present in all the skills that we learn and in symbols and sacraments of all kinds. These will include the sacraments of the cup of cold water and the washing of the disciples' feet. All these things will speak silently to the patients about God's love for them. So too will the whole planning and decoration of the building itself, thought out over a very long period with our architect and carried out by him with great insight and imagination. Especially, I think it will be shown in the planning of the chapel and in all the pictures, the symbols and the sculpture that are being created specially for us by artists who share this faith with us. It is very important that this message should be shown in these different ways. I have seen again and again how receptive patients are to the things they look at when they are not able to bear with talking any longer. Often it is important that very little should be said at all because it is so easy to interrupt a real message.

So much of our communication with people is done without words but I think this is especially so with the very ill. The patient who says soon after her admission 'It is marvellous to begin to feel safe again' has been met by the atmosphere and by the things she lies and looks at just as much as by the nursing and by the drugs and relief she is given. In a whole climate of safety she finds her own key and her own meeting. We will see patients able to listen, perhaps for the first time, to something that has been said to them all their lives but for which they have somehow never had time for real attention.

I have been impressed again and again at St. Joseph's by the way patients will lie and look at pictures or a crucifix and how much these can say to them. I believe that it is very important that these should be works created now, by artists who are interpreting these truths in the context of the world today. I am especially glad that this growing emphasis on art for St. Christopher's has given us connections with Poland once again, a link that has been there from the beginning and forged again and again.

'My bags are packed . . .'

I think all of us remember the words of Pope John when he said 'My bags are packed and I can leave with a tranquil heart at any moment.' I think that this is how we pray for all the patients who will come to us. We remember that some of them are already ill, frail, lonely or despairing and pray for them now. Others are busy and have no thought of calamity. Perhaps only in calamity are they going to find the meaning of the whole of the rest of their lives. I think that we should pray that we will be able to make it possible for them to pack their bags with the right things, pack them with what matters, with what *they* need; that while they are here they will find all that they should of reconciliation, fulfilment and meaning as they go through this last part of their lives.

. . . to be silent, to listen, to be there

I have tried to sum up the demands of this work we are planning in the words 'Watch with me.' Our most important foundation for St. Christopher's is the hope that in watching we should learn not only how to free patients from pain and distress, how to understand them and never let them down, but also how to be silent, how to listen and how just to be there. As we learn this we will also learn that the real work is not ours at all. We are building for so much more than ourselves. I think if we try to remember this we will see that the work is truly to the greater glory of God.

CARE OF THE DYING is a series of articles by Dr. Cicely Saunders, originally published in the *Nursing Times*, and reprinted as a booklet. Euthanasia, control of pain, mental distress, and Should a Patient Know? are among the subjects discussed.

Care of the Dying is obtainable from Macmillan (Journals) Ltd. Brunel Road, Basingstoke, Hants., 2s. 4d. by post.

4
Home-Based Care of the Older Patient

Mary Ann Forciea, MD, and Jean Yudin MSN, RNCS

Reproduced paper following commentary: Cherkasky M. Montefiore Hospital Home Care Program. Am J Pub Health 1949;39:163–166. Reprinted with permission from the American Public Health Association.

Commentary

Visiting an ill patient at home constituted the standard mode of practice from the earliest days of medical care. The practitioner brought his skills in diagnosis, treatment, and compassion with him and whatever his portable "tool kit" could contain. Even after the creation of inpatient hospitals, transfer to these facilities was rare, and often dreaded.

During the twentieth century, advances in diagnosis and treatment escalated. Many of these advances were linked to the use of new equipment, such as radiographs and microscopes. Improvements in hygiene made congregate settings safer. Treatments involving anesthesia became more commonplace. Patients began to come to the office or hospital. Patients could be seen more quickly, diagnosis became more accurate, and treatments more specific and effective. These advances in practice began to outweigh the appreciation of the patient in his social context that came from home visiting.

A small number of physicians maintained the tradition of home-based care, incorporating whatever advances were possible into that site of care. One of the leading programs in academic-based home-care programs was located at Montefiore Hospital in the Bronx, NY. In the article reproduced here, Martin Cherkasky described the goal of the home-care program: "to evaluate the patient as 'a whole in society' where a patient's environment may 'be more provocative in the origin of his disease than the germ isolated'." He described a program of integrated medical, nursing, social work, housekeeping, transportation, rehabilitation, and pharmacy services. Costs to maintain a patient in this program at that time were $3/day, which compared favorably with the $12 to $15/day of inpatient hospital costs. The benefits of individualized care at home contributed to high patient satisfaction.

Isadore Rossman and the Montefiore group published protocols of care for patients with cancer and cardiac diseases. Rossman described treatment modalities, such as transfusions, thoracentesis or paracentesis, oxygen, and physical therapy, that could be provided at home. Physicians visited patients weekly, and worked closely with visiting nurses, rehabilitation therapists, and pharmacists (1). In 1975, Bricker and coworkers described a lower Manhattan housecalls program targeted at older patients. Medical staff was based at a local hospital, with close link to social and community agencies. The authors estimated that approximately 70% of patients followed in their program would have required institutional care without the medical and social services provided in the program (2). An excellent review of modern housecalls programs was published by Loengard and Boal in 2004 (3). Sullivan et al. published home-based care curriculum guidelines for medical resident training in 1998 (4).

The last decade of the twentieth century and the first decade of the twenty-first have seen the resurgence of home-based care for older patients. Miniaturization of diagnostic technologies has allowed X-rays, ultrasounds, and EKGs to be easily performed at home. Home infusion therapies can be offered in most areas of the country. Community support services have become more available. Medical school curricula are again including information about home-based care. Improvements in Medicare reimbursement for house calls have enabled many primary care practitioners to return to visiting older patients at home. A national organization to support the practice needs of home-care practitioners is flourishing: the American Academy of House Call Practitioners. The Academy supports Web-based clinical and

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

professional materials. For frail, older, housebound patients and their caregivers, the medical support of a house call can allow the older patient to remain in her favorite site of care: her own home.

References

1. Rossman, Isadore Treatment of Cancer on a Home Care Program. JAMA 1954;156:827–830.
2. Bricker PW, Duque ST, Kaufman A, Sarg M, Jahre J, Maturlo S, Janeski JF. The Homebound Aged. Ann Int Med 1975; 82:1–6.
3. Loengard AU, Boal J. Home Care of the Frail Elderly. Clin Geriatr Med 2004;20:795–807.
4. Sullivan GM, Boling PA, Ritchie C, Levine S. Curriculum Recommendations for Resident Training in Home Care. J Am Geriatr Soc 1998;46:910–912.

Vol. 39

The Montefiore Hospital Home Care Program*

MARTIN CHERKASKY, M.D.

Home Care Executive, Montefiore Hospital, New York, N. Y.

ENUMERATING services which the Montefiore Hospital Home Care Program provides for its patients, and the gratifying results which we have achieved, would be telling only part of the story. The basis of the program, which carries a step further the philosophy which Dr. Bluestone. our Director at Montefiore Hospital, has been expounding for some three decades, may be of even greater significance. When I was in medical school, not so long ago, we were taught that it was important to think of a patient as a whole, and not just to examine a limb or an eye. We have now come to a point in the practice of medicine where we must broaden that point of view. When we think about a patient, we should think about him not only as an organic and spiritual whole, but also as a whole in society. It is no more fair or useful to separate a man from his environment than it is to divide him into separate and independent parts.

Our hospitals, despite their stress on scientific medicine which includes diagnostic machines, laboratory examinations, therapeutic procedures, and all of the other wonderful accomplishments which have raised the level of medical care, have held back from an understanding of sick human beings as social human beings. When a patient presents himself to a hospital with certain symptoms these days, he comes in as a stranger, his immediate illness is diagnosed, and he is relieved of his condition. Unfortunately, however, he too often returns to the same situation which may have given rise to his illness. To understand what caused this patient to become sick, it is necessary to know what sort of family he has, where he lives, what kind of clothes he wears, what food he eats, what kind of employment he has, and how he reacts to these factors. These and similar facts of life make up man as a social being, and may be more provocative in the origin of his disease than the germ which has been isolated from his sputum in the bacteriology laboratory. The hospital must seek a knowledge of these factors as eagerly as it seeks knowledge in the field of scientific medicine.

This introduction is, I believe, vital to an understanding of "Home Care." Our hospital, in extending its services into the home on an extramural basis, has begun to learn many things about the patient which can only be learned when he is in the bosom of his family. To the doctors on the program it has brought a new realization of the importance of social factors in disease. This is true of any disease. It is particularly true of long-term disease where the stress of illness brings about many changes in the relationship of the patient to his family, both emotionally and economically. Montefiore Hospital in New York has not only made its scientific machinery available to pa-

* Presented before a Joint Session of the Public Health Nursing and Medical Care Sections of the American Public Health Association at the Seventy-sixth Annual Meeting in Boston, Mass., November 10, 1948.

(Continued)

tients who live miles from its walls, it has also begun to learn new reasons why a patient becomes sick and why his illness is prolonged. This may indeed prove to be the most important contribution of the Home Care Program.

For the years 1947–1948, our Home Care Program has received $80,000 from the New York Cancer Committee, and $14,000 from the Greater New York Fund on an extra budgetary basis.

To illustrate the thinking which goes into the selection of a home care patient and what is done for him, it might be well to have you follow a typical case.

John J. was admitted to the hospital with some undetermined abdominal disease, and after thorough investigation by the clinicians and by the laboratories of the hospital, it was found that he had a cancer of the colon. He was subjected to major surgery, at which time it was found that the disease had progressed so far that the entire cancer could not be removed and he was left with a colostomy. Here is a patient who will ultimately die of his disease, but he may have six months, a year, two years or more to live. He requires nursing care, colostomy irrigations, watchful attention for complications, medication, and someone to help him with his food. He may be semi-ambulatory or bedridden. In any case, he is a sick man. But he may no longer require the special facilities of the hospital. Indeed, even if there were plenty of room for him in the hospital, he might do much better in some other environment.

The Department of Home Care was notified about this patient, and our doctor saw him for the purpose of determining whether we could provide him with a level of medical care at home which would be in conformity with our best hospital standards. In addition to being medically eligible, the patient was investigated and determined to be socially eligible. Every patient who is admitted to Montefiore Hospital has a social service "work up." When he is evaluated for Home Care, the social service worker reviews the patient's record, interviews him, interviews members of his family, and investigates the home. Since no patient is returned to his home unless this is in his best interests, it is obviously important that the family situation, the physical facilities of the home, and the patient's relationship with other members of his family should be such as to encourage the return to his home.

Many of us think of families and just naturally assume (at least when we are younger) that all parents love their children and that all children love their parents. Most of us live long enough to find out that this is not necessarily so. When return of a patient to his home is contemplated, it is important to know what bonds exist between him and his family—Are they still strong, after the disrupting effects of a long illness? These questions must be answered before it can be decided that it is best for a patient to return to his home. Some families seem not to want the patient back, but closer investigation reveals that the reason is not lack of love, but fear—fear of illness, fear of impending disaster, fear of inability to do what is required. If the fundamental attitudes are sound, all of these fears can be overcome by careful handling and good service. Some of the families who were doubtful proved to be among the best in our experience.

When it has been decided that a patient is medically and socially eligible, the patient goes on Home Care and receives the following services:

1. Medical service, around the clock, seven days a week. Specialists are available for the patient in his home, such as orthopedists, ophthalmologists, and surgeons. Many medical procedures such as abdominal taps and blood transfusions can readily be done in the home.

2. Social Service—The social worker who cared for the patient on the ward follows him into the home to help him and his family with any problems that may arise, and interprets the program to the family.

3. Nursing—The Visiting Nurse Service of New York, by contracts with us, visits each patient at least once, even in those cases where we do not foresee any need for Visiting Nurse Service, since their experience in the home will give us a good evaluation of the patient and of the patient's need for nursing. In addition, the nurses have two other important functions. They provide nursing and they teach. The teaching is, in some respects, the most important part of their job. They often teach a member of the family to become an expert nurse in the care of a particular patient.

4. Housekeeping service—We provide housekeeping service 5 to 10 hours per week. We find that this is very helpful since many of the patients who would otherwise have to remain in the hospital can well be taken care of at home if there is someone to help with the heavy housework. We have discovered that a woman is more than just a housekeeper in the home—that the mother, when she returns to the home, even though she no longer is able to do the dishes and wash the floors, can still be the rallying point for the entire family.

5. Transportation—Transportation to and from the hospital is provided, and there is a free interchange of patients between the hospital and the home. Dr. Bluestone pointed out [1] that Home Care is, in essence, an extension of the hospital into the home. There are none of the facilities of the hospital to which we cannot bring our patients by ambulance. The inconvenience to the patient is little greater than moving him from the 4th floor of the hospital to one of its laboratories.

6. Medication—We supply the patient with all medications, with hospital beds, wheelchairs, special mattresses, braces—anything that contributes to the welfare of the patient and which can be transported.

7. Occupational Therapy—We have a full-time occupational therapist who visits the patient in his home. This serves several purposes. First, it is a morale builder and certain corrective procedures can be taught to the patient. Second, for some patients it may in a small way alleviate the ever present financial difficulties.

8. Physical therapy — Our physical therapist also enters the home to treat the patient.

What are the results of our program? Let us consider the financial benefits first even though they may not be the most important. In the first twenty months of our program, we have provided something over 23,000 days of patient care. The average cost per patient day was less than $3 per day which compares quite favorably with the present cost of hospital care of $12 to $15 per day. It is, however, not of importance to have a product which is only cheaper. It must be as good or better. Home Care for patients who are suitable is not only "as good as" hospital care—it is infinitely better. If there were many empty hospital beds, a patient who is suitable for Home Care would still do much better in his home than he could possibly do in a hospital. In a hospital, a patient is one of many. He has to give up many of his own little private privileges and desires for the benefit of the group as a whole. In Home Care, we have provided the best of scientific medicine and the best in environment. He is an individual in his own bed with his own type of bedclothes, and he can have the window up or down as he sees fit. He can have his breakfast when he wants it and not when the dictates of hospital discipline compel. A patient on the ward in a hospital may be looked

(Continued)

at every day by a doctor, but he is not always "seen." When a doctor visits a patient in his home two, three, or four times a week, he is the sole recipient of the doctor's attention and care. A doctor on the ward may find greater interest in some patient three beds down the ward who is clinically more exciting or more interesting. Where the special facilities of the hospital are no longer needed, the rigidity and chilliness of a hospital can be profitably exchanged for the flexibility and warmth of the home.

Let me cite a case which illustrates the individualization of medical care and the well organized team which can be brought to bear on the patient in the home:

Jean J. had a growth involving her spine. An operation was performed and a large bony segment was removed. This happened about six years ago. During the intervening time Jean spent more than one year in a body cast and, because of the defect in her spine, was told she could never walk. She was seen in some of the best hospitals, but here was a patient permanently consigned to bed, a hard fate for a 29 year old girl to endure.

Eight months ago Jean came on the Home Care Program. She lived in a third story apartment with her widowed mother. The doctor seeing this young woman in her home, developed a much clearer insight into her hopes and desires than could a doctor on the ward where she was just one of a dozen patients bedridden for life. An orthopedist was called in, and after reviewing all the x-ray films, a special back brace was made for the patient. One day the visiting nurse met the doctor at Jean's home and helped her out of bed with under-the-arm crutches, and so began a long period with the doctor visiting three times a week, the visiting nurse three or four times a week, the physical therapist four times a week, massage, encouragement, new Swiss crutches, leg brace, and one day Jean got out of bed and walked to the bathroom for the first time in over five years. By using telephone books as an improvised stair, she was taught to walk up and down stairs. More than six months after coming on Home Care, Jean walked down two flights of stairs, got into a cab, came to our hospital and was presented to our clinical conference. Many of the doctors were surprised to see this "bedridden" patient come in under her own steam. Jean now is progressing toward walking without any supports.

We have salvaged a human being, and this by individualizing her care and by coördinating all the facilities of the hospital and community in their joint fight for health and against disease.

REFERENCE

1. See Home Care, An Extramural Hospital Function, *Survey Midmonthly*, Apr., 1948.

5
Education of Health Professionals

Kathy L. Egan, PhD

Reproduced paper following commentary: Libow Leslie L. A Fellowship in Geriatric Medicine. J Am Geriatr Soc 1972;20:580–584. Blackwell Publishing. Reproduced by permission of the publisher.

Palmore Erdman. Facts on Aging. Gerontologist. 1977;17:315–320. Copyright © The Gerontological Society of America. Reproduced by permission of the publisher.

Commentary

Interest in aging is probably as old as human self-reflection, but the body of published work on geriatrics education for physicians and other health care professionals in training is quite young. When geriatric medicine emerged as a distinct focus of medical study and practice in the twentieth century, it became necessary to define the body of knowledge and the education needed to prepare both clinicians and faculty for practice and research. In the mid-1970s, the American Geriatrics Society (AGS) led this effort through two national conferences that represented "the first major attempt at clarifying alternative models and strategies in relation to geriatric education" (1). The conference chose "not to recommend a new Board-certified specialty" but to suggest fellowship-level geriatrics-specific training in appropriate specialties, along with the infusion of geriatrics into undergraduate medical education, faculty development, and continuing medical education. These recommendations were supported by the Institute on Medicine and multiple specialty societies (2) and continue to shape geriatrics education.

The 1970s was thus a period of considerable development in geriatrics education. In 1972, Leslie Libow described the first full-time fellowship program in geriatric medicine in the United States (reproduced here). In 1977, Erdman Palmore's *Facts on Aging* Quiz (FAQ) gave educators in gerontology and geriatrics a much-needed tool for measuring and describing "the most common misconceptions about aging" among students (also reproduced here).

The fellowship in geriatric medicine that is the subject of Libow's 1972 report was developed at the Mount Sinai School of Medicine after some years of experience with medical student and resident education in geriatrics at that institution. Libow reported on a national AGS survey on training programs, which indicated that Mount Sinai had the first such formal program within an internal medicine department and residency. The curriculum set what would remain a high standard even today, including nine separate educational activities that spanned the inpatient, home, and community health program settings. The curriculum also involved interdisciplinary conferences and collaboration with geriatric physiatry and psychiatry. Fellows were also integrated into the daily conferences of the department of medicine. The inpatient setting for clinical training was an 80-bed "Geriatric Unit" that admitted patients from the other inpatient services or home care who were otherwise thought to be at risk for long-term care placement. The fellows were expected to follow patients through home visits, collaboration with visiting nurses, and office visits. Although the range of care settings seems familiar today, the average eight-week length of stay for the majority of patients on that unit would not. Libow's question about the financial support of fellowships is likely to resonate with today's readers: "How can the government put so much money into geriatric care, via Medicare and Medicaid programs and not put appropriate money into the development of specially trained physicians who could develop and lead the care and research programs in geriatrics?"

Although people have always desired to live long lives, few of us have been eager to care for those who have achieved length of years. In an 1848 book on aging, George Edward Day complained that "other physicians had little interest in caring for the ills of the aged" (3). Geriatrics education has struggled from the outset with the issues of negative attitudes and stereotypes related to aging and the elderly. How could geriatrics education overcome these subjective issues to attract learners to the field? How would educators know if they were being

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

successful in this endeavor? In 1977, Erdman Palmore provided a tool, called the "Facts on Aging, A Short Quiz." The Palmore instrument aimed to overcome the limitations of previous tests on aging by making the FAQ short (25 true-false items), by documenting the facts thoroughly, and by using only factual statements, some of which could serve as indirect indicators of bias. In his own presentation of the FAQ, however, Palmore seems to emphasize its utility in the classroom to stimulate discussion of misconceptions about aging and explore the various facts and their implications. The most frequent misconceptions among the undergraduates Palmore sampled in an early iteration would not be unusual today: that a large proportion of the aged are living in institutions, that a majority of the aged are frequently bored, and that more than 15% of the population are age 65 or over, though this last item will be true in the foreseeable future. The Facts on Aging Quiz proved so useful that by 1981 it spawned more than 40 studies across many disciplines. Palmore published a supplementary second version a few years later (4). It is particularly impressive that the FAQ has been and is still being used widely and researched as reported, for example in a paper as recently as 2002 on its application among nurses (5).

In the 1970s, the Veterans Administration established Geriatric Research Education and Clinical Centers for the "advancement and integration of research, education, and clinical achievements in geriatrics and gerontology into the total VA healthcare system (http://www1.va.gov/grecc/). In the 1980s, the federal government began funding of Geriatric Education Centers (GECs) to provide education in geriatrics to clinicians, students, and faculty across the healthcare professions. The national network of GECs grew to almost 50 by 2006 when funding was abruptly cut off and restored a year later. Despite periodic threats to funding, the 1990s and early years of the twenty-first century have seen substantial support for innovative geriatrics education programs and educational measurement from private foundations, such as the John A. Hartford Foundation, Inc., and the D.W. Reynolds Foundation. The Hartford Foundation has been notable for its support of interdisciplinary education. Fulmer and coworkers (6) reported on a comprehensive evaluation of a Hartford-funded, large-scale, interdisciplinary team-training program at eight sites nationally. This study showed that team training was successful in achieving desired attitudinal changes toward team care among medicine, nursing, and social work trainees, although the attitudes of medical trainees toward team care remained the least favorable throughout. The Hartford Foundation has also joined with the American Geriatrics Society to develop education programs across the medical and surgical specialties as reported by Potter and coworkers (7). Between 2001 and 2006, the D.W. Reynolds Foundation funded grants to 30 medical schools with a primary purpose of infusing geriatrics into the curriculum and a mandate to disseminate innovative curriculum models and educational materials (http://www.dwreynolds.org/Programs/National/Aging?AboutAging.htm). Perhaps the papers being written on those programs will be as significant as those by Libow and Palmore and find their ways into future collections of classic papers.

Thirty years later, the papers by Libow and Palmore anticipate current concerns in geriatrics education, such as the continued need to struggle for public support of health professions education, challenges in developing curricula that incorporate the full range of geriatric-specific clinical settings, such as nursing homes and home care, and the need for user-friendly but reliable and valid educational measures.

References

1. Reichel W. Proceedings of the American Geriatrics Society Conferences on Geriatric Education. J Am Geriatr Soc 1977;25:481–513.
2. Reichel W. Geriatric Medical Education: Development Since the American Geriatrics Society Conferences on Geriatric Education, 1976–77. J Am Geriatr Soc 1981;29:1–9.
3. Morley J. A Brief History of Geriatrics. J Gerontol Med Sci 2004;59A:1132–1152.
4. Palmore E. The Facts on Aging Quiz: Part Two. Gerontologist 1981;21:431–437.
5. Suefert RL, Carrozza MA. A Test of Palmore's Facts on Aging Quizzes as Alternate Measures. J Aging Studies 2002;16:279–294.
6. Fulmer T. Geriatric Interdisciplinary Team Training Program. J Aging Health 2005;17:443–470.
7. Potter JF et al. Geriatrics for Residents in the Surgical and Medical Specialties: Implementation of Curricula and Training Experiences. J Am Geriatr Soc 2005;53:511–515.

Web sites:

http://cas.umkc.edu/cas/AgingFactsQuiz.htm

JOURNAL OF THE AMERICAN GERIATRICS SOCIETY
Vol. XX, No. 12

A Fellowship in Geriatric Medicine*

LESLIE S. LIBOW, M.D.**

Elmhurst, New York

ABSTRACT: After several years of experience in training interns and residents in Geriatrics during their two- or three-month rotational period on this service, a full-time Fellowship program has now been established in Geriatric Medicine, with the cooperation of the Department of Medicine. It is expected that the Fellows will achieve: 1) a special knowledge of and adeptness with the illnesses and health needs of the elderly, and 2) special abilities to plan for community needs regarding its elderly. The base for the program is the 80-bed medical and surgical convalescent unit of this 1000-bed general teaching hospital. Two-thirds of the patients admitted return to the community within ten weeks. The Fellowship program includes participation in: 1) daily bedside rounds, 2) weekly staff conferences emphasizing an interdisciplinary approach to patients' problems, 3) weekly journal-club sessions on "current concepts" in geriatrics, 4) community-based health programs, 5) daily teaching conferences of the Department of Medicine, and 6) a research project related to the medication problems of the elderly. The first Fellow has been accepted and a two-year to three-year program is planned which will be divided between Internal Medicine and Geriatric Medicine.

Where in the United States can a physician obtain special graduate training in Geriatric Medicine? How many programs exist and to what extent does the federal government support such training? Before answering these questions, perhaps a clearer definition of Geriatric Medicine is required. Geriatric Medicine is a specialized field encompassing *gerontology* (the normal processes of aging) and *gero-medicine* (the pathology of the elderly in a social, psychiatric and medical sense). Perhaps the term Gerontologic Medicine makes the definition clearer. To obtain a solid base of such special knowledge, as well as a "gestalt" of the field, should take one to two years of fellowship training. This definition will be explored in the description of our Fellowship program.

In referring to the matter of graduate training, Dr. Irving Wright, in his Presidential address at the annual meeting of the American Geriatrics Society in April 1971, stated, "The experience of a senior medical student is pertinent; this year he has been seeking an internship and residency program which would provide him with some special training in the field of geriatrics but, so far, has been deeply frustrated by the lack of interest manifested in a number of major medical centers" (1).

Our geriatric unit communicated with this young physician, and after appropriate evaluations, he and another equally well-motivated and highly recommended intern were selected as our first two "trainee-fellows" in Geriatric Medicine. These two physicians, as well as most of the other men and women who have applied to our program, appear to see their geriatric training as coupled to, and part of the traditional training in internal medicine.

Subsequent to these events, the Research and Education Committee of the American Geriatrics Society conducted a survey involving 27 well known geriatricians at 24 established medical centers in the United States and Canada, to obtain more information about the availability of fellowship-residency programs in Geriatric Medicine (2).

* Presented at the 29th Annual Meeting of the American Geriatrics Society, New York, N. Y., April 5-7, 1972.

** Chief, Geriatric Medicine, Department of Medicine, Mount Sinai City Hospital Center at Elmhurst, New York; Assistant Clinical Professor of Medicine, Mount Sinai School of Medicine, City University of New York.

Address: Leslie Libow, M.D., Elmhurst City Hospital, 79-01 Broadway, Elmhurst, N. Y. 11373.

(Continued)

Twenty-four of the 27 geriatricians replied, and 16 of these 24 reported that no clinical training program was available. Eight programs do exist. Some of these programs are located in voluntary, long-term facilities, with well organized teaching programs; however, the medical residency training programs in these facilities are not "approved." One highly organized program in Baltimore utilizes a system of rotations through various levels of convalescent and long-term care, and appears to emphasize chronic disease as much or more than geriatrics. Another program in Baltimore, centered within a department of Family Practice rather than Internal Medicine, provides twelve to eighteen months of geriatric training as part of a four-year total program in Family Practice. Two Canadian programs are centered in geriatric units in general hospitals and offer approved training in Geriatric Medicine. Many of the 16 centers without any programs appear to have the interest and potential to develop them. It is probable that there are other programs in the United States which this survey did not uncover.

THE HISTORY OF FEDERAL FUNDING

Before describing our program, it is pertinent to review the history of federal funding for training in Geriatric Medicine within the United States. This is not a difficult task, since there is no funding, to the best of my knowledge.

The history of this lack of support appears fairly clear. The Administration on Aging (AoA) was created by the Older Americans Act of 1965 to focus on the "total needs of older people." One of their earliest efforts was to coordinate the training needs of the entire field. Assuming that medical training would be supported from other agencies, AoA has not supported training for the health and rehabilitation professions, except under unusual circumstances (3). AoA does support the training of administrators of long-term health facilities, planners, non-medical specialists in aging and teachers of gerontology at universities. Since funds for training social workers and psychiatrists do not come from AoA, they have come from the National Institute of Mental Health (4). Similarly, since funds for basic research do not come from AoA, they have come from the National Institute of Child Health and Human Development (5).

But where do the funds come from to support the training of physicians in Geriatric Medicine? To my knowledge, no federal agency supports such training at this point. In part, it was probably assumed, erroneously, that money would be made available from hospital-generated funds. Another probable assumption was that there would be neither a need nor a demand from young physicians or from society for such training. Such a view is now an anachronism. There is an obvious need, as well as a demand.

Other important but non-traditional areas of medicine have also struggled for federal "start-up" support. The field of rehabilitation medicine was fortunate in getting most of its fellowship money, not from classical government medical sources but from the federal Division of Vocational Rehabilitation. More recently, departments of community medicine are developing. American medicine is changing in structure, and many fellowships are being supported federally from varied directions within the Department of Health, Education, and Welfare.

How can the government put so much money into geriatric care, via Medicare and Medicaid programs and not put appropriate money into the development of specially trained physicians who could develop and lead the care and research programs in geriatrics? A lesson might be taken from private industry, where a good percentage of funds is invested in what is termed "development," based on the belief and the evidence that such investment leads to profitable advances.

FELLOWSHIP PROGRAM IN GERIATRIC MEDICINE

Our Fellowship program, in many respects, is an innovation in medical training.

For several years we have gained experience in geriatric training with our medical interns and residents during their two-month to three-month rotation on our geriatric unit. Recently, we have discerned a need (and possibly a demand) for a full-time trainee-Fellowship program in Geriatric Medicine within the department of Internal Medicine. With the cooperation of the chairman of the Department of Medicine, we have established a program which extends over one to three years. To start this program, faculty funds were redirected to Fellowship funds.

The goals are for the trainees to achieve: 1) a clinical competence in general internal medicine, with a special knowledge of the illnesses and health needs of the elderly; and 2) a community-health planning competence as applied to the needs of its elderly citizens. The clinical goal aims at developing classical diagnostic and therapeutic abilities in internal medicine, combined with a special skill in handling the key medical and social-psychiatric problems of the elderly. This includes emphasis on geriatric physiatry and geriatric psychiatry. The community-health goal includes familiarity with the health situations in the patient's home, the general hospital, the true extended care facility, the nursing home, the chronic disease hospital, and the state psychiatric hospital. In particular, our major effort is to emphasize the means whereby the individual may be maintained in the community. This necessitates the Fellow-trainee working closely with community resources such as the home care service, the visiting nurse service, nursing homes, administrative and health personnel of housing developments and local government and voluntary agencies.

The Fellows will participate in all of the following activities:

1. Regular bedside rounds on our Geriatric Unit, with the geriatricians-in-charge, interns, nurses, and the geriatrics coordinator. When necessary, the various specialty consultants participate in the clinical discussions.
2. A weekly meeting of the geriatricians and the social workers.
3. A weekly staff conference of 20 to 30 personnel from the following disciplines, in addition to Geriatric Medicine: nursing, physical medicine, social service, recreational therapy, visiting nurse service, administration, unit coordinator, house physicians, nurse supervisors from other parts of the hospital, and home care physicians. Five to 10 patients are discussed at these meetings, with new patients and family or friends participating. The meeting has varying goals. For the patients, it is a message of broad professional concern for their situation. It provides the patients with an unusual forum to express their feelings. For the staff, it is the occasion to develop a multidisciplinary therapeutic plan. It is also an excellent opportunity for the geriatricians to teach geriatrics from the real case situation, with all of its medical and psycho-social challenges.
4. Evaluation of patients on the acute disease services of this hospital and the home care service, as well as other community-residing persons, for their possible admission to the geriatric unit.
5. Planning and participating in community health programs for the elderly.
6. Seeing and following patients after their discharge from our unit. We use three methods for community support and health care of these patients: a) the Fellow will be making home visits to many of our discharged patients who cannot come in for follow-up care; other discharged patients will be followed by physicians of the home-care division of this hospital; b) the Fellow will also work closely with the visiting-nurse services, an integral part of geriatric care; it is the visiting nurse who makes it possible for most of the marginally compensated elderly to remain at home; and c) those of our discharged patients who are ambulatory will be seen in our geriatric follow-up clinic, located on our unit.
7. Special programs to emphasize training in geriatric physiatry and geriatric psychiatry.
8. A "current concepts" journal club, emphasizing both gerontology and geriatrics.
9. Daily conferences of the department of medicine, i.e., grand rounds, medical x-ray evaluations, electrocardiography, and subspecialties.

Depending upon the Fellow's prior training and goals, there will also be available one to two years of residency training in internal medicine on the general wards and subspecialty services of this hospital.

THE GERIATRIC UNIT

Our 80-bed "Geriatric Unit" is part of our 1000-bed municipal, general teaching hospital, The Mount Sinai City Hospital Center at Elmhurst, New York, a major teaching division of The Mount Sinai School of Medicine of the City University of New York. The earlier experience at this unit has been described previously (6). Most of our admissions are selected from the general services of the hospital and the home-care service. They are elderly patients whom we feel might never return home, or if at home, might not be capable of remaining at home unless given an

(Continued)

exposure to the treatment and support system of the Geriatric Unit. Two-thirds of all patients admitted are discharged to the community in an average of eight weeks. In a recent eight-week period in January and February of 1972, we admitted 66 patients, and simultaneously sent 40 home with support systems to be described. Eight additional patients went to long-term facilities.

An exception to these criteria is the admission of a limited number of alert and well oriented patients with advanced malignant disease, not necessarily terminal. Five to 8 beds are used for this purpose. The unit has developed some special abilities and the courage to deal directly with the needs of such patients and families. Our involvement has stemmed directly from the work of Dr. Elizabeth Kübler-Ross (7).

A Geriatric Unit is a concept that is somewhat new, though not unique, to United States hospitals. It is not a long-term facility, such as a nursing home or a chronic disease hospital, nor is it an infirmary or residence. It is an active therapeutic environment comprised of an interdisciplinary team, serving an entire hospital and a community by focusing on the marginally compensated elderly. The aim is to produce a coordination of the multiple services, in-hospital and community, needed by and available to the elderly.

Most older people admitted to the general medical wards of this hospital are evaluated by us for possible admission to our unit. Many of the older people admitted to our surgical services are also evaluated. Those who seem particularly marginal in their chances of getting home, because of physical, mental and/or social frailty, are given first choice for admission to our unit. Those not found suitable for, or not in need of, our unit are recommended for: 1) direct return home; 2) permanent care in a community nursing home; 3) care in a chronic disease hospital; or 4) care in a psychiatric hospital. Less marginal patients, e.g., those recovering from myocardial infarction, who are in need of one to two more weeks of convalescent care and not necessarily frail, are also admitted. Our unit also has 4 long-term beds.

ILLUSTRATIVE CASES

To make clearer what is done, 3 case reports are presented.

Case 1

R. P., is a 79-year-old widow, living alone in a low-income housing development. The hundreds of older people in this development have direct access to our unit, at the request of the health clinic located in the development. She was admitted to the general medical service because of shortness of breath due to congestive heart failure and possible pulmonary emboli. She also had arteriosclerotic heart disease and diabetes mellitus. She was selected for transfer to the Geriatric Unit, where anticoagulation therapy was continued. Emphasis was placed on re-ambulation and self-administration of medications, since a contributing factor in her heart failure seemed to be medication errors with digoxin and warfarin sodium (Coumadin). Each night she was mildly dyspneic, and anxiety seemed to be a factor. However, a diagnostic thoracentesis revealed a small hemorrhagic effusion; thus the presence of pulmonary emboli was more clearly established. She was very inaccurate with medications, and the nurses had to take over their administration. However, because she was not a candidate for vena cava ligation, and because she wanted to return home rather than go to a nursing home, we again tried to teach her to self-administer her medications accurately. She finally learned the system and has returned home under the supervision of the same visiting nurse, who is an active participant in our unit and who participated in the unit's decision about once again returning this patient to the community. She is doing well, with much support, i.e., frequent visits from the visiting nurse and the home-care physician. As changes in her health status occur, her case is discussed at our weekly staff conference.

Case 2

M. C., a 66-year-old married woman, attempted suicide by jumping from the third-floor roof of her building. She fractured her left humerus, and a bloody pleural effusion with pulmonary contusion also developed. After nineteen days on the orthopedic service, where psychiatric care was simultaneously given, we selected her for transfer to our unit. She was encouraged to walk again and to self-administer medications (the suicide effort notwithstanding). She and her husband were

dealt with in a open and direct way as regards her desperate effort, in the face of her son's alcoholism and dissolving marriage. She improved considerably and has returned to the community, being supported by our geriatric follow-up clinic and hospital psychiatric clinic.

Case 3

O. P., a 52-year-old mother of two teen-aged daughters, has had multiple sclerosis for nineteen years, and carcinoma of the breast with metastases for two years. Following admission to the general medical ward for the treatment of bone pain, she was evaluated by our unit for admission to one of the special beds mentioned previously. Her pain necessitated hormonal therapy and occasionally narcotics. She insisted upon returning home, though her family was only minimally able to render care. Interestingly, the social service department of this hospital and the visiting-nurse service of our community were in sharp conflict over the patient's ability to be maintained at home. At our staff meeting, the husband and two daughters openly talked of their ambivalent feelings about having the patient home again. The visiting nurse agreed to further care, after the family promised to participate to a greater extent. The patient returned home, aided by the presence of a homemaker from the visiting-nurse service. The pain increased, and this was reported at our staff meeting by the visiting nurse. Her private physician was told about the situation and was advised by the hospital chemotherapy specialist as to the proper manipulation of hormones. Fortunately, the pain diminished and the patient remained at home for two months. She has now been re-admitted and is receiving radiotherapy to the involved bony areas. Her condition is becoming worse. However, she is as alert as ever, still has the entire team of the geriatric unit involved in her care, and may soon return home again, with the multiple support system.[1]

Many of our patients remain at home permanently, but some are re-admitted frequently. Nevertheless, these patients are living in the community most of the time.

[1] During this hospitalization, diffuse bone metastases developed rapidly, and the patient died on April 23, 1972.

CANDIDATES AND FINANCES

As regards the interest in and "demand for" such Fellowship training, it is noteworthy that within three weeks of our advertisement of this position in the standard medical journals, we heard from 13 interested young physicians at different levels of training. Clearly, there are many candidates for such positions. The interest of young physicians in working with the elderly is certainly changing for the better.

A final note of optimisim may suggest something of the future of this field. After we had selected our first Fellowship candidate, the City of New York informed the hospital that there was a financial crisis in the hospital system. Ordinarily, we do not worry about such events, since this is normal for New York (perhaps for most big cities). However, as funds were cut back, our Fellowship funds suddenly disappeared for next year. In the midst of this potential disaster, a business executive and the son of a patient on our unit, hearing about the problem, generously donated the funds necessary to launch this program. Our hospital will support the program in the following year. I do not think that Fellowship training in Geriatric Medicine should depend upon individual philanthropy. Rather, such support should be federal or at least, hospital-generated. Nevertheless, private funds could be another source to nurture the growth of the embryonic field of Geriatric Medicine.

REFERENCES

1. Wright, I. S.: Geriatrics — The challenges of the seventies: rethinking and retooling for the future, J. Am. Geriatrics Soc. 19: 737 (Sept.) 1971.
2. Laird, R. C., Chairman, Research and Education Committee, Am. Geriatrics Soc., 1972: Personal communication.
3. Tibbitts, C.: Administration on Aging's Title V Training Grant Program, Gerontologist 10 (1): 55 (Spring) 1970.
4. Anderson, T. E., and Blank, M. L.: National Institute of Mental Health Training Programs in Aging, Gerontologist 10 (2): 153 (Summer) 1970.
5. Duncan, L. E.: National Institute of Child Health and Human Development Training Grant Programs, Gerontologist 10 (1): 62 (Spring) 1970.
6. Libow, L. S.; Viola, R. M., and Stein, M. F., Jr.: The extended care facility at Mount Sinai City Hospital Center, Elmhurst, New York: three-year experience, J. Am. Geriatrics Soc. 16: 1164 (Oct.) 1968.
7. Kübler-Ross, E.: On Death and Dying. New York, The Macmillan Co., 1969.

A short, factual, and documented quiz is developed and tested which covers the basic facts and frequent misconceptions about aging. Its uses include stimulating discussion, measuring levels of information and anti-aged bias, identifying the most frequent misconceptions, measuring the effects of courses, and measuring changes in public information or bias.

Facts on Aging
A Short Quiz[1]

Erdman Palmore, PhD[2]

Several tests or scales on aging have been developed and a few have been published (Golde & Kogan, 1959; Kogan, 1961; Tuckman & Lorge, 1952). However, these tests all share one or more of the following disadvantages:

(1) They tend to have 40 or 50 items and require several pages of print.

(2) They confuse factual statements (which may or may not be true) with attitudinal statements which are arbitrarily scored as being "favorable" or "unfavorable." For example, Kogan's statement "Most old people would prefer to continue working just as long as they can, rather than be dependent on anybody" is probably false, depending on what is meant by "most," "working," and "dependent." Yet, a "disagree" response is scored as showing an unfavorable attitude toward the aged. Unfortunately, some "negative stereotypes" about the aged are generally true and some of the "positive" statements are generally false.

(3) The factual statements are undocumented and we have nothing but the author's assertion that they are true or false.

The following quiz is designed to avoid these disadvantages. It is short (25 items requiring only one page and less than 5 minutes to complete) and confined to factual statements which can be documented by empirical research. It is designed to cover the basic physical, mental, and social facts and the most common misconceptions about aging. Before proceeding further, you are encouraged to try out the quiz to find out which facts you may be unaware of. Circle "T" for True, or "F" for False.

T F 1. The majority of old people (past age 65) are senile (i.e. defective memory, disoriented, or demented).
T F 2. All five senses tend to decline in old age.
T F 3. Most old people have no interest in, or capacity for, sexual relations.
T F 4. Lung capacity tends to decline in old age.
T F 5. The majority of old people feel miserable most of the time.
T F 6. Physical strength tends to decline in old age.
T F 7 At least one-tenth of the aged are living in long-stay institutions (i.e. nursing homes, mental hospitals, homes for the aged, etc.).
T F 8. Aged drivers have fewer accidents per person than drivers under age 65.
T F 9. Most older workers cannot work as effectively as younger workers.
T F 10. About 80% of the aged are healthy enough to carry out their normal activities.
T F 11. Most old people are set in their ways and unable to change.
T F 12. Old people usually take longer to learn something new.
T F 13. It is almost impossible for most old people to learn new things.
T F 14. The reaction time of most old people tends to be slower than reaction time of younger people.
T F 15. In general, most old people are pretty much alike.
T F 16. The majority of old people are seldom bored.
T F 17. The majority of old people are socially isolated and lonely.
T F 18. Older workers have fewer accidents than younger workers.

[1]Research for this article was supported in part by Grant AC-00364, NIA, USPHS. The author wishes to thank all the students and faculty who participated in the development and testing of this quiz. Jane Crosby did the tabulations for the tables.

[2]Professor of Medical Sociology and Senior Fellow at the Center for the Study of Aging and Human Development, Box 3003, Duke Univ. Medical Center, Durham 27710.

T F 19. Over 15% of the U.S. population are now age 65 or over.
T F 20. Most medical practioners tend to give low priority to the aged.
T F 21. The majority of older people have incomes below the poverty level (as defined by the Federal Government).
T F 22. The majority of old people are working or would like to have some kind of work to do (including housework and volunteer work).
T F 23. Older people tend to become more religious as they age.
T F 24. The majority of old people are seldom irritated or angry.
T F 25. The health and socioeconomic status of older people (compared to younger people) in the year 2000 will probably be about the same as now.

The key to the correct answer is simple: all the odd numbered items are false and all the even numbered are true. So far, no one taking the quiz has guessed this pattern of correct answers.

Documentation

(1) The majority of old people are not senile (i.e., defective memory, disoriented, or demented). Only about 2 or 3% of persons age 65 or over are institutionalized as a result of psychiatric illness (Busse & Pfeiffer, 1977). A series of eight community surveys found the prevalence of psychosis (of all types) to range from 4 to 8% (Riley & Foner, 1968). Thus, all the evidence indicates that there are less than 10% of the aged who are disoriented or demented. It is more difficult to get accurate estimates of the proportion with defective memories, partly because of the different types of memory defects and different methods of measuring it. However, most studies agree that there is little or no decline with age in short-term memory storage capacity (using the digit span test). Four studies did find large age differences in free recall of words, but two of them found no age differences in recognition of words in a list (Woodruff & Birren, 1975). As for long-term memory, various community surveys have found less than 20% of the aged who cannot remember such things as the past President of the United States, their correct age, birth date, telephone number, mother's maiden name, address, or the alphabet (Botwinick, 1976; Pfeiffer, 1975). Thus, it is clear that the majority of aged do not have such serious memory defects.

(2) All five senses do tend to decline in old age. Most studies agree that various aspects of vision, hearing, and touch tend to decline in old age. Some studies of taste and smell have not found a significant decline, but the best evidence indicates increases in taste and smell threshholds with age (Riley & Foner, 1968). Studies of structural atrophy in the tongue and nose with old age support the experimental evidence of decline in taste and smell (Birren, 1959).

(3) The majority of persons past age 65 continue to have both interest in, and capacity for, sexual relations. Masters and Johnson (1966) found that the capacity for satisfying sexual relations continues into the decades of the 70s and 80s for healthy couples. The Duke Longitudinal Studies found that sex continues to play an important role in the lives of most men and the majority of women through the seventh decade of life (Palmore, 1974).

(4) Lung capacity does tend to decline in old age. Both vital lung capacity (the volume of air that can be forcibly expelled in one breath) and maximum breathing capacity (the volume of air that can be moved in and out of the lungs in 15 seconds) declines on the average from age 30 onward (Shock, 1962).

(5) The majority of old people do not feel miserable most of the time. Studies of happiness, morale, and life satisfaction either find no significant difference by age groups or find about one-fifth to one-third of the aged score "low" on various happiness or morale scales (Riley & Foner, 1968). A recent national survey found that less than a fourth of persons 65 or over reported that "This is the dreariest time of my life"; while a majority said "I am just as happy as when I was younger" (Harris, 1975).

(6) Physical strength does tend to decline in old age. Studies of various kinds of muscular strength show declines in old age compared to young adulthood of 15 to 46% (Birren, 1959).

(7) Only 4.8% of persons 65 or over were residents of any long-stay institutions in 1970 (U.S. Census, 1970). Even among those age 75 or over only 9.2% were residents in institutions.

(8) Drivers over age 65 do have fewer accidents per person than drivers under age 65. Older drivers have about the same accident rate per person as middle-aged drivers, but a much lower rate than drivers under age 30

(Continued)

(National Safety Council, 1976). Older drivers tend to drive less miles per year and apparently tend to compensate for any declines in perception and reaction speed by driving more carefully.

(9) The majority of older workers can work as effectively as younger workers. Despite declines in perception and reaction speed under laboratory conditions among the general aged population, studies of older workers (the 12% who are able to continue employment) under actual working conditions generally show that they perform as well as young workers, if not better than younger workers, on most measures. When speed of reaction is important, older workers sometimes produce at lower rates, but they are at least as accurate and steady in their work as younger workers. Consistency of output tends to increase by age, as older workers perform at steadier rates from week to week than younger workers do. In addition, older workers have less job turnover, less accidents, and less absenteeism than younger workers (Riley & Foner, 1968).

(10) About 80% of the aged are healthy enough to engage in their normal activities. About 5% of those over age 65 are institutionalized and another 15% among the noninstitutionalized say they are unable to engage in their major activity (such as work or housework) because of chronic conditions. This leaves 80% who are able to engage in their major activity (National Center for Health Statistics, 1974).

(11) The majority of old people are not "set in their ways and unable to change." There is some evidence that older people tend to become more stable in their attitudes, but it is clear that most older people do change and adapt to the many major events that occur in old age such as retirement, children leaving home, widowhood, moving to new homes, and serious illness. Their political and social attitudes also tend to shift with those of the rest of society, although at a somewhat slower rate than for younger people (Cutler & Kaufman, 1975; Glenn & Hefner, 1972).

(12) Old people usually take longer to learn something new. Experiments have consistently shown that older people take longer than younger people to learn new material (Botwinick, 1967). Studies of on-the-job trainees also show that older workers tend to take somewhat longer to learn new jobs (Riley & Foner, 1968).

(13) But, it is not impossible for most old people to learn new things. The same studies (cited in #12) also show that most older persons can eventually learn new things about as well as younger persons, if given enough time and repetitions of the material to be learned.

(14) The reaction time of most old people tends to be slower than that of younger people. This is one of the best documented facts about the aged on record. It appears to be true regardless of the kind of reaction that is measured (Botwinick, 1967).

(15) Most old people are not pretty much alike. There appears to be at least as much difference between older people as there is at any age level; there are the rich and poor, happy and sad, healthy and sick, high and low intelligence, etc. In fact, some evidence indicates that as people age they tend to become less alike and more heterogeneous on many dimensions (Maddox & Douglas, 1974).

(16) The majority of old people are seldom bored. Only 17% of persons 65 or over say "not enough to do to keep busy" is a "somewhat serious" or "very serious" problem (Harris, 1975). Another survey found that two-thirds of the aged said they were never or hardly ever bored (Dean, 1962). The Duke Adaptation Study found that 87% of those 65 or over said they were never bored in the past week.

