The Compression of Morbidity

JAMES F. FRIES

Stanford University School of Medicine

IN THIS CENTURY WE ARE PROGRESSING THROUGH three separate eras with dramatically different characteristics of health and illness. We entered the century in an era of infectious disease, with tuberculosis the number one killer of our population, and smallpox, diphtheria, tetanus, and other infectious illnesses extremely prevalent. A reduction in mortality from these diseases of over 99 percent (Fries and Crapo 1981; Cooper 1982) has led to the present era, where the major burdens of illness of the United States are the chronic diseases. Atherosclerosis and its complications, neoplasia, emphysema, diabetes, cirrhosis, and osteoarthritis have increased in prevalence even as the infectious illnesses which preceded them declined. It is one thesis of this discussion that this chronic disease era in its turn will slowly decline in significance, leaving a third era in which the major health problems of the United States will be directly related to the process of senescence, and where the aging process itself, independent of specific disease, will constitute a major burden of illness for the United States.

The ultimate constraint within which we must develop health policy for the future is, of course, the limit of life itself. Man is mortal, and the limits to what presently may be accomplished in decreasing

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mortality are set by the life span of our species. In the following discussion, we will examine natural limits to the life span, develop an incremental model of chronic disease and of aging which focuses upon the postponement both of disease and of senescent change, and examine the implications of improvement of vitality in a finite world.

It is important to define some terms. The "maximum life potential" is approximated by the oldest age achieved by any human being. In the United States, this age is 113 years, 214 days (McWhirter 1980). This figure represents a point far out on the "tail" of a distribution of genetically different individuals. "Life expectancy" is the average length of life which we may expect, given current age-specific death rates, for an infant born today. This figure is 73 at present, approximately 70 for men and 77 for women. The "life span," on the other hand, represents the average longevity in a society without disease or accident. The life expectancy can rise toward, but cannot exceed, the life span. The human life span appears to be approximately 85 years, with a broad distribution in which natural longevity for individuals falls nearly entirely within the range of from 70 to 100 years. Much misunderstanding in discussion of this topic comes from confusion of the mean of a distribution with its extremes.

The Finite Life Span

National mortality figures demonstrate a smooth decline in number of deaths and a smooth increase in mortality rates as we move toward higher ages (Fries and Crapo 1981). The 41,000 deaths per year in the United States in the eighty-fifth year of age has decreased to 24 by age 110, in a smooth progression which shows no exceptions. The absence of exceptions carries important implications since it demonstrates that particular lifestyles or particular food or vitamin intakes which have been promoted as aides to longevity and which have been used extensively throughout the culture do not, in fact, prolong the genetically determined life span. If vitamin C or lifelong aerobic exercise extended the life span, we should expect to have seen exceptionally long lives (beyond 115) in at least a few practitioners of such habits. The ultimate limits appear to apply to the aerobically fit and to the megavitamin faddist, to the farm or city dweller, and to all societies.

Examination of mortality rates in different societies confirms the

actuarial "law" first proposed by Benjamin Gompertz (1825). Gompertz noted a linear increase in mortality rates with age when rates were plotted on a logarithmic scale. That is, the mortality rate increases exponentially with age, doubling approximately every eight years of age. Gompertz's law is an empirical observation which fits closely with the observation of smoothly and rapidly declining numbers of individuals alive at successive ages.

Different species, on the other hand, have very different life spans; the obvious fact that we tend to outlive our pets is not due to increased disease or accident among the animals, but rather to the difference in species life span (Rockstein 1958). Rodents live, at best, a few years, while some of the Galapagos tortoises which formed a part of Charles Darwin's observations about the origin of species were still alive on the 100th anniversary of Darwin's death.

While the process of senescence certainly has biochemical and cellular underpinnings, it is presently best understood by decline of maximal function of the vital organs (Finch 1976; Shock 1960; Strehler 1960). The organ reserve potential, greatest in early life, shows a functional decline which is essentially linear and which is roughly parallel for all major organs. The decline is in the "reserve power" of the particular organ, and, thus, is apparent on measurement of maximum performance well before it is clinically visible as a limitation to activity of the organism. Studies of this physiologic decline, about 1.5 percent per year, uniformly indicate that the decline begins early in life in healthy individuals—well before it is reasonable to postulate any specific chronic disease effects (figure 1).

Decline in the function of multiple organs may be considered in the context of preservation of homeostasis. Reserve function is required when the organism is stressed in order to restore the normal homeostatic equilibrium. As the reserve of individual organs declines in a linear fashion, the ability to maintain homeostasis in the face of a threat of a given magnitude declines exponentially (Strehler 1960); hence, the observations of physiology and the actuarial observations of Gompertz are reconciled (figure 2). Natural death must ensue, without disease, when the reserve function has declined below that point, probably about 20 percent above basal levels, at which routine daily perturbations cannot be weathered. A transition from premature death to natural death occurs as the characteristics of the host resistance (homeostatic reserve) become more important than the specific nature of the insult

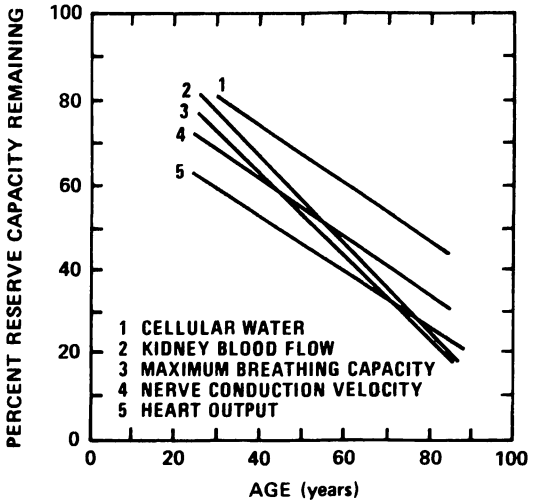

FIG. 1. Linear decline in organ function with age. Based on Shock 1960 and Strehler and Mildvan 1960. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

to the equilibrium. The concepts of premature death (due to disease or accident) and natural death (due to senescent frailty) are complementary rather than antagonistic, and any dividing line must be an arbitrary one.

Life expectancy from birth in this century in the United States has increased from 47 to 73 years (Fries 1980)—an increase of 26 years