(17) The majority of old people are not socially isolated and lonely. About two-thirds of the aged say they are never or hardly ever lonely (Dean, 1962), or say that loneliness is not a serious problem (Harris, 1975). Most older persons have close relatives within easy visiting distance and contacts between them are relatively frequent (Binstock & Shanas, 1976). About half say they "spend a lot of time" socializing with friends (Harris, 1975). About three-fourths of the aged are members of a church or synagogue (Erskine, 1964), and about half attend services at least three times per month (Catholic Digest, 1966). Over half belong to other voluntary organizations (Hausknecht, 1962). Thus, between visits with relatives and friends and participation in church and other voluntary organizations, the majority of old people are far from socially isolated.

(18) Older workers have fewer accidents than younger workers. Most studies agree this is true. For example, a study of 18,000 workers in manufacturing plants found that workers beyond age 65 have about one-half

the rate of nondisabling injuries as those under 65, and older workers have substantially lower rates of disabling injuries (Kossoris, 1948).

(19) Only 10.3% of the population were age 65 or over in 1975 and this will probably not increase to more than 12% by the year 2000, even if completed fertility drops to zero population growth levels (Current Population Survey, 1975).

(20) Most medical practioners tend to give low priority to the aged. A series of 12 empirical studies all found that most medical students and doctors, nursing students and nurses, occupational therapy students, psychiatry clinic personnel, and social workers tend to believe the negative stereotypes about the aged and prefer to work with children or younger adults rather than with the aged. Few specialize, or are interested in specializing, in geriatrics (Brown, 1967; Campbell, 1971; Coe, 1967; Cyrus-Lutz & Gaitz, 1972; DeLora & Moses, 1969; Gale & Livesley, 1974; Garfinkel, 1975; Gunter, 1971; Miller, Lowenstein, & Winston, 1976; Mills, 1972; Spence & Feigenbaum, 1968).

(21) The majority of persons 65 or over have incomes well above the poverty level. In 1975 there were only 15.3% of the aged below the official poverty level (about $2,400 for an aged individual or $3,000 for an aged couple). Even if the "near poor" are included, the total in or near poverty is only 25.4% (Brotman, 1976).

(22) Over three-fourths of old people are working or would like to have some kind of work to do (including housework and volunteer work). There are about 12% of persons 65 or over who are employed, 21% who are retired but say they would like to be employed, 17% who work as housewives, 19% who are not employed but do volunteer work, and another 9% who are not employed and not doing volunteer work but would like to do volunteer work (Harris, 1975). These percentages total to 78%.

(23) Older people do not tend to become more religious as they age. While it is true that the present generation of older persons tend to be more religious than the younger generations, this appears to be a generational difference (rather than an aging effect) due to the older persons' more religious upbringing. In other words, the present older generation has been more religious all their lives rather than becoming more religious as they aged. Longitudinal studies have found no increase in the average religious interest, religious satisfaction, nor religious activities among older people as they age (Blazer & Palmore, 1976).

(24) The majority of old people are seldom irritated or angry. The Kansas City Study found that over one-half the aged said they are never or hardly ever irritated and this proportion increases to two-thirds at age 80 or over. About three-fourths said they are never or hardly ever angry (Dean, 1962). The Duke Adaptation Study found that 90% of persons over age 65 said they were never angry during the past week.

(25) The health and socioeconomic status of older people (compared to younger people) in the year 2000 will probably be much higher than now. Measures of health, income, occupation, and education among older people are all rising in comparison to those of younger people. By the year 2000, the gaps between older and younger persons in these dimensions will probably be substantially less (Palmore, 1976).

Uses

There are several possible uses for this quiz which we will discuss and illustrate. The simplest use is as a stimulus for group discussion and clarification of misconceptions. Whenever I have presented the quiz to a group, it always stimulates many questions and considerable discussion of the basis for these facts and of their implications.

Table 1. Facts on Aging Scores for Undergraduates, Graduates, and Faculty.

Group	N	Mean % Right	Standard Deviation
Undergraduate students	87	65	11.2
Graduate students	44	80	7.5
Faculty	11	90	7.7

A second use is to measure and compare different groups' overall levels of information about aging. For example, Table 1 shows that a sample of Duke undergraduate students (in Introductory Sociology classes) got only two-thirds of the facts correct, compared to 80% correct among graduate students in human development (at Duke University and Pennsylvania State University), and 90% correct among faculty in human development (at Duke and Pennsylvania State). These differences also support the validity of the quiz. The only item on which more errors were

(Continued)

made by graduate students and faculty than by undergraduates is Item #22: "The majority of older people are working or would like to have some kind of work to do (including housework and volunteer work)." Apparently, most undergraduates believed correctly that the majority of aged do some kind of work or want to work, but there were 4 faculty and 5 graduate students who were unaware of how the various categories of working, housework, volunteer work, and wanting work or volunteer work, add up to well over half the elderly.

It would be interesting and useful to find out which age, sex, race, religious, regional, socioeconomic, and other groups have more or less correct information about aging.

A third use is to identify the most frequent misconceptions about aging. Table 2 shows that the most frequent misconceptions among the sampled undergraduates were that a large proportion of the aged are living in institutions (74% wrong); that a majority of the aged are frequently bored (74% wrong); that over 15% of the population are age 65 or over (86% wrong); and that a majority of older people have incomes below the poverty level (74% wrong). Almost half of even the faculty thought that a majority of the aged were in poverty. Notice that three of these frequent misconceptions are negative stereotypes and that the other one exaggerates the problem of the aged by exaggerating the numbers of aged.

Table 2. Percentage of Errors on Each Statement by Undergraduates, Graduates, and Faculty.

Statement #	% Errors by: Undergraduates	Graduates	Faculty
1	7	0	0
2	40	14	27
3	16	2	0
4	21	16	9
5	12	0	0
6	2	7	0
7	74	27	0
8	40	27	18
9	37	2	0
10	9	0	0
11	47	9	0
12	47	30	9
13	5	0	0
14	7	7	0
15	9	2	0
16	74	73	55
17	42	16	0
18	42	18	0
19	86	55	36
20	56	9	9
21	74	50	45
22	2	11	36
23	63	44	18
24	58	73	0
25	21	18	18

A fourth use is as an indirect measure of bias toward the aged. Errors on some of the items probably indicate a negative bias toward the aged: for example, if someone says it is true that a majority of old people are senile (#1), it probably indicates a negative image of the aged. On the other hand, errors on other items probably indicate a positive bias toward the aged: for example, if someone denies that the five senses tend to decline in old age (#2) it probably indicates an unrealistically favorable image of old age. We have classified sixteen items as indicating a negative bias if they are marked incorrectly: items numberd 1, 3, 5, 7, 8, 9, 10, 11, 13, 16, 17, 18, 21, 22, 24, and 25. On the other hand, we have classified five items as indicating a positive bias if they are marked incorrectly: items numbered 2, 4, 6, 12, and 14. Using these items, one can then compute a net anti-aged or pro-aged score by subtracting the percentage of errors on the negative bias items from the percentage of errors on the positive bias items. If the resulting score is negative, it indicates a net anti-aged bias; if it is positive, it indicates a net pro-aged bias. For example, 12 of the undergraduates had net anti-aged scores of 33 or more, and 5 had net pro-aged scores of 33 or more. Table 3 shows that the undergraduates and graduates tended to have more anti-aged errors than pro-aged errors, but that there was little difference in anti-aged and pro-aged errors among the faculty. About 2/3 of the undergraduates had net anti-aged bias. It would be useful to know which groups in the population tend to have high or low anti-aged bias.

Table 3. Pro- and Anti-Aged Errors for Undergraduate Graduates, and Faculty.

Group	N	Mean % Pro-Errors	Mean % Anti-Errors	% Pro minus % Anti
Undergraduate students	87	26	33	-7
Graduate students	44	15	20	-6
Faculty	11	9	11	-2

A final use of the quiz would be to measure the effects of lectures, courses, or other training experiences by comparing before and after scores; both total scores and the net anti-

aged scores. A longer test with multiple choice format might be a more sensitive measure, but this quiz has the advantage of requiring only a few minutes without taking much time away from the lecture or course itself. Similarly, periodic administrations of the quiz to representative samples of the public could be used to gauge changes in information levels and biases of the public as a whole.

Summary

Previous tests on aging tend to be long, undocumented, and confuse factual statements with attitudes. The present quiz is short (25 items on one page), documented, and consists of factual statements only. It is designed to cover the basic physical, mental, and social facts and the most frequent misconceptions about aging. It may be used to stimulate discussion; compare levels of information in different groups (undergraduates averaged 65% correct, graduates averaged 80%, and faculty averaged 90%); to identify frequent misconceptions; to measure anti-aged or pro-aged bias (there is usually more anti-aged than pro-aged bias); and to measure the effects of courses or training materials or to measure changes in information or biases over time.

References

Binstock, R., & Snanas, E. (Eds.) *Handbook of aging and the social sciences*. Van Nostrand, New York, 1976.

Birren, J. (Ed.) *Handbook of aging and the individual*. Univ. Chicago Press, Chicago, 1959.

Blazer, D., & Palmore, E. Religion and aging in a longitudinal panel. *Gerontologist*, 1976, *16*, 82-85.

Botwinick, J. *Cognitive processes in maturity and old age*. Springer, New York, 1967.

Brotman, H. Advance data on income in 1975 with revisions of published data for 1974. (Source: Bureau of Census), Oct., 1976. (mimeo)

Brown, M. *Nurses' attitudes toward the aged and their care*. Annual report to the Gerontology Branch, USPHS. USGPO, Washington, 1967.

Busse, E., & Pfeiffer, E. (Eds.) *Behavior and adaptation in late life*. Little, Brown, Boston, 1977.

Campbell, M. Study of the attitides of nursing personnel toward the geriatric patient. *Nursing Research*, 1971, *20*, 147-151.

Catholic Digest. Survey of religions in the U.S., 1966, *7*, 27.

Coe, R. Professional perspectives on the aged. *Gerontologist*, 1967, *7*, 114-119.

Current Population Survey. *Projections of the population of the U.S. by age and sex. 1975-2000*. Series P-25, #541, U.S. Census, USGPO, Washington, 1975.

Cutler, S., & Kaufman, R. Cohort changes in political attitudes. *Public Opinion Quarterly*, 1975, *39*, 69-81.

Cyrus-Lutz, C., & Gaitz, C. Psychiatrists' attitudes toward the aged and aging. *Gerontologist*, 1972, *12*, 163-167.

DeLora, J., & Moses, D. Specialty preferences and characteristics of nursing students in baccalaureate programs. *Nursing Research*, 1969, *18*, 137-144.

Dean, L. Aging and decline of affect. *Journal of Gerontology*, 1962, *17*, 440-446.

Erskine, H. The polls. *Public Opinion Quarterly*, 1964, *28*, 679.

Gale, J., & Livseley, B. Attitudes toward geriatrics; a report of the King's survey. *Age & Aging*, 1974, *3*, 49-53.

Garfinkel, R. The reluctant therapist. *Gerontologist*, 1975, *15*, 136-137.

Glenn, N., & Hefner, T. Further evidence on aging and party identification. *Public Opinion Quarterly*, 1972, *36*, 31-47.

Golde, P., & Kogan, N. A sentence completion procedure for assessing attitudes toward old people. *Journal of Gerontology*, 1959, *14*, 355-363.

Gunter, L. Students' attitudes toward geriatric nursing. *Nursing Outlook*, 1971, *19*, 466-469.

Harris, L. *The myth and reality of aging in America*. National Council on the Aging, Washington, 1975.

Hausknecht, M. *The joiners*. Bedminster Press, New York, 1962.

Kogan, N. Attitudes toward old people. *Journal of Abnormal Psychology*, 1961, *62*, 44-54.

Kossoris, M. Absenteeism and injury experience of older workers. *Monthly Labor Review*, 1948, *67*, 16-19.

Maddox, G., & Douglas, E. Aging and individual differences, *Journal of Gerontology*, 1974, *29*, 555-563.

Masters, W., & Johnson, V. *Human sexual response*. Little, Brown, Boston, 1966.

Miller, D., Lowenstein, R., & Winston, R. Physician's attitudes toward the ill aged and nursing homes. *Journal of American Geriatrics Society*, 1976, *24*, 498-505.

Mills, J. Attitudes of undergraduate students concerning geriatric patients. *American Journal of Occupational Therapy*, 1972, *26*, 200-203.

National Center for Health Statistics. *Health characteristics of persons with chronic activity limitation*. Series 10, #112, USGPO, Washington, 1974.

National Safety Council. *Accident facts*. National Safety Council, Chicago, 1976.

Palmore, E. *Normal Aging. II*. Duke Univ. Press, Durham, 1974.

Palmore, E. The future status of the aged. *Gerontologist*, 1976, *16*, 297-302.

Pfeiffer, E. A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. *Journal of American Geriatrics Society*, 1975, *23*, 433-441.

Riley, M., & Foner, A. *Aging and Society, Vol. One*. Russell Sage, New York, 1968.

Shock, N. The physiology of aging. *Scienfitic American*, 1962, *206*, 100-110.

Spence, D., & Feigenbaum, E. Medical students' attitudes toward the geriatric patient. *Journal of Gerontology*, 1968, *16*, 976-983.

Tuckman, J., & Lorge, I. The effect of institutionalization on attitudes toward old people. *Journal of Abnormal Psychology*, 1952, *47*, 337ff.

U.S. Census. *Persons in institutions and other group quarters*. Special Subject Report. USGPO, Washington, 1970.

Woodruff, D., & Birren, J. (Eds.) *Aging*. Scientific perspectives and social issues. Van Nostrand, New York, 1975.

6
Dementia

Jason H. Karlawish, MD

Commentary

Medicine is relentlessly antihistorical. Current practitioners care little that once upon a time we bled patients whose febrile countenance was overstimulated or that we distinguished between intermittent and remittent fevers. We care about facts that are true in the here and now. The past is for emeritus professors to ponder.

What then is the value of reading Alois Alzheimer's case report of the disease that bears his name? This case report shows how the way we look at a disease changes what we call that disease. Diseases have a history because we have a history. Review also shows how time past and time present are both perhaps present in time present.

Auguste D was a young woman when she presented to the insane asylum. At 51 years of age, she began accusing her husband of infidelity. Soon, she suffered a rapid loss of memory, becoming lost even in her own home. In time, she became suspicious, aggressive, and angry. She heard things that were not there. These problems waxed, and they waned. She had trouble reading, writing, and speaking. She no longer could use common objects. At the end, she was completely stuporous. She lay in her bed with her legs drawn up under her and, despite all precautions, she acquired decubitus ulcers. After four and one-half years of her peculiar disease, she died.

Her brain autopsy showed that her entire cortex scattered full of fibrils and miliary foci, especially in the upper layers. Alzheimer's point was that through careful correlation of clinical and anatomic data he had discovered a distinct disease. He wrote that "these observations show that we should not be satisfied to take a clinically unclear case and, by making great efforts, fit is into one of the known disease categories."

What Dr. Alzheimer saw was that the changes in his young patient were the same changes seen in a "senile brain," that is the brain of an old person. For years we would distinguish between senile dementia as a nonspecific syndrome seen in "normal aging" and Alzheimer's disease: the same clinical problems but occurring in a "younger person's" brain riddled with plaques and tangles. That concept of Alzheimer's disease lasted for some 60 years.

In time, as our values about normal aging and disease changed, we began to look at the data differently. Why make the distinction between young and old if both suffer alike and both have the same pathology? "Senile dementia" disappeared. The current criteria for Alzheimer's disease, published in 1984, include an age of onset from 40 to 90.

As we shift our definition of Alzheimer's disease, we are also shifting how we picture Alzheimer's disease. Now, we are discovering Alzheimer's disease by imaging the brain, sampling blood and spinal fluid, and measuring the electrical encephalogram waves. As we gather these data, we discover that the contours of Alzheimer's Disease are not as sharp as we once thought, that the thing we call Alzheimer's Disease may be many diseases contained within what we think is one disease. The more we explore, the more we reshape what is Alzheimer's Disease. The shadows move but the darkness never lifts.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

Testing in Dementia

"No measurement, no science." This pithy dictum sums up the challenge to medicine's claim as a scientific discipline. A doctor is more than a shaman in a white coat because a doctor measures health and disease.

These measures are not mere impressions or hunches. They are valid scales that a trained examiner uses to describe the patient. A heart is normal because the examiner hears, among other findings, no third heart sound, murmurs, or rubs. A person is not diabetic because his fasting glucose is below 120 milligrams per deciliter. And so on. Medicine has thousands of measures to diagnose disease, grade its severity, and assess the patient's response to treatments.

I would submit that the success of a discipline to lay claim to a disease and its successful treatment and in turn reap the rewards of reimbursement, grants, and public accolades is directly proportional to the degree to which that the field has a clear and coherent language of measurement. By "language of measurement" I mean a set of measures that the field uses to define what is a disease, how sick is the patient, and how well the patient responded to treatment. A language is clear and coherent when physicians in practice, researchers, and even patients readily understand what the measures mean. Think of blood pressure. Researchers, patients, and clinicians all have a reasonably consistent understanding of this measure.

The language of assessing cognition gained its basic grammar in 1975 when Marshal Folstein, Susan Folstein, and Paul R. McHugh published "Mini-Mental State: A Practical Method for Grading the Cognitive State of Patients for the Clinician." The MMS is called "mini" because it only assesses cognitive aspects of mental functioning. In the years to follow, the MMS would gain a vowel: "e" for exam.

Their results showed a method to "quantitatively estimate the severity of cognitive impairment, in serially documenting cognitive change, and in teaching residents a method of cognitive assessment." Their data came from hospitalized adults drawn from a variety of psychiatric diagnoses: dementia, affective disorder of the depressed type with cognitive impairment, and "neuroses." Their goal was a coherent language of cognition. The MMS "makes more objective what is commonly a vague and subjective impression of cognitive disability during an assessment of a patient." Psychiatric residents have "a standard set of questions replacing what is often a bewildering variety of individual approaches." Once a language is in the hands of the people who use it, the language itself has a way of transforming and changing the thing it was intended to describe. Heart failure is becoming a disorder of cardiac ejection fraction. The MMS has had the same history.

In time, the MMS score, that is, the number more so than the questions whose answers added up to that score, took hold of our scientific imaginations. What was developed as a standard set of questions to replace a bewildering variety of individual approaches to assessing the severity of cognitive symptoms turned into a number that labeled a patient's cognitive status. That is, MMS scores were used to classify patients into groups, such as demented or not demented. The transformation from an instrument to monitor the severity of symptoms to one to classify a person into a diagnostic category began even in the original paper. Four pages into the paper, the Mini-Mental *State* is called the Mini-Mental *Status*.

The MMS is now the foundation of the diagnostic assessment of cognitive complaints in ambulatory elderly. It has cut-points for "normal" scores. It is one of the key eligibility criteria for enrollment in Alzheimer's disease clinical trials. It is used to determine whether a patient with Alzheimer's Disease has benefited from a treatment. In the United States and Australia, MMSE scores are sometimes used to justify state reimbursement for medications to treat Alzheimer's disease.

On a Peculiar Disease of the Cerebral Cortex*

Alzheimer—Munich

A. reports a patient observed in the insane asylum in Frankfurt am Main, whose central nervous system had been given to him for investigation by Director Sioli.

Clinically the patient presented such an unusual picture that the case could not be categorized under any of the known diseases. Anatomically the findings were different from all other known disease processes.

*Translation of: Über eine eigenartige Erkrankung der Hirnrinde. *Allgemeine Zeitschrift für Psychiatrie und Psychisch-Gerichtlich Medicin* 64:146-148. 1907. (Also *Zentralblatt für Nervenheilkunde und Psychiatrie* 30:177-179, 1907.)

Arch Neurol—Vol 21, July 1969

(Continued)

A woman, 51 years old, showed jealousy toward her husband as the first noticeable sign of the disease. Soon a rapidly increasing loss of memory could be noticed. She could not find her way around in her own apartment. She carried objects back and forth and hid them. At times she would think that someone wanted to kill her and would begin shrieking loudly.

In the institution her entire behavior bore the stamp of utter perplexity. She was totally disoriented to time and place. Occasionally she stated that she could not understand and did not know her way around. At times she greeted the doctor like a visitor, and excused herself for not having finished her work; at times she shrieked loudly that he wanted to cut her, or she repulsed him with indignation, saying that she feared from him something against her chastity. Periodically she was totally delirious, dragged her bedding around, called her husband and her daughter, and seemed to have auditory hallucinations. Frequently, she shrieked with a dreadful voice for many hours.

Because of her inability to comprehend the situation, she always cried out loudly as soon as someone tried to examine her. Only through repeated attempts was it possible finally to ascertain anything.

Her ability to remember was severely disturbed. If one pointed to objects, she named most of them correctly, but immediately afterwards she would forget everything again. When reading, she went from one line into another, reading the letters or reading with a senseless emphasis. When writing, she repeated individual syllables several times, left out others, and quickly became stranded. When talking, she frequently used perplexing phrases and some paraphrastic expressions (milk-pourer instead of cup). Sometimes one noticed her getting stuck. Some questions she obviously did not comprehend. She seemed no longer to understand the use of some objects. Her gait was not impaired. She could use both hands equally well. Her pateller reflexes were present. Her pupils reacted. Somewhat rigid radial arteries; no enlargement of cardiac dullness; no albumin.

During her subsequent course, the phenomena that were interpreted as focal symptoms were at times more noticeable and at times less noticeable. But always they were only slight. The generalized dementia progressed however. After 4½ years of the disease, death occurred. At the end, the patient was completely stuporous; she lay in her bed with her legs drawn up under her, and in spite of all precautions she acquired decubitus ulcers.

The autopsy revealed a generally atrophic brain without macroscopic lesions. The large cerebral vessels were altered by arteriosclerosis.

In sections prepared with the Bielschowsky silver method, remarkable changes in the neurofibrils appeared. In the interior of a cell that otherwise appeared normal, one or several fibrils stood out due to their extraordinary thickness and impregnability. At a later stage, many fibrils appeared, situated side by side and altered in the same way. Then they merged into dense bundles and gradually reached the surface of the cell. Finally, the nucleus and the cell disintegrated, and only a dense bundle of fibrils indicated the site where a ganglion cell had been.

Since these fibrils could be stained with different dyes than normal, a chemical alteration of the fibrillar substance must have taken place. This then could be the reason why the fibrils survived the death of the cell. The alteration of the fibrils seemed to go hand in hand with the deposition in the ganglion cell of a pathological metabolic product not yet investigated further. About ¼ to ⅓ of all ganglion cells in the cerebral cortex showed such changes. Numerous ganglion cells, particularly in the upper cell layers, had disappeared entirely.

Scattered through the entire cortex, especially in the upper layers, one found miliary foci that were caused by the deposition of a peculiar substance in the cerebral cortex. It could be recognized without staining, but was very refractory to dyes.

The glia had formed abundant fibers. Furthermore, many glial cells exhibited large fat vesicles.

Infiltration of the vessels was entirely absent. However, one saw evidence of endothelial proliferation and also neovascularization in some places.

In summary, we are apparently confronted with a distinctive disease process. An increasing number of unusual diseases have been discovered during the past few years. These observations show that we should not be satisfied to take a clinically unclear case and, by making great efforts, fit it into one of the known disease categories. Undoubtedly there are many more psychiatric diseases than are included in our textbooks. Often a subsequent histological examination would show the peculiarity of the case. Then gradually we would be able to separate individual diseases clinically from the large classes of diseases in our textbooks and define their clinical characteristics more precisely.

(From our own correspondent)

J. psychiat. Res., 1975, Vol. 12, pp. 189–198. Pergamon Press. Printed in Great Britain.

"MINI-MENTAL STATE"

A PRACTICAL METHOD FOR GRADING THE COGNITIVE STATE OF PATIENTS FOR THE CLINICIAN*

MARSHAL F. FOLSTEIN, SUSAN E. FOLSTEIN
and
PAUL R. MCHUGH

Department of Psychiatry, The New York Hospital-Cornell Medical Center, Westchester Division, White Plains, New York 10605, U.S.A.
and
Department of Psychiatry, University of Oregon Medical School, Portland, Oregon 97201, U.S.A.

(*Received* 17 *December* 1973; *in revised form* 25 *November* 1974)

INTRODUCTION

EXAMINATION of the mental state is essential in evaluating psychiatric patients.[1] Many investigators have added quantitative assessment of cognitive performance to the standard examination, and have documented reliability and validity of the several "clinical tests of the sensorium".[2,3] The available batteries are lengthy. For example, WITHERS and HINTON's test includes 33 questions and requires about 30 min to administer and score. The standard WAIS requires even more time. However, elderly patients, particularly those with delirium or dementia syndromes, cooperate well only for short periods.[4]

Therefore, we devised a simplified, scored form of the cognitive mental status examination, the "Mini-Mental State" (MMS) which includes eleven questions, requires only 5–10 min to administer, and is therefore practical to use serially and routinely. It is "mini" because it concentrates only on the cognitive aspects of mental functions, and excludes questions concerning mood, abnormal mental experiences and the form of thinking. But within the cognitive realm it is thorough.

We have documented the validity and reliability of the MMS when given to 206 patients with dementia syndromes, affective disorder, affective disorder with cognitive impairment "pseudodementia"[5,6]), mania, schizophrenia, personality disorders, and in 63 normal subjects.

DESCRIPTION OF THE MMS

The MMS is shown in the appendix. Questions are asked in the order listed and scored immediately. The tester (psychiatric resident, nurse, or volunteer) is instructed first to make the patient comfortable, to establish rapport, to praise successes, and to avoid

*Reprint request to M.F.F. now at Department of Psychiatry and Behavioral Science, Johns Hopkins Hospital, Baltimore, Md. 21205.

(Continued)

pressing on items which the patient finds difficult. In this setting most patients cooperate, and catastrophic reactions are avoided.

The MMS is divided into two sections, the first of which requires vocal responses only and covers orientation, memory, and attention; the maximum score is 21. The second part tests ability to name, follow verbal and written commands, write a sentence spontaneously, and copy a complex polygon similar to a Bender–Gestalt Figure; the maximum score is nine. Because of the reading and writing involved in Part II, patients with severely impaired vision may have some extra difficulty that can usually be eased by large writing and allowed for in the scoring. Maximum total score is 30. The test is not timed. Detailed instructions for administration are given in the appendix.

METHODS

The MMS was given to two groups of people that we will refer to as Samples A and B. In Sample A (Table 1) are 69 patients chosen specifically as clear examples of clinical conditions (29 with dementia syndromes due to a variety of brain diseases, 10 with affective disorder, depressed type with clinically recognizeable cognitive impairment, 30 with uncomplicated affective disorder, depressed type) and 63 normal, elderly persons similar in age to the patients. All the patients were tested shortly after admission to the New York Hospital Westchester Division, a private psychiatric hospital and the normal subjects were tested at a Senior Citizens Center and at a retirement apartment complex. Thirty-three of the 69 patients in Sample A were retested after treatment. The patients with dementia were treated according to their clinical conditions. They occasionally received tricyclic antidepressants or phenothiazines as well as treatment for medical illnesses. The patients with depression were treated with antidepressants and/or ECT. They also may have received medical treatments.

Sample B (Table 2) is a patient group formed by taking consecutive admissions to the hospital and giving them the MMS shortly after admission. It was intended to be a standardization sample and came eventually to consist of 137 patients (9 patients with dementia, 31 patients with affective disorder, depressed type, 14 patients with affective disorder, manic type, 24 with schizophrenia, 32 with personality disorder with drug abuse, and 27 with neurosis). These diagnoses were made by M.F. on review of the hospital chart employing the diagnostic criteria described below and without knowledge of the MMS scores. Subsets of patients from both Samples A and B were extracted for age-matched studies (Table 1B) concurrent validity (Table 3) and test-retest reliability (Table 4).

The following diagnostic criteria were used for both Sample A and B:

Dementia. A global deterioration of intellect in clear consciousness.

Affective disorder, depressed type, with cognitive impairment. A sustained feeling of depression with an attitude of hopelessness, worthlessness or guilt accompanied by disturbances in orientation and memory which occurred after the onset of the depression.

Affective disorder, depressed type, uncomplicated. A sustained feeling of depression with an attitude of hopelessness, worthlessness or guilt and with no notable cognitive defect.

Affective disorder, manic type. A sustained feeling of elevated mood with an attitude of overconfidence or exaggerated self-importance.

Schizophrenia. Either Schneider's first rank symptoms in the absence of affective symp-

TABLE 1.

Sample A

A. Mini Mental State Scores on Admission

Diagnosis	N	Age	Sex M/F	MMS $\bar{x}$	MMS S.D.	MMS Range	Mann-Whitney U	P
Dementia	29	80.8	12/17	9.6	5.8	0-22	45	$<.001$
Depression with cognitive Impairment	10	74.5	7/3	19.0	6.6	9-27	65.8	$<.001$
Affective Dis., Depressed	30	49.8	9/21	25.1	5.4	8-30	1178 (Z=6)	$<.001$
Normal	63	73.9	27/36	27.6	1.7	24-30		

B. Mini Mental Scores on Admission: Age-Matched Sample

Diagnosis	N	Age	Age Range	Sex M/F	MMS $\bar{x}$	MMS S.D.	MMS Range	Mann-Whitney U	P
Dementia	8	76	75-79	2/6	6.9	4.7	1-14	4	$<.001$
Depression with cognitive Impairment	8	76	70-86	5/3	18.4	5.7	9-27	8.5	$<.006$
Affective Dis., Depressed	8	74	69-79	1/7	26.1	4.4	17-30		

C. Mini Mental State Scores of Patients Tested Before and After Treatment

Diagnosis	N	Age	Sex M/F	MMS $\bar{x}$	MMS S.D.	MMS Range	MMS $\bar{x}$	MMS S.D.	MMS Range	$\bar{x}$ days between tests	Wilcoxon T (1 tail)	P
Dementia	14	81.4	6/8	10.5	6.6	0-22	11.1	5.7	1-19	29	20	NS
Depression with cognitive Impairment	7	75.0	5/2	18.3	5.0	13-27	23.4	2.4	21-26	36	1.0	$<.025$
Affective Dis., Depressed	12	58.9	3/9	25.5	5.0	14-30	27.2	3.7	16-30	51	10.5	$<.025$

TABLE 2.

Sample B

Diagnosis	N	Age	Sex M/F	MMS $\bar{x}$	MMS S.D.	MMS Range
Dementia	9	74.4	3/6	12.2	6.7	1-22
Depression	31	50.7	16/15	25.9	4.2	9-30
Mania	14	39.5	6/8	26.6	3.5	20-30
Schizophrenia	24	44.6	14/10	24.6	6.6	1-30
Personality Disorder with Drug Abuse	32	34.3	17/15	26.8	2.5	19-30
Neurosis	27	25.6	15/12	27.6	2.4	21-30

toms or the presence of a personality deterioration associated with thought disorder and emotional incongruence without first rank symptoms.

(Continued)

Personality disorder with drug abuse. Absence of all above symptoms with a history of drug abuse, including alcohol.

Neuroses. Presence of psychological symptoms appearing to arise from the combination of a particular life situation and vulnerable character but with the specific absence of symptoms characteristic of the other syndromes.

TABLE 3.

Sample for MMS - IQ Correlation

Diagnosis	N	Age	Sex M/F
Dementia	7	78	3/4
Depression with cognitive Impairment	8	76	6/2
Depression	8	55	3/5
Schizophrenia	2	68	1/1
Neurosis	1	22	0/1

TABLE 4.

Test-Retest Reliability

Type of Reliability	Sample Composition	N	Age	Sex	MMS 1			MMS 2			# days between tests	Wilcoxon T (2 tail)	P	Pearson r	P
				M/F	$\bar{x}$	S.D.	Range	$\bar{x}$	S.D.	Range					
24 hr. retest (1 tester)	various types of depressive symptoms	22	41.2	3/19	24.2	7.1	2-30	25.3	7.0	1-30	1	45	NS	0.887	<.0001
24 hr. retest (2 testers)	various types of depressive symptoms	19	45.6	7/12	23.9	4.7	13-30	25.2	5.1	13-30	1	22	NS	0.827	<.0001
28 day retest clinically stable patients	dementia, depression, schizophrenia	23	74.1	6/17	19.3	10.0	1-30	19.2	9.2	1-29	27.7	42	NS	0.988	<.0001

RESULTS

Validity

The MMS separated the three diagnostic groups in Sample A from one another and from the normal group. Of a total possible score of 30, the mean score for patients with dementia was 9·7, depression with cognitive impairment 19·0, and uncomplicated affective disorder, depressed 25·1. The mean score for normals was 27·6. Thus, the MMS scores agreed with the clinical opinion of the *presence* of cognitive difficulty and as the cognitive difficulty is usually less in depression than in dementia the scores dispersed in a fashion agreeing with the *severity* of the difficulty.

To be sure that these scores were not due to age effects and unrelated to clinical conditions an age-matched group was drawn from Sample A and showed an identical dispersal of scores according to diagnosis (Table 1b). Mean initial Mini-Mental Status score for

patients with depression under 60 yr-of-age was 24·5 and for patients over 60 was 25·7. These scores were not significantly different.

Thirty-three patients in Sample A were tested prior to and after treatment appropriate to their conditions. Patients with dementia most of whom have uncorrectable brain disease could be expected to show little change in a valid test of cognitive state, whereas those with depression and an associated cognitive difficulty (pseudo dementia) should show a considerable gain with treatment. These expectations are borne out in the results. There is no significant change in the MMS of dementia, a small but significant increase in the depressed patients, and a large and significant increase in those depressed patients with symptoms of cognitive difficulty.

Graphs charting the change-over time in the Mini-Mental State in three patients with improving cognitive states illustrate its usefulness serially and are further examples of how the MMS changes with the clinical state. The examples include a patient recovering from a head injury (Fig. 1), a patient recovering from a metabolic delerium (Fig. 2), and a patient recovering spontaneously over 2½ months from a depression accompanied by severe cognitive impairment (Fig. 3).

Sample B was drawn in order to improve the impression of validity by standardizing the

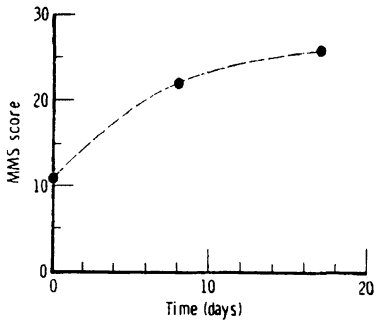


FIG. 1. Serial Mini-Mental State Scores of a patient recovering from a head injury.

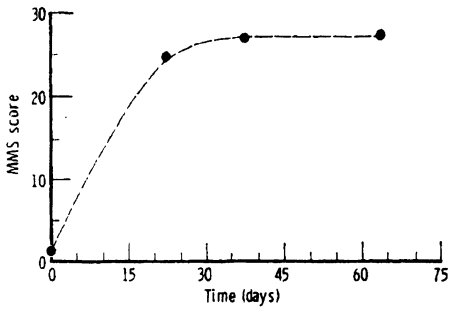


FIG. 2. Serial Mini-Mental State Scores of a patient recovering from a metabolic delerium.

(Continued)

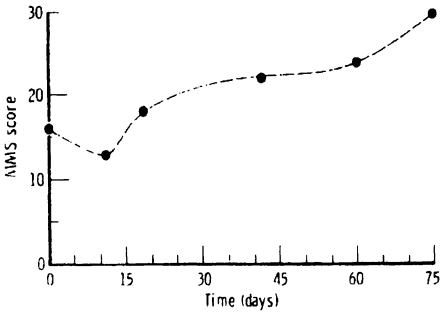


FIG. 3. Serial Mini-Mental State Scores of a patient recovering spontaneously from a depression accompanied by severe cognitive impairment.

MMS in a consecutive series of admission. One hundred and thirty-seven consecutive admissions were examined. Their mean MMS scores were: dementia 12·2; affective disorder, depressed 25·9; mania 26·6; schizophrenia 24·6; personality disorder with drug abuse 26·8; and neuroses 27·6. The minor differences in mean scores between Sample A and B for dementia and depression are not significant. In Sample B the means are similar for all diagnostic groups except dementia. However, amongst the groups with similar means those with depression and schizophrenia had a much wider range of scores than the other diagnostic groups or normal subjects in Sample A. Scores below 20 were found only in functional psychosis or dementia with but one exception; a score of 19 in a patient who had a history of drug abuse.

Concurrent validity was determined by correlating MMS scores with the Wechsler Adult Intelligence Scale, Verbal and Performance scores in a group of patients selected from Sample A and B because they had both a MMS and WAIS Performance in the same week. See Table 3 for the diagnostic and age distribution of this group. For Mini-Mental Status vs Verbal IQ, Pearson r was 0·776 ($p < 0·0001$). For Mini-Mental Status vs Performance IQ, Pearson r was 0·660 ($p < 0·001$).

Reliability

The MMS is reliable on 24 hr or 28 day retest by single or multiple examiners. When the Mini-Mental Status was given twice, 24 hr apart by the same tester on both occasions, the correlation by a Pearson coefficient was 0·887. Scores were not significantly different using a Wilcoxon T. To note examiner effect on 24 hr test retest reliability the MMS was given twice, 24 hr apart by two examiners. The Pearson r remained high at 0·827. The scores did not change; Wilcoxon T was not significant (Table 4). Thus the scores seem stable even when multiple examiners are used, the practice effect is small.

When elderly depressed and demented patients chosen for their clinical stability were given the Mini-Mental Status twice, an average of 28 days apart, there was no significant difference in these scores by the Wilcoxon T and the product moment correlation for test 1 vs test 2 was 0·98. (See Table 4.)

DISCUSSION

The MMS is a valid test of cognitive function. It separates patients with cognitive disturbance from those without such disturbance. Its scores follow the changes in cognitive state when and if patients recover. Its scores correlate with a standard test of cognition, the Wechsler Adult Intelligence Scale (WAIS).

Before considering its uses, it is an elementary but important point that as with any examination of cognitive performance, the MMS cannot be expected to replace a complete clinical appraisal in reaching a final diagnosis of any individual patient. Cognitive difficulties arise in a number of different clinical conditions. This is demonstrated by the overlapping of scores on the MMS in several categories here. Accurate diagnosis, including appraisal of the significance of cognitive disabilities documented in the MMS, depends on evidence developed from the psychiatric history, the full mental status examination, the physical status and pertinent laboratory data.

But the MMS does have a number of valuable features for clinical practice even though it cannot carry alone the diagnostic responsibility. As it is a quantified assessment of cognitive state of demonstrable reliability and validity, it makes more objective what is commonly a vague and subjective impression of cognitive disability during an assessment of a patient. It can provide this quantification easily requiring only a few minutes to complete. It can be repeated during an illness and shows little practice effect. Thus it is ideal for initial and for serial measurements of this important aspect of mental functioning and can demonstrate worsening or improvement of this feature over time and with treatment.

As with any other quantified assessment of cognitive function such as the WAIS with which it correlates so well, the MMS permits comparisons to be drawn between intellectual changes and other aspects of mental functioning. We have found it particularly useful in documenting the cognitive disability found in some patients with affective disorder (Post's pseudodementia) and the improvement of this symptom with appropriate therapy for the mood disorder. Other applications that demand a quantitative assessment of cognitive function might be expected.

The MMS as it is extracted from the clinical examination has an advantage in assessment of patients and clinical problems not so obvious in tests such as the WAIS that are designed for other purposes such as prediction of school or occupational performance. Thus failures in the MMS on orientation, memory, reading and writing have much clearer implications than do failures in digit symbol, picture completion or vocabulary subtests of the WAIS in terms of a patients capacity to care for himself. These implications from the MMS score are easily appreciated by other professionals such as lawyers, judges and social workers concerned with such issues as the patient's competency to manage his daily affairs. It can therefore aid in bringing to the patient the social supports that he needs.

Finally we have found the MMS useful in teaching psychiatric residents to become skilful in the evaluation of the cognitive aspects of the mental status. It provides them with a standard set of questions replacing what is often a bewildering variety of individual approaches. Those questions that it employs have obvious clinical pertinence and cover most of the categories of cognitive disability. Since it can be done quickly and gives a score it draws the resident's attention to global improvements or declines in cognitive state. It also though because special attention is focused on memory and language functions will reveal

(Continued)

the partial cognitive disabilities seen in the aphasic and the amnestic syndromes. As it becomes a routine, we have found an increase in resident interest and competence in assessing and managing the conditions that affect cognitive functioning such as dementia and delerium.

SUMMARY

A short, standardized form was devised for the serial testing of the cognitive mental state in patients on a neurogeriatric ward, as well as for consecutive admission to a hospital. It was found to be quick, easy to use, and acceptable to patients and testers.

When given to 69 patients with dementia, depression with cognitive impairment, and depression (Sample A), the test proved to be valid and reliable. It was able to separate the three diagnostic groups, it reflected clinical cognitive change, it did not change in patients thought to be cognitively stable, and it was correlated with the WAIS scores. Standardization of the test by administration to 63 normal elderly subjects and 137 patients (Sample B) indicated that the score of 20 or less was found essentially only in patients with dementia, delerium, schizophrenia or affective disorder and not in normal elderly people or in patients with a primary diagnosis of neurosis and personality disorder. The Mini-Mental Status was useful in quantitatively estimating the severity of cognitive impairment, in serially documenting cognitive change, and in teaching residents a method of cognitive assessment.

Acknowledgement—Supported in part by the general research funds, University of Oregon, Health Sciences Division.

REFERENCES

1. ROTH, M. The clinical interview and psychiatric diagnosis. Have they a future in psychiatric practice? *Comp. Psychiat.* 8, 427, 1967.
2. SHAPIRO, M. B., POST, F., LOFVING, B. and INGLES, J. "Memory Functions" in psychiatric patients over sixty, some methodological and diagnostic implications. *J. Ment. Sci.* 102, 233, 1956.
3. WITHERS, E. and HINTON, J. Three forms of the clinical tests of the sensorium and their reliability. *Br. J. Psychiat.* 119, 1, 1971.
4. HALSTEAD, H. A psychometric study of senility. *J. Ment. Sci.* 89, 363, 1943.
5. POST, F. *The Clinical Psychiatry of Late Life*. Pergamon Press, Oxford, 1965.
6. KILOH, L. G. Pseudo-dementia. *Acta psychiat. scand.* 37, 336, 1961.

APPENDIX

Patient.....
Examiner .....
Date .....

"MINI-MENTAL STATE"

Maximum Score	Score	
		ORIENTATION
5	( )	What is the (year) ?
		REGISTRATION
3	( )	Name 3 objects: 1 second to say each. Then ask the patient all 3 after you have said them. Give 1 point for each correct answer. Then repeat them until he learns all 3. Count trials and record.
		Trials

MINI-MENTAL STATE 197

ATTENTION AND CALCULATION

5 ( ) Serial 7's. 1 point for each correct. Stop after 5 answers. Alternatively spelt "world" backwards.

RECALL

3 ( ) Ask for the 3 objects repeated above. Give 1 point for each correct.

Note that the above appendix is intentionally incomplete. The sample questions provided are intended as examples only.

7
Delirium

Jerry C. Johnson, MD

Commentary

In "Delirium, A Syndrome of Cerebral Insufficiency," Engel and Romano presage modern-day precepts about delirium to an amazing degree. This paper, published in 1959 and a second published by the same authors 15 years earlier (1) offer some of the best clinical descriptions of delirium available to this day. In a style indicative of the time, meticulous clinical descriptions of cases were used to conclude that the neuropsychological impairment in delirium considered fundamental to this day is an attentional deficit. Referring to attentional deficits as "level of awareness," Engel and Romano clearly use awareness to refer to problems "focusing attention on the important precept." One has difficulty in "screening out interfering perceptions, whether they arise from the external environment or from within." In 1972, Chedru and Geschwind, neurologists, arrived at the same conclusion and further showed clinical correlations to varying degrees with other cognitive neurological elements (2). Over the five decades since this paper was published, the clinical criteria and the testing criteria (3) for delirium have remained grounded in the teachings of Engels and Romano.

Current clinicians would be mindful of the warnings in this paper. We are told that delirium is prevalent and underrecognized and that its causes are environmental (an acquired disease) and usually multifactorial. The authors reveal that hypo- and hyperactive cases are common and remind us that the behavioral and other psychological manifestations are variable and fluctuating. We are warned that isolated nighttime occurrences of cognitive impairment, often called "sundowning" in current parlance, are not to be equated with delirium, which also may be exacerbated by darkness, but persists throughout the day, a distinction first pointed out in 1908 by Hawley (4). Moreover, Engel and Romano believe that all delirious patients that are not stuporous are frightened (1), an emotion that may cause agitation and paranoia. This fear and paranoia may be exacerbated by bedside discussions that are misinterpreted. And of course, the use of restraints can provoke additional hyperactivity.

The article departs in some ways from current thinking. While current thinking endorses delirium as a metabolic syndrome, the use of the EEG as a marker is not widely accepted today. On the other hand, Engel and Romano viewed the EEG changes in delirium as pathognomonic with 100% sensitivity and specificity, test characteristics unfortunately too good to be true. They also explain the variable behavioral manifestations of delirium by the "ego" of the individual, and while this precept conforms to their training in psychiatry, it would not be accepted entirely today. And although treatment of the underlying disease, the use of family members as a calming influence, avoidance of sedatives, and control of noise and lighting are consistent with our current tenets of treatment, the use of a "tub bath" as an alternative to using a sedative drug is not feasible in most hospitals today, and perhaps unwise.

Some of the current challenges in need of additional research were raised by these authors: 1) to what extent does delirium presage dementia, versus being an isolated syndrome? 2) what is the cellular and biologic basis of the syndrome? and 3) what is the most effective treatment?

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

References

1. Romano J, Engel G. Physiologic and psychologic considerations of delirium. Med Clinic NA 1944;28:629–639.
2. Chedru F, Geschwind N. Disorders of higher cortical functions in acute confusional states. Cortex 1972;8:395–411.
3. Inouye SK, Van Dyck CH, Alessi CA, Balkin S, Siegal AP, Horwitz RI. Clarifying confusion: the confusion assessment method. A new method for detection of delirium. 1990; Ann Inter Med 113:941–948.
4. Hawley E. Manifestations of delirium in the night-time. Am J Nursing 1908;8:757–761.

DELIRIUM, A SYNDROME OF CEREBRAL INSUFFICIENCY

GEORGE L. ENGEL, M.D., AND JOHN ROMANO, M.D.,
ROCHESTER, N. Y.

From the Departments of Psychiatry and Medicine, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.

(Received for publication Dec. 1, 1938)

IT IS a curious fact that while most physicians have a strong bias toward an organic etiology of mental disturbances, at the same time they seem to have little interest in and, indeed, often completely overlook delirium, the one mental disorder presently known to be based on derangement of cerebral metabolism. This state of affairs persists in spite of the fact that many seriously ill hospitalized patients experience some degree of delirium during the course of their illness. The explanation for this state of affairs is not difficult to find. In the first place, the deficiencies in the education of many physicians ill equip them to recognize any but the most flagrant examples of delirium, much less to differentiate it from other types of psychologic disturbance ordinarily encountered among medically ill patients. Most delirious patients are considered either dull, stupid, ignorant, or uncooperative. It is only when their behavior and content of thought are grossly deviant that an abnormal mental state is recognized, although it is not always correctly identified as delirium. Only the latter type of patient, often a management problem on a medical or surgical service, is likely to result in a psychiatric consultation. The psychiatrist, on the other hand, is likely to see only the more disturbed delirous patients since he is generally called in consultation only when someone is disturbed by the patient's behavior. Hence, not only may his picture of delirium be a restricted one, but also, seeing the patient in the home territory of the "organic" specialists, he is less likely or able to pursue an understanding of the underlying physiologic derangements, which are generally conceived to be the proper domain of the internist. Unhappily, the unfortunate patient's malfunctioning brain rests in limbo, an object of attention and interest neither to the medical man nor to the psychiatrist.

This unsatisfactory state of affairs is not helped at all by how the matter is handled in either the medical or the psychiatric texts. The student who hopes to find some clarification of these matters in modern textbooks will gain only confusion. The more modern textbooks of psychiatry follow the official system of classification adopted by the American Psychiatric Association, whereby delirium is correctly included among the disorders caused by or associated with impairment of brain tissue function.[1,2] But these are then subdivided into acute

This work was supported in part by a grant from the Foundations Fund for Research in Psychiatry.

(Continued)

and chronic brain disorders, which are classified in etiologic terms, such as disorders due to or associated with infection, intoxication, trauma, circulatory disturbance, etc., as if each represented a clinical entity. The unwary student is plunged directly into a consideration of such supposed specific entities with either no or only a very sketchy consideration of the basic processes underlying and characterizing delirium. There is little or no indication of the frequency of this syndrome or consideration of the various ways in which brain function may become disordered in relationship to the physiologic derangements characterizing organic illness. By inference the reader is led all too easily to associate delirium with certain specific entities, e.g., meningococcal meningitis, chorea, encephalitis, bromides, alcohol, head trauma, myxedema, and so on, and to overlook the fact that in most instances multiple factors are operating, sometimes only indirectly related to the primary diagnosis.

The textbooks of medicine are even more cavalier in their treatment of this subject. In some it is mentioned only in passing as a complication of certain illnesses, as in yellow fever or typhoid. Perhaps the best discussion is found in Harrison's work,[3] but this is somewhat misleading because of the author's bias for the alcoholic patient as depicting most perfectly the picture of delirium. Further, it is surprisingly lacking in any consideration of the physiologic and metabolic factors.

The problem of delirium is far from an academic one. Not only does the presence of delirium often complicate and render more difficult the treatment of a serious illness, but also it carries the serious possibility of permanent irreversible brain damage. With increasing life expectancy and with improved survival through the influence of surgery and antibiotics, we are now beginning to see an increasing incidence of so-called senile and arteriosclerotic dementias. Do we know how often such developments are initiated during delirious episodes experienced in the course of serious illness?[4] Bedford[5,6] has recently shown that dementia not infrequently develops in old people following operations under general anesthesia and after shock in the course of serious illness. The physician who is greatly concerned to protect the functional integrity of the heart, liver, and kidneys of his patient has not yet learned to have similar regard for the functional integrity of the brain. This is a serious and, perhaps, tragic omission. It is our hope that this paper will stimulate among physicians in general, and among clinical investigators in particular, more frequent recognition of and greater interest in the syndrome of delirium.

DEFINITION

Before attempting to define delirium in clinical terms, we wish to establish firmly what we believe to constitute the basis for this syndrome. In the title the term "syndrome of cerebral insufficiency" is used. This draws attention to the basic etiology of all delirium and couches it in terms analogous to the more familiar concepts of renal insufficiency, hepatic insufficiency, cardiac insufficiency, etc. As with the more familiar types of organ insufficiency, this refers to what evolves when the function of the organ as a whole is interfered with, for whatever

reason. Whether applied to the liver, kidney, or brain, this can be reduced to two basic underlying processes, namely, the failure of metabolic processes to maintain the function of the organ or the loss through death of enough functioning units (cells) to render the function of the organ insufficient. It will be recognized that the first situation may evolve into the second and that except in situations of rapid and massive process, both must often co-exist. Further, the first situation may have a high potentiality of reversibility, a possibility not likely when there is cell death unless the organ has the capacity for regeneration of its functional units, which the brain does not. With respect to the brain, this also corresponds to the clinical distinction between delirium and dementia. Delirium refers to the more reversible disorder and dementia to the irreversible disorder. While such a distinction has some pragmatic value, it would be a mistake at this time in our knowledge to regard these states as any more than different degrees or stages of similar processes. This is especially so since clinically many patients manifest a lower threshold to delirium in response to some general physiologic derangement by virtue of having already suffered some loss of brain substance (i.e., dementia), although the latter may have been so well compensated as to have been clinically inapparent. Sometimes the concept of acuteness is associated with delirium and chronicity with dementia—but this is not correct. Some types of delirium, such as those associated with pernicious anemia and myxedema, may develop very slowly and last for weeks or months and yet be largely reversible when appropriate therapy is administered.[34,35] Similarly, an irreversible defect, a dementia, may develop abruptly, as after a severe head injury, prolonged anoxia, or carbon monoxide intoxication. If the cerebral insufficiency develops very abruptly, in a matter of a few seconds, and lasts only seconds, the resulting disorder is syncope, but again we should appreciate that the basic process is essentially the same as that in delirium.[7] Indeed, we know that if the unconsciousness of syncope persists beyond 30 to 60 seconds, the victim usually will pass through a period of delirium before complete recovery is achieved, whereas with the longer lasting faints, permanent damage and dementia may result. In other words, clinical convention leads us to delineate a number of syndromes associated with cerebral insufficiency, and it is well for the physician to appreciate the common denominator shared by delirium and these other syndromes. In all instances, the factors maintaining the metabolism, the functional integrity, and the life of the cell are of primary importance.

THE METABOLISM OF MAMMALIAN BRAIN

Further progress in the understanding of the etiology, treatment, and prevention of delirium is linked irrevocably with our advancing knowledge of cerebral metabolism. For a long time progress in this field has been slow and uncertain, and few have seriously interested themselves in it. The last decade has, however, seen a great surge of interest and progress, as indicated by the founding of the *Journal of Neurochemistry* in 1956 and the holding of two International Neurochemical Symposia. We will make no attempt even to summarize the important recent advances but would refer the interested reader to the two volumes con-

(Continued)

taining the proceedings of the two international conferences and to a recent review.[8-10]

It is misleading to speak of brain metabolism in any unitary fashion since it is becoming clear that not only may there be major differences in the metabolism of the neuron or nerve pathways compared to the supporting structures, but also that there are significant differences in the metabolic activity of the various regions of the brain. Lowry[11] has developed an elegant technique which makes feasible the measurement of enzyme concentrations in single cell bodies and related structures and has demonstrated significant differences in the activities of 9 enzymes in cell bodies and related structures taken from 6 different areas of the brain

There is need for caution in accepting too easily the classic generalization that "brain" derives its energy entirely from aerobic processes and that glucose is the only substrate. While clinical observation and in vivo studies leave no question that both oxygen and glucose are essential to maintain higher nervous (and mental) activity, the exclusive emphasis on this fact has tended to obscure other biochemical derangements that may take place in spite of adequate oxygen and glucose supplies. Even this dependence on aerobic processes to maintain the normal state of the brain may vary. Shapot,[12] for example, has shown that the susceptibility of the brain to hypoxia varies with the level of functional activity of the central nervous system. He claims that resistance to hypoxia is increased by inhibition and falls after severe excitation. If verified, this phenomenon is one which could have very great clinical importance in respect to the susceptibility to develop delirium. It is a common clinical observation that an organic syndrome (usually dementia) may develop rapidly after a period of psychologic stress. Shapot has also pointed out that the energy produced by the cell is utilized in two ways, namely, for "structural metabolism" (renewal of cell structures) and for specialized functional metabolism. Obviously such a distinction between structure and function is only a relative one, involving as it does relative rates of chemical reaction underlying various processes. Nonetheless this does introduce the possibility of competition between these two kinds of metabolism, as when there is an excessive need or an insufficient supply involving one or another system. In another study, Shapot demonstrated that, when rats were teased to exhaustion, methionine incorporation into brain proteins was much lower than in control animals, whereas, if the exhausted rats were left alone to sleep for 30 minutes, the rate of methionine incorporation was greatly increased as compared with the controls. He interprets these findings to mean that, since during the first minutes of sleep following intense excitation the cerebral respiration still proceeds at a high level and the energy requirements for functional activity are presumably minimal, this energy must therefore be for the requirements of the structural metabolism, which then restores the functional capacity of the nerve cells. Thereafter respiration again falls as the animal continues to sleep.

Other studies indicate that the amino acid and protein metabolism of brain is much more active than heretofore had been supposed.[13,14] Strecker[15] brings support for the idea that the glutamic acid-glutamine system can act as a homeo-

static, maintenance mechanism, controlling carbohydrate metabolism and the many metabolic systems which are interdependent with carbohydrate metabolism. Sporn, Dingman and Defalco[16] have recently demonstrated a rapid rate of uptake of intracisternally injected proline by brain protein and the conversion by brain of this proline to glutamic acid, aspartic acid, alanine, gamma-aminobutyric acid, ornithine, and arginine.

The alleged toxic effects of ammonia on the function of the central nervous system, with its suggested relationship to the delirium of liver disease, focuses attention on the processes of ammonia formation in the brain and the mechanisms of its removal, for which the glutamic acid-glutamine system is regarded as the most likely candidate. Weil-Malherbe[17] observed that brain cortex slices form ammonia at a fairly steady rate in a glucose-free medium. This finding is of particular interest in the light of Geiger's observation[18] that, if cerebral blood flow is increased two- or threefold over normal during a glucose-free perfusion lasting an hour or longer, the electrical activity (EEG) is maintained, reflexes can be elicited, and oxygen consumption of the brain is only slightly below that obtained with glucose. Geiger and his co-workers showed that during glucose-free perfusion structural components are used up by the brain while the preparation still maintains its physiologic functions. These experiments indicate that, given a fast enough cerebral blood flow, the brain can survive and maintain its excitability in the absence of glucose for over an hour by using some of its structural components. Geiger believes this can be explained on the assumption that the breakdown products resulting from noncarbohydrate metabolism in the brain are toxic, but that by speeding up blood flow these breakdown products of noncarbohydrate metabolism are quickly eliminated. This worker also showed that the brain ceases to oxidize glucose, accumulates a high concentration of lactic acid, and loses its physiologic activity in perfusion experiments after about 60 minutes of perfusion without the liver. The insertion of an isolated liver into the perfusion or the addition of fresh liver extract restores cerebral glucose oxidation. A depletion of the galactolipid and phospholipid content of the brain is observed during perfusion in the absence of the liver, but the addition of cytidin and uridin maintains glucose oxidation even in the absence of the liver and prevents the depletion of liquids.

These are but a few of the recent discoveries which invite the clinical investigator to turn his attention to the exploration of similar phenomena in the brain of the patient with delirium.

THE METABOLISM OF THE HUMAN BRAIN

The problem of investigating the metabolism of the intact human brain presents major difficulties. This subject is well reviewed in a recent paper by Kety[19] whose nitrous oxide technique has been the most extensively used method of studying the general metabolism of the brain in vivo. While it has yielded useful results, particularly in respect to the quantity and dynamics of cerebral blood flow, on the whole it has been of limited value in advancing our knowledge of cerebral metabolism in delirium. Some of the reasons for these limitations have been pointed out by Kety. For one thing, since it measures only the total

(Continued)

oxygen uptake of the total brain, it gives no information as to how or even where such energy is being utilized, a point already emphasized when we drew attention to the contrast between so-called structural metabolism and functional metabolism. Thus, it is not surprising that coma is the only situation in which marked depression in oxygen uptake is consistently found. With the lesser degrees of derangement characteristic of delirium, results have been inconsistent. This, we suspect, is more a reflection of a poor design of these studies than a deficiency of the blood flow method itself. The technique usually has been to select for cerebral metabolism studies patients with established diagnoses, such as congestive heart failure, pernicious anemia, hypothyroidism, hypertension, etc., but then to make only a crude evaluation of mental status, often with little understanding of what psychologic and behavioral characteristics could be expected to correlate with reduced cerebral metabolism. In some case reports it seems improbable that the patient was delirious and in others it is impossible to tell from the published material. Further, only a few investigators have made serial observations on the same patients, either during spontaneous recovery or during the application of specific measures which might be expected in any particular instance to improve cerebral metabolism. And in some of the instances in which this was done, the techniques used to evaluate the change in mental status were so inadequate as to limit greatly the significance of the results. In spite of these deficiencies, there is a quite suggestive correlation between the degree of decrease in cerebral oxygen consumption and the mental state in hypoglycemia, diabetes, pernicious anemia, and hypothyroidism.[20-22] It is our opinion that when more attention is paid to identifying and evaluating those aspects of mental function which are characteristic of delirium and when each patient is used as his own control, a much more consistent correlation between cerebral oxygen uptake and mental status will be established in delirium. Perhaps the main exceptions might be those situations in which increased oxygen uptake is not maintaining the function of the brain but some other processes, such as acute inflammatory reactions in encephalitis.

THE ELECTROENCEPHALOGRAM AS AN INDEX OF CEREBRAL INSUFFICIENCY

At present the electroencephalogram, as an index of functional metabolism, is by far the most sensitive and reliable indicator of cerebral insufficiency, as we have defined it. Indeed, in this respect, the electroencephalogram reflects cerebral insufficiency as reliably, if not more reliably, than the electrocardiogram reflects myocardial insufficiency. In spite of the considerable evidence that has accumulated over the past 15 years, this fact remains little known and even less used, either by investigators or by clinicians. Victor and Adams[3] state, "The value of the electroencephalogram in studying delirium has largely been limited by the difficulty of obtaining records free of movement artifact. There is some evidence that the EEG in severe delirium shows non-focal slow activity in the range of 5-7 per second, a state which rapidly returns to normal as the delirium clears. In milder degrees of delirium there is usually no abnormality of the EEG." As we shall show this statement is grossly inaccurate.

Actually, Gerard[23] demonstrated more than 20 years ago that EEG frequency could be expected to parallel changes in the metabolism of the cell, increasing with rise in metabolism and decreasing with fall in metabolism. Polarographic studies demonstrate a very close relationship in time between local reduction of oxygen availability, as measured by the electropolarograph and change in the EEG; the latter usually occurs 6 to 8 seconds after the local oxygen tension begins to fall.[24] Both experimental and clinical observations bear this out. For many years it has been known that both hypoxia and hypoglycemia produce slowing of the EEG.[25] These are two physiologic conditions under which it is well established that the metabolism of the brain cannot be successfully supported. With the application of a quantitative method of frequency analysis of EEG, it has been possible to demonstrate significant slowing of the EEG at ambient air pressures equivalent to 12,000 feet and at blood sugar levels below 60 mg. per 100 c.c., changes which increase progressively as oxygen saturation and blood sugar levels, respectively, are further reduced.[26,27] Indeed, changes in EEG frequency can be demonstrated before any change in psychologic performance becomes demonstrable and well before any change in total cerebral oxygen uptake can be measured by the Schmidt-Kety method. Similar results have been obtained using alcohol,[28] carbon monoxide,[29] and Freon (F12).[30] In all these experimental studies, the fundamental fact has been demonstrated that the psychologic changes correlating most precisely with the slowing of EEG frequency were those that had to do with awareness, attention, memory, and comprehension—that is, the cognitive functions. Observing on different occasions the effects of hypoxia, hypoglycemia, and alcohol on the same subjects, we have demonstrated that, for comparable degrees of slowing of the EEG, the degree of disturbance in cognition, as assayed by clinical tests, was essentially the same under each of these three circumstances.[27] On the other hand, the individual subjects differed appreciably as to their behavior, content of thought, and expression of affect, depending on whether they were under the influence of hypoxia, hypoglycemia, or alcohol. The next section will take up the more detailed delineation of these psychologic characteristics of delirium.

Such studies indicate an approach to the experimental production and study of delirium. We have demonstrated that drugs or physiologic processes that lead to slowing of the EEG also lead to a reduction in the level of consciousness and the efficiency of cognitive processes.[26-30] We have not observed any circumstance in which under experimental conditions diffuse slowing of the EEG failed to be correlated with such an alternation in cognition or vice versa. For example, the prolonged administration of ACTH or cortisone is occasionally associated with psychotic reactions. ACTH and cortisone produce no change in the EEG frequency of normal subjects and most psychotic reactions occurring during the administration of either ACTH or cortisone are unaccompanied by EEG slowing or by changes in cognitive functions characteristic of delirium.[31] In those few instances in which diffuse slowing does occur, a reduction in the level of awareness has been a consistent feature. Atabrine (quinacrine) administered to normal subjects produced heightened attention and vigilance and increased the frequency of the EEG, resulting in a syndrome which was clinically different from delirium.[32]

(Continued)

Utilizing this experimental technique, it has also been shown that the significant EEG finding is the degree of slowing rather than the absolute frequency.[27,28,33-5] Thus, if the EEG initially is fast or in the upper range of normal, a significant reduction in the level of consciousness and EEG frequency may be provoked by drugs, alcohol, hypoxia, etc. without the EEG frequency necessarily falling below the accepted normal range.[27,28,33] It is therefore possible to have a normal EEG in the presence of an appreciable degree of cerebral insufficiency and reduction in the level of awareness, as when a person whose premorbid alpha frequency is 11 to 12 per second shows a slowing to 8 to 9 per second during a moderate delirium. Both of the values still fall within the accepted normal range for the adult population.

The experimental data receive ample confirmation when one examines patients exhibiting varying degrees of disturbance in the level of consciousness and cognitive functioning (as established by the techniques of examination to be described) in the course of any somatic illness. When one studies patients who improve spontaneously or as the result of appropriate therapy, all patients show an increase in the frequency of EEG corresponding to the degree of recovery of the underlying clinical condition and the degree of improvement in the level of awareness.[34-37] As in the experimentally provoked deliria, a few clinically delirious patients exhibit EEG frequencies within the accepted range of normal, but these also show an increase in frequency on improvement, indicating that their premorbid EEG had been in the fast range. When physiologic derangements can be corrected, as with the administration of oxygen to the patient in congestive failure, the change from the recumbent to the sitting position in the patient with pulmonary edema, the administration of glucose to the patient with spontaneous hypoglycemia, or the transfusion of blood to a patient with very severe anemia, then changes in EEG frequency and in mental state may take place rapidly and always to a corresponding degree.[37] The majority of patients with untreated pernicious anemia have slow EEG's and reduced levels of awareness and both of these abnormalities respond rapidly to the administration of Vitamin B_{12} or liver extract.[34] Of interest is the fact that in pernicious anemia the improvement in EEG and in mental status corresponds in time with the beginning of the reticulocyte response and antedates by a considerable period the rise in hemoglobin. This we consider as evidence that the pernicious anemia process includes a cerebral metabolic defect independent of the oxygen-carrying capacity of the blood, a finding which may be related to Geiger's findings noted previously.[18] Addison's disease and hypothyrcidism are two other conditions in which such EEG abnormalities occur with great regularity and respond specifically to appropriate hormone therapy.[35,37]

All these clinical and experimental studies have revealed that the degree of slowing of the EEG corresponds best with the disturbance in consciousness; that these EEG changes are reversible to the extent to which the underlying pathophysiologic process is reversible; and that the character of the EEG change is independent of the specific underlying disease processes but is more related to its severity and duration. On the other hand, there is little correlation between

the degree of EEG change and the more personal aspects of behavior, as will be described.

THE CLINICAL SYNDROME: DELIRIUM

We thus arrive at the proposition that a derangement in functional metabolism underlies all instances of delirium and that this is reflected at the clinical level by the characteristic disturbance in cognitive functions and at the physiologic level by the characteristic slowing of the EEG. To express this slightly differently, a reduction in the level of cognition is a sine qua non of delirium; a relative generalized slowing of the EEG is a sine qua non of delirium, the clinical expression of a cerebral metabolic defect; the diagnosis of delirium is unequivocally established when it can be shown that the level of awareness correlates with changes in EEG frequency, reduction being accompanied by further slowing of the EEG and improvement by relative acceleration of the EEG.

Before describing in more detail the identifying psychologic characteries of delirium, it might be well to comment on the inadequate and confusing fashion in which delirium and the behavior of delirious patients are referred to in the literature. While there are virtually no papers on delirium per se, hospital records and the medical literature abound with case reports involving patients who are delirious by the criteria we have established. In some papers the evaluation of the mental state is an important correlate of other physiologic or pathologic processes that are the subject of investigation. Yet it is common to find such statements as: "The patient was disoriented but not delirious." "The patient was confused and agitated, almost delirious." "He was confused and lethargic and became delirious at night." "He was intermittently obtunded and hallucinating." "The patient became uncooperative and confused." "The patient was unable to cooperate because of lethargy" (this was a patient with ammonia intoxication).

Of a patient dying of hepatic and renal failure we read, "Throughout his hospital stay the patient had displayed varying degrees of emotional lability and alertness. During the last 4 weeks of his life he had fluctuating periods of lethargy, drowsiness, obtundity, and disorientation." In a discussion of the treatment of pneumococcal pneumonia, the case descriptions of patients who unquestionably were delirious include, "He was irritable and thrashing about." "For the first 3 days the patient was restless and almost maniacal." "On admission he was semistuporous, and although responsive to questioning, his answers were irrational. Eight hours after admission he was afebrile, completely rational, and was able to sit up and eat his meals." A good example of a hodgepodge description is the statement, "The retention of carbon dioxide occurring in patients with alveolar hypoventilation may produce a variety of mental manifestations, i.e., depression, anxiety, marked irritability, somnolence, confusion, delirium, and coma." These examples are picked at random and are completely typical of current writing when the mental status of the patient is mentioned at all, which often it is not. It is apparent that most physicians have only the vaguest comprehension of the phenomena they are attempting to report and therefore dispose of the matter by referring to whatever most forcibly strikes

(Continued)

their attention, whether it be the significant process or not. This is clearly an unsatisfactory situation, hardly conducive to clear understanding of the conditions being reported. It might be compared to reports of examinations of the heart which read "There were unusual sounds," or, "There was an exceptionally fast and disturbed rhythm," or, "An odd pause every now and then, clearly tantalizing but unhelpful descriptions even if vaguely correct.

We have intentionally delayed discussing the clinical picture of delirium until the case had been developed for delirium as a unified syndrome, a syndrome of cerebral insufficiency. If we have achieved this, we hope it will now be possible for the reader to comprehend in an orderly and logical fashion the manifestations of delirium. Hopefully this should lead to more frequent recognition and more accurate description of delirium.

The distinctive feature of delirium concerns the cognitive functions and the level of awareness, ranging all the way from coma, the most severe degree, to a disturbance which is detectable only by very exacting (and clinically impractical) psychologic testing. That there is a degree of metabolic insufficiency beyond which consciousness cannot be maintained is a quantitative rather than a qualitative consideration, as evidenced by the fact that all the characteristics of delirium can clearly be demonstrated if the patient can be studied before he becomes comatose or during the period of recovery from coma.

The reader may perhaps best orient himself as to the nature of the experience of mild delirium by his own personal experience. That most familiar is mild alcoholic intoxication. Also similar, although not strictly speaking "delirium" as we have defined it, is the experience of being awakened suddenly in the middle of the night, especially in a strange place. Subjectively one notes a slight blurring or haziness of perceptions so that what impinges on the sense organs is not so promptly or accurately identified or apperceived. The familiar motion picture stunt of depicting the experience of the drunken person by a blurred image which only gradually comes into focus is a good but perhaps caricatured demonstration of this. Not only does one have difficulty in focusing attention on the important percept, but one also has difficulty in screening out interfering perceptions, whether they arise from the external environment or from within. Similarly one notes a difficulty in marshaling one's thoughts logically, coherently, and appropriately. Desired memories and associations seem less readily accessible and often undesired or inappropriate memories and associations appear in their place. The execution of appropriate behavior and speech encounters the same difficulties.

With very mild delirium, such difficulties may be quite inapparent to the observer since, for the most part, they can be adequately overcome by heightened effort. Nonetheless the EEG at this point already will show some slowing as we have demonstrated in both experimental and clinical delirium. But, if the patient does not or cannot mobilize the increased effort, the observant physician will note vagueness, uncertainty, hesitancy in the patient's manner of speaking, and errors or contradictions of fact that the patient may or may not himself correct. At this point the patient may acknowledge that he is having some difficulty in thinking or remembering

With further advance of the delirium, this state becomes more manifest. Now the patient becomes unable to perform correctly, even with heightened effort. He begins to appear confused and even bewildered. His responses tend to be slow, uncertain, and often reveal errors in memory retention, and recall. The heightened effort may be revealed by the knitted brow, the expression of concentration, the slowness of the responses, and a tendency to look up or to look around when asked even relatively simple, straightforward questions. At this phase the patient is beginning to have significant difficulty in dealing with abstract concepts. One of the earliest expressions of this is the disturbance in time orientation, first in respect to the day of the week and later in respect to month and year. He shows increasing difficulty in retaining and grasping what is presented to him, so that he becomes less able to follow the directions of the doctors, nurses, or family. He gives up reading because he cannot comprehend or retain what he reads, and his conversation becomes increasingly limited, incoherent, and irrelevant.

Soon he manifests confusion in his orientation as to place, misidentifying his location, or identifying it correctly only by the most painstaking visual search to establish landmarks. He now may misidentify what he perceives, mistaking voices in the hall for those of familiar persons or pieces of furniture for people. His language becomes less coherent and understandable, and his capacity to maintain attention for any type of task progressively becomes diminished. Concurrently there tends to be progressive loss of motor control and of skills, with increasing difficulty in feeding, grooming, writing, etc. Incontinence of urine and feces, drooling, and spilling of food and water all are characteristic of more severe delirium. At first the patient may have the delusion that he is urinating or defecating in the proper place, as in a dream, and may feel guilty or ashamed later to discover that he had soiled his bed. As the condition deteriorates, however, the patient seems to lose all awareness of these social requirements, if he is aware of urinating or defecating at all.

In the final stages of delirium, before stupor or coma supervenes, the patient's speech usually becomes incoherent and muttering. He seems to be incapable of comprehending or responding to even the simplest questions, and he is totally disoriented for time and place. He may recognize only the most familiar persons in his life, often misidentifying strangers as members of the family. Picking, groping, grasping, and ataxic movements make their appearance. He becomes less and less responsive, lapsing into a stuporous state and finally into coma.

With each of these stages there is an increasing degree of slowing of the EEG. With the mildest degree of delirium the slowing may only be demonstrated by doing a frequency count, but as the disorder increases in severity the EEG becomes more obviously slow and irregular, reaching in progressive steps the very irregular and slow (1 to 3 per second) pattern of coma. Usually when the slowing has reached 5 to 6 per second or less, the characteristic disruption of the pattern by opening the eyes is no longer observed, a physiologic correlate of the reduced impact of externally derived perceptions.

This description in essence summarizes the sequence of changes in the level of consciousness characteristic of progressing delirium. Needless to say, the rate

(Continued)

and order in which such developments take place vary greatly from person to person. Thus one patient, following an overwhelming noxious stimulus, such as a severe head trauma, may become instantly comatose, and recovery may reverse the steps just described. In some patients the disturbance in consciousness may progress only to a mild or moderate degree, while in others it may fluctuate considerably, depending on variations in the severity of the underlying metabolic disturbance. In every case, however, the degree of slowing of the EEG correlates well with the degree of decrement in the level of consciousness as is manifest by the progressive changes described above.

This description adequately delineates the psychologic and behavioral changes that characterize all cases of delirium. It by no means, however, covers the full range of behavioral and psychologic aberration which may be noted in the course of delirium. These run the gamut of all varieties of psychopathology, and, in this respect, delirium may simulate any type of mental disorder, neurotic or psychotic. But, as will be discussed later, the presence of the disturbance in the level of awareness and the characteristic EEG changes provide the basis for differentiation of delirium from other psychopathologic entities. If we postulate that the effect of a cerebral metabolic derangement is to interfere with or damage mental processes or mechanisms in the reverse order in which they have been acquired (learned), then the polymorphous character of the clinical expressions of delirium become more understandable. In addition to interference with the systems whereby new and current stimuli are perceived and related to old experience, the systems that have to do with the recording in memory of such new experiences and their subsequent translation into appropriate thought and action are also affected. Further, the systems that have to do with the maintenance and focusing of attention seem to be particularly vulnerable. These are all functions of the ego, and one is justified in presuming that the ego is weakened as a consequence of this attack on its organic substrate.

This weakening of the ego is also manifest in the varieties of bizarre thoughts and fantasies which erupt into consciousness and in the primitive character of some of the defense mechanisms that may be used. With the more severe degrees of delirium, one sees a mental apparatus virtually stripped of all but the most primitive ego functions and barren of the social and cultural standards represented by the superego and the ego ideal. This means that, over and above the universal and characteristic disturbance in the level of awareness, the more personal and idiosyncratic characteristics of the behavior of the delirious patient will be determined by his own past development.

Accordingly, among delirious patients, we may expect to see varying degrees of anxiety, depending on the nature of past experience, the variety of ego defenses available, and the nature of support the patient receives from his environment. Some patients may experience a great deal of free-floating anxiety, with frightening thoughts, fantasies, and dreams and with all the behavioral and physiologic expressions of the anxiety reaction—tremor, sweating, tachycardia, and so on. Occasional patients succeed in overcoming this anxiety by denying or minimizing the extent of their intellectual defect, by withdrawing from or avoiding situations in which their defects may become manifest; by recourse

to sleep, or by confident dependence on the physician nurse, or family who will care for them. Patients with such successful ego defenses may reveal little manifest behavioral aberration and the fact of even a considerable degree of delirium may be inapparent unless the physician demonstrates by examination the reduction in the level of awareness. Many of these latter patients succeed in hiding their delirium by a façade of pleasantness, cooperativeness, or by parrying with humor or some aggression questions or tests which may expose their difficulties. Some patients with relatively well-developed and healthy ego function prior to illness may even lapse silently into coma with only a very attentive observer appreciating the development of delirium. At the other extreme are the more marginally compensated patients, psychologically speaking, in whom the metabolic derangement applies the coup de grace, so to speak; to the already weak ego. Among such patients one is more likely to see extreme degrees of anxiety and panic, with gross sense deceptions, hallucinations, and delusions of a psychotic character. Such patients are so grossly disturbed that they rarely escape the physician's attention, even though the diagnosis of delirium is not always correctly made. The chronic alcoholic seems particularly vulnerable to this type of development, perhaps as much the result of being the kind of person who becomes a chronic alcoholic as the result of the prolonged influence of alcohol or withdrawal from it. Such patients may injure themselves in panicky flights or may injure others in frantic attempts to ward off what they misinterpret as attacks.

Equally important are the varieties and expressions of depression in the delirious patient. A patient who has previously experienced depression or the patient whose earlier development renders him more vulnerable to depression may well respond to the perception of loss of his intellectual and mental functions with depression. Some patients may respond with excessive shame to their inability to maintain standards, especially in respect to soiling. And some patients may respond with guilt to the disturbing aggressive and or sexual impulses which surge up during the delirium. Some of these patients present serious suicide risks and it is sometimes only the severity to the underlying disease which renders them physically incapable to carrying out the suicide.

All varieties of neurotic and psychotic behavior may become manifest or accentuated in the course of delirium. In general, such developments constitute accentuations of previous tendencies which may have been latent up to this time.

An important clinical characteristic of delirium is the fluctuation in the manifestations. Many patients appear to be much more manifestly disturbed and delirious at some periods of the 24 hours than others. Careful examination, including EEG study, reveals that these fluctuations generally are not related to changes in the underlying metabolic disturbance, but rather to psychologic and environmental factors. Thus it is quite common for the delirious patient to appear more anxious and disturbed at night than during the day, a situation which finds its explanation in the fact that at night the patient has fewer sensory guides to help him in orientation. The darkness and the absence of familiar persons contribute to this. This accounts for the frequent report by doctors and nurses that a patient "suddenly became delirious" at night; most often the

(Continued)

patient already was delirious but did not manifest behavior that was recognized as disturbed until the additional stress of darkness brought forth more manifest anxiety and disturbed behavior.

THE EXAMINATION OF THE DELIRIOUS PATIENT

In the great majority of instances, the presence of delirium can be recognized and established in the course of taking the routine history. Once the physician becomes suspicious that there is a reduction in the level of consciousness, he can test this without the patient necessarily discovering that he is being so examined. For example, by repeating questions concerned with dates and names of places involved in the patient's illness, he may discover whether or not the patient is able to give consistent answers. By asking the patient to provide such factual data as are usually found on the front sheet of a hospital record or are easily ascertained from other sources, as the home address, telephone number, date of birth, date of marriage, ages and dates of birth of children, physician's name, address, and phone number, duration of residence, etc. it is usually relatively simple to demonstrate all but the milder degrees of disturbance in consciousness. The atmosphere of testing, which may be disturbing to some patients, may be eliminated if the physician simply frames his questions in terms of asking for information, even asking the patient for the date as if he, the physician, does not know it.

A number of relatively simple testing procedures are available if one wishes to establish the presence of a milder delirium or to follow serially the course of delirium. Perhaps the most useful is the serial subtraction of numbers, asking the patient to subtract 7 from 100 or 3 from 100 down to zero. Here one notes the speed, accuracy, the number and nature of the errors made and whether the patient perseverates or loses his place or simply errs in subtraction, or has to seek recourse to a concrete guide such as counting on his fingers. This procedure tests not only attention and concentration but also retention, memory, and the capacity to handle abstract concepts (numbers) without recourse to sensory guides. In evaluating this test, one must take into account the educational background and previous intellectual level of the patient. It cannot be used in patients who have not had at least a sixth grade education. Another useful procedure is to test the number of digits which the patient can retain and repeat forward or backward. The interpretation of familiar proverbs tests the capacity of the patient to deal with abstract concepts. With the more severe disturbances in consciousness, the patient may be unable to repeat the proverb accurately, much less interpret it. With somewhat less severe delirium, the patient may repeat the proverb in essentially the same terms, or give as its interpretation the concrete literal one. The increasing capacity to provide an abstract and generalized interpretation reflects improving function.

Other techniques of examination are described elsewhere.[36,38]

THE DIFFERENTIAL DIAGNOSIS OF DELIRIUM

The possibility of delirium should be considered in *any* patient who is seriously ill organically. Among such patients, however, it must be differentiated from

varieties of apathy, withdrawal, depression, and anxiety which may also accompany serious illness. These states, however, are not accompanied by the varieties of disturbances in the level of awareness which have just been described for delirium, and, if the patient's cooperation can be gained, the absence of such disturbances becomes clearly evident on examination. When in doubt, the issue can always be settled by an EEG, although it must be appreciated that a single, normal EEG does not necessarily rule our delirium since the premorbid record may have been faster. A repeat record after recovery will settle this.

Dementia.—The distinction between dementia and delirium is a somewhat arbitrary one, established by convention. Delirium has been defined as a reversible disturbance and dementia as an irreversible disturbance. Obviously there are many situations in which one cannot know whether or not the condition is reversible until after a period of observation. Thus if the underlying disorder, be it an infection, heart failure, or whatever, clears up and there is no significant improvement in either mental status or EEG, in all probability the condition was dementia and antedated the development of the more acute illness. More often, and especially in the older age group, we deal with patients with a pre-existing mild degree of dementia upon which is superimposed a further impairment in the course of the acute illness. In most instances, dementia develops either gradually over a matter of months or even years or very abruptly following some major cerebral insult, such as a head injury or a stroke from which recovery does not take place. Because the condition is more stable and chronic, there is more opportunity in the demented person for the development of compensatory psychologic devices, the presence of which are of some value in differentiating dementia from delirium. For example, the demented person is somewhat less likely to manifest the extremes of anxiety or panic noted in some delirious patients, although he may show considerable lability of expression of affect, with irritability, easy laughing and crying, and variations between expansive and depressive moods. The extent of the intellectual defect is often obscured by the use of denial, perseveration, and confabulation. Old memories, usually pleasant or successful in fact or in fantasy, are repeated endlessly as part of a pattern of recapturing the more successful past and denying the more restricted present. The attempts of the examiner to test current performance may be warded off by recourse to anecdotes and reminiscences. In general, the disturbance in retention, recent memory, and the capacity to use abstract concepts is less immediately obvious.

The EEG in dementia is less consistently slow as compared to that in delirium. Indeed a significant proportion of at least mildly to moderately demented patients have normal or borderline EEG's. This is probably explained by the fact that the abnormally slow potentials are arising from damaged neurons. In dementia we may presume that the neurons have died or at least are no longer effectively functioning. The electrical activity arising from the neurons still present may not deviate significantly from the normal.[39,40]

Depression.—Many depressed patients complain of difficulty in thinking and of memory loss. The general psychomotor retardation commonly accompanying depression may make it difficult to differentiate such manifestations from

(Continued)

those commonly seen in delirium. When in doubt the matter can be easily settled by an EEG, since depression is unassociated with any EEG change.

Schizophrenia.—As mentioned previously, the psychotic manifestations in delirium may occasionally simulate those of schizophrenia. Ordinarily in schizophrenia, tests for orientation and level of awareness will indicate no defect. However, if the patient is mute or uncooperative, an EEG will provide the differentiation, since diffuse slowing of the EEG is not a finding in schizophrenia.

Hysteria.—The hysterical psychosis (including the Ganser syndrome, pseudodementia, and prison psychosis) may simulate delirium.[41] In some cases the determinants for the choice of such manifestations were a true delirium in the past or identification with an important person who suffered from delirium. Clinical examination will usually readily distinguish hysterical psychosis from delirium. Generally the difficulties in memory, concentration, and other cognitive functions are either much too sweeping or are spotty. Thus a patient may manifest amnesia in many areas but excellent memory in others. Or the difficulty in intellectual performance may assume ludicrous proportions, such as being unable to answer correctly how many legs a three-legged stool has. Amnesia for one's own identity is almost always an hysterical phenomenon, practically never occurring during delirium. The severely delirious persons may be unable to give his name, but he is not likely to complain of this fact. In any event, the diagnosis is again readily settle by the EEG, which will reveal no abnormality in hysteria.

Excitements.—Manic and schizophrenic excitements may present considerable difficulty in diagnosis and differentiation from delirium, especially since excited persons are likely to be overmedicated, producing a superimposed delirium. The problem is usually clarified after a brief period of clinical observation.

Drugs.—A variety of drugs such as, mescaline, lysergic acid, quinacrine, and many others may produce acute psychologic disturbances which are different from delirium. In general these materials produce their effects probably by specific affinity for certain parts of the nervous system or even components of the neurons. They do not produce reductions in the level of consciousness, as we have defined this clinically, but rather certain types of heightened perception—sometimes with hyperalertness and sometimes with states of withdrawal. They are more likely to lead to excited states or states with vivid, complex visual or auditory hallucinations. While the patient may be preoccupied with such experiences, if one can secure the patient's attention, it will be found that he has no significant defect in cognitive function. In contrast to the delirious patient, he may have very vivid memory of these experiences after the disorder is over. It is perhaps significant that the EEG during such states does not show diffuse slowing as in delirium, but rather either diffuse acceleration (quinacrine),[32] sleep patterns in an alert state (atropine),[42] or focal spikes or paroxysmal slow activity (mescaline).[43] There are some grounds to suspect that delirium tremens, the more or less distinctive syndrome noted in alcoholics (which may be part of a withdrawal syndrome), has more in common with these states than with delirium, or at least is a mixture of the two. The basis for this suggestion is the prominence and consistency of hyperalertness, hypervigilance, and hallucinations in delirium

tremens, the clarity of the patient's memory of such experiences after recovery, and the relatively minor character of the EEG changes. Rather than showing diffuse slow activity, most patients with delirium tremens show low voltage fast or moderate voltage fast activity.[44] We believe it was their emphasis on alcoholic patients which misled Victor and Adams[3] to state that the EEG in delirium is usually normal.

Sensory Deprivation.—Experimentally, reduction in sensory input provokes certain psychologic disturbances of psychotic proportions even in some healthy individuals.[45] Hallucinations and distortions of the body image are particularly prominent and may provoke severe anxiety. It is now becoming evident that some psychotic episodes occurring among patients in the respirator and with bandaged eyes are of this origin rather than due to cerebral insufficiency. Obviously both factors may operate. In any event, the EEG again will provide the differential since slowing does not occur as a part of the sensory deprivation syndrome.

SUMMARY

The thesis is presented that a derangement in the general functional metabolism of the brain underlies all instances of delirium and that this is reflected at the clinical level by a characteristic disturbance in cognitive functions and at the physiologic level by a characteristic generalized slowing of the electroencephalogram. As background for this thesis are summarized the studies of a large number of patients exhibiting delirium in the setting of a wide variety of physiologic and biochemical derangements as well as instances of delirium experimentally induced by techniques known to affect cerebral metabolism adversely.

The clinical characteristics of delirium are carefully delineated and the basis established for the identification of delirium and its differentiation from other types of psychologic disturbance commonly seen in organically ill patients.

REFERENCES

1. Noyes, A. P., and Kolb, L. C.: Modern Clinical Psychiatry, ed. 5, Philadelphia, 1958, W. B. Saunders Company.
2. Ewalt, J. R., Strecker, E. A., and Ebaugh, F. G.: Practical Clinical Psychiatry, ed. 8, New York, 1957, McGraw-Hill Book Co., Inc.
3. Victor, M., and Adams, R. D.: Delirium, *in* Harrison, T.: Principles of Internal Medicine, ed. 2, New York, 1954, McGraw-Hill Book Co., Inc.
4. Engel, G. L.: Research Possibilities in the Dementias, Am. J. Psychiat. **106**:146, 1949.
5. Bedford, P. D.: Adverse Cerebral Effects of Anesthesia on Old People, Lancet **2**:259, 1955.
6. Bedford, P. D.: Cerebral Damage From Shock Due to Disease in Old People, Lancet **2**:505, 1957.
7. Engel, G. L.: Fainting: Physiologic and Psychologic Considerations, Springfield, Ill., 1950, Charles C Thomas, Publisher.
8. Waelsch, H. (ed.): Biochemistry of the Developing Nervous System, New York, 1955, Academic Press, Inc.
9. Richter, D. (ed.): Metabolism of the Nervous System, New York, 1957, Pergammon Press.
10. Fazekas, J. F.: Pathologic Physiology of Cerebral Dysfunction, Am. J. Med. **25**:89, 1958.
11. Lowry, O. H.: Enzyme Concentrations in Individual Nerve Cell Bodies, *in* Richter, D. (ed.): Metabolism of the Nervous System, New York, 1957, Pergammon Press, p. 323.
12. Shapot, V. S.: Brain Metabolism in Relation to the Functional State of the Central Nervous System, ibid., p. 257.
13. Waelsch, H.: Metabolism of Proteins and Amino Acids, ibid., p. 431.
14. Richter, D., and Gaitonde, M. K.: The Metabolism of ^{35}S-methionine in the Brain, ibid., p. 449.

(Continued)

15. Strecker, H. J.: Glutamic Acid and Glutamine, ibid., p. 459.
16. Sporn, M., Dingman, W. and Defalco, A.: A Method for Studying Metabolic Pathways in the Brain of the Intact Animal. I. The Conversion of Proline to Other Amino Acids, J. Neurochem. In press.
17. Weil-Malherbe, H.: Quoted by Strecker, H. J., [15] p. 463.
18. Geiger, A.: Chemical Changes Accompanying Activity of the Brain, *in* Richter, D. (ed.): Metabolism of the Nervous System, New York, 1957, Pergammon Press, p. 245.
19. Kety, S. S.: General Metabolism of the Brain in Vivo, ibid., p. 221.
20. Kety, S. S.: Circulation and Metabolism of the Human Brain in Health and Disease, Am. J. Med. 8:205, 1950.
21. Scheinberg, P.: Cerebral Blood Flow and Metabolism in Pernicious Anemia, Blood 6:213, 1951.
22. Scheinberg, P., Stead, E. A., Brannon, E. S., and Warren, J. V.: Correlative Observations on Cerebral Metabolism and Cardiac Output in Myxedema, J. Clin. Invest. 29:1139, 1950.
23. Libet, B., and Gerard, R. W.: Control of the Potential Rhythm of Isolated Frog Brain, J. Neurophysiol. 2:153, 1939.
24. Meyer, J. S., Faug, H. C., and Denny-Brown, D.: Polarographic Study of Cerebral Collateral Circulation, A.M.A. Arch. Neurol. & Psychiat. 72:296, 1954.
25. Gibbs, F. A., Williams, D., and Gibbs, E. L.: Modification of the Cortical Frequency Spectrum by Changes in Carbon Dioxide, Blood Sugar, and Oxygen, J. Neurophysiol. 3:49, 1940.
26. Engel, G. L., Romano, J., Ferris, E. B., Webb, J. P., and Stevens, C. D.: A Simple Method of Determining Frequency Spectra in the Electroencephalogram: Observations on Physiological Variations in Glucose, Oxygen, Posture, and Acid-Base Balance on the Normal Electroencephalogram, Arch. Neurol. & Psychiat. 51:133, 1944.
27. Engel, G. L., Webb, J. P., and Ferris, E. B.: Quantitative Electroencephalographic Studies of Anoxia in Humans; Comparison With Acute Alcoholic Intoxication and Hypoglycemia, J. Clin. Invest. 24:691, 1945.
28. Engel, G. L., and Rosenbaum, M.: Delirium, III. Electroencephalographic Changes Associated With Actual Alcoholic Intoxication, Arch. Neurol. & Psychiat. 53:44, 1945.
29. Ryder, H. W., Engel, G. L., Stevens, C. D., and Ferris, E. B.: The Effect of Carbon Monoxide Hemoglobinemia of Approximately 20 Per Cent on the Cerebral Blood Flow, Gas Exchange, and Electroencephalogram in Human Beings, National Research Council, CMR, Committee on Aviation Medicine, Report #25, Nov. 10, 1942.
30. Engel, G. L., Romano, J., Ferris, E. B., and Kehoe, R.: Unpublished data.
31. Friedman, S. B., and Engel, G. L.: Effect of Cortisone and Adrenocorticotropin on the EEG of Normal Adults: Quantitative Frequency Analysis, J. Clin. Endocrinol. & Metab. 16:839, 1956.
32. Engel, G. L., Romano, J., and Ferris, E. B.: Effect of Quinacrine (Atabrine) on the Central Nervous System: Clinical and Electroencephalographic Studies, Arch. Neurol. & Psychiat. 58:337, 1947.
33. Engel, G. L., Romano, J., and Goldman, L.: Delirium. IV. Quantitative Electroencephalographic Study of a Case of Acute Arsenical Encephalography, Arch. Neurol. & Psychiat. 56:659, 1946.
34. Samson, D. C., Swisher, S., Christian, R. H., and Engel, G. L.: Some Observations on the Mechanism of Delirium in Pernicious Anemia, J. Clin. Invest. 30:669, 1951.
35. Browning, T. B., Atkins, R. W., and Weiner, H.: Cerebral Metabolic Disturbances in Hypothyroidism. Clinical and EEG Studies of the Psychoses of Myxedema and Hypothyroidism, A.M.A. Arch. Int. Med. 93:938, 1954.
36. Romano, J., and Engel, G. L.: Studies of Delirium. I. Electroencephalographic Data, Arch. Neurol. & Psychiat. 51:356, 1944.
37. Engel, G. L., and Romano, J.: Studies of Delirium. II. Reversibility of the Electroencephalogram With Experimental Procedures, Arch. Neurol. & Psychiat. 51:378, 1944.
38. Romano, J., and Engel, G. L.: Psychologic and Physiologic Considerations of Delirium, M. Clin. North America, 28:629, 1944.
39. Arnold, W., Friedman, S., and Engel, G. L.: The EEG in Delirium and Dementia. Unpublished.
40. Weiner, H., and Schuster, D. B.: The EEG in Dementia. Some Preliminary Observations and Correlations, EEG Clin. Neurophysiol. 8:479, 1956.
41. Weiner, H., and Braiman, A.: The Ganser Syndrome: A Review and Addition of Some Unusual Cases, Am. J. Psychiat. 3:767, 1955.
42. Funderburk, W. H., and Case, T. J.: The Effect of Atropine on Cortical Potentials, EEG Clin. Neurophysiol. 3:213, 1951.
43. Wikler, A.: Clinical and EEG Studies on the Effects of Mescaline, N-Allylnormorphine and Morphine in Man, J. Nerv. & Ment. Dis. 120:157, 1954.
44. Engel, G. L.: Unpublished observations.
45. Solomon, P., Leiderman, P. H., Mendelson, J., and Wexler, D.: Sensory Deprivation: A Review, Am. J. Psychiat. 114:357, 1957

8
Falls

Lesley S. Carson, MD

Reproduced paper following commentary: Tinetti ME, Inouye SK, Gill TM, Doucette JT. Shared Risk Factors for Falls, Incontinence, and Functional Dependence: Unifying the Approach to Geriatric Syndromes. JAMA;273:1348–1353.

Commentary

Geriatrics is a discipline characterized by the study of a spectrum of functions determined by interrelationships between environmental and physiologic factors. "Falls" is an entity considered a geriatric syndrome, as it is not only common in the older age group, but it also has no single, well-defined pathophysiologic mechanism.

In the article reproduced here, Tinetti and coworkers identify a set of impairments that predispose patients to incontinence and falling as geriatric syndromes and, furthermore, try to define whether incontinence and falling per se contribute to functional dependence and vice versa. That is, they discuss whether functional dependence contributes to falling and incontinence. The clarification of these relationships could lead to interventions that target more than one syndrome and promote independent function more effectively and efficiently.

A community sample of 1103 persons was given face-to-face interviews at time zero; 84% of these individuals were then interviewed again a year later. Cognitive, emotive, and physical impairments were quantified; medications (prescription and nonprescription) and substance use were identified; chronic health conditions were elicited; and a falls calendar was kept. Slow timed chair stands (lower extremity impairment), decreased arm strength (upper extremity impairment), vision and hearing decreases (sensory impairment), and anxiety/depression (affective impairment) were the four factors identified as independent risk factors for incontinence, falls, and functional dependence. As the number of risk factors increased the relative risk of falls rose from 1.0 with zero risk factors to 5.7 with more than or equal to three risk factors. Functional dependence, which seemed to be significantly related to incontinence, was less strongly related to falls.

This type of information paved the way toward such approaches as the use of Tai Chi to abort falls (1). The identification of orthostatic hypotension as a contributing factor to recurrent falls is another modifiable variable (2). The data that falls and injuries from falls lead to nursing home placement and increased functional dependence emphasize the accompanying cost in dollars and diminished quality of life (3). Physical restraints, once common in nursing homes to prevent falls, are now used as a last resort given the demonstrated increased serious injury rate (4). There is also a suggestion that hip protectors might have a role in certain settings to minimize the occurrence of hip fractures from falls and subsequent functional dependence (5).

The significance of a fall still goes underappreciated, and the etiology is usually difficult to define. Healthcare providers cannot ignore a fall and claim to promote independence and quality of life. Further instruction in the subtleties and existence of this syndrome is essential, and landmark papers by Tinetti and others have offered an approach to recognizing and understanding the complex etiologies involved as well as potential interventions.

References

1. Wolf SL, Barnhart HX, Kutner NG, McNeely E, Coogler C, Xu T. Reducing Frailty and Falls in Older Persons: An Investigation of Tai Chi and Computerized Balance Training.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

Atlanta FICSIT Group. Frailty and Injuries: Cooperative Studies of Intervention Techniques. J Am Geriatr Soc 1996;44: 489–497.
2. Ooi WL, Hossain M, Lipsitz LA. The Association Between Orthostatic Hypotension and Recurrent Falls in Nursing Home Residents. Am J Med 2000;08:106–111.
3. Tinetti ME, Williams CS. Falls, Injuries Due to Falls, and the Risk of Admission to a Nursing Home. New Engl J Med 1997;337:1279–1284.
4. Capezuti E, Maislin G, Strumpf N, Evans LK. Side Rail Use and Bed-Related Fall Outcomes Among Nursing Home Residents. J Am Geriatr Soc 2002;50:90–96.
5. Kannus P, Parkkari J, Niemi S, et al. Prevention of Hip Fractures in Elderly People with Use of a Hip Protector. New Engl J Med 2000;343:1506–1513.

Shared Risk Factors for Falls, Incontinence, and Functional Dependence

Unifying the Approach to Geriatric Syndromes

Mary E. Tinetti, MD; Sharon K. Inouye, MD, MPH; Thomas M. Gill, MD; John T. Doucette, PhD

Objective.—To determine whether a set of factors representing impairments in multiple areas could be identified that predisposes to falling, incontinence, and functional dependence.

Design.—Population-based cohort with a 1-year follow-up.

Setting.—General community.

Participants.—A total of 927 New Haven, Conn, residents, aged 72 years and older who completed the baseline and 1-year interviews.

Main Outcome Measures.—At least one episode of urinary incontinence per week, at least two falls during the follow-up year, and dependence on human help for one or more basic activities of daily living.

Results.—At 1 year, urinary incontinence was reported by 16%, at least two falls by 10%, and functional dependence by 20% of participants. The four independent predisposing factors for the outcomes of incontinence, falling, and functional dependence included slow timed chair stands (lower extremity impairment), decreased arm strength (upper extremity impairment), decreased vision and hearing (sensory impairment), and either a high anxiety or depression score (affective impairment). There was a significant increase in each of incontinence, falling, and functional dependence as the number of these predisposing factors increased. For example, the proportion of participants experiencing functional dependence doubled (7% to 14% to 28% to 60%) (χ^2=119.8; P<.001) as the number of predisposing factors increased from zero to one to two to at least three.

Conclusions.—Our findings suggest that predisposition to geriatric syndromes and functional dependence may result when impairments in multiple domains compromise compensatory ability. It may be possible to restore compensatory ability and prevent or delay the onset of several geriatric syndromes and, perhaps, functional dependence by modifying a shared set of predisposing factors. Perhaps it is time to take a more unified approach to the geriatric syndromes and functional dependence.

(JAMA. 1995;273:1348-1353)

FALLING, urinary incontinence, and delirium are examples of health conditions often referred to as geriatric syndromes. Geriatric syndromes are experienced by older—particularly frail—persons, occur intermittently rather than either continuously or as single episodes, may be triggered by acute insults, and often are linked to subsequent functional decline.[1] To date, the individual geriatric syndromes have been addressed in both the research and clinical settings as separate entities. Consider, for example, falling and urinary incontinence, two of the more thoroughly studied geriatric syndromes. Until recently, both entities were thought to result solely from distinct anatomic and/or physiologic abnormalities within discrete organ systems.[2,3] While progressing our understanding, these anatomic and physiologic classification schemes fail to completely explain the occurrence of these two clinical syndromes. Many elderly persons with the presumed physiologic and/or anatomic lesions do not manifest falls or incontinence, while, conversely, many fallers and incontinent individuals do not possess readily identifiable lesions within the neuromuscular or genitourinary systems, respectively.

For editorial comment see p 1381.

Falling and incontinence, along with other geriatric syndromes such as delirium, are believed to often result from the accumulated effect of impairments in multiple domains.[4-6] Individuals maintain postural stability and urinary continence, for example, because of complex networks of sensory, motor, and central integrative systems.[3,7] Each of these systems, in turn, has inputs from other systems and each has many levels of organization. This complexity and redundancy result in a large reserve capacity and a wide repertoire of responses, thus allowing the individual to respond appropriately to challenges and insults.[8] Redundancy also enables the individual to compensate for impairments. However, as multiple impairments accumulate, this redundancy and thus compensatory capability are reduced, making the individual vulnerable to additional insults or challenges.[4,9] This increased vulnerability resulting from impairments in multiple systems defines frailty, thus explaining why the subset of frail elderly persons are at particular risk of experiencing geriatric syndromes.

This postulated relationship between chronic impairments and geriatric syndromes is supported by epidemiologic evidence. Both falling and delirium, for example, have been shown to increase in frequency with the number of predisposing impairments possessed.[4,6,10-12] Furthermore, investigators have identified many of the same predisposing factors for different geriatric syndromes. Cognitive impairment, impaired gait and

From the Departments of Internal Medicine (Drs Tinetti, Inouye, and Gill) and Epidemiology and Public Health (Dr Doucette), Yale University School of Medicine, New Haven, Conn.

Reprint requests to Department of Internal Medicine, Yale University School of Medicine, 333 Cedar St, PO Box 208025, New Haven, CT 06520-8025 (Dr Tinetti).

(Continued)

mobility, depression, sedative-hypnotic use, and the use of multiple medications have all been identified in separate studies as predisposing factors for falling, incontinence, and delirium.[4-6,9-13]

The geriatric syndromes, in turn, are closely linked to the development of functional dependency,[14,15] an important adverse outcome among frail older persons. The relationship between the geriatric syndromes and functional dependence is likely to be both direct—falling, delirium, or incontinence may, for example, contribute to the development of functional dependence—and indirect—many of the shared predisposing factors just noted for falls, delirium, and incontinence are also risk factors for functional dependence. These relationships suggest the possibility of a common predisposition to geriatric syndromes and functional dependence resulting from the accumulated effect of impairments in multiple domains.

The purpose of this study, using data from a large cohort of community-living elderly persons, was to provide empiric evidence of the relationships among predisposing impairments, geriatric syndromes, and functional dependency. The specific aims were to (1) determine whether a set of predisposing factors, representing impairments in multiple areas, could be identified that were associated with both falling and urinary incontinence; (2) determine whether this "shared set of predisposing factors" for falling and incontinence also identified persons at risk of functional dependence; and (3) determine whether falling and incontinence were additional independent risk factors for functional dependence. Falling and incontinence were chosen for study because they represent the two most common geriatric syndromes among elderly persons in a community, rather than hospital or institutional, setting.

METHODS

Subjects

The participants were members of a previously described probability sample of community-living persons older than 71 years.[15] Among the 1391 persons who met eligibility criteria, 1103 (79%) agreed to participate, were enrolled, and completed the baseline face-to-face interview. Enrollment occurred between October 1989 and August 1990. Of these individuals, 927 (84%) completed the 1-year face-to-face interview. Reasons for noncompletion of the 1-year interview included refusal (n=110), death (n=59), and other reasons (n=7). There were no significant differences between persons who refused the 1-year interview and those who participated in the interview with respect to falling, incontinence, or functional dependence at baseline. Persons who died were more likely than persons who completed the 1-year interview to have had functional dependence at baseline (29% vs 15%) and to have fallen at least twice in the previous year (22% vs 17%), but were slightly less likely to have reported incontinence (6% vs 14%).

Descriptive Data

Sociodemographic data were ascertained during the baseline interview in participants' homes. Urinary incontinence and functional dependence present at the baseline interview were ascertained using the same questions at the 1-year interview. The number of falls experienced in the previous year was assessed. Participants were asked, in addition to standard questions concerning self-perceived health, whether a physician had ever told them they had any of the following: myocardial infarction, stroke, Parkinson's disease, cancer, arthritis, or diabetes mellitus. These chronic conditions were not considered potential predisposing factors as they likely affect functioning through the impairments described herein.

Potential Predisposing Factors

Candidate predisposing factors, representing chronic impairments from the cognitive, affective, upper and lower extremity physical performance, sensory, and nutritional domains, were ascertained during the baseline interview. Medications also were included as potential predisposing factors. Cognitive status was ascertained using the Folstein Mini-Mental State Examination (MMSE),[16] depressive symptoms were ascertained using the Center for Epidemiologic Studies-Depression (CES-D) test,[17] and anxiety trait was ascertained using the Spielberger State Trait Anxiety Inventory (STAI).[18] Physical performance skills were assessed through a series of qualitative and timed tests.[15,19] Shoulder abduction, grip strength, hip flexion, and knee flexion and extension were tested manually with the participant seated and were graded as normal (full range of motion against full resistance) or less than normal. Total arm and leg strength scores were the combination of the appropriate bilateral tests. The balance maneuvers included side-by-side, sternal nudge, tandem, and one-leg stands, while the gait maneuvers included path deviation, turning, step continuity, and step symmetry. These maneuvers were combined into a balance and gait score, which ranged from 0 to 22.[19] The timed physical performance testing included the time required to tap the foot back and forth 10 times between side-by-side circles (foot taps); get up from and sit down in a chair three times in a row (timed chair stands); turn a full circle; walk 10 feet, turn around, and walk back first at the usual pace, then at a rapid pace; and pick up a pencil, complete a signature, and put the pencil back down on the table. Corrected near visual acuity was assessed with the Rosenbaum card, and percentage of visual impairment was calculated.[20] Hearing was assessed by the Whisper test.[21] Body mass index (BMI) was calculated as the self-reported weight in kilograms divided by the square of height in meters. Alcohol consumption was assessed using a standard questionnaire.[22] Interviewers recorded the participants' medications directly from the bottles and containers. Both prescription and nonprescription medications were ascertained. The Iowa coding and categorization system for medications was used to classify types of medications.[23]

Incontinence, Falling, and Functional Outcome Data

The presence of urinary incontinence was ascertained during the 1-year interview, conducted in participants' homes, by response to the following question: "In a typical week, how often would you say you lose control of urine and wet yourself?" Responses were categorized as never vs one or more times per week. The occurrence and frequency of falls were ascertained throughout the study year by a "fall calendar," described in detail elsewhere.[15] For purposes of this study, fallers were defined, as in previous investigations,[10] as persons reporting at least two falls during the year. Functional dependence was defined as self-report of need for human help with one of the following basic activities of daily living: eating, grooming, bathing, dressing, transferring from bed to chair, and walking around the house.[24]

Analysis

The first step in analysis entailed identifying the bivariate association between the candidate predisposing factors and the outcomes of incontinence, falling, and functional dependence. To facilitate clinical interpretation and allow for the determination of relative risks (RRs), all categorical and continuous variables were dichotomized. Variables were dichotomized at the clinically acceptable cutoff point when one existed (eg, CES-D score ≥16[17]; Spielberger STAI >32[18]; and balance and gait score >12/22—the cutoff point used in previous analyses of this data set[19]). For continuous variables, if no relevant cutoff point existed, we looked at risk gradients within both deciles and quartiles.[25] Participants were dichotomized at the worst vs other quartiles unless the worst vs other deciles created better risk gradients (eg, Folstein MMSE

Table 1.—Sociodemographic Characteristics and Predisposing Factors Associated With Falling, Incontinence, and Functional Dependence Among Community-Living Older Persons

Factor	Prevalence, %	RR (95% CI)* Incontinence	Falling†	Functional Dependence
Sociodemographic characteristics				
Age ≥80 y	46	1.5 (1.2-2.0)	1.5 (1.0-2.1)	2.2 (1.7-2.9)
Female	73	1.7 (1.1-2.5)	1.1 (0.7-1.7)	1.4 (1.0-2.0)
African American	14	1.2 (0.9-1.9)	1.2 (0.7-2.0)	1.3 (0.9-1.8)
Education <12 y	61	1.5 (1.0-2.3)	0.7 (0.5-1.0)	1.2 (0.9-1.6)
Income <$7000/y	37	1.2 (0.8-1.7)	1.3 (0.8-2.0)	1.4 (1.0-1.9)
Health-related/chronic diseases				
Fair or poor self-rated health	44	1.5 (1.1-2.1)	1.8 (1.2-2.6)	2.0 (1.5-2.6)
Chronic dizziness	29	1.3 (0.9-1.8)	1.7 (1.1-2.5)	1.4 (1.0-1.8)
≥2 Chronic conditions	41	1.5 (1.1-2.0)	1.9 (1.3-2.8)	1.7 (1.3-2.2)
Diabetes	15	1.2 (0.8-1.8)	1.2 (0.7-2.0)	1.6 (1.2-2.2)
Past stroke	6	1.6 (0.9-2.6)	1.4 (0.7-2.8)	2.1 (1.5-3.0)
Arthritis	52	1.3 (1.0-1.8)	1.4 (1.0-2.1)	0.8 (0.5-1.1)
Predisposing factors				
Cognitive				
Folstein Mini-Mental State Examination score <20	11	1.4 (1.0-2.2)	2.6 (1.7-4.0)	3.2 (2.5-4.0)
Affective				
Center for Epidemiologic Studies–Depression score ≥16	22	1.4 (1.0-2.0)	1.6 (1.0-2.6)	1.5 (1.1-2.0)
Spielberger State Trait Anxiety Inventory ≥32	49	1.7 (1.2-2.4)	1.4 (1.0-2.2)	1.5 (1.1-2.1)
Physical performance				
Lower extremity				
Balance and gait score <12/22	41	1.9 (1.4-2.6)	3.0 (1.9-4.6)	2.9 (2.2-4.0)
Usual gait speed <0.42 m/s	25	2.0 (1.5-2.7)	2.2 (1.5-3.2)	5.0 (3.8-6.5)
Rapid gait speed <0.57 m/s	25	2.3 (1.7-3.2)	2.7 (1.8-4.1)	4.7 (3.4-6.3)
10 Foot taps >6.6 s	26	1.6 (1.2-2.2)	2.7 (1.9-4.0)	2.7 (2.1-3.5)
Three chair stands >10 s	24	2.0 (1.5-2.7)	2.9 (2.0-4.2)	4.3 (3.4-5.6)
Upper extremity				
Signature >13.5 s	25	1.4 (1.0-1.9)	1.7 (1.1-2.5)	2.4 (1.9-3.2)
Arm strength impairment	25	1.5 (1.0-2.0)	2.2 (1.5-3.2)	2.5 (2.0-3.2)
Sensory				
Vision >50% Impaired	36	1.5 (1.1-2.0)	1.6 (1.1-2.4)	2.1 (1.6-2.7)
>5 Whispered words missed	23	1.5 (1.1-2.0)	1.3 (0.9-2.0)	1.6 (1.2-2.1)
Vision and hearing impaired	11	2.0 (1.4-2.8)	1.8 (1.1-2.9)	2.2 (1.6-2.9)
Nutritional				
Body mass index worst quartiles	50	1.5 (1.1-2.0)	1.0 (0.7-1.5)	1.6 (1.2-2.2)
Medications/ingestions				
Any alcohol use	62	1.1 (0.8-1.5)	1.1 (0.9-1.4)	1.9 (1.4-2.7)
≥5 Medications	35	1.3 (1.0-1.8)	1.3 (1.1-1.6)	1.6 (1.3-2.1)
Nitrates	12	1.7 (1.1-2.4)	1.3 (1.0-1.7)	1.2 (0.9-1.7)
Any antihypertensive	38	0.9 (0.6-1.2)	1.2 (1.0-1.4)	1.1 (0.9-1.5)
Any psychotropic	12	1.4 (0.9-2.0)	1.4 (1.1-1.8)	1.3 (0.9-1.8)
Loop diuretic	14	1.4 (0.9-2.0)	1.1 (0.7-1.9)	1.8 (1.3-2.4)
Thiazide diuretic	24	1.1 (0.8-1.5)	0.5 (0.3-0.8)	1.0 (0.7-1.3)
Insulin	4	1.4 (0.8-2.6)	2.2 (1.2-4.1)	2.1 (1.4-3.1)
β-Blocker	17	0.8 (0.5-1.3)	0.4 (0.2-0.9)	0.7 (0.5-1.0)
Other geriatric syndromes				
Baseline urinary incontinence	14	...	1.9 (1.2-2.9)	2.4 (1.8-3.1)
At least two falls in previous year	17	1.8 (1.2-2.2)	...	1.6 (1.2-2.2)
Functional dependence at baseline	15	1.9 (1.4-2.9)	2.0 (1.3-3.1)	...

*Unadjusted relative risks (RRs) and 95% confidence intervals (CIs).
†Falling was defined as two or more falls during the follow-up year.

score <20). The only exception to this decision was BMI, which showed a U-shaped relationship with each of falling, incontinence, and functional dependence. Therefore, for this variable, the highest and lowest quartiles were combined and compared with the middle two quartiles of participants. Unadjusted RRs of falling, incontinence, and functional dependence were determined for each candidate predisposing factor.

The predisposing factors were grouped into conceptual domains using clinical judgment, as shown in Table 1. If, within a conceptual domain, one factor clearly displayed the strongest RR for each outcome of falling, incontinence, and functional dependence, that variable was selected for multivariable modeling. If no best candidate existed within a domain, individual factors were either combined into a single factor if this combination made clinical sense (eg, vision and hearing impairment were combined into the sensory impairment factor) or entered into a stepwise logistic model to select the factor with the best combination of adjusted RRs for each of the three outcomes of falling, incontinence, and functional dependence. The factors from the relevant domains were selected using both backward and forward stepwise selection procedures. The same factors were identified by both modeling techniques.

The candidate factors from each domain selected as described herein were entered into three preliminary models, one for each of the three outcomes. Adjusted RRs were estimated, through binomial models, using generalized linear interactive modeling as presented by Wacholder[26] and modified by Risch et al.[27] Factors significant at P less than .10 in each of the three models were retained. To determine if either incontinence or falling was associated with functional dependence independent of the predisposing factors, each geriatric syndrome was added to the final model for functional dependence. We also examined the risk of experiencing each outcome of incontinence, falling, and functional dependence according to the number of predisposing factors possessed. Finally, we reran the three multivariable models, including only the subset of participants who did not report the relevant condition at the baseline interview, to determine whether the final set of predisposing factors was associated with both incident and prevalent cases.

RESULTS

The mean age of the 927 participants who completed the 1-year face-to-face interview was 79.7 years (SD, 5.2 years), with a range of 71.8 to 99.9 years. At the time of the 1-year interview, 22% of subjects were married and 68% lived alone. Participants experienced a mean of 1.4 (SD, 1.0) self-reported chronic conditions, took an average of 3.8 (SD, 2.6) medications, and scored a mean of 24.9 (SD, 3.9) on the Folstein MMSE. Among the 927 subjects, 188 (20%) reported functional dependence at 1 year, including 95 who had not and 93 who had reported dependence at the baseline interview. During the year of follow-up, 96 subjects (10%) reported the occurrence of two or more falls, the fall-related outcome used in the current study. Fifty-six of these subjects had not reported the occurrence of two or more falls in the year before the study, while 39 subjects had. Urinary incontinence was reported by 146 subjects (16%)

(Continued)

at the 1-year interview, 82 of whom had not reported incontinence at baseline.

Most of the factors listed in Table 1 were associated, at least marginally, with at least one of the three outcomes, while many of the factors were related to all three. The variables in the lower extremity physical performance domain showed the strongest relationship with each of incontinence, falling, and functional dependence. Usual and rapid gait speed, balance and gait score, and timed chair stands appeared to be the strongest risk factors for each of the three outcomes. Because these three factors were highly correlated, timed chair stands was selected to represent lower extremity performance in the multivariable models as it performed the best overall.

As can be seen in Table 1, only a few of the individual medications showed a significant relationship with the two geriatric syndromes or functional dependence. Nitrates was the only category of medications associated with incontinence; psychotropic medications and insulin were associated with falling; and loop diuretics and insulin were associated with functional dependence. The use of five or more medications was at least marginally associated with each of the geriatric syndromes and functional dependence.

Identification of Shared Predisposing Risk Factors

Candidate factors for the initial models were selected following the criteria described in the "Methods" section. The candidate factors included Folstein MMSE score in the worst decile, which corresponded to a score of less than 20 (cognitive domain), timed chair stands in the worse quartile (lower extremity physical performance domain), arm strength impairment (upper extremity physical performance domain), vision and hearing impairment (sensory domain), high or low BMI (nutritional status domain), and use of at least five prescription medications (medication domain). Since depression and anxiety performed similarly in bivariate analyses, both factors from the affective domain were entered into separate models to determine which showed the strongest independent relationship with each outcome of incontinence, falling, and functional dependence.

The four predisposing factors remaining in the final models, as shown in Table 2, included timed chair stands, vision and hearing impairment, arm strength impairment, and presence of anxiety. Timed chair stands showed the strongest relationship with each of incontinence, falling, and dependence, while vision and hearing impairment was associated strongly with incontinence and was marginally significant for falling and functional dependence. Conversely, arm strength was significant for falling and functional dependence and marginal for incontinence. Anxiety performed somewhat better than depression and was thus selected for the final models (Table 2). In the model substituting depression for anxiety, the adjusted RRs associated with depression were 1.3 (95% confidence interval [CI], 0.9 to 1.7), 1.5 (95% CI, 1.0 to 2.3), and 1.3 (95% CI, 1.0 to 1.7) for incontinence, falling, and functional dependence, respectively. The risk estimates for the other three predisposing factors were similar regardless of whether depression or anxiety was included as the affective factor.

Table 2.—Shared Set of Predisposing Factors for Incontinence, Falling, and Functional Dependence

	Adjusted RR (95% CI)*				
			Functional Dependence†		
Factor	Incontinence	Falling	Model 1	Model 2	Model 3
Chair stands (lower extremity)	1.7 (1.3-2.4)	2.6 (1.7-3.9)	3.6 (2.7-4.8)	3.3 (2.5-4.4)	3.6 (2.7-4.7)
Arm strength (upper extremity)	1.2 (0.8-1.6)	1.6 (1.1-2.5)	1.7 (1.3-2.2)	1.6 (1.3-2.0)	1.6 (1.3-2.1)
Vision and hearing (sensory)	1.6 (1.1-2.3)	1.4 (0.8-2.2)	1.3 (0.9-1.8)	1.3 (1.1-1.6)	1.2 (0.9-1.6)
Anxiety (affective)	1.6 (1.1-2.2)	1.4 (0.9-2.1)	1.3 (1.0-1.7)	1.3 (1.0-1.8)	1.3 (1.0-1.8)
Incontinence	...	...	...	1.4 (1.1-1.7)	...
Falling	...	...	...	...	1.2 (0.9-1.5)

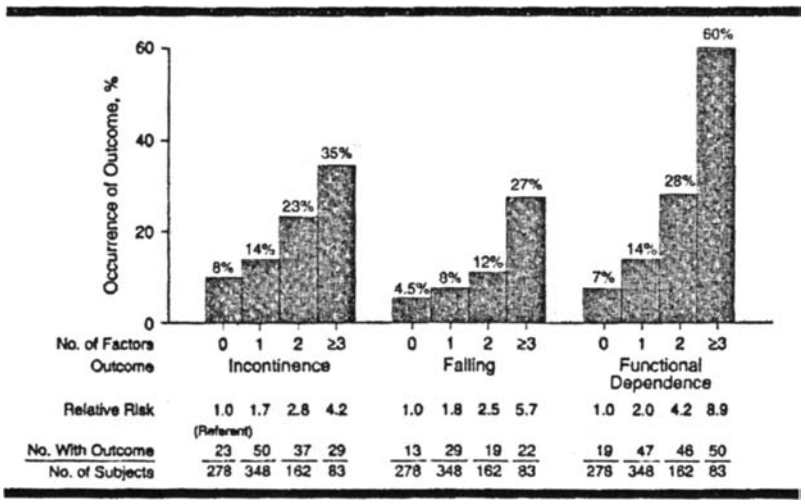
*Relative risks (RRs) and 95% confidence intervals (CIs), adjusted for other factors in the model.
†The first model for functional dependence included only the four predisposing factors; the second model, the predisposing factors plus incontinence; and third model, the predisposing factors plus falling.

The next step in the analysis was to determine whether the two geriatric syndromes were associated with the occurrence of functional dependence the independent of the identified set of predisposing factors. As can be seen in Table 2, incontinence was associated independently with functional dependence (adjusted RR, 1.4; 95% CI, 1.1 to 1.7), while falling displayed a trend toward significance. For both geriatric syndromes, the RR estimates for the other factors remained stable, suggesting that the association between these syndromes and functional dependence was independent of the predisposing factors.

The proportion of subjects reporting each outcome of incontinence, falling, and functional dependence according to the number of the four predisposing factors possessed is displayed in the Figure. A significant increase was seen in incontinence (Mantel-Haenszel χ^2=36.6; P<.001), falling (Mantel-Haenszel χ^2=34.4; P<.001), and functional dependence (Mantel-Haenszel χ^2=119.8; P<.001) as the number of predisposing factors increased. This relationship was particularly striking for functional dependence: the proportion of persons experiencing functional dependence doubled (7% to 14% to 28% to 60%) as the number of risk impairments increased from none to one to two to three or more.

Because of concern about the reliability of self-reports of incontinence, falling, and functional dependence among the cohort members with cognitive impairment, the final three models were repeated including only those subjects with Folstein MMSE scores greater than 24. The risk factors selected and the risk estimates, other than for anxiety for falling and dependence, were similar to those identified in the entire cohort, although the CIs were somewhat wider because of the smaller sample size (RR for anxiety was 1.1 [95% CI, 0.6 to 1.9] for falls and 1.0 [95% CI, 1.0 to 1.1] for functional dependence; other results are available from the authors on request).

We also reran the final models for incontinence, falling, and functional dependence, this time including only those participants who did not report the relevant condition at the baseline interview. The RR estimates, with two exceptions, were similar to those presented in Table 2, although the CIs were wider because of the smaller sample sizes. For falling, the RR for chair stands was 2.0 (95% CI, 1.2 to 3.4); for arm strength, 1.6 (95% CI, 0.9 to 2.8); for sensory, 2.0 (95% CI, 1.1 to 3.7); and for anxiety, 1.2 (95% CI, 0.7 to 2.1). For functional dependence, the RR for chair stands was 3.8 (95% CI, 2.6 to 5.5); for arm strength, 1.8 (95% CI, 1.2 to 2.7); for sensory, 1.6 (95% CI, 1.1 to 2.5); and for anxiety, 1.0 (95% CI, 0.7 to 1.4). For falling and functional dependence, depression performed better than anxiety in the multivariable models. For falling, depression had an RR of 1.6 (95% CI, 1.0 to 2.7); and for functional dependence, depression had an RR of 1.3 (95% CI, 0.9 to 1.9). Arm strength, which displayed a trend toward significance in the model displayed in Table 2, showed no relationship with incontinence in the model restricted to participants not reporting incontinence at the baseline interview. The RR for arm strength was 0.9 (95% CI, 0.6 to 1.6); for chair stands, 1.4 (95% CI, 0.9 to 2.3); for sensory, 2.3 (95% CI, 1.4 to 3.8); and for anxiety, 1.4 (95% CI, 0.9 to 2.2). Using depression in place of anxiety gave an RR of 1.2 (95% CI, 0.7 to 1.8.)



Occurrence of outcomes of incontinence, falling, and functional dependence according to the number of predisposing factors among the 827 participants (94%) who had complete predisposing and outcome data. The factors included decreased hearing and vision (sensory impairment), anxiety (affective impairment), decreased arm strength (upper extremity impairment), and slowed chair stands (lower extremity impairment). There was a significant increase in the occurrence of incontinence (Mantel-Haenszel χ^2=36.6; P<.001), falling (Mantel-Haenszel χ^2=34.4; P<.001), and functional dependence (Mantel-Haenszel χ^2=119.8; P<.001) as the number of predisposing factors increased.

COMMENT

In this cohort study of a representative sample of community-living elderly persons, four predisposing factors, namely, vision and hearing impairment, anxiety, upper extremity impairment, and lower extremity impairment, were associated with two common geriatric syndromes—incontinence and falling, and with the occurrence of functional dependence—one of the most important health outcomes among elderly persons. The risk of each of the geriatric syndromes and of functional dependence increased with the number of the four predisposing factors possessed. Furthermore, incontinence and, to a lesser extent, falling were associated with the occurrence of functional dependence, independent of the four predisposing factors. These results suggest that, as stated herein, the geriatric syndromes may contribute both indirectly—through the shared risk factors—and directly to the occurrence of functional dependence.

The shared predisposing risk factors identified in the study represent four important domains relative to functioning, namely, sensory, psychological or affective, and upper and lower extremity physical performance. Finding that the occurrence of the two geriatric syndromes and functional dependence increased with the number of functional domains impaired supports the multifactorial etiology of the syndromes and suggests that the loss of compensatory ability may be an important etiologic mechanism for the development of the geriatric syndromes and functional dependence.

The model tested in our analyses is obviously an oversimplification of the relationships among the predisposing impairments, geriatric syndromes, and functional dependence. First, the loss of compensatory ability through the accumulated effect of multiple impairments is not the only pathway to the geriatric syndromes. Falls and incontinence in many elderly persons may result from a specific disease or a single physiologic or structural abnormality as postulated by the more traditional pathophysiologic model of disease. Studies of large numbers of elderly persons, representing the full spectrum from health and frailty, are needed to compare, contrast, and eventually reconcile these two etiologic models. Second, for simplicity, we merely identified the presence or absence of four impairments. It is likely that other characteristics of these risk impairments, such as severity or chronicity, may be important. Certainly, impairments other than the four we selected also predispose to geriatric syndromes and, as suggested by study results, individual impairments are more or less strongly associated with specific geriatric syndromes. Taken to its full measure, our conceptual model would suggest that impairment in any domain could contribute—albeit to different degrees—to any geriatric syndrome or to functional dependence. Third, for this study, we limited the description of the postulated relationships among the predisposing factors, geriatric syndromes, and functional dependence to a single direction. It is just as likely, however, that functional dependence predisposes to geriatric syndromes, such as falling or incontinence. The multidirectionality and complexities of the relationships need to be tested in study populations with a larger number of outcomes and longer follow-up. Finally, because we only studied incontinence and falling, we cannot comment on whether our shared set of impairments also identifies persons predisposed to other important geriatric syndromes, such as delirium or pressure sores. However, we and others have previously shown that, like incontinence and falling, the frequency of delirium in hospitalized patients increases with the number of risk factors possessed.[6,12]

Cognitive impairment, which is a known risk factor for our three outcomes, had a strong relationship with each of our outcomes in bivariate analyses and was the final factor to exit each of the three multivariable models. We did not report power calculations because the bivariate relationships were significant, and we are unaware of methods for calculating power for multivariate relationships. Insufficient sample size, however, was likely at least a partial explanation for the exclusion of cognitive impairment as a predisposing factor. Certainly, cognitive impairment needs to be included in any future studies.

As is necessary for large longitudinal studies of community-living elderly persons, all three outcomes were ascertained by self-report. The reliability of self-report among community-living elderly persons, however, has been shown to be good for the outcomes of incontinence,[28,29] falling,[10] and functional dependence.[30] To further assess the reliability of our findings, we repeated the analyses in the subset of cohort members who were cognitively intact. The concurrence of our results in this subgroup with the entire cohort strengthens the credibility of our results.

While we ascertained falls for 3 years and conducted a 3-year telephone interview during which we repeated the questions on incontinence and functional dependence, we elected to limit analyses to conditions reported at 1 year. We felt 3 years was too long a lag time between assessment of the predisposing factors and the ascertainment of the geriatric syndromes and functional dependence.

In our primary analyses, we included all subjects who reported the occurrence of falling, incontinence, or functional dependence at 1 year regardless of whether they had also reported the condition during the baseline interview for several reasons. First, as noted in the introduction and supported by our findings, the geriatric syndromes—and to an extent functional dependence—are intermittent and recurrent in nature, making it difficult to discern the point of onset that allows one to differentiate incident from prevalent cases. Indeed, given the intermittency of these conditions, it may not be appropriate to think in terms of incidence and

(Continued)

prevalence. While beyond the scope of these analyses, further exploration of the intermittent nature of these syndromes may divulge further clues to etiology and treatment. Second, the risk estimates for most of the predisposing factors were similar whether prevalent cases were included or excluded; their inclusion increased our power and reduced the width of the CIs around the estimates. Finally, our analyses were meant to be exploratory, to determine whether sufficient preliminary evidence exists to warrant further investigation of our postulated etiologic model of geriatric syndromes and functional dependence.

While we established an association among the predisposing impairments, the geriatric syndromes, and functional dependence, we could not determine any cause-effect relationships, and, as noted herein, that was not our intent. The strongest support of a cause-effect relationship would be a controlled trial showing that modification of the predisposing impairments leads to a reduction in the occurrence of the geriatric syndromes and functional dependence. Indeed, we recently reported such a trial for the geriatric syndrome of falling.[31]

If verified in other large cohorts of elderly persons and with other geriatric syndromes, our results would have important clinical and research implications. At the very least, our study provides empiric evidence to support the concept of frailty—increased vulnerability to insults or challenges resulting from impairments in multiple domains that compromise compensatory ability. This increased sensitivity to acute insults or challenges among vulnerable persons has been shown for two geriatric syndromes, namely, serious fall injuries[32] and delirium.[33] In both cases, the occurrence of the syndrome increased in frequency in the presence of acute challenges (environmental and activity related in the case of fall injury and medical exposures in the case of delirium) as the number of predisposing risk impairments increased. The shared set of risk factors thus could be used to identify those elderly persons at risk for developing geriatric syndromes or functional dependence in the face of new insults or challenges. Efforts could then be made to decrease exposure to avoidable insults, such as sedatives, or to more accurately weigh the risks vs benefits of necessary treatments, such as essential medications, surgery, or hospitalizations.

Conceivably, the identification of key shared predisposing impairments or risk factors also could serve as the focus of a highly efficient and effective geriatric assessment and intervention strategy. The key shared predisposing impairments could be used to identify who to target and, perhaps more importantly, what to target. The four predisposing impairments identified in the current study are all potentially modifiable with various combinations of medical, surgical, psychological, rehabilitative, and environmental interventions. By intervening, and thus modifying, these shared predisposing impairments, it may be possible to restore compensatory ability, in turn decreasing vulnerability and, consequently, preventing the onset or recurrence of several geriatric syndromes and, perhaps, even functional dependence.

While much additional work is necessary to verify our findings and to elucidate whether a definitive set of predisposing shared factors or impairments exists, our preliminary results provide empiric evidence for the loss of compensatory ability as a mechanism for frailty and provide clues to investigating the mechanisms of pathologic aging at the whole organism (ie, human) level to complement work being conducted at the cellular and molecular level.[34] Results suggest that a more cohesive and coordinated approach, both clinically and investigationally, is needed in defining, preventing, and treating the various geriatric syndromes, functional dependence, and ultimately, frailty. Perhaps it is time to stop looking at diseases, impairments, and syndromes in isolation and begin considering a more unified approach.

This study was supported in part by grant AG07449 and by the Claude D. Pepper Older American Independence Center (P60AG10469), both from the National Institute on Aging. Dr Inouye is the recipient of Academic Award K08AB00524 from the National Institute on Aging.

We are indebted to Margaret Carlson and Linda Herzman for assistance in the preparation of the manuscript.

References

1. Reuben DB. Geriatric syndromes. In: Beck AC, ed. *Geriatrics Review Syllabus*. 2nd ed. New York, NY: American Geriatrics Society; 1991:117-231.
2. Nutt AG, Marsden CD, Thompson PD. Human walking in higher level gait disorders, particularly in the elderly. *Neurology*. 1993;43:268-279.
3. Resnick NM, Yalla SV, Laurino E. The pathophysiology of urinary incontinence among institutionalized elderly persons. *N Engl J Med*. 1989;320:1-7.
4. Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. *N Engl J Med*. 1988;319:1701-1707.
5. Houston KA. Incontinence and the older woman. *Clin Geriatr Med*. 1993;9:157-171.
6. Inouye SK, Viscoli CM, Horwitz RI, Hurst LD, Tinetti ME. A predictive model for delirium in hospitalized elderly medical patients based on admission characteristics. *Ann Intern Med*. 1993;119:474-481.
7. Alexander NB. Postural control in older adults. *J Am Geriatr Soc*. 1994;42:93-108.
8. Lipsitz LA, Goldberger AL. Loss of complexity and aging. *JAMA*. 1992;267:1806-1809.
9. Resnick NM. Urinary incontinence in older adults. *Hosp Pract*. October 15, 1992:552-566.
10. Nevitt MC, Cummings SR, Kidd S, Black D. Risk factors for recurrent nonsyncopal falls: a prospective study. *JAMA*. 1989;261:2663-2668.
11. Robbins AS, Rubenstein LZ, Josephson KR, Schulman BL, Osterweil D, Fine G. Predictors of falls among elderly people: results of two population-based studies. *Arch Intern Med*. 1989;149:1628-1633.
12. Francis J, Martin D, Kapoor W. A prospective study of delirium in hospitalized elderly. *JAMA*. 1990;267:827-831.
13. Marcantonio ER, Goldman L, Mangione CM, et al. A clinical prediction rule for delirium after elective noncardiac surgery. *JAMA*. 1994;271:134-139.
14. Siu AL, Beers MH, Morgenstern H. The medical and public health imperative revisited. *J Am Geriatr Soc*. 1993;41:78-84.
15. Tinetti ME, Liu W-L, Claus EB. Predictors and prognosis of inability to get up after falls among elderly persons. *JAMA*. 1993;269:65-70.
16. Folstein MF, Folstein SE. Mini-Mental State: a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975;12:189-198.
17. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas*. 1977;1:385-401.
18. Spielberger CD, Gorsuch RI, Lushene RE. *The STAI Manual for the State-Trait Anxiety Inventory*. Palo Alto, Calif: Consulting Psychologists Press; 1970.
19. Tinetti ME, Douchette JT, Claus EB, Marottoli R. Risk factors for serious injury during falls by community elderly persons. *J Am Geriatr Soc*. In press.
20. Spaeth EB, Fralick FB, Hughes WF. Estimates of loss of visual efficiency. *Arch Ophthalmol*. 1955;54:462-468.
21. MacPhee GJA, Crowther JA, McAlpine CH. A single screening test for hearing impairment in elderly patients. *Age Ageing*. 1988;17:347-351.
22. Cahalan D, Cisin IH, Crossley HM. *American Drinking Practices: A National Study of Drinking Behavior and Attitudes*. New Brunswick, NJ: Rutgers Center of Alcohol Studies; 1969.
23. Cornoni-Huntley J, Brock DB, Ostfeld AM, Taylor JO, Wallace RB. *Established Populations for Epidemiologic Studies of the Elderly: Resource Data Book*. Washington, DC: National Institute on Aging; 1986. DHHS publication NIH 86-2443.
24. Branch LG, Katz S, Kniepmann K, Papsidero HA. A prospective study of functional status among community elders. *Am J Public Health*. 1984;74:266-268.
25. Hosmer DW, Lemeshow S. *Applied Logistic Regression*. New York, NY: John Wiley & Sons Inc; 1989.
26. Wacholder S. Binomial regression in GLIM: estimating risk ratios and risk differences. *Am J Epidemiol*. 1986;123:174-184.
27. Risch HA, Weiss NS, Clarke EA, Miller AB. Risk factors for spontaneous abortion and its recurrence. *Am J Epidemiol*. 1988;128:420-430.
28. Herzog AR, Fultz NH. Prevalence and incidence of a urinary incontinence in community-dwelling populations. *J Am Geriatr Soc*. 1990;38:273-281.
29. Resnick NM, Beckett LA, Branch LG, Scherr PA, Wetle T. Short-term variability of self report of incontinence in older persons. *J Am Geriatr Soc*. 1994;42:202-207.
30. Smith LA, Branch LG, Scherr PA, et al. Short-term variability of measures of physical function in older people. *J Am Geriatr Soc*. 1990;38:993-998.
31. Tinetti ME, Baker DI, McAvay G, et al. A multifactorial intervention to reduce the risk of falling among elderly people living in the community. *N Engl J Med*. 1994;331:821-827.
32. Tinetti ME, Doucette JT, Claus EB. The contribution of predisposing and situational risk factors to serious fall injuries. *J Am Geriatr Soc*. In press.
33. Marcantonio ER, Juarez G, Goldman L, et al. The relationship of postoperative delirium with psychoactive medications. *JAMA*. 1994;272:1518-1522.
34. Martin GR, Danner DB, Holbrook NJ. Aging: causes and defenses. *Annu Rev Med*. 1993;44:419-429.

9
Urinary Incontinence

Mary Ann Forciea, MD

Reproduced paper following commentary: Reprinted from the J Urol, volume 38, Foley, Frederic EB, A Self-Retaining Bag Catheter for Use as an Indwelling Catheter for Constant Drainage of the Bladder, 140–143, copyright (1937), with permission from the American Urological Association.

Commentary

The unintended loss of urine in amounts sufficient to cause embarrassment or functional limitation remains one of the most common problems in the practice of geriatric medicine. The prevalence of urinary incontinence varies by definition, site of care, and gender. Frail populations such as nursing home residents and housebound patients may have prevalence rates of 50% to 70%, while office patients have prevalence figures from 5% to 40%. Urinary incontinence is more common in women than men. All studies have shown significant underreporting by patients due to embarrassment or low expectations of therapy.

Complete understanding of continence required advances in the anatomy of not only the bladder, but also of the autonomic and peripheral nervous systems. Lack of understanding of mechanism did not limit attempts at therapy: drawings from ancient Egypt illustrate techniques of bladder catheterization presumably from overflow incontinence. Benjamin Franklin offered a design for a bladder catheter among his catalog of inventions (1). Advances in synthetic materials, such as Latex, allowed Frederic Foley to develop an indwelling catheter (report reproduced here) which "would give positive self-retention, comfort to the patient and completely satisfactory performance to say nothing of the urologic blessing of relief from adhesive tape fixation." He goes on to relate that this catheter would allow "full activity" of patients suffering from urinary problems that could not be corrected by surgery. The therapeutic use of indwelling Foley-type catheters remains one option in the management of some types of incontinence today.

The rehabilitation of bladder and perineal muscles was another avenue of therapy for incontinence, especially of the "stress" type. Kegel, in the first article describing his technique of postpartum perineal muscle contractions (2), mentions that Aristotle advocated hot douches to stimulate perineal muscles and that Soranus in 110 AD advocated support for the pelvic floor with the hand. Kegel refined his perineal exercise technique for improvement in urine control for postmenopausal women and postprostatectomy men.

Special attention to urinary incontinence in the elderly began to develop in the last half of the twentieth century. Ouslander, in 1981, published the first review of problems of urinary incontinence specific to older patients (3). Resnick and coworkers subsequently focused attention on the pathophysiology of urinary incontinence in nursing home residents (4). They described the causes of urinary incontinence in this frail, old population: 38% of patients had detrusor overactivity as their predominant bladder dysfunction, while in 35% of the patients, mixed types of dysfunction were identified. The classic textbook on urinary incontinence is written by Diane Newman (5) and provides an excellent resource for teaching.

Modern treatment of urinary incontinence continues to stress accurate diagnosis, perineal muscle strengthening exercises, judicious use of medications, and occasionally surgery or chronic catheterization. None of these can be effective if we fail to carefully inquire about urinary leakage in our older patients.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

References

1. Kimbrough HM Jr. Benjamin Franklin (1706–2790). Investigative Urol 1975;12:509–510.
2. Kegel AH. Progressive Resistance Exercise in the Functional Restoration of the Perineal Muscles. Am J Obst Gyn 1948;56: 238–248.
3. Ouslander JG. Urinary Incontinence in the Elderly. W J Med 1981;135:482–491.
4. Resnick NM, Yalla SV, Laurino E. The Pathophysiology of Urinary Incontinence among Institutionalized Elderly Persons. New Engl J Med 1989;320:1421–1422.
5. Newman D. Managing and Treating Urinary Incontinence. Baltimore: Health Professions Press. 2002.

A SELF-RETAINING BAG CATHETER

For Use as an Indwelling Catheter for Constant Drainage of the Bladder

FREDERIC E. B. FOLEY, M.D.

Saint Paul

A "hemostatic bag catheter" was devised by the writer in 1927. The present fully perfected form of the device is described in another communication in this issue of this JOURNAL (see page 134).

Chief use of the "hemostatic bag catheter" was for control of bleeding following transurethral resection of the prostate. The distended "hemostatic bag" was found to retain the catheter in position perfectly and made unnecessary any other means of retaining it in place—such as adhesive tape fixation to the skin. It was noted that even considerable traction would not dislodge it and that it occasioned little or no discomfort.

From this early experience with the hemostatic bag catheter it was at once evident that a modified form of the device would make an ideal self-retaining indwelling catheter for routine use in constant drainage of the bladder. It was felt that no existing form of "self-retaining" catheter was actually self-retaining and certainly that none of them was eminently satisfactory. The prospect was that the proposed device would give positive self-retention, comfort to the patient and completely satisfactory performance to say nothing of the urologic blessing of relief from adhesive tape fixation.

With the excellent coöperation of Mr. Raymond C. Albright of American Anode, Inc., a thoroughly satisfactory "self-retaining bag catheter" has been perfected and is now available through American Cystoscope Makers, Inc., distributors of "Ameran" (American Anode) catheters.

The design of the catheter is shown in the schematic perspective and sectional drawing figure 1 and needs no description beyond this. The one piece construction and perfect smoothness of the catheter are well shown in the photographs figure 2.

Indicated uses of the catheter are clearly evident without extensive elaboration: it may be used where ever constant drainage by an indwelling catheter is required. When fixation of the ordinary catheter by adhesive tape, or otherwise, is unsatisfactory, difficult or impossible for

(Continued)

any reason (redundant prepuce, phimosis, genital infantilism, obesity or ulceration or other inflammatory change of skin) and where interval catheterization otherwise would be required the self-retaining bag catheter permits constant drainage to be maintained with perfect facility and comfort. Non-hospitalization, ambulatory condition and even full activity of the patient as may be desirable in permanent retention of urine due to disturbances of innervation not amenable to correction are not contraindications to its use. Indeed situations made by some of these conditions find their only satisfactory solution in use of the catheter. It is of course usable in the female as well as in the male for any condition requiring constant drainage.

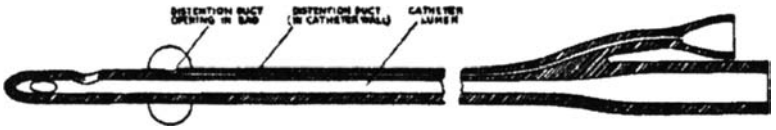


FIG. 1. Schematic, perspective and sectional drawing showing the design of the "self-retaining bag catheter." The distention duct lies in the catheter wall, opens within the bag cavity and has a proximal end extension for convenience in attaching a syringe to distend the bag

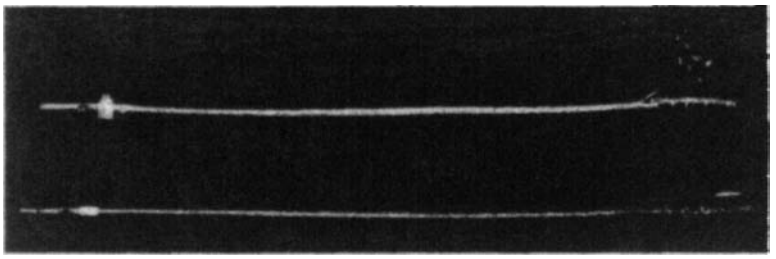


FIG. 2. Retouched photographs showing the "self-retaining bag catheter." Below: Bag collapsed. Above: Bag distended (5 cc.). Note the perfectly smooth surfaces, absence of cemented joints, two eyelets and proximal end extension of the distention duct for convenience in connecting syringe and applying clamp.

Installation of the catheter is a simple and easy procedure. It is passed as any catheter is passed and is advanced well up into the bladder. Five cubic centimeters of sterile water are injected into the bag with a piston syringe and a small clamp is applied to the distention duct or it may be bent double and bound with a rubber band. The catheter is then drawn down until contact of the bag with the vesical neck is felt. Apart from distention of the bag no other fixation whatever is necessary or desirable under ordinary circumstances. In restless or

disoriented patients the proximal portion of the catheter shaft may be fixed to the thigh to avoid undue traction on the retaining bag.

A considerable experience in routine use of the self-retaining bag catheter has emphasized a number of its advantages. Shaving of neighboring skin is not required or desirable. Very definitely it is tolerated better than the ordinary catheter fixed in place by adhesive or other means. This has appeared due to absence of movement of the vesical end of the catheter as occurs with the ordinary catheter in response to

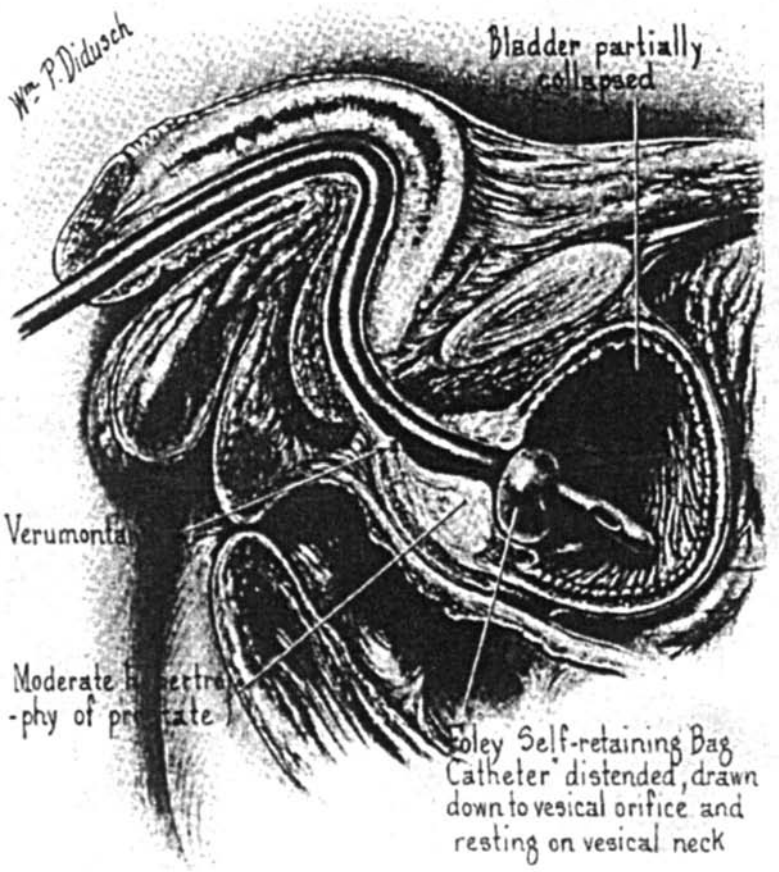


FIG. 3. Anatomic drawing of mid-sagittal section through prostate. The self-retaining bag catheter is shown in place with the distended retaining bag resting on the vesical neck.

(Continued)

movements of the skin and parts to which it is fixed. Also it has appeared that Latex is tolerated by the urethral mucosa better than the ordinary red rubber catheter. Certainly the absence of adhesive tape or other means of external fixation and freedom from purulent material retained around the meatus permits the patient to be clean in a degree not possible with the ordinary indwelling catheter. The catheter may be left in place almost indefinitely or until spontaneous rupture of the bag finally occurs—an event unaccompanied by any undesirable consequence other than displacement of the catheter.

10
Osteoporosis

Robert J. Pignolo, MD, PhD

Commentary

Paleopathologic examinations of old skeletons confirm that osteoporosis likely existed throughout history (1), but it has been only within the last 200 years that the term was coined and that the pathology distinguished it from osteomalacia (2). As human lifespan has increased over the late nineteenth and early twentieth centuries, osteoporosis has taken on major importance as a clinical problem.

In 1940, Fuller Albright, in a communication to the Association of American Physicians, described and introduced the term "postmenopausal" osteoporosis. In that landmark publication (reproduced here), he distinguished postmenopausal osteoporosis from other forms of bone loss that were known at the time, including disuse, senile, and so-called "idiopathic" osteoporosis, the later involving primarily the spine and to a lesser extent the pelvis. He evaluated 42 individuals with idiopathic osteoporosis who were less than 65 years of age, only two of whom were men. He also observed that several premenopausal women who were affected had undergone a surgical menopause. Thus, he correctly renamed this category of idiopathic bone loss as postmenopausal osteoporosis.

In associating estrogen deprivation with postmenopausal osteoporosis, Albright acknowledged in his 1940 paper the previous work of Kyes and Potter (3), who reported that male pigeons had osteoporotic bones compared to those of ovulating females and that ossification in the skeleton of female pigeons was proportional to the size of their ovarian follicles. Albright also acknowledged the work of Pfeiffer and Gardener (4), who injected male pigeons with estrogen and corrected the deficit in bone formation relative to female birds. In the same paper Albright treated three women with estrogen therapy and noted that there was "a markedly positive calcium and phosphorus balance . . . (which) continued as long as estrogen was administered." Based on these findings, Albright concluded that osteoporosis is a failure of osteoblasts to form adequate bone matrix.

In a subsequent paper in 1941, Albright attempted to elaborate differences between postmenopausal osteoporosis and other metabolic bone diseases (5). He made the observation that postmenopausal and senile osteoporosis rarely involves the skull, a feature which distinguishes them from mild hyperparathyroidism and osteomalacia.

Contrary to Albright's original idea that osteoblast dysfunction mediates bone loss associated with menopause, an increase in bone resorption (and not a decrease bone formation) appears primarily to underlie osteoporosis in estrogen deficient states (6,7). However, the production of new bone seen in response to mechanical loading is attenuated with estrogen deficiency, suggesting that estrogen is both an anticatabolic as well as an anabolic hormone in bone tissue (8).

References

1. Berg E. Paleopathology: Bone Lesions in Ancient Peoples. Clin Orthop Rel Res 1972;82:263–267.
2. Schapira D, Schapira C. Osteoporosis: The Evolution of a Scientific Term. Osteoporos Int 1992;2:164–167.
3. Kyes P, Potter TS. Physiological Marrow Ossification in Female Pigeons. Anat Rec 1934;60:377–379.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea
© Humana Press, Totowa, NJ

4. Pfeiffer CA, Gardener WU. Skeletal Changes and the Serum Calcium Level in Pigeons Receiving Estrogens. Endocrinology 1938;23:485–491.
5. Albright F, Smith PH, Richardson AM. Postmenopausal Osteoporosis: Its Clinical Features. JAMA 1941;116:2465–2471.
6. Parfitt AM, Villanueva AR, Foldes J, Rao DS. Relations between histologic indices of bone formation: implications for the pathogenesis of spinal osteoporosis. J Bone Miner Res 1995;10:466–473.
7. Ebeling PR et al. Bone Turnover Markers and Bone Density Across the Menopausal Transition. J Clin Endocrinol Metab 1996;81:3366–3371.
8. Lee K, Jessop H, Suswillo R, Zaman G, Lanyon L. Endocrinology: Bone Adaptation Requires Oestrogen Receptor-Alpha. Nature 2003;424.

POST-MENOPAUSAL OSTEOPOROSIS*

By FULLER ALBRIGHT, M.D.

AND (By Invitation)

ESTHER BLOOMBERG, B.S. AND PATRICIA H. SMITH, M.D.

BOSTON, MASS.

(From the Medical Service of the Massachusetts General Hospital and the Department of Medicine of the Harvard University Medical School)

MR. President, members of the Association and guests:

Our paper will be divided into three parts: (*a*) What is osteoporosis? (*b*) Why is osteoporosis? (*c*) What can one do about it?

To conserve time we will delete all "ifs" and "buts."

What is Osteoporosis? Adult bone (see Fig. 1*A*) is composed of an organic matrix in which is deposited a calcium-phosphate-carbonate complex. Covering the bone meshes are two types of surfaces: one where bone is being laid down; one where it is being resorbed. Bone deposition, furthermore, is composed of two processes: the laying down of the organic matrix by the osteoblasts and the deposition in this matrix of the calcium complex.

Now, one can have too little bone, either because bone resorption is too great, or because deposition is too little. The former process leads to osteitis fibrosa (see Fig. 1*D*), but does not concern us here. The latter process—too little bone formation—can be due to either one of two abnormalities. Thus, there may be a failure of the osteoblasts to lay down an organic matrix or the calcium complex may fail to be deposited in the organic matrix. The former condition is osteoporosis (see Fig. 1*B*); the latter is osteomalacia (see Fig. 1*C*).

Why is Osteoporosis? There is considerable circumstantial evidence that the stimulus for the osteoblasts to lay down an organic matrix is mechanical stresses and strains. Hence, one of the most clear-cut causes of osteoporosis is lack of such stresses and strains, which leads to "atrophy of disuse." Furthermore, just as very

* This work was aided by a grant from the Committee for Research in Problems of Sex of the National Research Council.

(Continued)

elderly people have atrophy of their hair, skin, and tissues in general so do they have atrophy of their bones. This is "senile osteoporosis." But we are concerned here with a condition which, until recently, we have been forced to call idiopathic osteoporosis. This condition involves primarily the spine and pelvis, to a much lesser extent the long bones, and least of all the skull. A survey of 42 such cases sixty-five years or under showed that 40 were women after the menopause; there were only 2 males; there were no cases in women before the menopause. This form of osteoporosis was found in several women of the pre-menopause age, who had undergone a surgical menopause. In brief, it is our belief that idiopathic osteoporosis is post-menopausal osteoporosis.

What Can One Do About It? Hens, when they lay eggs have high serum calcium values.[1] So do doves.[2] Bones of male doves are osteoporotic as compared with those of female doves.[3] Estrin therapy in male doves produces a marked increase in the density of the bones by stimulating the osteoblasts.[4] Therefore, it was decided to try estrin therapy on patients with menopausal osteoporosis.

RESULTS. In Figures 2, 3 and 4 are shown the metabolic data on 3 patients with post-menopausal osteoporosis who received estrin

---

LEGEND FOR FIG. 1, *A*, *B*, *C* AND *D*.

FIG. 1.—Schematic diagrams to show authors' conception of differences between normal, osteoporosis, hyperparathyroidism with osteitis fibrosa generalisata and osteomalacia. *A*, body limits; *B*, body fluid; *C*, body serum, a compartment of body fluid easy to tap for analysis; *D*, bone mass with two surfaces, one where bone is being resorbed and one where it is being laid down; *E*, arrow indicating by its size rate of Ca and P resorption; *F*, osteoclast; *G*, rate of Ca and P deposition; *H*, osteoblast laying down osteoid (*I*); *J*, Ca and P entering body from gastro-intestinal tract; *K*, Ca and P leaving body by kidney or other exits; *L*, syringe obtaining serum for analysis; *M*, blood values (n, normal; +, high; −, low).

A. Normal: Note that Ca and P going into bone equals that coming out of bone; that part of that which comes out goes back in.

B. Osteoporosis: Note decrease in bone mass (D_1); primary hypoplasia of osteoblasts (H_1); decreased deposition of osteoid (I_1); decreased Ca and P deposition (G_1); increased Ca and P excretion (K_1); and normal blood values (M_1).

C. Osteomalacia: Note decreased bone mass (D_2); hyperplasia of osteoblasts because of increased stresses and strains (H_2); increased deposition of osteoid which is inadequately calcified because of serum Ca and P values; decreased Ca and P deposition (G_2); primary difficulty in absorbing Ca and P from gastro-intestinal tract (J_2); and abnormal blood values (Ca normal or low, P low, phosphatase high).

D. Osteitis Fibrosa Generalisata complicating Hyperparathyroidism: Note increased Ca and P excretion in urine (K_3); increased Ca and P resorption (E_3); increase of osteoclasts (F_3); decreased bone mass (D_3); increased bone formation by osteoblasts (I_3) because of increased stresses and strains; increased Ca and P deposition (G_3) because serum is not undersaturated in respect to calcium phosphate (*i. e.*, serum Ca is sufficiently high to almost offset low serum P); and high phosphatase level (M_3).

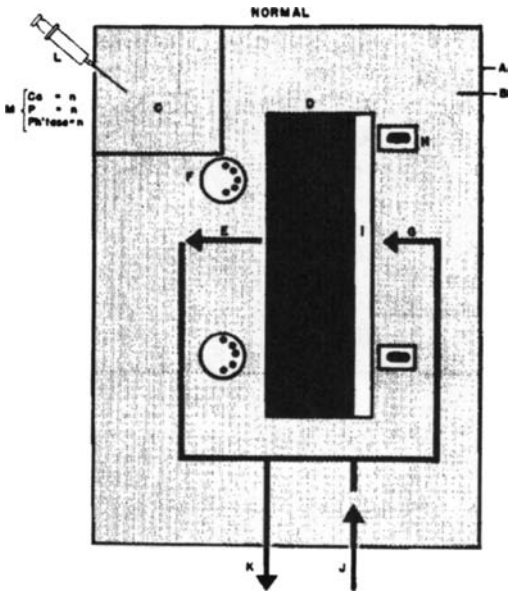


FIG. 1 *A*

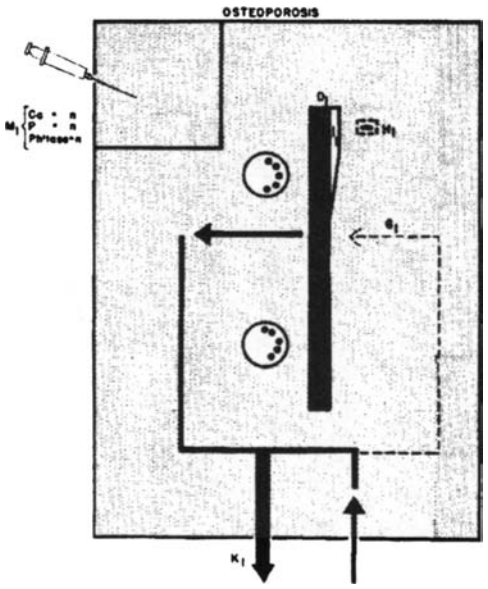


FIG. 1 *B*

(Continued)

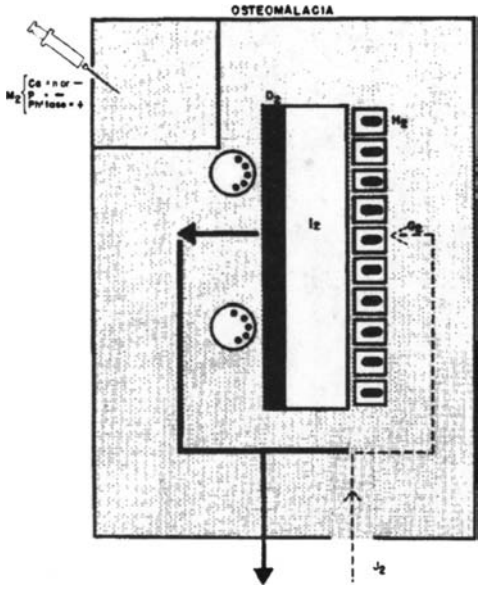

OSTEOMALACIA

FIG. 1 *C*

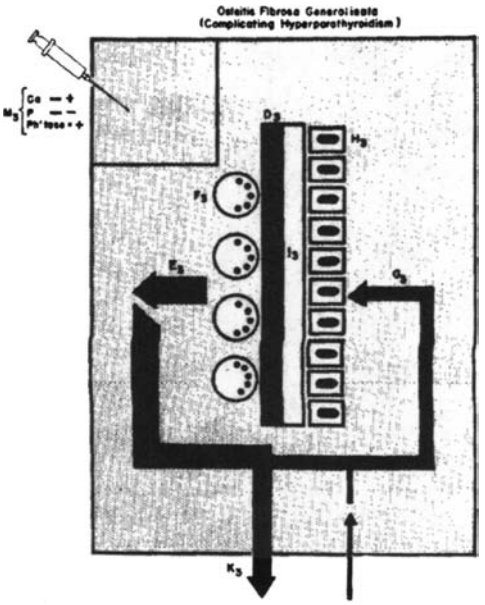

Osteitis Fibrosa Generalisata
(Complicating Hyperparathyroidism)

FIG. 1 *D*

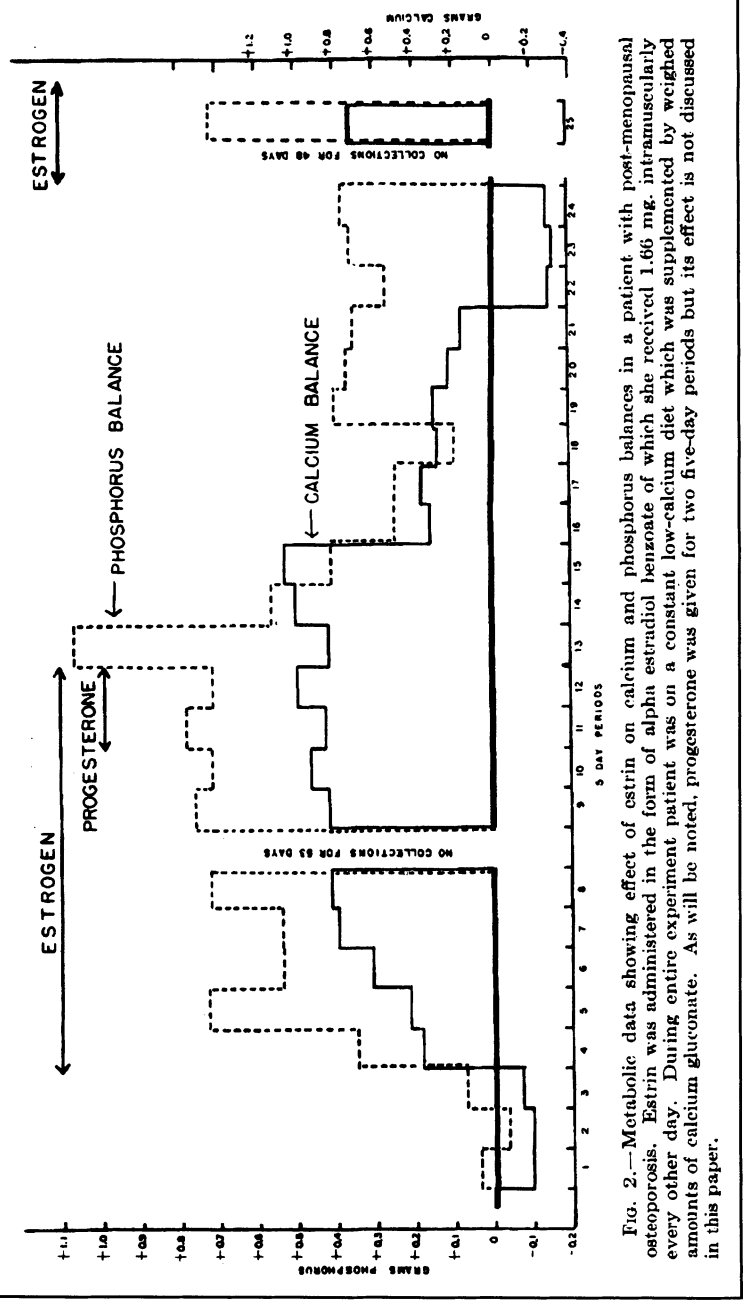


FIG. 2.—Metabolic data showing effect of estrin on calcium and phosphorus balances in a patient with post-menopausal osteoporosis. Estrin was administered in the form of alpha estradiol benzoate of which she received 1.66 mg. intramuscularly every other day. During entire experiment patient was on a constant low-calcium diet which was supplemented by weighed amounts of calcium gluconate. As will be noted, progesterone was given for two five-day periods but its effect is not discussed in this paper.

(Continued)

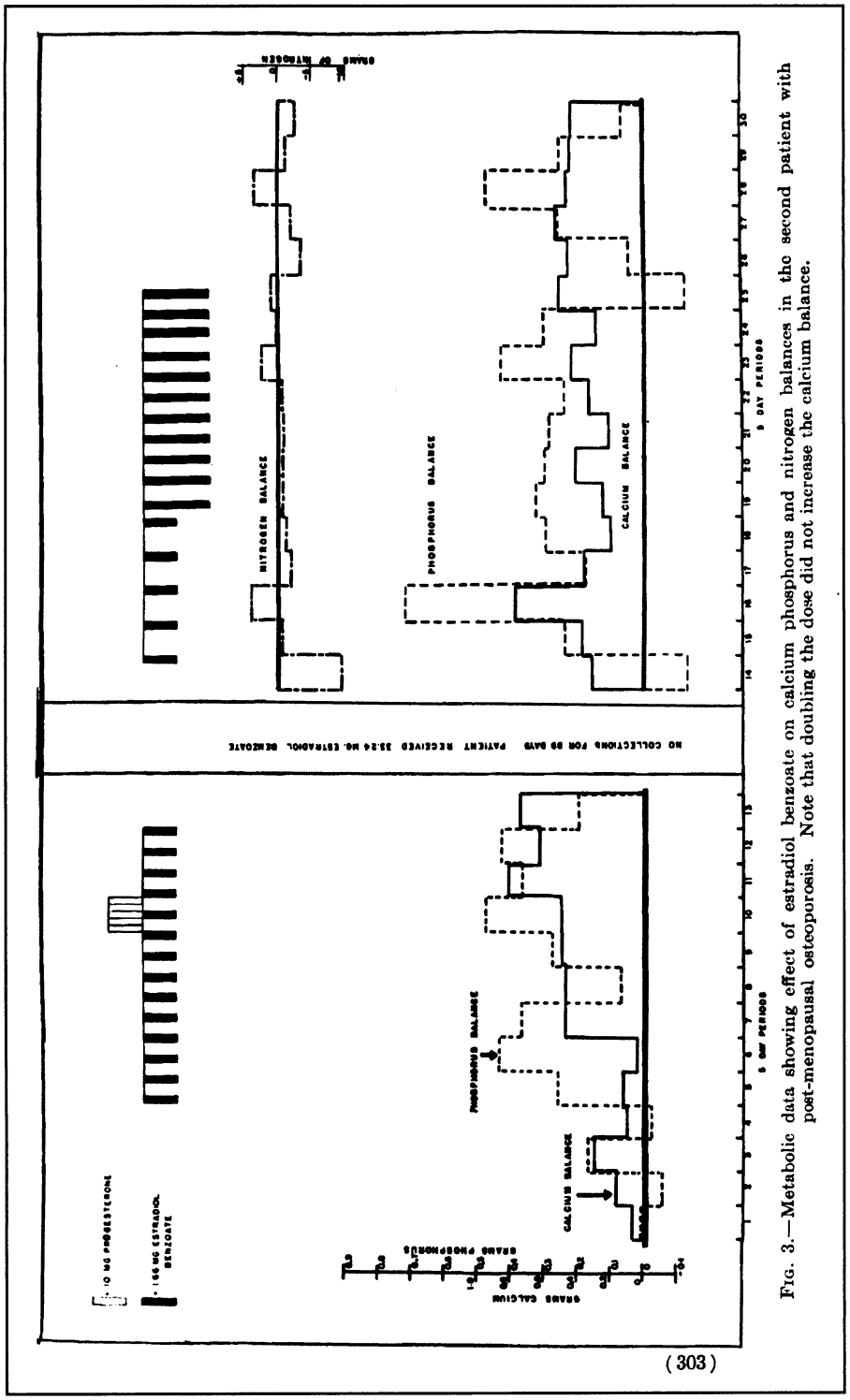


FIG. 3.—Metabolic data showing effect of estradiol benzoate on calcium phosphorus and nitrogen balances in the second patient with post-menopausal osteoporosis. Note that doubling the dose did not increase the calcium balance.

therapy. It will be noted in Figure 2 that on the administration of estrin the patient went into a markedly positive calcium and phosphorus balance; that the maximum effect was not reached until after about twenty days of therapy; that the effect then continued as long as estrin therapy was administered (ninety-eight days); that following cessation of the treatment there was very little change for about fifteen days after which there was a slow reversal

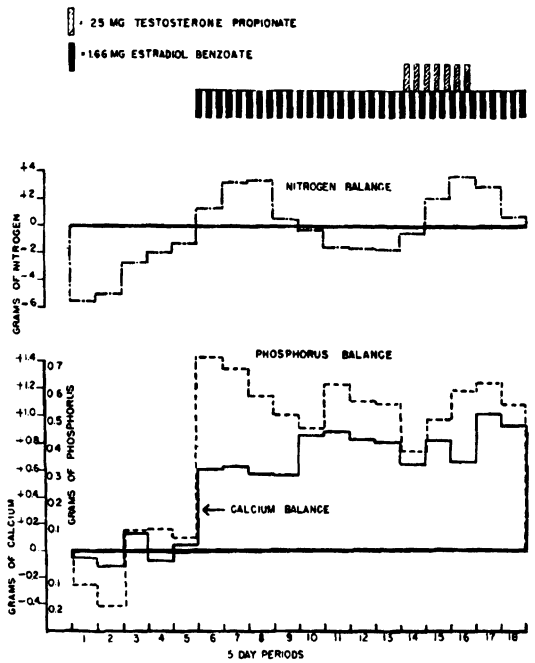


FIG. 4.—Metabolic data showing the effect of estradiol benzoate on the calcium, phosphorus and nitrogen balances on a third patient. Note that testosterone propionate was also given for two periods but its effect is not discussed in this paper.

to the pre-medication findings; and finally that the patient again returned to a positive calcium and phosphorus balance after a second course of medication. Figure 3 shows essentially the same findings much less convincingly in a second patient. Figure 4 shows a very convincing estrin effect in the third patient.

SUMMARY. We shall summarize by stating two conclusions and one reservation:

(Continued)

Conclusion 1. After the menopause, and hence after the time when a reserve supply of calcium is needed for the production of babies, women are prone to develop osteoporosis of their spines and pelves. We have designated this condition post-menopausal osteoporosis.

Conclusion 2. Estrin therapy has a very marked ability to put patients with post-menopausal osteoporosis into a positive calcium and phosphorus balance.

Reservation. Whether the effect of estrin on the calcium balance in post-menopausal osteoporosis is sufficient to make its administration of practical clinical value, it is as yet too soon to say.

The authors wish to thank Dr. Erwin Schwenk of the Schering Corporation, Bloomfield, N. J., for supplying the large amounts of estradiol benzoate (Progynon-B) used in this investigation.

BIBLIOGRAPHY

1. Hughes, J. S., Titus, R. W., and Smits, B. L.: The Increase in the Calcium of Hens' Blood Accompanying Egg Production, Science, **65**, 264, 1927.
2. Riddle, O., and Rhinehart, W. H.: Studies on the Changes of Physiological Reproduction in Birds, Am. Jour. Physiol., **76**, 660, 1926.
3. Kyes, P., and Potter, T. S.: Physiological Marrow Ossification in Female Pigeons, Anat. Rec. **60**, 377, 1934.
4. Pfeiffer, C. A., and Gardner, W. U.: Skeletal Changes and the Serum Calcium Level in Pigeons Receiving Estrogens, Endocrinology. **23**, 485, 1938.

11
Nutrition

Joan Weinryb, MD, CMD

Commentary

Before the percutaneous endoscopic gastrostomy (PEG) tube was invented, installing a feeding tube required laparotomy and general anesthesia. The use of feeding tubes was generally limited to young people with hopes of recovery. On June 12 1979, pediatrician M. Gauderer and endoscopist J. Ponsky implanted the first PEG tube in a 10-week-old child with a swallowing impediment. Because PEG placement required no open surgery and no general anesthetic, the early 1980s were marked by the increased use of PEG tubes in adults, starting with stroke victims and extending to other patients with impaired swallowing, including terminal cancer patients and elderly people with dementia. In 1988, the annual number of PEG procedures reimbursed by Medicare was 61,000; by 1995, approximately 121,000 procedures were reimbursed, by 2005, almost 300,000. In 1999, 34% of severely cognitively impaired nursing home residents had PEGs. Use of the technology had shifted from emergency care of young patients to indications including head and neck cancers, neuromuscular disorders, acute stroke, gastrointestinal obstruction, and dementia.

Various factors drove the increasingly common insertion of PEGs. Patients with advanced dementia often had difficulty with eating. Families were distressed by what they perceived as a withholding of nutrition from loved ones. Physicians may have recommended PEG placement due to fear of litigation or from reluctance to delve into difficult end-of-life issues. Nursing homes sometimes required PEG placement for admission to the facility because of staffing, regulatory, or legal concerns. Religious groups actively challenged living wills and surrogate decisions that called for incapacitated patients to die without a feeding tube, describing this as tantamount to euthanasia.

Dr. Finucane's article abstracted the data available for patients with advanced dementia, providing medical evidence that PEG tube placement did not provide the advantages previously thought, did cause various complications and, thus, was not medically indicated for patients with advanced dementia. Finucane found no evidence that tube feeding in persons with advanced dementia prolonged survival, prevented aspiration pneumonia, reduced the risk of pressure sores or infections, improved function, or provided comfort. Use of a feeding tube in a demented patient was associated with significant increases in restraint use, utilization of the emergency department, and hospitalization.

Finucane's article provided physicians with the data to educate themselves, long-term care and regulatory professionals, and families. It offered a framework to show that PEG placement was not only not ethically indicated, but was medically contraindicated for patients with advanced dementia. He mustered the evidence that lent credence to careful hand feeding of the patient with advanced dementia as the preferred way to maintain patient comfort.

References

1. Mitchell S, Buchanan JL, Littlehale S, Hamel MB. Tube-Feeding Versus Hand–Feeding Nursing Home Residents with Advanced Dementia: A Cost Comparison. JAMA 2004;5(suppl):S23.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

2. Mitchell S et al. Conical and Organizational Factors Associated with Feeding Tube Use Among Nursing Home Residents with Advanced Cognitive Impairment. JAMA 2003;290: 73.
3. Hague T. Percutaneous Endoscopic Gastroscopy for Enteral Nutrition in Patients with Stroke. Scand J Gastrol 2003;9: 962–966.
4. Gillick MR. Rethinking the Role of Tube Feeding in Patients with Advanced Dementia. New Engl J Med 2000;342: 206–210.
5. Gauderer M. Twenty Years of Percutaneous Endoscopic Gastrostomy: Origin and Evolution of a Concept and Its Expanded Applications. Surg Endoscopy 1999;50:882.

SPECIAL COMMUNICATION

Tube Feeding in Patients With Advanced Dementia

A Review of the Evidence

Thomas E. Finucane, MD
Colleen Christmas, MD
Kathy Travis, MD

Patients with advanced dementia frequently develop eating difficulties and weight loss. Enteral feeding tubes are often used in this situation, yet benefits and risks of this therapy are unclear. We searched MEDLINE, 1966 through March 1999, to identify data about whether tube feeding in patients with advanced dementia can prevent aspiration pneumonia, prolong survival, reduce the risk of pressure sores or infections, improve function, or provide palliation. We found no published randomized trials that compare tube feeding with oral feeding. We found no data to suggest that tube feeding improves any of these clinically important outcomes and some data to suggest that it does not. Further, risks are substantial. The widespread practice of tube feeding should be carefully reconsidered, and we believe that for severely demented patients the practice should be discouraged on clinical grounds.

JAMA. 1999;282:1365-1370 www.jama.com

PATIENTS WITH ADVANCED DEmentia commonly develop difficulty eating, often when they become bedridden and dependent in all activities of daily living. They may resist or be indifferent to food, fail to manage the food bolus properly once it is in the mouth (oral phase dysphagia), or aspirate when swallowing (pharyngeal phase dysphagia). Enteral tube feeding is intended to prevent aspiration pneumonia, forestall malnutrition and its sequelae, including death by starvation, and provide comfort. We reviewed data about whether any type of tube feeding can accomplish these goals in this group of patients. Studies limited to patients with cancer, burns, trauma, dysphagic stroke, mechanical obstruction, critical illness, pediatric patients, or patients receiving ventilatory assistance were not considered. We did not include discussion of ethical issues, since our focus was on clinical evidence.

We searched MEDLINE from 1966 through March 1999 and found no relevant randomized clinical trials comparing tube feeding with oral feeding in the severely demented. Thus, a meta-analysis was not possible; rather, we have presented a summary of the data available. In each section, we describe how articles were identified and summarize the findings. Our goal is to present the relevant data in a way that is useful to clinicians, patients, families, and perhaps policy makers.

For editorial comment see 1380.

DOES TUBE FEEDING PREVENT ASPIRATION PNEUMONIA?

Aspiration pneumonia is often an imprecise diagnosis both conceptually and clinically. Mendelson[1] described a group of parturient women who underwent ether anesthesia and vomited and aspirated gastric contents. All developed tachypnea, wheezing, rales, and cyanosis and all recovered uneventfully in a few days. Some authors use "aspiration pneumonia" to refer to this syndrome, a pneumonitis that follows aspiration and resolves spontaneously without antibiotics.[2] The term is also used to describe pulmonary infection due to misdirection of contaminated pharyngeal contents, especially oral secretions, into the airway. This syndrome is usually insidious in onset, associated with fever, and when a microbiologic diagnosis can be made, polymicrobial. Infection probably results when normally nonpathogenic organisms arrive in high enough inoculum to overcome host defenses.

Tube feeding cannot be expected to prevent aspiration of oral secretions, and no data show that it can reduce the risk from regurgitated gastric contents. In fact, in children[3] and in animal models,[4] gastrostomy tube placement may reduce lower esophageal sphinc-

Author Affiliations: Division of Geriatric Medicine and Gerontology, Johns Hopkins Bayview Medical Center, Baltimore, Md.
Corresponding Author and Reprints: Thomas E. Finucane, MD, Johns Hopkins Geriatrics Center, 5505 Hopkins Bayview Cir, Baltimore, MD 21224 (e-mail: tfinuc@jhmi.edu).

(Continued)

ter pressure and increase the risk of gastroesophageal reflux, with "a change in the gastroesophageal angle (as) the suspected mechanism."[4] No comparable studies have been reported in the elderly.

A 1996 review of tube feeding to prevent aspiration pneumonia conducted by 1 of the authors (T.E.F) and Bynum[5] found that "No randomized trials of the intervention have been done, and some data suggest ineffectiveness." A MEDLINE search from 1966 through March 1999 using the same search terms as that article, *enteral nutrition*, *deglutition disorders*, and *aspiration pneumonia*, confirmed these observations. Three additional case-control studies identified tube feeding as a risk factor for aspiration pneumonia and demonstrated high rates of pneumonia and death in tube-fed patients.[6-8] In a nonrandomized, prospective study,[9] orally fed patients with oropharyngeal dysphagia had significantly fewer major aspiration events than those fed by tube. The authors conclude, "Artificial feeding does not seem to be a satisfactory solution for preventing pneumonia in elderly prandial aspirators." Jejunostomy is not associated with lower rates of pneumonia than gastrostomy.[10,11] We found no published studies suggesting that tube feeding can reduce the risk of aspiration pneumonia.

DOES TUBE FEEDING PREVENT THE CONSEQUENCES OF MALNUTRITION?

Demented patients with problems eating frequently lose weight and develop other abnormal markers of nutritional status such as lowered serum albumin levels or total lymphocyte count, diminished triceps skin fold or body mass index, or impaired skin-test reactivity. Tube feeding may then be initiated to try to prevent or correct consequences of malnutrition including pressure ulcers, infection, debility, and death.

However, in several clinical situations, provision of increased nutrients to patients with abnormal markers of nutritional state had no effect on meaningful clinical outcomes. For 40 patients receiving tube feeding in long-term care (the majority due to neurologic impairment), "adequate calories and protein were provided . . . still, subjects showed weight loss and severe depletion of lean and fat body mass. . . . Despite administration of apparently adequate formula, micronutrient deficiencies and marasmic malnutrition exist in chronically ill patients."[12] In 2 additional clinical situations, patients with abnormal markers of nutritional status did not benefit from increased administration of nutrients. Of 17 trials studying patients with advanced cancer, most of whom were emaciated, no trial showed a survival benefit from parenteral nutrition.[13] Megestrol acetate in patients with acquired immunodeficiency syndrome (AIDS)–cachexia improved intake and nutritional markers; however, death rates in each of 4 treatment groups were more than double that of placebo controls.[14,15] For wasting disorders associated with AIDS and cancer, a 1997 conference sponsored by the National Institutes of Health, the American Society for Parenteral and Enteral Nutrition, and the American Society for Clinical Nutrition concluded that "there are no published observations providing direct evidence that wasting is a cause of death or that reversal of wasting improves outcome."[16]

For patients with advanced dementia and eating difficulties, the relationships among nutritional intake, markers of nutritional status, and clinically meaningful outcomes remain uncertain. For some patients with catabolic illness, delivery of additional nutrients may not provide benefit. For others, additional nutrients might provide benefits, but these may be outweighed by adverse effects of tube feeding. The relevant clinical question is whether tube feeding improves outcomes putatively ascribed to malnutrition.

IS SURVIVAL IMPROVED BY TUBE FEEDING?

We conducted a MEDLINE search of the terms *survival* and *enteral nutrition* from 1966 through March 1999 as well as the bibliographies of many articles related to these topics. Four lines of evidence undermine the apparently commonsense practice of tube feeding emaciated, demented patients to prevent death due to starvation.

First, survival of very low-weight, hand-fed demented patients can be substantial. Survival of demented and nondemented patients was not different in a long-term care facility with a program of careful feeding by hand.[17] A 2-year prospective observation of 71 demented patients in long-term care found similar mortality rates among 4 groups: those who fed themselves, those who required assistance but otherwise had no eating difficulties, those who refused food, and those who coughed and choked on food. Only 1 patient was tube fed.[18]

Second, feeding tube placement itself can cause death. Mortality during percutaneous endoscopic gastrostomy (PEG) tube placement ranges from 0% to 2%[19,20] and perioperative mortality ranges from 6% to 24%.[21-25] In a study of 882 fluoroscopic nasogastric tube placements, 3 patients died of arrhythmia during the procedure.[26]

Third, mortality among tube-fed patients is substantial. Several retrospective studies describe survival after feeding tube placement in patients with eating difficulties, although none are restricted to those with dementia. A review of studies of PEG tubes, each comprising more than 50 patients, found mortality rates of 2% to 27% at 30 days and 50% or more at 1 year.[27] Mortality data from articles not included in that review show 1-month mortality rates ranging from 8% to 67%, and median survival appears to be well under 1 year (TABLE 1). The 2 largest studies included 7369 and 81 105 patients, respectively. The former reported that median survival after PEG tube placement was 7.5 months.[25] The latter found that 63% of patients had died by 1 year after PEG or surgical gastrostomy tube placement and 81.3% were dead by 3 years.[23]

Finally, nonrandomized, retrospective observations of nursing home residents have found no survival advantage with tube feeding. No difference in sur-

vival was found between groups treated with and without tube feeding among 1386 patients with recent progression to severe cognitive impairment. This finding persisted after adjustment for age, prior history of pulmonary aspiration or stroke, presence of swallowing disorder, decubitus ulcer, functional state, resuscitation wishes, and cognitive status.[37] A separate article based on the same data set described 5266 residents with chewing and swallowing problems and reported a significant increase in 1-year mortality among tube-fed patients (risk ratio, 1.44).[38]

We found no published studies suggesting that tube feeding can prolong survival in demented patients with dysphagia.

ARE PRESSURE ULCERS PREVENTED OR IMPROVED BY TUBE FEEDING?

Data linking poor nutrient intake or abnormal markers of nutritional status to pressure ulcers are extremely limited. In a 1995 review[39] that excluded orthopedic and spinal cord injury patients, 13 studies found very weak associations between nutritional status and pressure sores. Data relating nutrient intake to pressure sores were similarly inconclusive. No prospective trials of tube feeding were found, and retrospective studies found only an increased risk or no benefit associated with tube feeding.[39] A MEDLINE search of *enteral nutrition* and *decubitus ulcer* from 1966 through March 1999 found no controlled clinical trials of tube feeding in those with or at risk for pressure ulcers. Two studies that used an administrative database of more than 800 patients during 6 months of follow-up reported that tube feeding was not associated with healing of preexisting pressure sores,[40] nor with protection from new pressure sores.[41]

Bedfast, incontinent patients with dementia who are tube fed are more likely to be restrained[42] and will probably make more urine and stool. Pressure sore outcomes could be worsened. We found no published studies suggesting that tube feeding can improve pressure sore outcomes.

IS THE RISK OF OTHER INFECTIONS REDUCED BY TUBE FEEDING?

Aspiration pneumonia and pressure ulcers, conditions that are sometimes infectious, have already been considered. We searched MEDLINE from 1966 through March 1999 using the terms *enteral nutrition* and *infection* and limited our search to studies involving humans. We found no studies of tube feeding to reduce the risk of other infections—eg, urinary tract, viral, gastrointestinal, or eye infections. In contrast, feeding tubes can cause infection. Nasogastric tubes predispose to infections of the sinuses and middle ear. Gastrostomy tubes have been associated with diarrhea (infectious and noninfectious), cellulitis and abscess (at a rate of 3% to 8%), and rarely with necrotizing fasciitis and myositis.[43] Enteral feeding solutions can be contaminated with bacteria, perhaps leading to gastrointestinal symptoms.[44] Case reports have described streptococcal bacteremia following insertion of a PEG tube[45] and contaminated enteral solution causing nosocomial bacteremia.[44,46,47] We found no published studies suggesting that tube feeding can reduce the risk of infection in dysphagic patients with dementia.

CAN TUBE FEEDING IMPROVE FUNCTIONAL STATUS?

Providing an emaciated patient with artificial feeding is sometimes intended to improve strength, function, or self-care. We reviewed a MEDLINE search of the terms *function*, *functional status*, *recovery of function*, *strength*, or *activi-*

Table 1. Mortality After Feeding Tube Placement: Observational Studies*

Study, y	Intervention	Type of Patient, No.	Outcome
Heimbach,[28] 1970	Surgical feeding tube	Neurogenic, 100	63% Mortality by 1 mo
Matino,[29] 1981	Jejunostomy tube	Neurogenic, 54	33% Mortality by 1 mo, 50% mortality among survivors by 6 mo
Golden et al,[30] 1997	PEG tube	Mixed population, 102	24% Mortality by 6 mo, 55% mortality by 2 y
Kaw and Sekas,[31] 1994	PEG tube	Mixed population, 46	20% Mortality by 1 mo, 59% mortality by 18 mo
Hull et al,[19] 1993	PEG tube	Mixed population, 49	8% Mortality by 1 mo, mean survival <6 mo
Kohli and Block,[20] 1995	PEG tube (review of 4 studies)	Mixed population, 612	16%-30% Mortality by 1 mo
Nevins,[21] 1989	PEG tube or gastrostomy tube	Neurogenic, 22	41% Mortality by 3 wks
Fay et al,[32] 1991	PEG vs nasoenteric tube	Mixed population, 109	50% Mortality by 4 mo for both populations
Hassett et al,[22] 1988	Gastrostomy tube	Neurogenic, 87	20% Mortality by 1 mo, 40% mortality by 1 y
Grant et al,[23] 1998	PEG tube or gastrostomy tube	Mixed population, 81 105	24% Mortality by 1 mo, 63% mortality by 1 y, 81.3% mortality by 3 y
Finocchiaro et al,[24] 1997	PEG tube	Mixed population, 136	9.5% Mortality by 1 mo, 58% mortality by 1 y, 65% mortality by 2 y
Loser et al,[33] 1998	PEG tube	Mixed population, 210	66% Mortality by 1 y
Fisman et al,[34] 1999	PEG tube	Mixed population, 175	18% Mortality by 30 d, 61% mortality by 1 y
Light et al,[35] 1995	PEG tube	Mixed population, 416	9% Mortality by 1 mo
Bergstrom et al,[36] 1995	Gastrostomy tube	Mixed population, 77	21% Mortality by 1 mo, 64% mortality by 1 y

*Neurogenic indicates dementia, cerebrovascular accident, trauma, anoxic brain injury, Parkinson disease, Guillain-Barré syndrome, or motor neuron disease; PEG, percutaneous endoscopic gastrostomy; and mixed population, patients with neurogenic mechanical disorders and cancer.

(Continued)

ties of daily living, and *enteral nutrition* from 1966 through March 1999. In stroke patients, emaciation may be associated with slower functional improvement,[48,49] but we found no study in which a nutritional intervention facilitated recovery of function. Among 100 frail nursing home residents, oral protein supplements produced no improvement in measures of strength or function unless combined with resistance strength training.[50] A retrospective review found that no nursing home patients had improvement in functional status as measured by the Functional Independence Measurement scale during 18 months after PEG tube placement.[31] We found no published studies suggesting that tube feeding can improve function or mitigate its decline in dysphagic demented patients.

DOES TUBE FEEDING IMPROVE PATIENT COMFORT?

We searched MEDLINE from 1966 through March 1999 using the terms *palliative care* and *enteral nutrition*. For many demented patients, data about symptoms and symptom control can be based only on inference. In a prospective observation of palliative care for terminally ill patients with anorexia, primarily with cancer or stroke, few experienced hunger or thirst. Of those who did, relief was achieved with small amounts of food and fluids or by ice chips and lip lubrication.[51]

Patients with amyotrophic lateral sclerosis and dysphagia who had feeding tubes placed continued to cough, have difficulty managing oral secretions, and develop aspiration pneumonia. Hunger and nausea often began or increased after tube placement, and human contact was diminished.[52] Tube-fed patients may be denied the pleasure of eating or made uncomfortable by the tube or frequent repositioning; some require restraints. We found no published studies suggesting that tube feeding makes dysphagic demented patients more comfortable.

ADVERSE EFFECTS

We searched MEDLINE from 1966 through March 1999 using the terms *complication* and *enteral nutrition* and limited our search to studies of humans age 65 years or older. The many adverse effects of tube feeding have been divided into 4 major categories: local or mechanical, pleuropulmonary, abdominal, and other (TABLE 2). The most common adverse effect associated with all types of tube feeding is aspiration pneumonia (0%-66.6%[5]). For PEG tubes, common adverse effects are tube occlusion (2%-34.7%[19,31,57]), leaking (13%-20%[31,32]), and local infection (4.3%-16%[19,31,32,60]). Approximately two thirds of nasogastric tubes require replacement.[32,68]

CONSERVATIVE ALTERNATIVES

Discontinuing nonessential medications may reduce eating difficulties. Among psychiatric patients, swallowing dysfunction and choking have been associated with certain medications, especially those with anticholinergic effects.[71,72] Several drugs cause inattention (eg, sedatives), movement disorders (eg, major tranquilizers), xerostomia (eg, anticholinergics), esophagitis (eg, alen-

Table 2. Burdens and Complications Associated With Tube Feeding

Adverse Effect Category	Type of Tube		
	Nasogastric	Gastrostomy and/or Jejunostomy	Both
Local/mechanical	Erosion/necrosis, bleeding of nose, pharynx, and/or esophagus[52,53,56]; postcricoid perichondritis[54]; tube misplacement into lung or brain[43,66]; high extubation rate; otitis media; sinusitis	Wound dehiscence; bleeding at insertion site; closure or stenosis of stoma; skin excoriation; hematoma; erosion of bumper into abdominal wall	Knotting of tube; tube malfunction[64]; tube migration; discomfort from tube; tube placement failure
Pleuropulmonary	Tracheoesophageal or bronchopleural fistula[55]; hemothorax, hydrothorax, pneumothorax[53,56,57]; tracheobronchial perforation; pneumonitis, lung abscess; pneumomediastinitis; airway obstruction; infusion into lung	Erosion of tube into pleural cavity	Aspiration of feeding
Abdominal	Perforation of esophagus or duodenum; esophageal stricture; esophageal bezoar[58]; reflux esophagitis	Gastric perforation[60]; gastric prolapse; gastrocolic fistula[65]; pneumoperitoneum; pneumatosis intestinalis[61]; prolonged ileus; evisceration[62]; acute gastric dilatation[66]; intussusception[66]; gastric wall defects[66]; laceration of esophagus[64]; peritonitis[54,60,64,67,68]; cellulitis[60,62]; necrotizing fasciitis; abdominal or subphrenic abscess[67]	Diarrhea; gastrointestinal bleeding[62,67]; bowel obstruction[64]; nausea[62]; vomiting; promotion of gastroesophageal reflux[70]
Other	Agitation[62,68]; requirement for frequent repositioning; increased secretions or frequent suctioning	Arrhythmia[26,62]; laryngospasm; shock; mediastinitis[62]	Fluid overload; increased skin moisture; death; use of restraints[53,68,69]; weight loss[53]; metabolic disturbance[53]; loss of gustatory pleasure; anorexia; loss of dignity; loss of social aspects of feeding; altered cosmesis[43,59]

dronate), or anorexia (eg, nonsteroidal anti-inflammatory drugs). Careful attempts to limit use of such medications may yield small but critical increments in eating ability.

Several conservative feeding strategies have been tried. In nursing home patients who were previously less than 80% of ideal body weight, an 8-week trial including staff education, ad lib diets, medication adjustment, assistive devices, changes in the environment, dental care, swallowing evaluations, and augmented energy intake during illness demonstrated that 50% of patients gained an average of 4.5 kg without feeding tubes.[73]

While body position during feeding is poorly studied in patients with dementia, supine (vs semirecumbent) position and length of time supine are risk factors for aspiration of gastric contents in patients receiving ventilatory assistance who are fed by nasogastric tube.[74] Potentially useful techniques include the use of finger foods and preferred foods,[75] strong flavors, hot or cold rather than tepid food, gravy or juices, and enrichers such as cream.[54,76,77] Other helpful techniques are reminders to swallow and swallow multiple times per bolus,[75,77] gentle coughs after each swallow,[77] bolus size of less than 1 teaspoon,[77] liquid supplements,[75] and facilitation techniques such as vibration, gentle brushing, and icing of the cheeks and neck.[52] Additional methods include increasing personal assistance with meals[75]; altering size and frequency of meals; evaluating for other illnesses, especially depression[75]; placing food and fluid well into the mouth[52]; and modifying environmental aspects such as noise level and the company of disruptive patients. These techniques require increased staff time and have not been rigorously studied. They do offer less invasive alternatives to tube feeding.

CONCLUSIONS

We identified no direct data to support tube feeding of demented patients with eating difficulties for any of the commonly cited indications. Tube feeding is a risk factor for aspiration pneumonia; to our knowledge, it has never been shown to be an effective treatment, and neither regurgitated gastric contents nor contaminated oral secretions can be kept out of the airways with a feeding tube. Survival has not been shown to be prolonged by tube feeding. Periprocedure mortality is substantial and prolonged survival of very underweight, dysphagic, demented patients without tube feeding is common. Feeding tubes have not been shown to improve pressure sore outcomes, and in fact, the relationship between nutrient intake and pressure sores is tenuous at best. Improved delivery of nutrients via tube has not been shown to reduce infection, but, on the contrary, feeding tubes have been shown to cause serious local and systemic infection. Functional status has not been improved and demented patients are not made more comfortable with tube feeding while dozens of serious adverse effects have been reported. Conservative measures are available although these are not well studied. Randomized clinical trials of this intervention in this population would be tremendously complex both ethically and clinically.

Several factors likely contribute to the widespread use of tube feeding in elderly patients with dementia. Artificial sustenance retains special status in some discussions about life-sustaining treatment. The apparent validity of tube feeding is very persuasive; if patients have trouble eating, it seems sensible to feed them by any means. Several other factors probably also contribute—administrative convenience, ease of use by nursing staff, and misunderstanding by health care professionals and family members.

A demented patient with eating difficulty can present formidable clinical challenges. We believe that a comprehensive, motivated, conscientious program of hand feeding is the proper treatment. If the patient continues to decline in some clinically meaningful way, tube feeding might be considered as empirical treatment; however, all who help make the decision should be clearly informed that the best evidence suggests it will not help.

REFERENCES

1. Mendelson CL. The aspiration of stomach contents into the lungs during obstetric anesthesia. *Am J Obstet Gynecol*. 1946;52:191-204.
2. DePaso WJ. Aspiration pneumonia. *Clin Chest Med*. 1991;12:269-284.
3. Grunow JE, Al-Hafidh AS, Tunell WP. Gastroesophageal reflux following percutaneous endoscopic gastronomy in children. *J Pediatr Surg*. 1989; 24:42-45.
4. Canel D, Vane B, Golto S. Reduction of lower esophageal sphincter pressure with Stamm gastrostomy. *J Pediatr Surg*. 1987;22:54-58.
5. Finucane TE, Bynum JP. Use of tube feeding to prevent aspiration pneumonia. *Lancet*. 1996;348:1421-1424.
6. Pick N, McDonald A, Bennett N, et al. Pulmonary aspiration in a long-term care setting: clinical and laboratory observations and an analysis of risk factors. *J Am Geriatr Soc*. 1996;44:763-768.
7. Bourdel-Marchasson I, Dumas F, Pinganaud G, Emeriau JP, Decamps A. Audit of percutaneous endoscopic gastrostomy in long-term enteral feeding in a nursing home. *Int J Qual Health Care*. 1997;9:297-302.
8. Langmore SE, Terpenning MS, Schork A, et al. Predictors of aspiration pneumonia: how important is dysphagia? *Dysphagia*. 1998;13:69-81.
9. Feinberg MJ, Knebl J, Tully J. Prandial aspiration and pneumonia in an elderly population followed over 3 years. *Dysphagia*. 1996;11:104-109.
10. Lazarus BA, Murphy JB, Culpeper L. Aspiration associated with long-term gastric versus jejunal feeding: a critical analysis of the literature. *Arch Phys Med Rehabil*. 1990;71:46-53.
11. Fox KA, Mularski RA, Sarfati MR, et al. Aspiration pneumonia following surgically placed feeding tubes. *Am J Surg*. 1995;170:564-566.
12. Henderson CT, Trumbore LS, Mobarhan S, Benya R, Miles TP. Prolonged tube feeding in long-term care: nutritional status and clinical outcomes. *J Am Coll Nutr*. 1992;11:309-325.
13. Koretz RL. Nutritional support: how much for how much [review]? *Gut*. 1986;27(suppl):85-95.
14. Oster MH, Enders SR, Samuels SJ, et al. Megestrol acetate in patients with AIDS and cachexia. *Ann Intern Med*. 1994;121:400-408.
15. Von Roenn JH, Armstrong D, Kotler DP, et al. Megestrol acetate in patients with AIDS-related cachexia. *Ann Intern Med*. 1994;121:393-399.
16. Klein S, Kinney J, Jeejeebhoy K, et al. Nutrition support in clinical practice: review of published data and recommendations for future research directions. *Am J Clin Nutr*. 1997;66:683-706.
17. Franzoni S, Frisoni GB, Boffelli S, Rozzini R, Trabucchi M. Good nutritional oral intake is associated with equal survival in demented and nondemented very old patients. *J Am Geriatr Soc*. 1996;44:1366-1370.
18. Volicer L, Seltzer B, Rheaume Y, et al. Eating difficulties in patients with probable dementia of the Alzheimer type. *J Geriatr Psychiatry Neurol*. 1989;2:189-195.
19. Hull MA, Rawlings J, Murray FE, et al. Audit of outcome of long-term enteral nutrition by percutaneous endoscopic gastrostomy. *Lancet*. 1993;341: 869-872.
20. Kohli H, Block R. Percutaneous endoscopic gastrostomy: a community hospital experience. *Am Surg*. 1995;61:191-194.
21. Nevins MA. Gastrostomy tube-feeding. *N J Med*. 1989;10:779-780.
22. Hassett JM, Sunby C, Flint L. No elimination of aspiration pneumonia in neurologically disabled patients with feeding gastrostomy. *Surg Gynecol Obstet*. 1988;167:383-388.
23. Grant MD, Rudberg MA, Brody JA. Gastrostomy placement and mortality among hospitalized Medicare beneficiaries. *JAMA*. 1998;279:1973-1976.

(Continued)

24. Finocchiaro C, Galletti R, Rovera G, et al. Percutaneous endoscopic gastrostomy: a long-term follow-up. *Nutrition*. 1997;13:520-523.
25. Rabeneck L, Wray NP, Petersen NJ. Long-term outcomes of patients receiving percutaneous endoscopic gastrostomy tubes. *J Gen Intern Med*. 1996; 11:287-293.
26. Gutierrez ED, Balfe DM. Fluoroscopically guided nasoenteric feeding tube placement: results of a 1-year study. *Radiology*. 1991;178:759-762.
27. Cowen ME, Simpson SL, Vettese TE. Survival estimates for patients with abnormal swallowing studies. *J Gen Intern Med*. 1997;12:88-94.
28. Heimbach DM. Surgical feeding procedures in patients with neurological disorders. *Ann Surg*. 1970; 172:311-314.
29. Matino JJ. Feeding jejunostomy in patients with neurologic disorders. *Arch Surg*. 1981;116:169-171.
30. Golden A, Beber C, Weber R, Kumar V, Musson N, Silverman M. Long-term survival of elderly nursing home residents after percutaneous endoscopic gastrostomy for nutritional support. *Nurs Home Med*. 1997;5:382-389.
31. Kaw M, Sekas G. Long-term follow-up of consequences of percutaneous endoscopic gastrostomy (PEG) tubes in nursing home patients. *Dig Dis Sci*. 1994;39:738-743.
32. Fay DE, Poplansky M, Gruber M, Lance P. Long-term enteral feeding: a retrospective comparison of delivery via percutaneous endoscopic gastrostomy and nasoenteric tube. *Am J Gastroenterol*. 1991;86:1604-1609.
33. Loser C, Wolters S, Folsch UR. Enteral long-term nutrition via percutaneous endoscopic gastrostomy (PEG) in 210 patients: a four-year prospective study. *Dig Dis Sci*. 1998;43:2549-2557.
34. Fisman DN, Levy AR, Gifford DR, Tamblyn R. Survival after percutaneous endoscopic gastrostomy among older residents of Quebec. *J Am Geriatr Soc*. 1999;47:349-353.
35. Light VL, Slezak FA, Porter JA, Gerson LW, McCord G. Predictive factors for early mortality after percutaneous endoscopic gastrostomy. *Gastointest Endosc*. 1995;42:330-335.
36. Bergstrom LR, Larson DE, Zinsmeister AR, Sarr MG, Silverstein MD. Utilization and outcomes of surgical gastrostomies and jejunostomies in an era of percutaneous endoscopic gastrostomy: a population-based study. *Mayo Clin Proc*. 1995;70:829-836.
37. Mitchell SL, Kiely DK, Lipsitz LA. The risk factors and impact on survival of feeding tube placement in nursing home residents with severe cognitive impairment. *Arch Intern Med*. 1997;157:327-332.
38. Mitchell SL, Kiely DK, Lipsitz LA. Does artificial enteral nutrition prolong the survival of institutionalized elders with chewing and swallowing problems? *J Gerontol*. 1998;53A:M1-M7.
39. Finucane TE. Malnutrition, tube feeding and pressure sores: data are incomplete. *J Am Geriatr Soc*. 1995; 43:447-451.
40. Berlowitz DR, Brandeis GH, Anderson J, Brand HK. Predictors of pressure ulcer healing among long-term care residents. *J Am Geriatr Soc*. 1997;45:30-34.
41. Berlowitz DR, Ash AS, Brandeis GH, Brand HK, Halpern JL, Moskowitz MA. Rating long-term care facilities on pressure ulcer development: importance of case-mix adjustment. *Ann Intern Med*. 1996;124: 557-563.
42. Quill TE. Utilization of nasogastric feeding tubes in a group of chronically ill, elderly patients in a community hospital. *Arch Intern Med*. 1989;149:1937-1941.
43. Keymling M. Technical aspects of enteral nutrition. *Gut*. 1994;51:S77-S80.
44. Fernandez-Crehuet NM, Jurado D, Guillen JF, Galvez R. Bacterial contamination of enteral feeds as a possible risk of nosocomial infection. *J Hosp Infect*. 1992;21:111-120.
45. Tsai CC, Bradley SF. Group A streptococcal bacteremia associated with gastrostomy feeding tube infections in a long-term care facility. *J Am Geriatr Soc*. 1992;40:821-823.
46. Thurn J, Crossley K, Gerdts A, Maki M, Johnson J. Enteral hyperalimentation as a source of nosocomial infection. *J Hosp Infect*. 1990;15:203-217.
47. Levy J, van Laetham J, Verhaegen G, Perpete C, Butzler JP, Wenzel RP. Contaminated enteral nutrition solution as a cause of nosocomial bloodstream infection: a study using plasmid fingerprinting. *JPEN J Parenter Enteral Nutr*. 1989;13:228-234.
48. Davalos A, Ricart W, Gonzalez-Huix F, et al. Effect of malnutrition after acute stroke on clinical outcome. *Stroke*. 1996;27:1028-1032.
49. Finestone HM, Greene-Finestone LS, Wilson ES, Teasell RW. Prolonged length of stay and reduced functional improvement rate in malnourished stroke rehabilitation patients. *Arch Phys Med Rehabil*. 1996; 77:340-345.
50. Flatarone MA, O'Neill EF, Ryan ND, et al. Exercise training and nutritional supplementation for physical frailty in very elderly people. *N Engl J Med*. 1994; 330:1769-1775.
51. McCann RM, Hall WJ, Groth-Juncker A. Comfort care for terminally ill patients: the appropriate use of nutrition and hydration. *JAMA*. 1994;272:1263-1266.
52. Scott AG, Austin HE. Nasogastric feeding in the management of severe dysphagia in motor neurone disease. *Palliat Med*. 1994;8:45-49.
53. Sullivan RJ. Accepting death without artificial nutrition or hydration. *J Gen Intern Med*. 1993;8:220-224.
54. Vreugde S. Nutritional aspects of dysphagia. *Acta Otorhinolaryngol Belg*. 1994;48:229-234.
55. Miller KS, Tomlinson JR, Sahn SA. Pleuropulmonary complications of enteral tube feedings. *Chest*. 1985;88:230-233.
56. Stolke D, Winkelmuller W. Perforating craniocerebral trauma as a complication of a nasogastric feeding tube [in German]. *Anasth Intensivther Notf Med*. 1982;17:104-105.
57. Roubenoff R, Ravich WJ. Pneumothorax due to nasogastric feeding tubes. *Arch Intern Med*. 1989; 149:184-188.
58. Blasco Navalpotro MA, Zaragoza Crespo R, Malaga Lopez A, Alfonso Moreno V. Esophageal bezoar: an exceptional complication of enteral nutrition [in Spanish]. *Rev Clin Esp*. 1998;198:487-488.
59. Park RH, Allison MC, Lang J, et al. Randomized comparison of percutaneous endoscopic gastrostomy and nasogastric tube feeding in patients with persisting neurological dysphagia. *BMJ*. 1992;304:1406-1409.
60. Larson DE, Burton DD, Schroeder KW, DiMagno EP. Percutaneous endoscopic gastrostomy: indications, success, complications, and mortality in 314 consecutive patients. *Gastroenterology*. 1987;93:48-52.
61. Zern RT, Clarke-Pearson DL. Pneumatosis intestinalis associated with enteral feeding by catheter jejunostomy. *Obstet Gynecol*. 1985;65(3 suppl):81S-83S.
62. Wasiljew BK, Ujiki GT, Beal JM. Feeding gastrostomy: complications and mortality. *Am J Surg*. 1982; 143:194-195.
63. Peck A, Cohen CE, Mulvihill MN. Long-term enteral feeding of aged, demented nursing home patients. *J Am Geriatr Soc*. 1990;38:1195-1198.
64. Adams MB, Seabrook GR, Quebbeman EA, Condon RE. Jejunostomy: a rarely indicated procedure. *Arch Surg*. 1986;121:236-238.
65. Ghosh S, Eastwood MA, Palmer KR. Acute gastric dilatation—a delayed complication of percutaneous endoscopic gastrostomy. *Gut*. 1993;34:859-860.
66. Levine CD, Handler B, Baker SR, et al. Imaging of percutaneous tube gastrostomies: spectrum of normal and abnormal findings. *AJR Am J Roentgenol*. 1995;164:347-351.
67. Cogen R, Weinryb J, Pomerantz C, Fenstemacher P. Complications of jejunostomy tube feeding in nursing facility patients. *Am J Gastroenterol*. 1991; 86:1610-1613.
68. Ciocon JO, Silverstone FA, Graver LM, Foley CJ. Tube feedings in elderly patients: indications, benefits, and complications. *Arch Intern Med*. 1988;148: 429-433.
69. Baeten C, Hoefnagels J. Feeding via nasogastric tube of percutaneous endoscopic gastrostomy: a comparison. *Scand J Gastroenterol*. 1992;27:95-98.
70. Russell GN, Yam PC, Tran J, et al. Gastroesophageal reflux and tracheobrachial contamination after cardiac surgery: should a nasogastric tube be routine? *Anesth Analg*. 1996;83:228-232.
71. Craig TJ. Medication use and deaths attributed to asphyxia among psychiatric patients. *Am J Psychiatr*. 1980;137:1366-1373.
72. Craig TJ, Richardson MA. "Cafe coronaries" in psychiatric patients [letter]. *JAMA*. 1982;248:2114.
73. Abbasi AA, Rudman D. Undernutrition in the nursing home: prevalence, consequences, causes, and prevention. *Nutr Rev*. 1994;52:113-122.
74. Torres A, Serra-Batlles J, Ros E, et al. Pulmonary aspiration of gastric contents in patients receiving mechanical ventilation: the effect of body position. *Ann Intern Med*. 1992;116:540-543.
75. Morley JE. Dementia is not necessarily a cause of undernutrition. *J Am Geriatr Soc*. 1996;44:1403-1404.
76. Boylston E, Ryan C, Brown C, Westfall B. Preventing precipitous weight loss in demented patients by altering food texture. *J Nutr Elder*. 1996;15:43-48.
77. Horner J, Massey EW, Riski JE, Lathrop DL, Chase KN. Aspiration following stroke: clinical correlates and outcome. *Neurology*. 1988;38:1359-1362.

12
Health Screening and Disease Prevention

Vivian S. Argento, MD

Commentary

It is generally accepted that health screening to prevent and identify disease early enough to permit cure is an appropriate medical goal. The research on disease prevention, however, excludes older adults and focuses on mortality end points that may not be appropriate measures for successful aging where function and quality of life are paramount. By minimizing morbidity and mortality of disease and compressing it into the very end of a person's lifespan, the patient can maximize function, productivity, and quality of life despite the presence of disease (1).

Various organizations publish guidelines to help physicians offer appropriate screening and prevention services to their patients. However, the heterogeneity of an aging cohort makes it difficult to generalize and create guidelines for effective screening. In 1984, the U.S. Public Health Service created the U.S. Preventative Services Task Force (USPSTF). This organization, which since 1998 has been sponsored by the Agency for Healthcare Research and Quality (AHRQ), provides evidence-based reviews of healthcare screening strategies and is considered the "gold standard" for screening and prevention recommendations (2). Its recommendations for healthcare screening in the geriatric population are often limited by the lack of research on the effectiveness of screening in this population. Consequently, the decisions of when to screen and when to stop screening need to be individualized to the specific patient based on their preferences, life expectancy, and competing risks for death and functional impairment.

To date, the best summary of the decision-making process for screening and prevention in the elderly can be found in an article (reproduced here) by Walter and Covinsky. Although this article focuses on cancer screening, the decision-making process can be extrapolated to other screening and prevention modalities. Using equations developed and published by Beck in 1982, they suggest beginning with a determination of a patient's life expectancy and risk of dying from the disease being screened for to arrive at an approximation of the patient's specific mortality (3, 4). Taking the data tabulated by Welch and colleagues in 1996, physiologic age, rather than chronologic age (5), can be combined with current life expectancy tables that aggregate competing risk (6); and the physician can estimate the patient's risk of dying from the condition for which screening is being performed. The physician can use this information to arrive at an idea of the overall magnitude of the screening's effectiveness. Then, taking into account a patient's values and preferences, the physician can discuss the risks and benefits of the proposed procedures and the impact of the screening on the patient's function, quality of life, and mortality. The physician-patient team is, in this way, able to arrive at a reasonable personalized approached to screening and prevention.

The number of older adults is increasing greatly. This cohort is very diverse in health and function. Until further research clarifies the exact effectiveness of screening and prevention for older patients, physicians must rely on expert opinion and extrapolate from the limited information currently available.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

References

1. Fries JF. The Compression of Morbidity. Milbank Mem Fund Q—Health Soc. 1983;61:397–419. (Reproduced in: Fries JF. The compression of morbidity. Milbank Q 2005;83:801–823).
2. USPSTF: Guide to Clinical Preventative Services; http://www.ahrq.gov/clinic/cps3dix.htm, accessed June 16, 2006.
3. Beck JR, Kassirer JP et al. A Convenient Approximation of Life Expectancy (the DEALE). I. Validation of the Method. Am J Med 1982;73:883–888.
4. Beck JR, Pauker SG. A Convenient Approximation of Life Expectancy (the DEALE). II. Use in Clinical Decision-Making. Am J Med 1982;73:889–897.
5. Welch GH, Albertsen PC et al. Estimating Treatment Benefits for the Elderly: The Effect of Competing Risks. Ann Intern Med 1996;24:577–584.
6. National Vital Statistics Report for 2003 (preliminary data) with life expectancy table for 2003; http://www.cdc.gov/nchs/data/nvsr/nvsr53/nvsr53_15.pdf, accessed June 16, 2006.

SPECIAL COMMUNICATION

Cancer Screening in Elderly Patients
A Framework for Individualized Decision Making

Louise C. Walter, MD
Kenneth E. Covinsky, MD, MPH

Considerable uncertainty exists about the use of cancer screening tests in older people, as illustrated by the different age cutoffs recommended by various guideline panels. We suggest that a framework to guide individualized cancer screening decisions in older patients may be more useful to the practicing clinician than age guidelines. Like many medical decisions, cancer screening decisions require weighing quantitative information, such as risk of cancer death and likelihood of beneficial and adverse screening outcomes, as well as qualitative factors, such as individual patients' values and preferences. Our framework first anchors decisions through quantitative estimates of life expectancy, risk of cancer death, and screening outcomes based on published data. Potential benefits of screening are presented as the number needed to screen to prevent 1 cancer-specific death, based on the estimated life expectancy during which a patient will be screened. Estimates reveal substantial variability in the likelihood of benefit for patients of similar ages with varying life expectancies. In fact, patients with life expectancies of less than 5 years are unlikely to derive any survival benefit from cancer screening. We also consider the likelihood of potential harm from screening according to patient factors and test characteristics. Some of the greatest harms of screening occur by detecting cancers that would never have become clinically significant. This becomes more likely as life expectancy decreases. Finally, since many cancer screening decisions in older adults cannot be answered solely by quantitative estimates of benefits and harms, considering the estimated outcomes according to the patient's own values and preferences is the final step for making informed screening decisions.

JAMA. 2001;285:2750-2756 www.jama.com

CURRENTLY CONSIDERABLE UNcertainty exists about the best use of cancer screening tests in older people.[1] Part of this stems from a lack of randomized controlled trials of screening interventions that have included patients older than 75 years. This requires physicians to extrapolate data about the effectiveness of screening in younger patients and apply it to older patients. Even if the effectiveness of screening is similar in the elderly population, uncertainty remains about how to apply data from randomized trials to an individual elderly patient. Trials show average effectiveness of an intervention, but they generally do not address individual patient characteristics, such as comorbid conditions or functional status, which may change the likelihood of receiving benefit or harm from screening. Care in applying data from trials to individuals is especially important for older adults, since individual variability in health status and disability increases with age.[2]

The important issues that need to be considered when making individualized cancer screening decisions in elderly patients are not addressed by the often conflicting recommendations made by guideline panels and organizations that base their recommendations primarily on age. For example, for mammography, the US Preventive Services Task Force recommends that screening cease at age 70 years,[1] the American College of Physicians discourages screening after age 75 years,[3] the American Geriatrics Society recommends possible discontinuation at age 85 years,[3] and the American Cancer Society recommends annual screening for all women older than 40 years with no upper age limit.[4] Although most health care professionals would agree that clinical judgment should supersede age-cutoff guidelines when the potential harms or benefits from a screening test strongly weigh in a particular direction, there is little guidance about how to apply clinical judgment to screening decisions in older people.

We propose that a conceptual framework to guide cancer screening decisions in older patients may be more useful than age guidelines to the practicing clinician. Frameworks for weighing the benefits and harms of screening have been developed,[5-7] but none specifically address how to organize informed decision making for elderly patients that include consideration of an individual patient's characteristics and preferences. Like many medical decisions, informed screening decisions are best made by weighing quantitative estimates of benefits and risks with more subjective qualitative judgments of val-

For editorial comment see p 2776.

Author Affiliation: Division of Geriatrics, San Francisco Veterans Affairs Medical Center and Univeristy of California, San Francisco.
Corresponding Author and Reprints: Louise C. Walter, MD, Division of Geriatrics, VA Medical Center 111G, 4150 Clement St, San Francisco, CA 94121 (e-mail: louisew@itsa.ucsf.edu).

(Continued)

ues and preferences. Our framework first anchors decisions through quantitative estimates of life expectancy, risk of cancer death, and screening outcomes based on published data. Our framework then concludes with qualitative consideration of the estimated benefits and harms based on a patient's unique values and preferences.

Risk of Dying

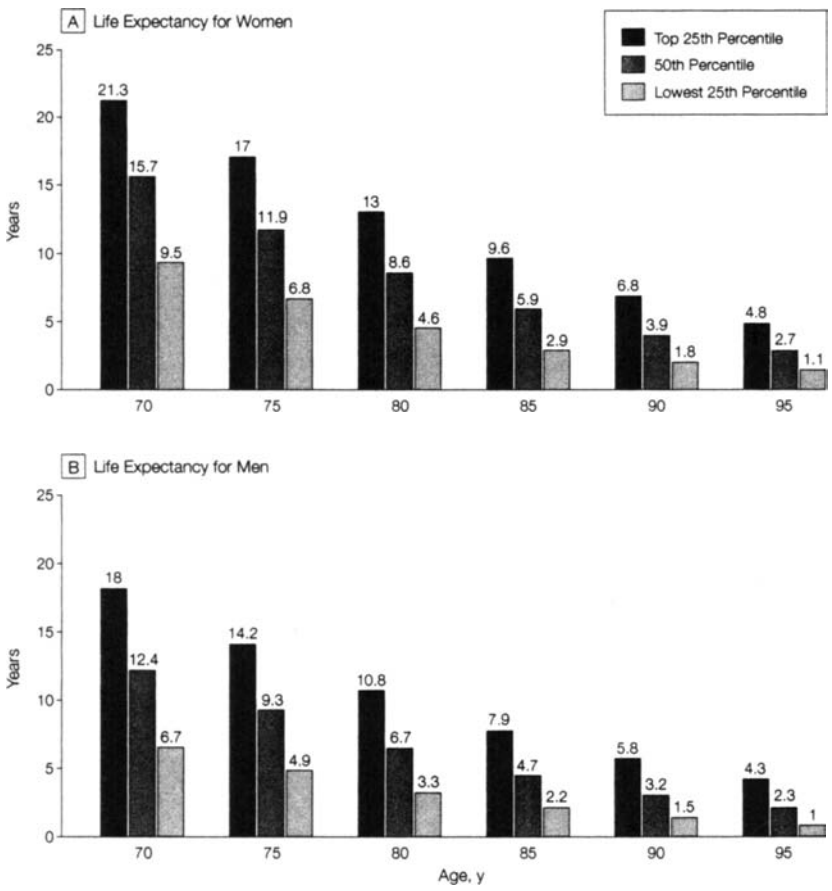
Our framework starts with considering the risk of dying of a screen-detectable cancer since the maximum potential benefit of screening is defined by a person's risk of dying of a screen-detectable cancer, not his or her risk of being diagnosed as having cancer. Finding an asymptomatic cancer in a person who will die of something else before the cancer would become symptomatic does not benefit the patient. The risk of death due to cancer can be estimated by considering the life expectancy of the individual and the age-specific mortality rate of the particular cancer. With advancing age, the mortality rates of most cancers increase,[8] but overall life expectancy decreases.[9] The need to weigh these 2 opposing factors makes cancer screening decisions in the elderly complex.

Median life expectancies of persons in the United States are summarized in tables of vital statistics by age and sex, but there is great variation in life expectancy within each age-sex subgroup.[10] Therefore, although it is useful to know median life expectancies, it is more helpful to have a general idea of the distribution of life expectancies at various ages. For example, when making screening decisions about a 75-year-old woman, it is useful to know that approximately 25% of 75-year-old women will live more than 17 years, 50% will live at least 11.9 years and 25% will live less than 6.8 years.[9] The FIGURE presents the upper, middle, and lower quartiles of life expectancy for the US population according to age and sex and illustrates the substantial variability in life expectancy that exists at each age. Although it is impossible for physicians to predict the exact life expectancy of an individual patient, it is possible for physicians to make reasonable estimates of whether a patient is likely to live substantially longer or shorter than an average person in his/her age cohort. For example, Fried et al[11] prospectively stratified elderly community-living persons into groups whose 5-year mortality ranged from 2% for the healthy patients to 39% for those with multiple cardiovascular risk factors. Such estimates of life expectancy would allow for better estimations of potential benefits and risks of screening than focusing on age alone.

There are many variables physicians can use to estimate whether an older patient is typical of someone at the middle of their age-sex cohort or is more like someone in the upper or lower quartiles. For example, the number and severity of comorbid conditions and functional impairments are strong predictors of life expectancy in older people.[12,13] Congestive heart failure (CHF), end-stage renal disease, oxygen-dependent chronic obstructive lung disease, or severe functional dependencies in activities of daily living are examples of risk factors that would cause an elderly person to have a life expectancy substantially below the average for his/her age.[14] The absence of significant comorbid conditions or presence of functional status considerably better than age-group averages identifies older adults who are likely to live longer than average.

Life expectancy estimates can be used to approximate the risk of dying of a screen-detectable cancer, which is useful in deciding whether a person is likely to benefit from screening. Consider an

Figure. Upper, Middle, and Lower Quartiles of Life Expectancy for Women and Men at Selected Ages



Data from the Life Tables of the United States.[9]

80-year-old woman with class IV CHF who is considering screening mammography. Although the median life expectancy of an 80-year-old woman is 8.6 years, this patient, because of her severe comorbidity, is probably in the lower quartile of life expectancy and is likely to live less than 5 years. Next, the risk of dying of breast cancer can be approximated by multiplying life expectancy by the age-specific breast cancer mortality rate. The Surveillance, Epidemiology, and End Results Program (SEER)[15] reports that women aged 80 to 84 years have an annual breast cancer mortality rate of 157/100000. Therefore, the risk of dying of breast cancer for an 80-year-old woman who is expected to live less than 5 years is estimated to be less than $5 \times 157/100000 = 0.8\%$.

TABLE 1 presents population-based estimates of the risk of dying of the 3 major screened cancers according to sex, age, and life expectancy. These risks were calculated by multiplying life expectancy by published age-specific cancer mortality rates and should be viewed as estimates that provide some quantitative knowledge of the average risk of dying of a screen-detectable cancer to help anchor screening decisions. Table 1 emphasizes the importance of considering life expectancy when making screening decisions, as illustrated by the example that an 85-year-old woman in the upper quartile of life expectancy has more chance of benefiting from cancer screening than a 75-year-old woman in the lower quartile. Some patients may have additional factors that increase their risk for dying of certain cancers, such as family history or race, requiring individualized tailoring of our baseline estimates. However, many risk factors become less important relative to older age and life expectancy.[17]

Benefits of Cancer Screening

The next step is to consider the potential benefits of screening for specific cancers. If screening were 100% effective at preventing cancer death, the patient's benefit would approximate his/her risk of dying of a screen-detectable cancer. However, the actual likelihood of benefit from screening will always be substantially less than this value, since screening may miss early-stage malignancies, detect disease too advanced or aggressive to respond to treatment, or detect indolent cancers that are not likely to produce clinical symptoms.[1]

Even screening, effective in early detection, may not benefit patients with short life expectancies since the benefit from screening is not immediate. For example, in the randomized controlled trials of fecal occult blood testing (FOBT)[18-20] and mammography[21,22] the cancer-specific survival curves between the screened and unscreened groups do not separate significantly until at least 5 years after the start of screening. This period could be even longer for patients older than 70 years since some evidence suggests that the length of time that a screen-detectable cancer remains clinically asymptomatic increases with advancing age for both breast and colorectal cancer.[23-25] The reason for the delay between the onset of screening and a survival benefit is probably because screening results in benefit by detecting cancers that would have resulted in death after more than 5 years. Cancers destined to result in death before 5 years may be too aggressive for patients to benefit from early detection and treatment. This suggests that older patients who have life expectancies of less than 5 years will not derive survival benefit from cancer screening.

For patients with estimated life expectancies greater than 5 years, it is important to consider what is known about the absolute benefit of cancer screening tests. The absolute benefit of a screening test can be conveyed by the absolute risk reduction (the absolute difference in proportions of patients with a given outcome from 2 treatments or actions), or more effectively by calculating the number needed to screen (NNS), which is the reciprocal of the absolute risk reduction.[26,27] Considering patients at average risk for developing a screened cancer, the approximate NNS to prevent 1 cancer-specific death is listed in TABLE 2 for screening tests that have been shown to be effective in reducing cancer-specific mortality. Although prostate-

Table 1. Risk (Percentage) of Dying of Cancer in Remaining Lifetime for Men and Women at Selected Ages and Life Expectancy Quartiles*

	Age 50 y			Age 70 y			Age 75 y			Age 80 y			Age 85 y			Age 90 y		
	Life Expectancy of Women, y																	
	40	33	24.5	21.3	15.7	9.5	17	11.9	6.8	13	8.6	4.6	9.6	5.9	2.9	6.8	3.9	1.8
Cancer																		
Breast	4.4	3.1	2.0	3.3	2.2	1.2	2.8	1.8	0.9	2.4	1.5	0.7	1.9	1.2	0.6	1.4	0.8	0.4
Colorectal	3.8	2.2	1.0	3.5	2.0	0.9	3.3	1.9	0.9	3.0	1.8	0.8	2.5	1.6	0.8	1.8	1.0	0.5
Cervical	0.34	0.26	0.18	0.22	0.15	0.08	0.19	0.12	0.07	0.15	0.10	0.05	0.12	0.07	0.04	0.08	0.05	0.02
	Life Expectancy of Men, y																	
	36	28.5	19.6	18	12.4	6.7	14.2	9.3	4.9	10.8	6.7	3.3	7.9	4.7	2.2	5.8	3.2	1.5
Cancer																		
Colorectal	4.1	2.3	1.0	3.8	2.1	0.9	3.5	1.9	0.8	3.2	1.8	0.8	2.7	1.6	0.8	2.0	1.1	0.5

*Life expectancy quartiles correspond to upper, middle, and lower quartiles as presented in the Figure. Data are presented as percentages. Risks for 50-year-old patients are included for comparison. Risks were calculated by multiplying life expectancy by age-specific cancer mortality rates from Surveillance, Epidemiology, and End Results (SEER) *Cancer Statistics Review 1973-1996*.[15] Since cancer screening in the United States among elderly patients remains low, these cancer mortality risks approximate those expected for patients who have not received regular cancer screening.[16] For example, to estimate the risk of dying of breast cancer for an 80-year-old woman with a life expectancy of 8.6 years, we multiplied the annual breast cancer mortality rate for women aged 80 to 84 years (157/100 000) by 5 = 0.785%. Next we multiplied the annual mortality rate for women older than age 80 years (200.5/100 000) by 3.6 = 0.722% and added these numbers to get the overall risk of 1.5%.

(Continued)

specific antigen (PSA) testing is frequently performed, we did not include it in the table because no compelling evidence currently demonstrates that PSA testing reduces prostate cancer mortality. We calculated the numbers in Table 2 by applying the reported risk reduction of each screening test to the baseline risks for dying from a screen-detectable cancer from Table 1. All the numbers in Table 2 assume a 5-year delay between the onset of screening and survival benefit.[31] The numbers are presented according to age and life expectancy since life expectancy defines the potential number of years available for screening. For example, 240 very healthy 80-year-old women would have to be screened with mammography during their remaining lifetime to prevent 1 death from breast cancer. This value is similar to the NNS of 226 for screening 50-year-old women with mammography for 24.5 years since the mortality rate from breast cancer increases with age and healthy older women have substantial life expectancies. The values in Table 2 illustrate that the NNS dramatically increases as life expectancy decreases from the upper to the lowest quartile.

Our estimates are based on published data of cancer mortality rates and screening efficacy, but the strength of the evidence that cancer screening is effective in older adults is limited by the small number of older patients included in screening trials. Likewise, for elderly patients who have received regular screening in the past, there are no data about whether some benefit from screening persists for several years after stopping regular screening. However, based on the relative risk reductions seen in clinical trials in younger patients, our estimates can be tailored to reflect that the baseline risk of dying from breast cancer may be reduced by approximately 26% in patients who have received regular screening mammography in the past, and the baseline risk of dying from colorectal cancer may be reduced by 18% in patients who have received regular FOBT screening.[19,20,22] For cervical cancer, decision models suggest that elderly women who have had repeated normal Pap smears during their reproductive years do not benefit from continued Pap testing.[32,33]

There is not a fixed NNS for each screening test at each age. There are several factors, beyond chronological age, that determine the NNS, which include the individual's estimated baseline risk of dying from a screen-detectable cancer, the relative risk reduction of the screening test, and the life expectancy over which the patient is expected to be screened. By remembering which factors determine the NNS, we can better estimate the likelihood that an elderly patient might derive survival benefit.

Harms of Cancer Screening

Considering the potential harms of screening is the third step in our framework. All cancer screening tests potentially pose direct and indirect harms. Harms that would be accepted to treat a symptomatic patient with known disease are less acceptable when they are caused by screening tests, which benefit only a few individuals but expose all screened individuals to the harms. In our framework, harms from each round of

Table 2. Number Needed to Screen (NNS) Over Remaining Lifetime to Prevent 1 Cancer-Specific Death for Women and Men at Selected Ages and Life Expectancy Quartiles*

	RRR (95% CI)	Age 50 y			Age 70 y			Age 75 y			Age 80 y			Age 85 y			Age 90 y		
		Life Expectancy of Women, y																	
		40	33	24.5	21.3	15.7	9.5	17	11.9	6.8	13	8.6	4.6	9.6	5.9	2.9	6.8	3.9	1.8
Screening test																			
Mammography	0.26 (0.17-0.34)†	95	133	226	142	242	642	176	330	1361	240	533	...	817	2131	...	1066	...	...
Papanicolaou smear	0.60‡	533	728	1140	934	1521	4070	1177	2113	8342	1694	3764	...	2946	15 056	...	7528	...	...
Fecal occult blood	0.18 (0.01-0.32)§	145	263	577	178	340	1046	204	408	1805	262	581	...	455	2326	...	1163	...	...
		Life Expectancy of Men, y																	
		36	28.5	19.6	18	12.4	6.7	14.2	9.3	4.9	10.8	6.7	3.3	7.9	4.7	2.2	5.8	3.2	1.5
Screening test																			
Fecal occult blood‖	0.18 (0.01-0.32)§	138	255	630	177	380	1877	207	525	...	277	945	...	554	...	...	2008	...	...

*Life expectancy quartiles correspond to upper, middle, and lower quartiles as presented in the Figure. The NNS is based on the baseline risk of dying of a screen-detectable cancer (Table 1), the relative risk reduction (RRR) of the screening test, and the life expectancy over which the patient is expected to be screened. Patients with life expectancies of less than 5 years are unlikely to derive any survival benefit from cancer screening, which is denoted by ellipses. The numbers for 50-year-old patients are included for comparison. For example, we first estimated the risk of dying of breast cancer for an 80-year-old woman with a life expectancy of 8.6 years who has regular mammography screening during this period. We assumed a 5-year lag before mortality benefit starts. We mutiplied the annual breast cancer mortality rate for women aged 80 years to 84 (157/100 000) by 5, which equals 0.785%. Next we multiplied the annual rate for women older than age 85 years (200.5/100 000) by 3.6 and reduced this number by 26% (the RRR of mammography), which equals 0.534%. Adding these numbers together gives an estimated risk of 1.319%. Since the estimated risk of dying of breast cancer without screening is 1.5068% (Table 1), the absolute risk of dying of breast cancer without screening is 1.5068% minus 1.319%, which is 0.1878%. The NNS is 1/0.001878 and equals 533.

†RRR estimate for breast cancer mortality from a meta-analysis of screening mammography in women aged 50 to 74 years.[22]

‡RRR estimate represents mid point of reported mortality reductions from population-based studies of screening papanicolaou smears in women aged 20 through 79 years since no randomized controlled trials have been done.[28,29]

§RRR estimate for colorectal cancer mortality from a randomized study of screening biennial fecal occult blood testing (nonrehydrated) in people aged 45 to 75 years.[19]

‖Alternative methods for colorectal cancer screening, such as colonoscopy, would have lower numbers to treat since the RRR of these tests are probably higher than that of fecal occult blood testing.[30] Fecal occult blood testing is presented since it is the only test for colorectal cancer screening that has been studied in randomized controlled trials.

screening are considered according to the likelihood of 3 types of adverse effects: (1) complications from additional diagnostic procedures due to inaccurate test results, (2) identification and treatment of clinically unimportant cancers, and (3) psychological distress from screening.

For screening mammography approximately 77 to 86 per 1000 women older than 70 years will require additional testing after screening, and about 86% of these women will not have invasive cancer.[34] Thus, there is approximately 1 false-positive mammogram result for every 15 mammographies performed. The most common test following an abnormal result is diagnostic mammography, followed by biopsy. There is a small complication rate from biopsy, which includes infection and scarring.[35] In terms of colon cancer screening, the standard workup for an abnormal FOBT result is a colonoscopy. Approximately 89 to 96 per 1000 patients older than 65 years will require additional workup after FOBT,[36] but this number increases with advancing age and slide rehydration to as high as 160 per 1000 for patients older than 80 years who undergo rehydrated FOBT.[37] Approximately 86% to 98% of patients with a positive FOBT will not have an early-stage cancer. Complications of colonoscopy include perforation (1/1000), serious bleeding (3/1000), and cardiorespiratory events from intravenous sedation (5/1000).[38]

Individuals who were found not to have cancer after workup of an abnormal screening result clearly have experienced burdens due to screening. However, what is often forgotten is that in elderly patients some of the greatest harms from screening occur by finding and treating cancers that would never have become clinically significant. As life expectancy decreases, the probability of identifying an inconsequential cancer increases.[39] The risk of identifying a clinically insignificant lesion also depends on the likelihood that screening will detect certain neoplasms that are unlikely to progress to symptoms in elderly patients, such as ductal carcinoma in situ (DCIS). For women older than 70 years, there is roughly a 1 in 1000 chance that screening mammography will identify DCIS that would not have been found without screening.[40] Only 7% to 25% of DCIS lesions progress to invasive cancer within 5 to 10 years.[41-43] Yet, due to the inability to distinguish which lesions will progress to invasive cancer, many older women with DCIS undergo mastectomy or lumpectomy combined with radiation.[44] Women who have surgery for DCIS that would have never become symptomatic in their lifetime have suffered serious harm.

Besides the physical harms, the psychological distress caused by cancer screening may be substantial for some elderly patients and caregivers.[45,46] Potential psychological harms range from the emotional pain of a diagnosis of cancer in patients whose lives were not extended by screening through the alarm of false-positive results to the stress of undergoing the screening test itself. Since cancer is one of the most feared diseases in the Western world,[47] some of the greatest psychological harm from screening occurs when a clinically insignificant cancer is identified. False-positive results can also lead patients to "temporarily experience the diagnosis of cancer,"[48] and often these anxieties, once aroused, cannot be allayed easily. A study[49] of women aged 50 through 74 years found that 47% of women who had false-positive "high-suspicion" mammogram results reported mammography-related anxiety even 3 months after learning that they did not have cancer. Similarly, substantial anxiety and discomfort may occur while undergoing the screening test itself or further diagnostic studies, especially among patients with a high predisposition to anxiety.[50] Also, elderly patients may have cognitive, physical, or sensory problems that make screening tests and further workup particularly difficult, painful, or frightening.[51] Considering factors that increase the likelihood of harm is vital to making appropriate screening decisions.

Values and Preferences

The final component of our framework is to assess how patients view the potential harms and benefits we have detailed and how to integrate patients' values and preferences into screening decisions. Cancer screening decisions have traditionally followed the public health strategy in which experts weigh the risks and benefits of an intervention and decide what is appropriate for certain populations. However, this strategy omits patient preferences and values. Since many cancer screening decisions in older adults will not be answered solely by quantitative assessment of benefits and harms, talking to older patients about screening preferences and values is especially important.

The degree to which individuals will discuss their preferences or be involved in screening decisions will vary.[52-54] Ideally, physicians over time should learn about patients and their families and come to understand their values and preferences. The value placed on different health outcomes will vary among patients, as will preferences for screening.[55,56] For example, some women undergoing screening mammography report "peace of mind" after a negative screening result; whereas, women with dementia may receive no such comfort.[57] Physicians should also consider a patient's usual approach to medical decision making to decide how to approach the discussion of screening. In some cases the physician will need to find out the patient's values, apply them to the known risks and benefits of screening, and make a formal recommendation. For other patients, the physician will want to discuss the risks and benefits with the patient and allow the patient to apply his/her values to the problem and come to a decision together. For patients with dementia, discussion about preferences should be held with an involved caregiver. However, it should be remembered that despite being unable to articulate consent, many patients with dementia can still effectively communicate refusal.[58] If a patient is frightened or agitated by a screening test, the caregiver and physician should consider forgoing it. Also, there should be a general discussion prior to screening about the possible procedures and treatments that may be required after an abnormal screening result. Patients who would not

(Continued)

want further workup or treatment of an abnormal result should not be screened.

Where there is patient misperception of cancer risk or screening efficacy physicians should provide information to facilitate informed decision making. Effective risk communication depends on qualitative assessment of patient values, emotional receptivity, communication styles, and intellectual abilities as well as quantitative understanding of benefits and harms of screening options.[59] Although there is evidence that patients are more inclined to overestimate the probability of risks and benefits presented in relative terms compared with those presented in absolute terms (absolute risk reduction or NNS), there is little research on the use of NNS as a communication tool.[60] Our framework uses NNS to present quantitative information since it is a single number that indicates in absolute terms the effort required to achieve a particular goal.[26,61]

Communicating quantitative information and integrating it with patient values is often difficult and will require time during a busy office visit.[62] Our framework is intended to help physicians by providing an organizational system to think through these often complex decisions. In addition, physicians can provide patients with decisional aids, such as pamphlets, videos, or interactive computers, as time-saving supplements to their own discussions with patients.[63]

Case Scenarios

To illustrate the application of our framework consider the following cases:

Case 1. Ms A is a 75-year-old white woman with diabetes, severe dementia, and functional dependency in all activities of daily living. She lives with her daughter and has no prior history of any cancer screening tests.

Case 2. Ms B is an 80-year-old white woman who is widowed, living with her sister. She has no comorbid conditions, walks 3 miles a day, and cooks and cleans for her older sister. She has no prior history of any cancer screening tests.

Our framework starts with estimating the risk of dying of cancer according to estimated life expectancy and cancer-specific mortality rates. Although Ms A is younger than Ms B, her estimated life expectancy is much less. Ms A's severe dementia and functional dependency place her in the lowest quartile of life expectancy for her age. Ms B, on the other hand, has no comorbid conditions and much better functional status than an average 80-year-old woman. She probably falls in the upper quartile of life expectancy for her age, which would give her an estimated life expectancy of 13 years. She, therefore, is at higher risk for dying of a currently occult cancer than Ms A.

The next step is to consider the probability of benefiting from screening according to the patient's estimated risk of dying of cancer and the efficacy of the screening test. Since it takes at least 5 years after starting screening to see a survival benefit between screened and unscreened groups, Ms A is unlikely to derive benefit from any cancer screening test. On the other hand, 80-year-old women with similar life expectancies to Ms B have approximately a 1 in 240 chance for survival benefit from screening mammography, a 1 in 262 chance of survival benefit from screening FOBT, and a 1 in 1694 chance of survival benefit from screening Pap smears. For comparison, it is estimated that 2500 40-year-old women would need to have regular screening mammography for 10 years to prevent 1 death by age 80 years.[31]

But the harms of screening also need to be considered. Ms A has significant dementia and may not understand why her breasts need to be squeezed during mammography or why a speculum needs to be inserted into her vagina to do a Pap smear, so her psychological distress may be substantial. Also, her family members are unsure whether they would want to pursue any type of surgery if a screening result were abnormal, since their main goal is to prevent her from suffering. Ms B, on the other hand, voices her concern about her risk for cancer. She accepts the risks of false-positive examinations and finding clinically insignificant disease. However, she reports that having the tests would give her "peace of mind."

This leads to the final step in our framework, which is the assessment of the patient's values and preferences. Discussion with Ms A's family members shows that preserving her quality of life is their most important goal. Ms A has avoided physicians all her life and does not like undergoing tests. She becomes agitated if anything interrupts her daily routine. Discussion with Ms B reveals that she worries about her health and wants to have a mammogram, Pap smear, and FOBT.

The decision to recommend against cancer screening for Ms A is clear given her low likelihood of benefit, increased likelihood of harm, and her preferences for focusing on quality-of-life issues and avoiding medical testing. Determining whether to screen Ms B may be a "close-call" if one only compares her potential screening benefits with its harms, but the decision to recommend screening becomes clear after she states her preferences.

Of course, it is more difficult when patients with limited life expectancies want screening examinations that offer them little chance of benefit. Recent evidence suggests that patients will frequently withdraw requests for unhelpful treatments when the rationale is discussed with them.[64] Our framework can help stimulate discussions with patients and promote informed cancer screening decisions.

CONCLUSION

We present a framework for guiding physicians and elderly patients to more informed cancer screening decisions by detailing the benefits and harms that need to be weighed when making screening decisions. Patient preferences then act like a moveable fulcrum of a scale to shift the magnitude of the harms or benefits that are needed to tip the decision toward a screening option.

Our framework illustrates potential difficulties for reimbursement and quality assessment systems that apply guidelines based on administrative data to decisions that involve estimating life expectancy and weighing potential benefits and harms according to patient val-

ues and preferences. Third-party payers who wish to provide high-quality care may need to forgo oversimplified guidelines that do not allow for the application of clinical judgment. Similarly, optimizing cancer screening decisions requires systems that reimburse physicians for the complexity and time requirements of these discussions.

Cancer screening discussions and decisions will often be difficult tasks. However, understanding potential risks and benefits of medical interventions and being aware of patient wishes are core principles of good medical practice and should be applied to cancer screening decisions.

Author Contributions: *Study concept and design:* Walter, Covinsky.
Analysis and interpretation of data: Walter, Covinsky.
Drafting of the manuscript: Walter.
Critical revision of the manuscript for important intellectual content: Walter, Covinsky.
Funding/Support: Dr Walter was supported in part by a grant from the John A. Hartford Foundation, University of California, San Francisco Geriatrics Center of Excellence, and a T-32 Training Grant (Research Training in Geriatric Medicine) from the National Institute on Aging. Dr Covinsky was supported in part by an independent scientist award (K02HS00006-01) from the Agency for Healthcare Research and Quality and is a Paul Beeson Faculty Scholar in Aging Research.

REFERENCES

1. US Preventive Services Task Force. *Guide to Clinical Preventive Services*. 2nd ed. Alexandria, Va: International Medical Publishing; 1996.
2. Robinson B, Beghe C. Cancer screening in the older patient. *Clin Geriatr Med*. 1997;13:97-118.
3. Weingarten S. Using practice guideline compendiums to provide better preventive care. *Ann Intern Med*. 1999;130:454-458.
4. Smith RA, Mettlin CJ, Davis KJ, Eyre H. American Cancer Society guidelines for the early detection of cancer. *CA Cancer J Clin*. 2000;50:34-49.
5. Eddy DM. Comparing benefits and harms: a balance sheet. *JAMA*. 1990;263:2493-2505.
6. Harris R, Leininger L. Clinical strategies for breast cancer screening. viewed as estimates of potential screening benefit that provide some quantitative context for thinking about screen. *Ann Intern Med*. 1995; 122:539-547.
7. Barratt A, Irwig L, Glasziou P, et al. Users' guides to the medical literature, XVII: How to use guidelines and recommendations about screening. *JAMA*. 1999; 281:2029-2034.
8. Yancik R, Ries LA. Cancer in older persons: magnitude of the problem—how do we apply what we know? *Cancer*. 1994;74:1995-2003.
9. National Center for Health Statistics. Life Tables of the United States, 1997. Available at: http://www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/lewk3.htm. Accessed July 18, 2000.
10. Welch HG, Albertsen PC, Nease RF, Bubolz TA, Wasson JH. Estimating treatment benefits for the elderly: the effect of competing risks. *Ann Intern Med*. 1996;124:577-584.
11. Fried LP, Kronmal RA, Newman AB, et al. Risk factors for 5-year mortality in older adults: the cardiovascular health study. *JAMA*. 1998;279:585-592.
12. Covinsky KE, Justice AC, Rosenthal GE, Palmer RM, Landefeld CS. Measuring prognosis and case mix in hospitalized elders: the importance of functional status. *J Gen Intern Med*. 1997;12:203-208.
13. Inouye SK, Peduzzi PN, Robison JT, Hughes JS, Horwitz RI, Concato J. Importance of functional measures in predicting mortality among older hospitalized patients. *JAMA*. 1998;279:1187-1193.
14. Standards and Accreditation Committee Medical Guidelines Task Force. *Medical Guidelines for Determining Prognosis in Selected Non-cancer Diseases*. 2nd ed. Arlington, Va: National Hospice Organization; 1996.
15. Ries LAG, Kosary CL, Hankey BF, Miller BA, Clegg L, Edwards BK, eds. *SEER Cancer Statistics Review, 1973-1996*. Bethesda, Md: National Cancer Institute; 1999.
16. Costanza ME. The extent of breast cancer screening in older women. *Cancer*. 1994;74:2046-2050.
17. Kerlikowske K, Carney PA, Geller B, et al. Performance of screening mammography among women with and without a first-degree relative with breast cancer. *Ann Intern Med*. 2000;133:855-863.
18. Mandel JS, Bond JH, Church TR, et al. Reducing mortality from colorectal cancer by screening for fecal occult blood. *N Engl J Med*. 1993;328:1365-1371.
19. Kronborg O, Fenger C, Olsen J, Jorgensen OD, Sondergaard O. Randomised study of screening for colorectal cancer with faecal-occult-blood test. *Lancet*. 1996;348:1467-1471.
20. Hardcastle JD, Chamberlain JO, Robinson MHE, et al. Randomised controlled trial of faecal-occult-blood screening for colorectal cancer. *Lancet*. 1996; 348:1472-1477.
21. Tabar L, Fagerberg G, Chen H, et al. Efficacy of breast cancer screening by age. *Cancer*. 1995;75: 2507-2517.
22. Kerlikowske K, Grady D, Rubin SM, Sandrock C, Ernster VL. Efficacy of screening mammography: a meta-analysis. *JAMA*. 1995;273:149-154.
23. Prevost TC, Launoy G, Duffy SW, Chen HH. Estimating sensitivity and sojourn time in screening for colorectal cancer: a comparison of statistical approaches. *Am J Epidemiol*. 1998;148:609-619.
24. Tabar L, Fagerberg G, Duffy SW, Day NE, Gad A, Grontoft O. Update of the Swedish two-county program of mammographic screening for breast cancer. *Radiol Clin North Am*. 1992;30:187-210.
25. Moskowitz M. Breast cancer: age-specific growth rates and screening strategies. *Radiology*. 1986;161: 37-41.
26. McQuay HJ, Moore RA. Using numerical results from systematic reviews in clinical practice. *Ann Intern Med*. 1997;126:712-720.
27. Sackett DL, Haynes RB, Guyatt GH, Tugwell P, eds. *Clinical Epidemiology: A Basic Science for Clinical Medicine*. 2nd ed. Boston, Mass: Little Brown & Co; 1991.
28. Laara E, Day NE, Hakama M. Trends in mortality from cervical cancer in the Nordic countries. *Lancet*. 1987;1:1247-1249.
29. Bergstrom R, Sparen P, Adami HO. Trends in cancer of the cervix uteri in Sweden following cytological screening. *Br J Cancer*. 1999;81:159-166.
30. Selby JV, Friedman GD, Quesenberry CP, Weiss NS. A case-control study of screening sigmoidoscopy and mortality from colorectal cancer. *N Engl J Med*. 1992;326:653-657.
31. Salzmann P, Kerlikowske K, Phillips K. Cost-effectiveness of extending screening mammography guidelines to include women 40 to 49 years of age. *Ann Intern Med*. 1997;127:955-965.
32. Eddy DM. Screening for cervical cancer. *Ann Intern Med*. 1990;113:214-226.
33. Cruickshank ME, Angus V, Kelly M, McPhee S, Kitchener HC. The case for stopping cervical screening at age 50. *Br J Obstet Gynaecol*. 1997;104:586-589.
34. Welch HG, Fisher ES. Diagnostic testing following screening mammography in the elderly. *J Natl Cancer Inst*. 1998;90:1389-1392.
35. Dixon J, Chetty U, Forrest A. Wound infection after breast biopsy. *Br J Surg*. 1988;75:918-919.
36. Lurie JD, Welch HG. Diagnostic testing following fecal occult blood screening in the elderly. *J Natl Cancer Inst*. 1999;91:1641-1646.
37. Ransohoff DF, Lang CA. Screening for colorectal cancer with the fecal occult blood test: a background paper. *Ann Intern Med*. 1997;126:811-822.
38. Winawer SJ, Fletcher RH, Miller L, et al. Colorectal cancer screening. *Gastroenterology*. 1997;112: 594-642.
39. Satariano WA, Ragland DR. The effect of comorbidity on 3-year survival of women with primary breast cancer. *Ann Intern Med*. 1994;120:104-110.
40. Kerlikowske K, Salzmann P, Phillips KA, et al. Continuing screening mammography in women aged 70 to 79 years. *JAMA*. 1999;282:2156-2163.
41. Page DL, Dupont WD, Rogers LW, Landenberger M. Intraductal carcinoma of the breast: follow-up after biopsy only. *Cancer*. 1982;49:751-758.
42. Eusebi V, Foschini MP, Cook MG, Berrino F, Azzopardi JG. Long-term follow-up of in-situ carcinoma of the breast with special emphasis on clinging carcinoma. *Semin Diagn Pathol*. 1989;6:165-173.
43. Fonseca R, Hartmann LC, Peterson IA, Donohue JH, Crotty TB, Gisvold JJ. Ductal carcinoma in situ of the breast. *Ann Intern Med*. 1997;127:1013-1022.
44. Ernster VL, Barclay J, Kerlikowske K, Grady D, Henderson C. Incidence of and treatment for ductal carcinoma in situ of the breast. *JAMA*. 1996;275:913-918.
45. Wardle J, Pope R. The psychological costs of screening for cancer. *J Psychosom Res*. 1992;36:609-624.
46. Marshall KG. Prevention. How much harm? How much benefit? 3. *CMAJ*. 1996;155:169-176.
47. Hughes JE. Psychological and social consequences of cancer. *Cancer*. 1987;6:455-475.
48. Warren R. The debate over mass mammography in Britain. *BMJ*. 1988;297:969-972.
49. Lerman C, Trock B, Rimer BK, Boyce A, Jepson C, Engstrom PF. Psychological and behavioral implications of abnormal mammograms. *Ann Intern Med*. 1991;114:657-661.
50. Essink-Bot M, de Koning HJ, Nijs HGT, Kirkels WJ, van der Maas PJ, Schroder FH. Short-term effects of population-based screening for prostate cancer on health-related quality of life. *J Natl Cancer Inst*. 1998; 90:925-931.
51. Sox HC. Screening for disease in older people [editorial]. *J Gen Intern Med*. 1998;13:424-425.
52. Bennahum DA, Forman WB, Vellas B, Albarede JL. Life expectancy, comorbidity, and quality of life: a framework of reference for medical decisions. *Clin Geriatr Med*. 1997;13:33-53.
53. Degner LF, Kristjanson LJ, Bowman D, et al. Information needs and decisional preferences in women with breast cancer. *JAMA*. 1997;277:1485-1492.
54. Redelmeier DA, Rozin P, Kahneman D. Understanding patients' decisions. *JAMA*. 1993;270:72-76.
55. Mazur DJ, Hickam DH. Patient preferences: survival vs quality-of-life considerations. *J Gen Intern Med*. 1993;8:374-377.
56. Pignone M, Bucholtz D, Harris R. Patient preferences for colon cancer screening. *J Gen Intern Med*. 1999;14:432-437.
57. Ransohoff DF, Harris RP. Lessons from the mammography screening controversy: can we improve the debate? *Ann Intern Med*. 1997;127:1029-1034.
58. Cassel CK. Breast cancer screening in older women. *J Gerontol*. 1992;47(special issue):126-130.
59. Fischhoff B. Why (cancer) risk communication can be hard. *Monogr Natl Cancer Inst*. 1999;25:7-13.
60. Malenka DJ, Baron JA, Johansen S, Wahrenberger JW, Ross JM. The framing effect of relative and absolute risk. *J Gen Intern Med*. 1992;92:121-124.
61. Schwartz LM, Woloshin S, Black WC, Welch HG. The role of numeracy in understanding the benefit of screening mammography. *Ann Intern Med*. 1997; 127:966-972.
62. Bogardus ST, Holmboe E, Jekel JF. Perils, pitfalls, and possibilities talking about medical risk. *JAMA*. 1999;281:1037-1041.
63. Pignone M, Harris R, Kinsinger L. Videotape-based decision aid for colon cancer screening: a randomized, controlled trial. *Ann Intern Med*. 2000;133: 761-769.
64. Gonzales R, Steiner JF, Lum A, Barrett PH. Decreasing antibiotic use in ambulatory practice. *JAMA*. 1999;281:1512-1519.

13
Osteoarthritis

Edna P. Schwab, MD

Commentary

Osteoarthritis is the most common disabling rheumatic disease in patients over the age of 55 years old and its incidence rate increases with age (1). Up to the middle of the twentieth century, physicians thought that osteoarthritis was a disease that inevitably occurred with aging, yet the etiology and features of the joint affected by osteoarthritis were not fully described. In 1934, a landmark paper written by Chester Keefer, Frederic Parker, Walter Myers, and Ralph Irwin enlightened the medical community as to the pathologic features of osteoarthritis and questioned the etiology of this disease. After examining 100 knee joints taken from 77 cadavers, these authors demonstrated that damage from osteoarthritis occurred in cartilage and, in some severe cases, bone was exposed. Their findings demonstrated erosions and thinning of cartilage in regions that involved areas of greatest movement and weight bearing and included activity such as walking. They also observed that these changes occurred with increasing frequency in joints of patients with advancing age but with equal frequency in men and women. The causation between aging and osteoarthritis was not examined. Consistent with current teaching on osteoarthritis, risk factors related to the development of arthritis were identified by them and included strain, weight-bearing, injury, aging tissue, wear and tear, trauma, occupation, and static deformities. Interestingly, they found that no correlation existed between the extent of the lesions in the joint and symptoms which patients reported.

In 1942, a subsequent study examined the pathogenesis of osteoarthritis and confirmed that articular cartilage was the site of this pathological process. In addition, this work demonstrated that cartilage senescence contributed to this pathological process (2).

Present-day consensus on the pathological features of osteoarthritis include deterioration and erosions of articular cartilage found on weight-bearing surfaces, along with new bone formation found in adjacent bone and capsular attachments. Inflammation is found to occur as a secondary event. This concept of impaired bone remodeling, where new bone is laid down at the joint margins as a result of cartilage erosions was first introduced by Lent Clifton Johnson in 1956 (3). The initial events which lead to these changes in the joint occur at the cellular and molecular level and are the focus of present-day clinical investigation (4). Research in chondroprotection and tissue engineering may alter the course of this disease. The role of identifying biochemical markers such as aggrecanase and matrix metalloproteinases from cartilage, or markers from synovium and bone may serve to monitor disease severity or become targets for potential pharmacological intervention and subsequent disease modification (4,5). Further research will provide for more interventions in this potentially modifiable disease.

References

1. Schwab EP, Albert D. Arthritis in the Elderly. In: Treatment of Rheumatic Disease, Second Edition. Weeisman W, Weinblatt ME (eds). Philadelphia: W.B. Saunders. 2001.
2. Bennett GA, Waine H, Bauer W. Changes in the Knee Joint at Various Ages with Particular Reference to the Nature and Development of Degenerative Joint Disease. New York: Commonwealth Fund. 1942.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

3. Johnson LC. Kinetics of Osteoarthritis. Lab Invest 1959;8: 223–1238.
4. Sokoloff L. Some Highlights in the Emergence of Modern Concepts of Osteoarthritis. Semin Arthritis Rheum 2001;31: 71–107.
5. Garnero P, Rousseau JC, Delmas PD. Molecular Basis and Clinical Use of Biochemical Markers of Bone, Cartilage and Synovium in Joint Diseases. Arthritis Rheum. 2000;43: 953–968.

Archives of Internal Medicine

VOLUME 53 MARCH, 1934 NUMBER 3

RELATIONSHIP BETWEEN ANATOMIC CHANGES IN KNEE JOINT WITH ADVANCING AGE AND DEGENERATIVE ARTHRITIS

CHESTER S. KEEFER, M.D.

FREDERIC PARKER, JR., M.D.

WALTER K. MYERS, M.D.

AND

RALPH L. IRWIN, M.D.

BOSTON

The acquirement of knowledge concerning arthritis has been delayed by a lack of detailed information regarding the changes which may be encountered with advancing age. Such information is highly desirable in order to assist in defining the limits of normal for a given age period. It is of further importance from the point of view of the ultimate changes that may occur in a joint which has been the site of an injury resulting from invasion by infective agents, and, finally, it provides knowledge regarding the early stages of disorders of the joints. For these reasons we have made a systematic study of 100 knee joints removed at consecutive necropsies, and in this article we detail the gross anatomic findings and discuss their significance.

MATERIAL AND METHODS

One hundred knee joints were removed from 77 cadavers. The cases were not selected on the basis of previous clinical diagnosis or the presence of symptoms in the joints. The only cases excluded from the group were those in which there was definite infection of the joints with various organisms, as determined by bacteriologic examination of the fluid found in the joints at necropsy. A study of these joints will be presented separately. Whenever possible, the whole knee joint was removed together with its capsule and a portion of the femur and tibia. When this was not feasible, the joint was opened by cutting across the inferior patellar ligament and reflecting the patella. The articular surfaces of the patella, femur and tibia were then removed in such a way that the surfaces could be reconstructed and studied. The gross changes were noted, and material was taken for histologic section.

In this paper the term degenerative arthritis is used synonymously with hypertrophic arthritis and osteo-arthritis.

From the Thorndike Memorial Laboratory, Second and Fourth Services (Harvard), the Department of Pathology, Boston City Hospital, and the Department of Medicine, Harvard Medical School.

(Continued)

ANATOMIC CONSIDERATIONS

It is not our purpose to comment in detail on the anatomy of the knee joint. It is necessary to know some of the salient facts before one can clearly understand the changes which were found. The relevant facts are recalled herewith.

The articulations of the knee joint are divided into the following groups: (1) the articulation of the patella with the femur, and (2) the articulations of the two condyles of the femur with the corresponding meniscus and condyle of the tibia.

The articulation of the patella with the femur is partly a gliding one, but as the articular surfaces are not mutually adapted to each other, the movement is not a simple gliding one. This fact will become obvious when the various movements of the knee joint are analyzed in detail.

For the purpose of describing the movements of the patella and its articular surface, it may be divided roughly into seven facets: the upper, middle and lower horizontal pairs and a median perpendicular facet.

The surface of the femur which articulates with the patella is named the patellar or anterior surface. It presents a median groove which extends downward to the intercondyloid fossa and two convexities, the lateral one of which is broader and more prominent and extends farther upward than the medial. In the extended position of the leg, when the quadriceps femoris is relaxed, the patella lies loosely on the front of the lower end of the femur. When the knee is forcibly flexed the median perpendicular facet is in contact with the semilunar surface of the lateral part of the medial condyle of the femur; this semilunar surface is a backward prolongation of the medial part of the patellar surface of the femur. As the leg is carried from the flexed to the extended position, first the upper, then the middle and finally the lowest pair of horizontal facets is successively brought into contact with the patellar surface of the femur. In the process of walking, in which the leg is alternately extended and semiflexed, the part of the patella which is brought into contact with the femur is the median pair of horizontal facets. The importance of this will be stressed presently.

The lower and posterior parts of the articular surface of the femur constitute the tibial surfaces for articulation with the corresponding condyles of the tibia and menisci. These surfaces are separated from one another by the intercondyloid fossa and from the patellar surface by faint grooves which extend obliquely across the two condyles. The lateral groove is better marked; it runs lateralward to form a triangular depression.

The articular surface of the tibia consists of two facets, the central portions of which articulate with the condyles of the femur, while their peripheral portions support the menisci of the knee joint which here

intervene between the two bones. Between the two facets is the intercondyloid eminence, surmounted on either side by a prominent tubercle, onto the sides of which the articular facets are prolonged. The lateral condyle is usually somewhat smaller than the medial and is nearly circular.

In the position of extension, when the subject is standing erect, the weight of the trunk is transmitted from the head of the femur in a vertical line which passes through the external condyle of the femur and tibia.

When full extension of the leg is reached and maintained the lateral part of the groove of the lateral condyle of the femur is pressed against the anterior part of the corresponding meniscus, while the medial part of the groove rests on the articular margin in front of the lateral process of the tibial intercondyloid eminence. Into the groove on the medial condyle is fitted the anterior part of the medial meniscus, while the anterior crucial ligament and the articular margin in front of the medial process of the intercondyloid eminence are received into the forepart of the intercondyloid fossa of the femur.

In full flexion, the posterior parts of the femoral condyles rest on the corresponding parts of the meniscotibial surfaces, and the semilunar area of the median condyle articulates with the median vertical facet of the patella. As the limb moves from flexion to extension the posterior two thirds of the tibial articular surfaces of the two femoral condyles gradually shift forward and the patella moves upward in the manner previously described.

In walking, greater weight is placed on the lateral condyle of the tibia than on the median, and the inferior surface of the femoral condyles articulates with the corresponding articular surfaces of the tibial condyles. With these facts borne in mind, the results of our investigation are detailed.

Sex and Age.—Of the 100 joints, 67 were obtained from males and 33 from females. From tables 1, 2 and 3, it is seen that the changes observed were as common in women as in men. No qualitative difference in the type of anatomic alterations in the joints was observed. For these reasons the gross changes in the two sexes are considered together.

The age distribution of the patients whose joints were studied is given in figure 1. In figure 2 the total percentage of joints showing changes at various age periods is shown, and it is obvious that the anatomic deviations from normal increase with advancing age.

Occupation.—As degenerative arthritis is sometimes explained on the basis of a person's occupation or at least is aggravated by certain occupations that require the excessive use of the joints, an analysis was made of the patients' occupations. They are summarized in table 4.

(Continued)

TABLE 1.—*Site of Observed Anatomic Changes in the Knee Joints in Various Decades of Life (Males)*

Age, Years	Number of Joints	Patella	Femur			Tibia	
			Interpatellar Groove	Median Condyle	Lateral Condyle	Median Condyle	Lateral Condyle
0- 9	2	0	0	0	0	0	0
10-19	0	0	0	0	0	0	0
20-29	0	0	0	0	0	0	0
30-39	4	1	1	0	0	2	2
40-49	8	4	3	1	0	2	4
50-59	14	13	9	7	4	9	9
60-69	17	16	13	7	3	10	11
70-79	16	15	15	11	11	10	12
80+	6	5	3	2	2	4	4
Total	67	54	44	28	20	37	42

TABLE 2.—*Site of Observed Anatomic Changes in the Knee Joints in Various Decades of Life (Females)*

Age, Years	Number of Joints	Patella	Femur			Tibia	
			Interpatellar Groove	Median Condyle	Lateral Condyle	Median Condyle	Lateral Condyle
0- 9	1	0	0	0	0	0	0
10-19	2	0	0	0	0	0	0
20-29	1	0	0	0	0	0	0
30-39	2	1	1	0	0	1	1
40-49	1	0	0	0	1	0	0
50-59	6	5	4	4	4	4	6
60-69	11	12	10	6	6	7	8
70-79	3	4	3	2	2	2	2
80+	6	5	3	3	3	4	5
Total	33	27	21	15	16	18	22

TABLE 3.—*Total Changes in Both Sexes, Showing Site of Anatomic Alterations*

Age, Years	Number of Cases	Number of Joints	Patella	Femur			Tibia	
				Median Condyle	Lateral Condyle	Interpatellar Groove	Median Condyle	Lateral Condyle
0- 9	3	3	0	0	0	0	0	0
10-19	2	2	0	0	0	0	0	0
20-29	1	1	0	0	0	0	0	0
30-39	5	6	2	0	0	2	3	3
40-49	7	9	4	1	1	3	2	4
50-59	14	20	18	11	8	13	13	15
60-69	23	28	28	13	9	23	17	19
70-79	15	19	19	13	13	18	12	14
80+	7	12	10	5	5	6	8	9
Total	77	100	81	43	36	65	55	64

TABLE 4.—*Occupations of the Patients*

Laborers	30
Housewives	21
Artisans	17
Cooks and waiters	4
Workers in offices	4
Professional worker	1

In a further analysis of the extent of anatomic change and occupation we were unable to determine any relationship between the extent of the damage and the occupation, but data concerning the actual details of the patients' life-long daily careers were not available.

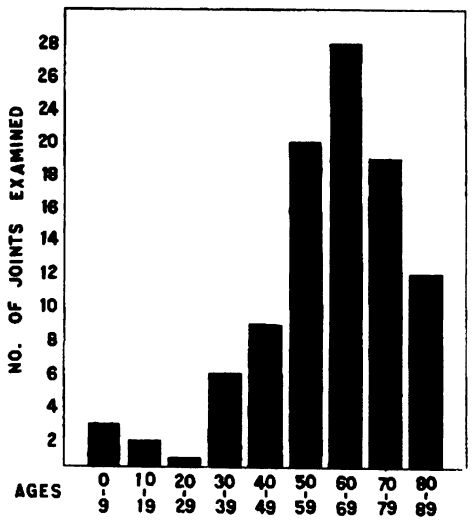


Fig. 1.—Age distribution of the 77 patients whose knee joints were examined.

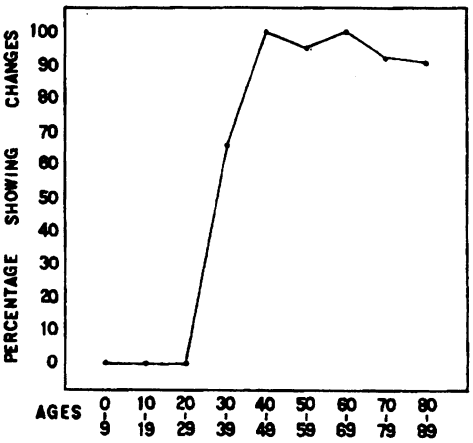


Fig. 2.—The percentage of cases showing anatomic changes in the knee joints at different age periods.

Cause of Death.—The diseases from which the patients died are listed in table 5. From this table it is seen that no one disease or group of diseases could be held responsible for the changes observed.

Relationship Between Anatomic Lesions and Symptoms Referable to the Joints.—There were no symptoms recorded in the clinical his-

(Continued)

tories of 66 patients, and pain and stiffness were present in only 11 of the 77 patients. When symptoms referable to the joints are present in a patient with anatomic changes in the joints such as we have observed, it is necessary to look for other factors in order to explain the symptoms. From our observations it is manifest that pathologic changes in the joints were present with equal frequency in both sexes, that they increased in frequency with advancing age and that neither the cause of death nor the occupation of the patients was of significance in accounting for the condition of the joints.

Changes in the Patella.—The articular surface of the patella showed distinctive alterations in 81 of the joints examined. The sites of the changes are recorded in table 6. In most cases the median surface

TABLE 5.—*Diseases Responsible for Death*

Lobar pneumonia and bronchopneumonia	20
Arteriosclerotic heart disease	15
Cancer (miscellaneous)	10
Cerebral hemorrhage	6
Tuberculosis	8
Peritonitis (generalized)	3
Pyelonephritis	5
Rheumatic heart disease	4
Syphilitic aortitis with aneurysm or aortic insufficiency	2
Pernicious anemia	1
Aplastic anemia	1
Osteogenic sarcoma	1
Bacterial endocarditis	1
Total	77

TABLE 6.—*Position of Changes in the Patella*

	Median Surface	Lateral Surface
Superior horizontal facets	14	17
Median horizontal facets	75	61
Inferior horizontal facets	22	19

showed alterations more often than the lateral surface, and the median horizontal facets were involved more often than the other two pairs. This is significant because this is the part of the patella which comes in contact with the patellar surface of the femur most frequently in such movements as walking. The inferior pair were changed more often than the superior, and again this pair of facets comes in contact with the femur more often than the superior pair.

The character and degree of the erosions varied tremendously in different cases. Figure 3 illustrates the kind of alterations observed. It is seen that there may be simple fibrillation of the cartilage, with irregularity of the surface or loss of varying amounts of cartilaginous substance so that in some cases the underlying bone is exposed. In the places where the bone was exposed it was usually very dense and, in some cases, shiny, giving an appearance of a highly polished surface, the so-called eburnated bone.

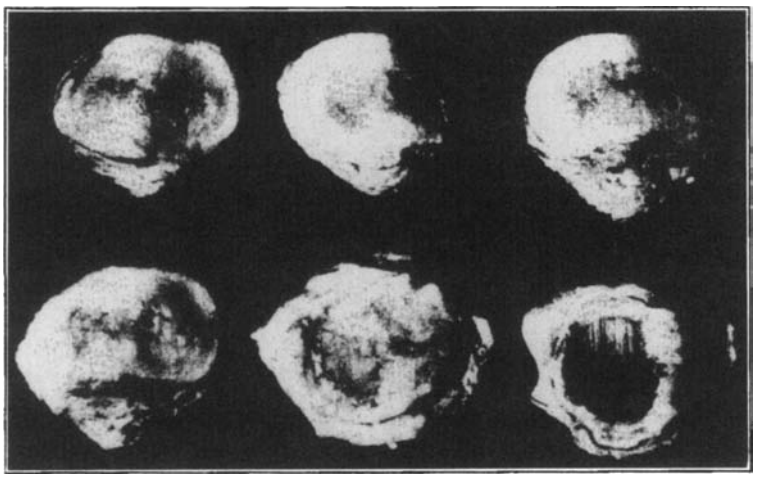


Fig. 3.—Photographs of the articular surface of the patella, showing the type of anatomic alterations observed. Various degrees of changes are present. In some cases there is only fibrillation of the cartilage; in others the underlying bone is exposed.

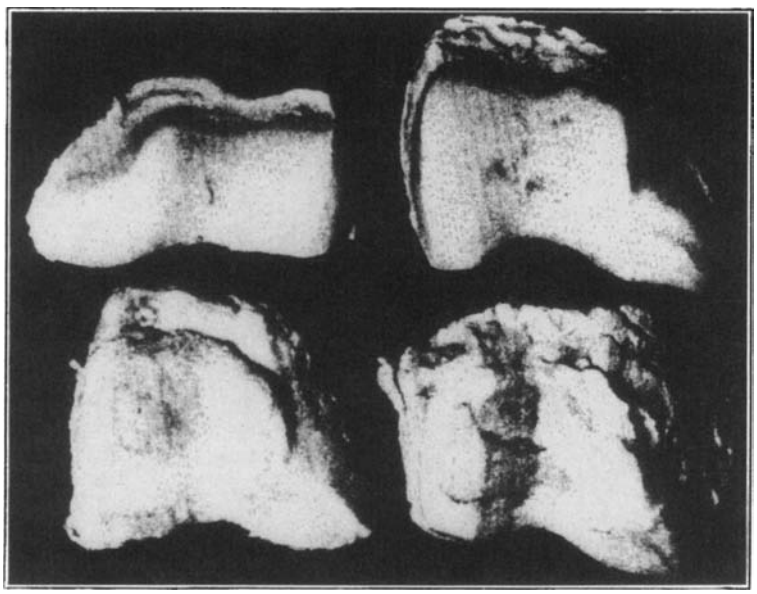


Fig. 4.—Photographs of the patellar groove of the femur, illustrating the type of lesion observed. The lesion is progressively more extensive in the specimens shown.

(Continued)

Femur.—For purposes of discussion we have divided the articular surfaces of the femur into the patellar groove, the anterior and posterior articular surfaces of the condyles and the median surface of the median condyle.

Patellar (Intercondyloid) Groove: This area of the femur was altered in 65 cases. In every case the erosion was situated at the point where the patella comes in contact with the femur. In most instances the eroded area was in the center of the groove; in others, it was at the right or left, depending on the size, shape and position of the patella when in contact with the femur. Figure 4 illustrates the site and type

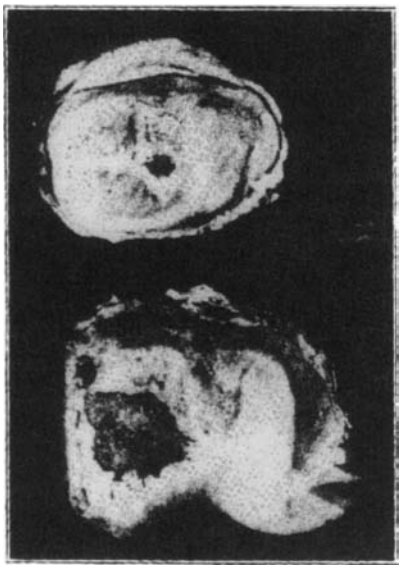


Fig. 5.—Photograph of the articular surface of the patella and the patellar groove of the femur, showing the erosions on the articulating surfaces.

of the erosions which were seen. In every case in which changes were observed in the interpatellar groove there was an irregular or eroded area on the patella at the point where it came in contact with the femur. This is illustrated in figure 5. The recognition of this fact is highly important in interpreting the findings.

Anterior Articular Surface of Femoral Condyles: It is to be recalled that the medial and lateral anterior articular surfaces of the femoral condyles come in contact with the tibial condyles and the corresponding menisci. The median condyle showed erosions in 46 joints and the lateral condyle in 36. These areas were most conspicuous at the point where the condyles were in contact with the central portion

of the tibia which was uncovered by the lateral or median meniscus. An example of these erosions is shown in figure 6.

Aside from the lesions on the anterior articular surface, erosions were observed on the median surface of the median condyle in 9 cases. They are illustrated in figure 7 and require special comment. It has already been pointed out that when the leg is in full extension the medial process of the intercondyloid eminence of the tibia and the anterior cruciate ligament are received into the forepart of the intercondyloid fossa of the femur. When a large number of these joints are examined, it becomes apparent that in some cases this eminence is high and promi-

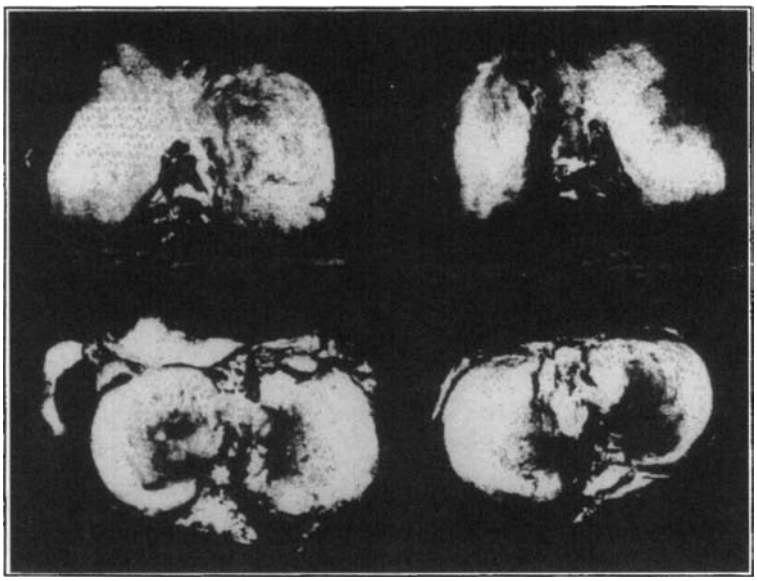


Fig. 6.—Photograph of the inferior articular surfaces of the femur and the corresponding articular surfaces of the tibia, showing the type and position of the irregularities and erosions.

nent and is in direct contact with the lateral surface of the median condyle, especially during flexion and extension. In every case in which we observed an erosion of the femur in this area the intercondyloid eminence was prominent and came in close contact with the femur, especially during the movements of semiflexion and extension. We are of the opinion that this observation is of importance in explaining the development of erosions in the area mentioned.

The posterior parts of the femoral condyles, which rest on the corresponding tibial surfaces during full flexion, showed erosions seven times on the medial condyles and five times on the lateral condyles. As

(Continued)

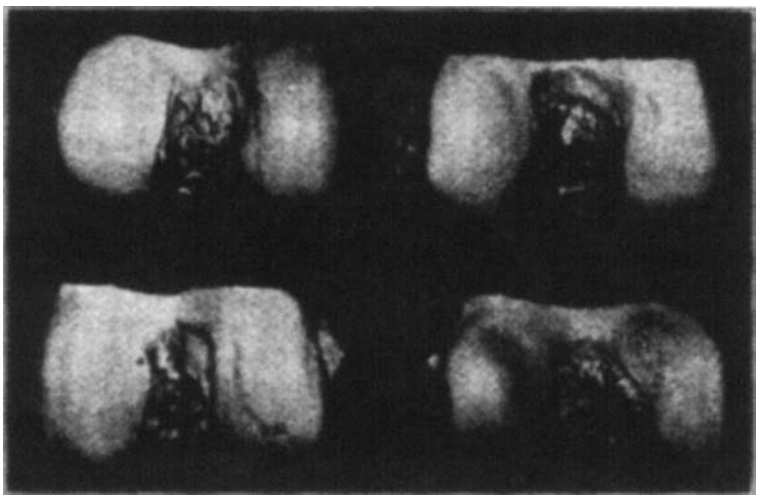


Fig. 7.—Photograph of the femoral condyles, showing erosions on the median aspect of the median condyle of the femur. This is the area of the femur which comes in contact with the intercondyloid tubercle of the median condyle of the tibia.

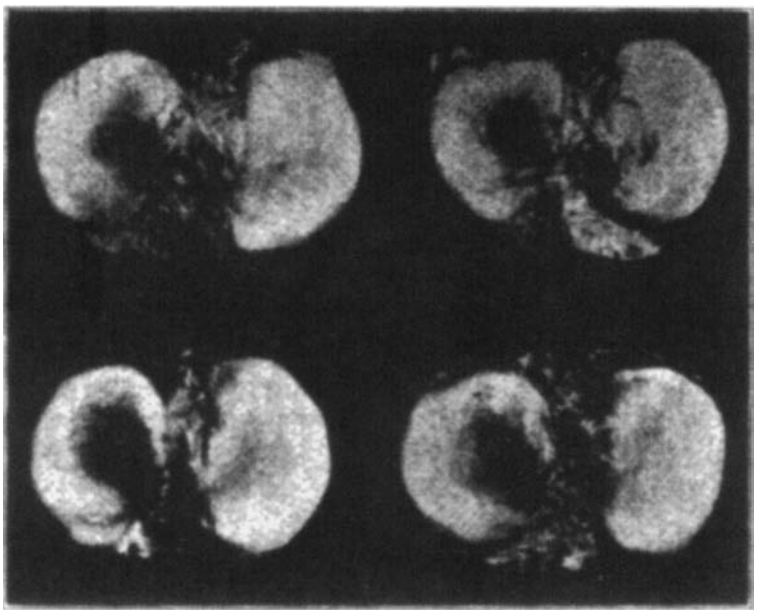


Fig. 8.—Photograph of the tibial condyles, showing areas of erosion. Note the erosions in the central part of the tibial condyles or the part of the surface which is uncovered by the semilunar cartilage.

the leg is in full flexion less frequently than in the other positions, these areas are in contact with one another less often. This probably accounts for the small number of erosions in this area.

Tibia.—The central portion of the lateral condyle of the tibia showed erosions or marked thinning of the cartilage in 64 cases, and the median condyle in 55 cases. The area of the tibial condyles which was covered by the menisci never showed erosions or thinning when the menisci were normal and were everywhere intact. When they were torn or displaced in such a way that they no longer protected the periphery of the tibia, erosions were frequently observed. Figure 8 illustrates the areas of the tibia usually involved. Here again it often was observed that the area of the tibia which was involved was opposite to an erosion on the corresponding articular surface of the femur.

Synovia.—The synovial membrane was smooth and glistening in 44 cases. In the remaining cases it ordinarily showed villous projections from the surface. It was not unusual to see the synovial membrane extending over the medial edge of the patella and, in many instances, projecting into the depressions made by the erosions in the cartilage. The villous projections increased in frequency with advancing age and were most striking in the patients with the most extensive alterations in joint structure.

COMMENT

From the data presented certain facts are clear. In the first place, anatomic changes in the knee joint were observed with increasing frequency with advancing age. The articular surface of the patella showed changes most often, and these changes were most conspicuous on the median articular facets. As we have said before, this is the part of the patella which comes in contact with the femur most often during such movements as walking. The interpatellar groove of the femur, or the area over which the patella glides, was the second most frequent site of erosion; then, in order, the lateral articular surface of the tibia, the median articular surface of the tibia and the median and the lateral condyles of the femur. On careful analysis it was obvious that the areas usually affected were those subjected to the greatest movement or weight, and this was particularly true of the points at which the articular surface came in contact during movement.

Previous observations have been made on the changes in the knee joints of patients who were not considered to be suffering from arthritis. Rimann [1] found changes in the joints of 67 of 100 persons between the ages of 15 and 80 who were examined at necropsy, and Beitzke,[2]

1. Rimann, H.: Arb. a. d. path. Inst. zu Berlin z. Feier **1**:139, 1906.

2. Beitzke, H.: Ueber die sogenannte Arthritis deformans atrophica, Ztschr. f. klin. Med. **74**:215, 1912.

(Continued)

on examining 200 bodies, found only 35 without alterations in some of the joints. Six showed anatomic changes of ordinary arthritis deformans, and in 16 there was evidence of gout. Of the remaining 178, visible changes were seen in the joints of 143. These changes were alike in the two sexes. Between the ages of 20 and 40 years 60 per cent of the joints showed changes; between 40 and 50 years, 95 per cent, and in older persons, 100 per cent. Clark [3] studied the lesions in the joints in 1,100 cases which came to autopsy in Panama; he found gross lesions in the joints in 15.6 per cent of the patients, and of these less than 1 per cent were considered to be suffering from arthritis. He emphasized syphilis, arteriosclerosis and hard work as important etiologic factors. The most recent observations are those of Heine,[4] who described the changes in 1,994 knee joints of patients varying from 15 to 89 years of age. Here again the changes increased with advancing age and varied from 1.5 per cent in the age period from 15 to 19 years to 100 per cent in the older age groups. The patella was involved most frequently and, as in our observations, the commonest point was the central or median facets. On the femur the interpatellar groove showed erosions most often, and the tibia was altered almost as frequently as the patella, the lateral condyle being involved more often than the median. In 490 cases, or 49 per cent, the changes were equal on both sides; in 35 per cent the right side showed greater changes than the left, and in 16 per cent the left side showed more changes than the right. The lesions occurred with equal frequency in males and females. It is evident, therefore, that in spite of the fact that the number of cases which we have observed is relatively small, lesions were observed in practically the same locations and with the same frequency by other investigators. Meyer [5] and Keyes [6] noted changes in the knee joints of cadavers which were similar to those observed by us, and Bennett and Bauer [7] made similar observations in patients who had no symptoms referable to the joints during life.

What has been said regarding the knee joint is true for other joints as well. According to Heine, the joints which show lesions most fre-

3. Clark, H. C.: Etiologic Factors in Gross Lesions of the Large Joints: Observations from 1,100 Consecutive Necropsies, J. A. M. A. **69**:2099 (Dec. 22) 1917.

4. Heine, J.: Ueber die Arthritis deformans, Virchows Arch. f. path. Anat. **260**:521, 1926.

5. Meyer, A. W.: Further Observations upon Use-Destruction in Joints, J. Bone & Joint Surg. **4**:491, 1922.

6. Keyes, E. L.: Erosions of the Articular Surfaces of the Knee Joint, J. Bone & Joint Surg. **15**:369, 1933.

7. Bennett, G. A., and Bauer, W.: A Systematic Study of the Degeneration of Articular Cartilage in Bovine Joints, Am. J. Path. **7**:399, 1931.

quently with increasing age are the knee, acromioclavicular, elbow, hip and metatarsal joints, the joints of the spine and the sternoclavicular and shoulder joints. Zöllner [8] and Smith-Petersen [9] have shown the same to be true in regard to the sacro-iliac joint. Sievers [10] studied the 77 acromioclavicular joints and obtained similar results. Ely [11] observed changes in the sternoclavicular joints at necropsy, although he did not state whether the alterations increased in frequency with advancing age.

From the recorded observations and from our own, it is difficult to escape the conclusion that the changes which are commonly observed in the joints with advancing age are precisely the same as those described as being characteristic of so-called degenerative arthritis. It is necessary, therefore, to inquire into some of the factors which have been held responsible for this disorder.

Degenerative arthritis is frequently considered to develop as the result of certain occupations. One of the first investigators to emphasize this point of view was Lane.[12] Since 1886 his observations have been confirmed repeatedly. Recently Fischer [13] and others made the interesting observation that degenerative arthritis commonly develops in the elbow and metacarpal and shoulder joints of men who work with compressed air hammers. The changes in the joints appear anywhere from three to ten years after work with these tools and Fischer was of the opinion that repeated small traumas from the constant jarring of the hammer are mainly responsible for the development of this disorder. On the other hand, Fischer was unable to demonstrate that arthritis in the knee was more common in any particular occupation. He pointed out, however, that workers who carry heavy weights and who work in a kneeling position get arthritis, but he considered that occupation alone was the cause in only a few cases. Of greater importance in accounting for the changes in the knee are static, functional

8. Zöllner, F.: Untersuchungen über die Erscheinungsformen der Arthritis deformans in den Sacro-Iliacalgelenken, Virchows Arch. f. path. Anat. **277**:817, 1930.

9. Smith-Petersen, M. N.: Traumatic Arthritis, Arch. Surg. **18**:1216 (April) 1929.

10. Sievers, R.: Arthritis deformans des Akromioklavikulargelenks: zugleich ein Beitrag zur traumatischen Entstehung der Arthritis deformans chronica, Virchows Arch. f. path. Anat. (supp. 1) **226**:123, 1919.

11. Ely, L. W.: A Study of the Sterno-Clavicular Joint, in Ely, L. W., and Cowan, J. F.: Bone and Joint Studies, Stanford University, Calif., Stanford University Press, 1916, p. 121.

12. Lane, W. A.: Some Points in the Physiology and Pathology of the Osseous Systems of Trunk and Shoulder Girdle, Guy's Hosp. Rep. **28**:321, 1886.

13. Fischer, A.: Rheumatismus als Berufskrankheit, Acta rheumatol. **4**:24, 1932.

(Continued)

or traumatic factors. Flatfeet and rachitic deformities of the hips or knees (coxa vara or genu valgum) were found to be of great importance in producing arthritis of the joints of the lower extremities. From a study of the occupation of our patients it was not possible to show any correlation between the changes in the knee joints and the type of work done by the patients, although complete details of the work are lacking. As the changes were the same in both sexes, it would appear that the alterations in the knee joints which we studied will have to be explained on some basis other than occupation. Heine was of the opinion that factors other than the type of occupation were accountable for the changes in the joints which he observed, but Pommer,[14] on the other hand, has repeatedly expressed the opinion that occupation is of great importance in degenerative arthritis, and he has quoted a number of observers who support this belief.

Of more importance than occupation in explaining the changes in the knee joints is the question of mechanical factors due to trauma, static defects and incongruities of the articular surfaces. The mechanico-functional theory of the production of the degenerative arthritis has received support from the observations of many investigators. Beneke,[15] as a result of his studies in spondylitis deformans, concluded that the primary change in this disorder was degeneration of the intervertebral disks and that the changes in the vertebra resulted from continuous trauma caused by the degeneration. It is known now that the intervertebral disk becomes less elastic with advancing age, as is shown by the decreasing water content of the nucleus pulposus and the narrowing of the intervertebral space. This allows the anterior part of the vertebral bodies to come in contact during movement so that lipping and the characteristic arthritic changes in the spine occur. These changes in the spine have been produced experimentally in dogs by Keyes and Compere [16] by reducing the intervertebral space so that the vertebral bodies impinged on one another during movement.

To Preiser [17] belongs the credit for having emphasized the great importance of static defects in producing degenerative arthritis. As a

14. Pommer, G.: Ueber die mikroskopischen Kennzeichen und die Entstehungsbedingungen der Arthritis deformans (nebst neunen Beiträgen zur Kenntnis der Knorpelknötchen), Virchows Arch. f. path. Anat. **263**:434, 1927.

15. Beneke, R.: Zur Lehre von der Spondylitis deformans, Versamml. d. deutsche Naturf. u. Aerzte, Braunschweig, 1897, p. 109.

16. Keyes, O. C., and Compere, E. L.: The Normal and Pathological Physiology of the Nucleus Pulposus of the Inter-Vertebral Disc, J. Bone & Joint Surg. **14**:897, 1932.

17. Preiser, Georg: Statische Gelenkerkrankungen, Stuttgart, Ferdinand Enke, 1911; Ueber die Arthritis deformans coxae; ihre Beziehungen zur Roser Nélatonschen Linie und über den Trochanter Hochstand, Hüftgesunder infolge abnormaler Pfannenstellungen, Deutsche Ztschr. f. Chir. **89**:591, 1907.

result of numerous clinical observations he demonstrated that static deformities cause incongruities of the articular surfaces which lead to abnormal pressure and weight in certain parts of the joint and to the subsequent changes due to the trauma caused by weight and movement. Pommer,[14] on the basis of extensive microscopic studies of the joints, proposed what is generally known as the "functional theory of Pommer." Instead of accepting the idea that the primary lesion in the joint is a degeneration of the cartilage or osteoporosis of the subchondral bone, he concluded that the first thing that happens is damage to the cartilage from trauma, with a loss of its elasticity, or that, if the cartilage has lost its elasticity as a result of the process of involution, it is more readily injured. When the elasticity of the cartilage is lost, the subchondral bone and its marrow are no longer protected from the irregular, localized effects of weight, pressure and impacts. This lack of protection and the subsequent trauma cause increased vascularization and ossification of the bone, with thickening and all the characteristic changes of degenerative arthritis. This theory of the development of degenerative arthritis has attracted widespread attention and interest, and there is ample clinical and experimental evidence available at present to support it. A few of the relevant facts are recalled.

There are numerous clinical examples of degenerative arthritis following trauma to the joints, with resulting incongruities of surface, static defects, abnormal weight bearing and pressure. A few of the more important ones may be cited.

When arthritis follows direct injury to a joint it is commonly called traumatic arthritis. When this occurs the clinical and pathologic features of the changes produced are sometimes indistinguishable from definite cases of degenerative arthritis in which no violent injury has occurred. For example, compressed fractures of one or both condyles of the tibia, with resulting genu valgum or varum, are invariably followed by changes in the knee joint characteristic of degenerative arthritis. In these cases the abnormal distribution of weight on one or the other tibial condyle, together with displacement of the patella, causes degenerative changes. Another example of such an occurrence is arthritis of the elbow joint developing as a result of fractures of the head or neck of the radius, with displacement of the head. If the displacement is not corrected or if the fractured bone is not removed, arthritis always appears sooner or later.

A further example of arthritis developing after an injury is its appearance following Legge-Perthes' disease or osteo-arthritis deformans juvenilis. As a result of the extensive researches of Lang [18] and the

18. Lang, F. S.: Osteo-Arthritis Deformans Contrasted with Osteo-Arthritis Deformans Juvenilis, J. Bone & Joint Surg. **14**:563, 1932.

(Continued)

recent studies of Freund [19] and of Miltner and Hu,[20] there seems to be little doubt that this disorder of the hip joint occurs as a result of direct or indirect traumatic damage to the cartilage, particularly at the osteochondral junction. This seems to be particularly striking if the blood supply through both the ligamentum teres and the periosteum is interfered with at the same time. The histologic picture is not unlike that seen in adults with osteo-arthritis of the hip joint, and with healing the deformity of coxa vara is commonly observed. The sequence of events in the healing of the process has recently been studied in detail by means of the roentgen ray by Freund.[19] Key [21] called attention to the deformity of the hip joint following the slipping of the upper femoral epiphysis in adolescence. In these cases the head of the femur is displaced so that it no longer forms a perfect ball and socket joint, and sooner or later, usually in adult life, pain and limitation of motion in the hip appear as a result of the changes following the mechanical defect.

Fractures of bones other than the ones mentioned may be followed by arthritis, for example, arthritis of the ankle joints following fracture of the os calcis and arthritis of the wrist joint following fractures of the lower end of the radius.

Arthritis following an injury to the hip joint may be seen in cases of posttyphoid coxitis. It is well recognized that when typhoid fever is complicated by arthritis the spine and hip are most commonly involved. When the hip is affected, subluxation or partial dislocation frequently occurs. This leads to a deformity of the head of the femur and acetabulum, and changes not unlike those seen in degenerative arthritis follow.

Charcot's Joints.—The alterations in so-called Charcot's joints must be regarded as nothing more or less than the results of frequent traumas to joints which have been rendered insensitive to pain by loss of the sensation of pain and the sense of position. There is overwhelming experimental and clinical evidence to support this view . The excellent review of the clinical aspects of the lesions in the joints caused by nerve lesions, as detailed by Shands,[22] and the recent thorough histologic study of Charcot's joints by Moritz [23] leave no doubt that the lesions are

19. Freund, E.: Zur Deutung des Röntgenbildes der Perthesschen Krankheit, Fortschr. a. d. Geb. d. Röntgenstrahlen **42**:435, 1930.

20. Miltner, L. J., and Hu, C. H.: Osteochondritis of the Head of the Femur; An Experimental Study, Proc. Soc. Exper. Biol. & Med. **30**:416 (Jan.) 1933.

21. Key, J. A.: Traumatic Arthritis and the Mechanical Factors in Hypertrophic Arthritis, J. Lab. & Clin. Med. **15**:1145, 1930.

22. Shands, A. R., Jr.: Neuropathies of the Bones and Joints, Arch. Surg. **20**: 614 (April) 1930.

23. Moritz, A. R.: Tabische Arthropathie, Virchows Arch. f. path. Anat. **267**:746, 1928.

the result of repeated trauma to the joints. It should be emphasized that in many instances the changes in the joints are extensive owing to the excessive trauma that follows the loss of the sensation of pain. It is only natural to find less extensive changes following trauma to a joint in a person with intact sensation of pain for the reason that once the joint has been injured it is not used so much as a joint devoid of the sensation of pain. In some cases the changes produced by trauma are further exaggerated by a secondary invasion of the joint by bacteria. This occurs particularly if there are decubitus ulcers around the joints, such as the ankle joints.

Hemophilia.—One of the outstanding features of hemophilia is hemorrhage into the joints. This commonly follows slight trauma. Keefer and Myers [24] have emphasized that the changes brought about by repeated trauma and hemorrhage into the joints in patients with hemophilia were often indistinguishable from those of degenerative arthritis. In these cases, there is little doubt that the influences of the hemorrhage resulting from slight injury are capable of producing the derangements of the joints.

Aside from these clinical observations, there are a number of carefully controlled experiments on animals which support the hypothesis that degenerative arthritis results from frequent traumas to opposing articular surfaces. They are reviewed briefly.

Experimental Degenerative Arthritis.—Numerous attempts have been made to reproduce the gross and microscopic changes of degenerative arthritis in animals. A variety of methods have been used, but the favorite ones have been those which damage the cartilage and subchondral bone. Other methods, such as the production of a decreased blood supply and of increased pressure on the surfaces of the joints, and the displacement of articular surfaces so that they are no longer mutually adapted to one another have been employed.

The most recent work in America has been done by Key [21] and by Bennett, Bauer and Maddock.[25] They studied the changes in the joints following the production of defects in the articular cartilage and noted alterations which were similar to those occurring in degenerative arthritis. Bennett, Bauer and Maddock were able to produce this picture with absolute regularity only when the patella became displaced following operation. When the defects in the cartilage of the femur were produced and the patella did not become displaced, the joints

24. Keefer, C. S., and Myers, W. K.: Hemophilic Arthritis, New England J. Med. **208**:1183 (June 8) 1933.

25. Bennett, G. A.; Bauer, W., and Maddock, J. J.: A Study of the Repair of Articular Cartilage and the Reaction of Normal Joints of Adult Dogs to Surgically Created Defects of Articular Cartilage; "Joint Mice" and Patellar Displacement, Am. J. Path. **8**:499, 1932.

(Continued)

remained essentially normal, except for the area damaged by the operation. These experiments are of the highest importance in showing the effect of incongruities of articular surfaces in producing degenerative arthritis. Other observers, notably, Axhausen [26] and Ely and Cowan,[27] produced changes in cartilage and bone similar to those seen in degenerative arthritis by destroying the cartilage with chemicals or with a knife. In some cases border exostoses were produced, but never with regularity. Other forms of trauma, such as freezing the joints and injury by percussion with a rubber hammer, have been used to produce arthritic changes in the joints, with reasonable success.

The effect of abnormal pressure on articular surfaces has been investigated by Müller.[28] He sutured the humeri of rabbits to the scapula and immobilized the shoulder so that the tendon of the biceps was in contact with the cartilage of the humerus. The result of these experiments was an erosion of the cartilage and changes in the bone caused by pressure. He did not observe the characteristic alterations of degenerative arthritis and concluded that the functional factors involved in movement following trauma were of importance in the production of lesions. When he produced loosening of the epiphysis and subluxation of the head of the femur in different animals a necrotizing and repair process on the head of the femur appeared, with the typical findings of degenerative arthritis.

Key [21] tested the effect of abnormal strain on joints by attempting to produce knock-knee in rabbits. In the animals in which the patella was displaced outward so that it rested on the femoral condyle chronic arthritis developed, with border exostoses. In these animals other disturbances than displacement of the patella had been produced during the manipulations; the lower end of the femur had been fractured, the epiphysis had slipped or the cruciate ligaments had been torn. In other words, damage to the joints as well as the mechanical strain produced by the lack of mutually adapted articular surfaces were important in producing the picture.

Aside from the fact that trauma, mechanical strain and surface irregularities are essential for the experimental production of arthritic changes in the joints, movement of the damaged joint is of the highest importance in determining the anatomic picture. This feature has been investigated at length by Burckhardt.[29] He injured the cartilage of

26. Axhausen, Georg: Arch. f. klin. Chir. **99**:519, 1912.

27. Ely, L. S., and Cowan, J. F.: Bone and Joint Studies, Stanford University, Calif., Stanford University Press, 1916, p. 38.

28. Müller, W.: Experimentelle Untersuchungen über Druckusuren an Gelenkenden für die Pathogenese der Arthritis deformans, Deutsche Ztschr. f. Chir. **180**:203, 1923.

29. Burckhardt, H.: Experimentelle Untersuchungen über die Beziehungen der Gelenkfunktionen zur Arthritis deformans, Arch. f. klin. Chir. **132**:706, 1924.

the joints with phenol and studied the changes which developed in those which were immobilized by paralysis of the extremities by cutting the nerve plexus; he compared them with joints which were similarly injured but which were allowed to move. The final picture was different. In the joints which were immobilized the cartilage was gradually replaced by connective tissue and new cartilage which tended to fill the joint cavity and produce ankylosis. The connective tissue (pannus) grew into the articular space from the periphery of the joint, and exostoses were never observed. In the joints which were not immobilized exostoses developed, and the picture was similar to that seen in typical degenerative arthritis.

The experiments of Müller, cited previously, likewise emphasize the importance of movement in producing the characteristic changes in the joints. As he never produced characteristic lesions in immobilized joints he emphasized the importance of the functional aspect of disease of the joints.

In view of these observations, it seems well established that the lesions of degenerative arthritis may be reproduced in animals by a variety of methods which injure cartilage and bone and alter normal articular function. The picture which is characteristic of degenerative arthritis, then, is the result of the process of repair to damaged tissues of the joint.

From our observations and from those of other investigators, it seems difficult to escape the conclusion that the changes which are seen in the joints with increasing frequency with advancing age are identical with those which have been previously described as characteristic of degenerative arthritis. If this is true, there is justification for the belief that degenerative arthritis is a process associated with the aging of the tissues of the joints. This conception is essential for a complete understanding of the pathogenesis of this disorder. Added to the process of involution, such factors as gross trauma, hemorrhage and static deformities exaggerate the condition. The end-result depends on the summation of these factors.

SUMMARY AND CONCLUSIONS

In a study of 100 knee joints from 77 consecutive patients who died of various diseases the following facts were determined:

1. Anatomic changes were noted with increasing frequency with advancing age.

2. The patella showed alterations in 81 per cent of the cases, the interpatellar groove in 65 per cent, the lateral condyle of the tibia in

(Continued)

64 per cent, the medial condyle of the tibia in 55 per cent, the medial condyle of the femur in 43 per cent and the lateral condyle in 36 per cent.

3. The erosions were commonest over the areas of contact which were subjected to the greatest movement, strain, weight-bearing and injury.

4. The changes were identical in males and females, and there was no relationship between the extent of the lesions in the joints and the symptoms referable to the joints.

5. There was no correlation between the lesions in the joints and the degree of arteriosclerosis or any other particular type of disease process.

6. The gross anatomic changes were indistinguishable from those previously described in degenerative arthritis.

7. The various factors which are of importance in the development of degenerative arthritis are discussed. They include the aging of tissue, wear and tear, strain, trauma, occupation and static deformities.

14
Pressure Ulcers

Mary Ann Forciea, MD

Reproduced paper following commentary: The National Pressure Ulcer Advisory Panel: Pressure Ulcers Prevalence, Cost, and Risk Assessment: Consensus Development Conference Statement. Decubitus, volume 2 (2) pp. 24–28, 1989. Permission from Lippincott, Williams & Wilkins.

Commentary

Perhaps no physical sign is more greatly associated with frailty and dependency in the minds of clinicians, patients, and families than is the decubitus ulcer (pressure sore, pressure ulcer). Derived from the Latin roots "decubere" (to lie down) and "ulcus" (sore or disruption), decubitus ulcers develop over bony prominences or other areas of pressure in patients who are immobile and often thin, anemic, malnourished, and/or dehydrated. In an effort to improve clarity for education, the term "pressure ulcer" replaced "decubitus ulcer" in the 1980s. The efforts to treat pressure ulcers have led to a wide variety of dressings or covers and to the growth of industries manufacturing special surfaces on which patients may recline. The development, severity, and progression of pressure ulcers are used as measures of quality of care in many settings.

The standard of description of pressure ulcers involves some method of measurement (length × width, tracings, and photographs), with comments on the depth of tissue involvement. This depth is described in stages: I: nonblanchable erythema, II: disruption of the skin surface, III: disruption through the dermis, and IV: muscle invasion. Although standardized in the final report of a consensus conference from the National Pressure Ulcer Advisory Panel in 1989 and reproduced here, this depth of involvement staging system is credited to the paper by Irvine et al (1). In this study by Irvine and coworkers, 395 patients admitted to an inpatient geriatric unit were randomly assigned to receive the anabolic steroid norethandrolone or placebo in an effort to improve nitrogen balance and prevent pressure ulcer development. The authors describe a method of depth of involvement to help quantify their results. While their intervention showed no benefit, their method of quantification lives on today.

Because of the high morbidity and mortality associated with the appearance of pressure ulcers, many efforts have focused on prevention. Several risk assessment instruments that can be used by nursing staff have appeared. The best known of these is the Braden Scale for Predicting Pressure Sore Risk. This instrument has 6 subscales, which focus on sensory perception, skin moisture, activity, mobility, friction/shear, and nutritional status. (2). The Braden Scale itself is available as part of the Web site of AHCPR Supported Clinical Practice Guideline: Pressure Ulcers in Adults: Prediction and Prevention (see reference list). The Guideline itself is an excellent review of the evidence base which supports our current principles of screening, prevention, and treatment. Another excellent resource for practice and teaching is the volume on pressure ulcers developed by Clinics in Geriatric Medicine (3).

References

1. Irvine RE, Memon AH, Shera AS. Norethandrolone and prevention of Pressure-Sores. Lancet 1961;2:1333–1334.

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

2. Bergstron N, Braden BJ, Laguzza A, Holman V. The Braden Scale for Predicting Pressure Sore Risk. Nursing Res 1987;36:205–210.
3. Thomas DR, Allman RM (eds). Clinics in Geriatric Medicine —Pressure Ulcers. Philadelphia: W.B. Saunders. 1997.

Web sites

AHCPR Supported Clinical Practice Guideline 3- Pressure Ulcers in Adults: Prediction and Prevention http://www.ncbi.nlm.nih.gov/books

PRESSURE ULCERS PREVALENCE, COST AND RISK ASSESSMENT: CONSENSUS DEVELOPMENT CONFERENCE STATEMENT

The National Pressure Ulcer Advisory Panel

Introduction

For most Americans, a bed sore is a remote problem confined to elderly, immobile persons. However, a 16-year-old who has been in an accident, a veteran with a spinal cord injury or a child in a cast all are potential victims, as well.

Medically known as pressure ulcers, these wounds are serious and cause suffering, disability and even death. A pressure ulcer occurs when soft tissue is compressed over a period of time. The precise number of people who have pressure ulcers is unknown. However, conservative estimates indicate that well over a million persons in hospitals and nursing homes suffer from pressure ulcers. One large study found that one in four Americans who died in 1987 had a dermal ulcer. The prevalence of pressure ulcers is almost certain to grow due to increasing numbers of fragile elderly and survivors of serious trauma.

Lack of research, funding or even interest in pressure ulcers hinders both prevention and treatment. Disagreements about such basic matters as definition makes it difficult to analyze and interpret many of the studies which have been published. Moreover, the common practice of ascribing ulcers to poor care adversely affects comprehensive and accurate reporting. Better understanding of the epidemiology, costs, and risk factors associated with pressure ulcers is essential to developing effective prevention strategies and treatments.

This consensus conference evolved as an initiative of the National Pressure Ulcer Advisory Panel (NPUAP) to focus the attention of researchers, clinicians, health policy makers and financing organizations on several important questions. In May 1988, the panel, a multidisciplinary group of experts in the prevention and management of pressure ulcers, formulated a set of questions that are, as yet, inadequately explored.

On March 1-3, 1989, the National Pressure Ulcer Advisory Panel sponsored a National Consensus Conference on pressure ulcers in Washington, D.C. After a day and a half of presentations by researchers from multiple professional perspectives, a consensus panel consisting of experts in geriatric medicine, nursing, plastic surgery, spinal cord injury, health policy, health care economics, epidemiology, and a consumer advocate considered the evidence and developed responses to the following questions:

1. What is the prevalence and incidence of pressure ulcers in acute, long-term and home care settings?
2. How can the epidemiology of pressure ulcers in the United States be determined?
3. What is the cost of prevention and/or care of pressure ulcers?
4. What is the appropriate methodology for determining costs of pressure ulcers?
5. What should be included in risk assessment for pressure ulcer prevention?
6. Can risk assessment tools be effectively used to decrease pressure ulcers?
7. What are the needs for future

(Continued)

CONFERENCE STATEMENT

research related to pressure ulcers?

1. What is the prevalence and incidence of pressure ulcers in acute, long-term, and home care settings?

Pressure ulcers are localized areas of tissue necrosis that tend to develop when soft tissue is compressed between a bony prominence and an external surface for a prolonged period of time. These wounds have been referred to by many names including decubitus ulcers, bedsores, pressure sores, and pressure ulcers. Since pressure is an essential factor in the development of these wounds, the term pressure ulcers seems to be most accurate in describing these lesions.

Pressure ulcers represent a continuum from an erythematous soft tissue lesion to an open wound extending into the deep tissues. A number of different systems have been developed for classifying pressure ulcers based on their depth. Each of these systems has utility, but they are not interchangeable. This leads to difficulty when trying to compare existing studies. It is important that clinicians and researchers are clear about the system used to classify pressure ulcers in order to compare studies of prevalence, incidence, efficacy of treatment, and costs of prevention.

The following classification system combines several of the most commonly used staging systems and is offered to serve as a step in the evolution of a universally accepted classification system. While the Stage I lesion has different anatomic and treatment implications than Stages II through IV it represents an important clinical lesion. The classification system is as follows:

Stage I: Non-blanchable erythema of intact skin; the heralding lesion of skin ulceration.

Stage II: Partial thickness skin loss involving epidermis and/or dermis. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage III: Full thickness skin loss involving damage or necrosis of subcutaneous tissue which may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.

Stage IV: Full thickness skin loss with extensive destruction, tissue necrosis or damage to muscle, bone, or supporting structures (e.g., tendon, joint capsule, etc.)

Using additional descriptions of these wounds, particularly surface size and characteristics, would further clarify assessment of these lesions.

There are several methodological limitations in assessing the incidence and prevalence of pressure ulcers in various healthcare settings. These can be broadly grouped into problems with the comparability of various sample populations, sources of data, and methods of study. Any of these may confound interpretation of the estimates.

Most studies have been undertaken in single sites, and are thus

CONFERENCE STATEMENT

not generalizable to other institutions or populations. Much of the recent incidence data in acute care settings is derived from patients hospitalized in tertiary care hospitals and may not reflect the patient population in community hospitals. Likewise, studies in individual nursing homes may be unique to their specific case mix.

The type of data utilized varies widely, from observational studies in a single nursing home, to large national data sets. There are constraints in using any of these to predict incidence or prevalence. For example, hospital discharge data bases contain only five to seven diagnoses which may lead to an underestimation of the prevalence of pressure ulcers in acute care settings.

Studies also vary with regard to the unit of analysis. Some researchers describe the incidence of individual pressure ulcers, while others focus on the number of patients affected. Patients often have multiple ulcers, thus depending on which approach is used, studies may report two- or three-fold differences in rates for the same population. Clarity in the unit of analysis is necessary.

It is imperative that researchers be precise in the use of the terms incidence and prevalence. Incidence refers to new cases occuring over a given time period, while prevalence includes new and old cases and is usually assessed on a cross-sectional one-time basis. Further, to assure comparability of prevalence and incidence data, it is important for reports to specify whether Stage I lesions are included or excluded in these calculations. Adherence to methodological rigor will allow more useful interpretation of studies.

Reliability of estimates is also compromised by inconsistency in report methods and exclusion criteria employed by researchers. One source of confusion relates to the composition of study populations; while some studies include all admissions, others exclude certain groups such as pediatric, maternity and ambulatory surgical patients. The implication of pressure ulcers as an indicator of quality of care appears to be a deterrent to accurate reporting. Additionally, because patients transfer among different healthcare settings, data needs to be interpreted carefully.

Acute care settings

Estimates of the *prevalence* pressure ulcers range between 3% and 14% among hospitalized patients depending upon the source of data inclusion of Stage I lesions and the sample population. Most studies report a range of 5%-11%. On the other hand, *incidence* among hospitalized patients from several studies was found to range between 1% and 5%.

Long term care settings

The prevalence of pressure ulcers among patients on admission to skilled nursing facilities ranges between 15% and 25%. The incidence depends upon length of stay and is difficult to generalize since it is also institution-specific and is influenced by the patient's acuity level.

These statistics are complicated by the heterogenity of facilities that are considered long-term care. Facilities vary in their proportions of skilled and intermediate beds and different case mixes.

Home care settings

Since persons with pressure ulcers receiving professional home healthcare are a subset of the larger population of persons with pressure ulcers who are living at home, it would be helpful to consider both groups. However, data is available only on those receiving home health care services. Two recent studies have revealed pressure ulcer prevalence rates of 7% and 12% in this group. National surveys may help determine estimates for the total home dwelling population.

2. How can the epidemiology of the pressure ulcers in the United States be determined?

The epidemiology of pressure ulcers requires a systematic attempt to understand the natural history of the condition and its numerous treatment modalities. Because of the various stages of pressure ulcers, settings in which they are treated, and populations afflicted, consideration of study design should include careful assessment of the generalizability of the results. Studies concentrated in single facilities are limited in utility. It is advisable to undertake multi-center studies to identify independent risk factors and the natural course of the disorder.

3. What is the cost of prevention and/or care of pressure ulcers?

Based upon what is known about other medical problems, it can be speculated that prevention of pressure ulcers will be less expensive (i.e., more cost effective) than treatment of ulcers. However prevention implies careful surveillance which involves staff training and time, (i.e., nurse, dietician, pharmacist, physician) and in some cases prevention also implies intervention targeted at the reduc tion of risk factors such as malnu trition and immobility. Simple measures such as turning patients on a regular or frequent schedule may involve changes in staffing patterns which may be costly to an institution. Both patient assessment and reduction of risk factors involve staff time that needs to be calculated when analyzing the cost of pressure ulcer prevention.

Without knowing the precise incidence/prevalence of pressure ulcers, it is impossible to predict their total national cost. Also, few estimates are available of per case treatment costs in acute, long-term or home care settings. Estimates of average per case financial cost of pressure ulcer treatment in acute

(Continued)

CONFERENCE STATEMENT

care settings range widely, from approximately $2,000 to $30,000. Lower estimates are typically for cases in which "pressure ulcer" is not the primary diagnosis. The wide range of costs for care of pressure ulcers reported in the literature may be related to inconsistencies or inaccuracies in data collection on ulcer assessment and management cost. Data providing information on ulcer treatment by stage, concomitant health problems, or patient demographic characteristics are lacking. In addition, the cost of hospitalization is not typically broken down to identify multiple services, (e.g., surgeons, dieticians, nurses) that are involved in the treatment of pressure ulcers. In many cases, too, hospital costs reflect total patient care including management of chronic illness which is likely to predispose one to ulcer formation. Thus the specific cost of ulcer treatment is difficult to separate.

Less is known about the actual cost of pressure ulcer management in long-term care facilities. This may be particularly difficult given that that the presence of a pressure ulcer may necessitate transfer of the patient back to the acute care facility. The cost of home care management of pressure ulcers is unknown. Regardless of the patient management site, the cost of the use of protective devices (e.g., specialized mattresses, heel protectors) appears to be escalating and deserves study.

4. What is the appropriate methodology for determining costs of pressure ulcers?

Cost measurement should focus on the incremental (added) costs due to ulcer care (for example, hospital room costs should be excluded if the patient's primary diagnosis is not the ulcer). Major cost elements to be considered in such analyses include physician fees, the cost of devices and equipment, major supplies, laboratory services, drugs, and the room and board charges associated with longer lengths of stay, where applicable. Whether or not nursing care time is included in the analysis depends on the underlying assumptions which should be stated. In addition, important related costs such as those associated with appropriate nutrition and mobility should also be considered.

5. What should be included in risk assessment for pressure ulcer prevention?

A number of clinical factors have been implicated in increased risk of pressure ulcers. Those most consistently described include:

- Immobility
- Inactivity
- Malnutrition
- Fecal and urinary incontinence
- Decreased level of consciousness

Other factors that have been reported as a predictive for some

CONFERENCE STATEMENT

groups and certain settings include:

- Advanced age
- Fracture
- Chronic systemic illness

There may be other important clinical, functional, and demographic variables which need further clarification.

Caregiver and institutional variables such as staffing patterns, levels of nursing education, etc., have not been well studied but would appear to be of great importance and thus merit attention.

Also the quantifiable biochemical, anatomical, and biophysical assessment of risk and of pressure ulcers themselves appears to hold great promise for better understanding and deserves further independent research.

6. Can risk assessment tools be effectively used to decrease pressure ulcers?

A risk assessment tool is not an end unto itself. It is only potentially useful insofar as it is linked to intervention. To date, a number of practical instruments have been developed to identify patients at increased risk for pressure ulcer development. These instruments contain variable elements of assessment. Many are sufficiently promising as to warrant further evaluation as screening tools. An ideal instrument for clinical use should have good predictive value, high sensitivity and specificity, and ease of use.

Evidence suggests that use of such instruments by trained individuals can help to identify high-risk patients. Early intervention strategies may then be targeted to these individuals. In addition, regular use of any assessment scale may be beneficial by making staff more aware of the problem of pressure ulcers. Work needs to be done to determine the populations and settings appropriate for each scale.

7. What are the needs for future research related to pressure ulcers

- Better understanding of underlying mechanisms in the etiology and natural history of pressure ulcers is needed.
- Instruments for assessing risk need refinement and testing as they apply to various populations and settings.
- To obtain accurate information on the incidence and prevalence of pressure ulcers, existing national surveys should be explored for potential utility in generating more valid and reliable data.
- Multisite studies are needed to determine the epidemiology and cost of treating pressure ulcers by stage, setting (e.g., hospitals, nursing home) and other factors (e.g., acuity level, functional status, morbidity). Particular attention should be paid to high-risk groups such as spinal cord injured patients, the frail elderly population and those who are immobilized or chronically debilitated.
- Studies to determine cost-benefit and cost effectiveness of preventive strategies and treatments are needed.
- Systematic studies are needed for validation of identified risk factors and existing scales thought to predict occurrence. Potential interventions for prevention of pressure ulcers also need systematic study.
- The role of nutrition in the etiology, prevention and treatment of pressure ulcers needs special attention.
- Education of healthcare providers and family caregivers regarding early identification and prevention of ulcers in the high-risk patient should be explored.

Conclusions and recommendations

- Pressure ulcers are a significant and increasing source of considerable human suffering.
- Minimum cost estimates indicate that the cost of pressure ulcer treatment contributes significantly to the financial burden of patients, families, institutions, and healthcare payment systems. Additional research must be funded to better determine the costs of prevention and treatment.
- There is a need for a major investigative intitiative into pressure ulcers due to the paucity of reliable, generalizable data.
- Current classifications and descriptions of pressure ulcers are confusing and inconsistently utilized in much of the current pressure ulcer literature. The panel recommends adoption of a uniform staging system.
- Expert opinion suggests that lack of reimbursement for prevention adversely influences the performance of risk assessment and early intervention.
- Clinicians and researchers must work together to explore practical methods for prevention and treatment of pressure ulcers.
- Systematic surveillance methodologies and reporting mechanisms which are appropriate for specific types of healthcare institutions need to be developed.
- Current developmental bioengineering methodologies should be explored for their potential application to preventive and treatment aspects of the pressure ulcer.
- More detailed discussions, possibly in future consensus conferences, are needed to address in sufficient detail, the current state of knowledge on preventive and treatment aspects of pressure ulcers.

15
Depression

Monica K. Crane, MD

Reproduced paper following commentary: Kraepelin E. (1904) Lecture I: Introduction: Melancholia. Lectures on Clinical Psychiatry, pp.4–10. New York: William Wood.

Commentary

More than two millennia ago, ancient Greek and Roman physicians first recognized the high prevalence of depression or melancholia in older adults. In the fourth century BC, Hippocrates made an early reference to distress and melancholia or "an excess of black bile." He defined melancholia (black bile) as a state of "aversion to food, despondency, sleeplessness, irritability and restlessness." Later, Galen (131–201 AD) described melancholia as a manifestation of "fear and depression, discontent with life and hatred of all people." Subsequent Greco-Roman medicine not only recognized the symptoms of melancholia in the form of fear, suspicion, aggression, and death wishes, but also referred to environmental contributions to melancholia as immoderate consumption of wine, perturbations of the soul due to passion, and disturbed sleep cycle.

At the turn of the twentieth century, several attempts were made to clarify the concept of melancholia and bring it closer to what would now be equated with depression. In 1904, Emil Kraepelin, a pioneer in experimental psychiatry (1), published his landmark Clinical Lecture series, in which he was one of the first authors to view melancholia as a disease independent of normal aging. In Lecture I: Melancholia (reproduced here in English translation), the professor attempts to define the causation, nature, and presentation of melancholia (2). He distinguishes himself in his approach to melancholia (depression) and other mental disorders in that he bases his classification on symptom patterns rather than on simple similarity of major symptoms, as did his predecessors (3).

In *Lectures I*, Kraepelin chose three exemplary cases to describe the different symptom patterns of depression. Kraepelin described melancholia as a pattern of symptoms: "an illness which began gradually . . . without any assignable cause . . . loss of sleep and appetite . . . stomach-ache and head troubles." Kraepelin also comments on prognosis and that the "termination of the illness is generally pretty favorable." He reported that "all three patients are of considerable age. This is not an accident. Melancholia, as we have described it here, sets in principally, or perhaps exclusively, at the beginning of old age in men, and in women from the period of the menopause onwards."

Kraepelin was one of the few to remark upon the differences between mental disease in the old and young. Given that he believed that biological processes were the underpinning of the major psychiatric disorders, he attempted to distinguish depression appearing in later life from earlier onset "insanity." He was confident that brain pathology and genetics were central to psychiatry, and he collaborated with Nissl and Alzheimer who discovered the pathologic basis of what is now known as Alzheimer's disease.

Although Kraepelin's symptom list of depression is very similar to that found in the *Diagnostic and Statistical Manual of Disorders*, his treatment regimen (paraldehyde, alcohol, and opium) was well before the advent of present antidepressants. It was not until the 1950s that treatments for depression were fortuitously discovered; iproniazid (a monoamine oxidase inhibitor) was developed for treatment of tuberculosis and imipramine (a tricyclic antidepressant) was developed as an antihistamine (4).

Great progress in the study of geriatric depression and psychiatry only occurred after World War II. Dr. Felix Post, one of the first geropsychiatrists, noted that "old age" depression was better defined in the German psychiatric literature and was not recognized within American and British psychiatry, where symptoms of

From: *Aging Medicine: Classic Papers in Geriatric Medicine with Current Commentaries*
Edited by: Robert J. Pignolo, Monica K. Crane, Mary Ann Forciea

depression were assumed to be part of a dementia in old age rather than a specific entity in their own right (5).

References

1. Meyer A. Emil Kraepelin, M.D. Am J Psychiatry 1927;83: 748–755.
2. Kraepelin E. Lecture I: Introduction: Melancholia. Lectures on Clinical Psychiatry, pp 4–10. New York: William Wood. 1904.
3. Kraepelin E. Classification of Mental Diseases. Lectures on Clinical Psychiatry, pp xv) New York: William Wood. 1904.
4. Slattery DA, Hudson AL, Nutt DJ. The Evolution of Antidepressant Mechanisms. Fundamental Clin Pharmacol 2004;(18): 1–21.
5. Post F. Interview by Professor Margot Jeffreys. In Oral History of Geriatrics as a Medical Specialty. 1991. Retrieved from http://cadensa.bl.uk/uhtbin/cgisirsi/WKWUp2qbPB/267030007/9Also: cadensa.bl.uk/uhtbin/cgisirsi/sQDTDSuI2F/204950006/9.

CLASSIFICATION OF MENTAL DISEASES

T. JOHNSTONE.

(Continued)

INTRODUCTORY LECTURES ON CLINICAL PSYCHIATRY

LECTURE I

INTRODUCTION: MELANCHOLIA

GENTLEMEN,—The subject of the following course of lectures will be the Science of Psychiatry, which, as its name implies, is that of the treatment of mental disease. It is true that, in the strictest terms, we cannot speak of the mind as becoming diseased, whether we regard it as a separate entity or as the sum total of our subjective experience. And, indeed, from the medical point of view, it is disturbances in the *physical foundations* of mental life which should occupy most of our attention. But the incidents of such diseases are generally seen in the sphere of psychical events, a department with which the art of medicine has dealt very little as yet. Here we are not so much concerned with physical changes in size, shape, firmness and chemical composition, as with disturbances of comprehension, memory and judgment, illusions, hallucinations, depression, and morbid changes in the activity of the will. With the help of the ideas you have derived from general pathology, you will usually be able to find your way in a new department of medicine without any serious difficulty. But here you will be utterly perplexed at first by the essentially peculiar phenomena of disease with which you will meet, until you have gradually learned to a certain extent to master the special symptomatology of mental disturbances. Of course, you will sometimes have met with isolated conditions of mental disease in everyday life, or in other hospitals—intoxication, fever delirium, and delirium tremens, or even imbecility

and idiocy—but they may have impressed you more as strange and incomprehensible curiosities than as adding to your stock of medical ideas.

Insanity works a change in the mental personality, that sum of characteristics which, to our minds, represents a man's real being in a far higher degree than his physical peculiarities. Hence, our patient's whole relation to the outside world is affected in the most comprehensive way. The knowledge of all these disturbances is a fruitful field for the investigation of mental life, not only revealing many of its universal laws, but also giving a deep insight into the history of the development of the human mind, both in the individual and in the race. It also provides us with the proper scale for comprehending the numerous intellectual, moral, religious, and artistic currents and phenomena of our social life.

But it is not these variously branching scientific relations to so many of the most important questions of human existence which make a knowledge of psychical disturbances indispensable to the physician ; it is rather their extraordinary *practical importance.* Insanity, even in its mildest forms, involves the greatest suffering that physicians have to meet. Only a comparatively small percentage of mental cases are permanently and completely cured in the strictest sense of the word. And the number of the insane, which will hardly be exaggerated if we estimate it as amounting at the present moment to 200,000 in Germany alone, is apparently increasing with the most unfortunate rapidity. This increase may depend, to a great extent, on our fuller knowledge of insanity, on the more highly-developed care of the insane, and on the increasing difficulty of treating them at home, and so may be only apparent. But, considering that from one-quarter to one-third of the cases admitted to our asylums are due to the abuse of alcohol or to syphilitic infection, and that these are causes of which the extension is certainly not diminishing, we cannot but suppose that the number of the insane is increasing, not only in itself, but also in its proportion to the population. The growing degeneration of our race in the future may therefore still be left an open question, but certainly it might be very greatly promoted by both these causes.

All the insane are dangerous, in some degree, to their neighbours, and even more so to themselves. Mental derangement is the cause of at least a third of the total number of suicides,

(Continued)

while sexual crimes and arson, and, to a less extent, dangerous assaults, thefts, and impostures are often committed by those whose minds are diseased. Numberless families are ruined by their afflicted members, either by the senseless squandering of their means, or because long illness and inability to work have gradually sapped the power of caring for a household. Only a certain number of those who do not recover succumb at once. The greater part live on for dozens of years, imbecile and helpless, imposing a heavy and yearly increasing burden on their families and communities, of which the effects strike deeply into our national life.

For all these reasons, it is one of the physician's most important duties to make himself, as far as possible, acquainted with the nature and phenomena of insanity. Even though the limits of his power against this mighty adversary are very narrow, opportunity enough is afforded to every practical physician to contribute his share to the prevention and alleviation of the endless misery annually engendered by mental disease. Alcoholism and syphilis undoubtedly offer the most profitable points of attack, together with the abuse of morphia and cocaine, which so clearly owes its fatal significance to the action of medical men. Family physicians, again, can often help to prevent the marriage of the insane, or of those who are seriously threatened with insanity, and to secure a proper education and choice of occupation for children predisposed to disease. But it will be their special province to recognise dangerous symptoms in time, and, by their prompt action, to prevent suicides and accidents, and obviate the short-sighted procrastination which so often keeps patients from coming under the care of an expert alienist until the time for practically useful treatment has long been past. Even in those numerous cases which never become insane in the narrower sense, the physician who has been trained in alienism will have such an understanding of the recognition and treatment of psychical disturbances as will amply repay him for the trouble of his years of study. Even in my own experience it has happened very often that older physicians have regretted their defective knowledge of alienism, and complained that it was only in practical life that they learned how great a part is played, in the daily round of ordinary medical practice, by the correct diagnosis of more or less morbid mental incidents. I need hardly mention that, for various reasons, such a diagnosis is in

4 Lectures on Clinical Psychiatry

constant demand by public authorities, courts of law, and trade societies.

Of course, an intimate knowledge of Psychiatry, as of every other separate branch of medicine, can only be acquired by long and thorough occupation with the subject. Yet, even in a short time, it is possible to cast at least a general and superficial glance over the commonest forms of mental disturbance. Personal investigation and continuous observation of the greatest possible number of different cases are indispensable to this, and it is only too true that, even after one or two terms of zealous clinical study, there will still be many cases which the beginner is unable to interpret correctly by means of the knowledge with which he has been furnished or which he has acquired for himself. But one important advantage to be gained comparatively quickly is a recognition of the great *difficulties* of the subject and the correction of that simple-minded ignorance, still so widely spread, which assumes that even a non-expert may give an opinion on mental cases without any more ado.

After these introductory remarks, let us turn to the consideration of our patients. I will first place before you a farmer, aged fifty-nine, who was admitted to the hospital a year ago. The patient looks much older than he really is, principally owing to the loss of the teeth from his upper jaw. He not only understands our questions without any difficulty, but answers them relevantly and correctly; can tell where he is, and how long he has been here; knows the doctors, and can give the date and the day of the week. His expression is dejected. The corners of his mouth are rather drawn down, and his eyebrows drawn together. He usually stares in front of him, but he glances up when he is spoken to. On being questioned about his illness, he breaks into lamentations, saying that he did not tell the whole truth on his admission, but concealed the fact that he had fallen into sin in his youth and practised uncleanness with himself; everything he did was wrong. "I am so apprehensive, so wretched; I cannot lie still for anxiety. O God, if I had only not transgressed so grievously!" He has been ill for over a year, has had giddiness and headaches. It began with stomach-ache and head troubles, and he could not work any longer. "There was no impulse left." He can get no rest now, and fancies silly things, as if someone were in the room. Once it seemed to him that he had seen the Evil One: perhaps he would be carried off. So things seemed to him.

(Continued)

As a boy, he had taken apples and nuts. "Conscience has said that that is not right ; conscience has only awakened just now in my illness." He had also played with a cow, and by himself. "I reproach myself for that now." It seemed to him that he had fallen away from God, and was now as free as a bird. His appetite is bad, and he has no stools. He cannot sleep. "If the mind does not sleep, all sorts of thoughts come." He has done silly things too. He fastened his neckerchief to strangle himself, but he was not really in earnest. Three sisters and a brother were ill too. The sisters were not so bad ; they soon recovered. "A brother has made away with himself through apprehension."

The patient tells us this in broken sentences, interrupted by wailing and groaning. In all other respects, he behaves naturally, does whatever he is told, and only begs us not to let him be dragged away—"There is dreadful apprehension in my heart." Except for a little trembling of the outspread fingers and slightly arhythmic action of the heart, we find no striking disturbances at the physical examination. As for the patient's former history, he is married, and has four healthy children, while three are dead. The illness began gradually seven or eight months before his admission, without any assignable cause. Loss of appetite and dyspepsia appeared first, and then ideas of sin. His weight diminished a little after his admission, but has now slowly risen again 7 kilogrammes.

The most striking feature of this clinical picture is the *apprehensive depression.* At first sight, it resembles the anxieties of a healthy person, and the patient says that he was always rather apprehensive, and has only grown worse. But there is not the least external cause for the apprehension, and yet it has lasted for months, with increasing severity. This is the diagnostic sign of its morbidity. It is true that the patient himself refers to the sins of his youth as the cause of the apprehension, but it is clear that, even if they were ever really committed, they did not particularly disturb him before his illness ; his conscience has only awakened now. His actions now appear to him in an entirely different and fatal light, and those morbid symptoms become prominent which are known as "*delusions of sin.*" The patient's ideas that the Evil One was in the room, that he would be carried off, and that he had fallen away from God, must be regarded as a result of his apprehension. There is no question of real hallucinations in these statements ; it only *seemed so* to

the patient. He also has a strong feeling that some great change has come over him, and that he is " not the same as before." He is certainly not in a condition to form a correct conception of the morbidity of his ideas of sin and of his fears in detail.

We give the name of *melancholia* to this condition, in which we see the gradual development of a state of apprehensive depression, associated with more or less fully-developed delusions. The most common of these are ideas of sin, which generally have a religious colouring. Such are the ideas of having fallen away from God and being forsaken, or of being possessed by the devil. Hypochondriacal ideas—of never being well again, never having a stool again, etc.—are also far from uncommon. Together with these there is often apprehension of poverty, of having to starve, of being thrown into prison, of being brought before a court, or even of execution.

As a consequence of this mental unrest and these tormenting ideas, the wish to have done with life develops almost invariably, and patients very often become suicidal. Our first patient only made a rather feeble attempt at suicide, but I will now show you a widow, aged fifty-four, who has made very serious efforts to take her own life. This patient has no insane history. She married at the age of thirty, and has four healthy children. She says that her husband died two years ago, and since then she has slept badly. Being obliged to sell her home at that time, because the inheritance was to be divided, she grew apprehensive, and thought that she would come to want, although, on quiet consideration, she saw that her fears were groundless. She complained of heat in her head and uneasiness at her heart, felt weak and excited, and was tired of life, especially in the morning. She says she could get no sleep at night, even with sleeping-powders. Suddenly the thought came to her, " What are you doing in the world now? Try to get out of it, so as to be at rest. It's no good any longer." Then she hung herself up behind the house with her handkerchief, and became unconscious, but her son cut her down and brought her to the hospital.

Here she was quite collected, and was orderly in thought and behaviour. She understood the morbidity of her condition, but feared that she would never be well again. She said she could not bear it any longer, and could not stay here ; she was driven to despair. She was very fond of talking about her condition, and loudly lamented that she was so apprehensive, asking for a

(Continued)

clergyman to come and drive out the Evil One. At this she was seized with violent trembling in her whole body, and declared that she had no peace; she could not rest, her heart beat so; her head was bursting, she could not live any longer; she wished to die at home; thoughts of suicide tormented her unceasingly. Her sleep and appetite were bad, but no other physical disturbance could be discovered. In the course of the first few months her mental condition improved fairly quickly, and, at the urgent desire of her relations, leave of absence was granted with the family of her daughter. But the apprehension and thoughts of suicide became so marked that she had to be brought back to the hospital within a fortnight. Here her condition is still improving, though very slowly and with many fluctuations. Her recovery has been much delayed by a carious affection of the right parietal bone and the left wrist, which necessitated repeated interference, but is now in a tolerably healthy condition.

This patient, too, is quite clear as to her surroundings, and gives connected information about her condition. She has no real delusions, apart from fear that she will never be well again. Indeed, we find that the real meaning of the whole picture of disease is only permanent *apprehensive depression*, with the same accompaniments as we see in mental agitation in the sane—*i.e.*, loss of sleep and appetite, and failure of the general nutrition. The resemblance to anxiety in a sane person is all the greater because the depression has followed a painful external cause. But we can easily see that the severity, and more especially the duration, of the emotional depression have gone beyond the limits of what is normal. The patient herself sees clearly enough that her apprehension is not justified by her real position in life, and that there is absolutely no reason why she should wish to die.

This sense of the morbid nature of the apprehension, or "insight into the disease," is not always present in melancholia. In those cases, more especially, in which there are marked *delusions* this important symptom may be altogether absent for a long time together. As an example, I will show you a widow, aged fifty-six, who nursed her son when he was ill of typhus two and a quarter years ago. She then had a feverish illness herself, presumably also typhus, and lost her husband suddenly a few months later. Very soon after this she began to be apprehensive, and to reproach herself with not having taken proper care

of her husband. Strongly-marked delusions of sin quickly developed. She had never done anything properly, and had allowed herself to be led away by the wicked fiend. Her prayers had been no good, only she did not know that before. Her husband absolutely married the devil, and could not go to heaven, and she and her children were damned on account of her former unchristian life. Great restlessness and almost complete sleeplessness now came on. The patient lamented, shrieked, and wept persistently, her appetite quite failed, and she soon had to be brought to the hospital.

Here she was collected and clear about her surroundings, but gradually passed into a very severe state of apprehensive excitement, which found expression in monotonous and almost intolerable shrieks. She could only be interrupted for a short time by asking her questions, which she always answered. She also expressed a quantity of the most fantastic ideas. She had been the serpent in paradise, had led astray her husband, who was called Adam, and had made herself and her children accursed, and everyone unhappy. Therefore she was burning, was already in hell, and saw her fearful sins in the abyss. The firmament had fallen; there was no more water or money or food; she had ruined everything, and was guilty of the downfall of the world—" The whole world lies upon my soul." She accused herself of all these transgressions in a written document, addressed to the District Court, and begged to be taken to prison. She wrote her name on a label as " Devil."

In spite of all this, you are soon convinced that, even while she is senselessly shrieking and expressing delusionary ideas of this kind, the patient knows where she is quite well, knows the doctors, and gives broken but relevant answers to questions about the circumstances of her home. She also does sums correctly, though she returns at once to her monotonous lamentations. As the result of sleeplessness and insufficient nourishment, she is physically quite run down, but shows no other sign of illness. She has three healthy sons, while three of her children died in childhood. Her father is said to have been temporarily insane.

At first sight this clinical picture of disease seems different from the other and simpler forms. But it is easily shown that the variations are only a matter of degree. Both in the development of the delusions and in the strength and manifestations of the apprehension we meet with every conceivable transition,

(Continued)

from the form first described and generally known as melancholia simplex to the present morbid condition, and to even more marked cases. Often enough the same patient presents first one and then another type of symptoms at different times. It is therefore impossible to lay down any reliable clinical line of division in these cases.

All three patients are of considerable age. This is not an accident. Melancholia, as we have described it here, sets in principally, or perhaps exclusively, at the beginning of old age in men, and in women from the period of the menopause onwards. We might regard it as a morbid expression of the feeling of growing inadequacy, usually more or less noticeable in healthy people of the same age. Those who are morbidly disposed by nature of course become melancholic most easily, as is shown by our examples, and women seem more inclined to the disease than men. Of external influences, emotional shocks, and especially the death of near relations, often figure as the exciting cause, although they cannot be regarded as the original cause, on account of their absence in other cases. The termination of the illness is generally pretty favourable.* About a third of the patients make a complete recovery. In severe and protracted cases, emotional dulness may remain, with faint traces of the apprehensive tendency. Judgment and memory may also undergo considerable deterioration. The course of the disease is always tedious, and usually continues, with many fluctuations, for from one to two years, or even longer, according to the severity of the case.

The treatment of the malady cannot, as a rule, be carried out, except in an asylum, as thoughts of suicide are almost always present. Patients who show such tendencies require the closest watching, day and night. They are kept in bed and given plenty of food, though this is often very difficult, on account of their resistance. Care is also taken to regulate their digestion, and, as far as possible, to secure them sufficient sleep by means of baths and medicines. Paraldehyde is generally to be recommended, or, under some circumstances, alcohol, or occasional doses of trional. Opium is employed to combat the apprehension, in gradually increasing doses, which are then by degrees

* The first of these three patients has been well for more than five and a quarter years, and the second for a year. The third is still under treatment, but has improved after two and a half years' illness.

reduced. This remedy has often done very good service with our first two patients, while with the third we have had better results from small doses of paraldehyde. Great care is needed in discharging patients. If this is done too soon, as in the case of our second patient, serious relapses may result, with attempted suicide. Visits from near relations have a bad effect up to the very end of the illness.

Index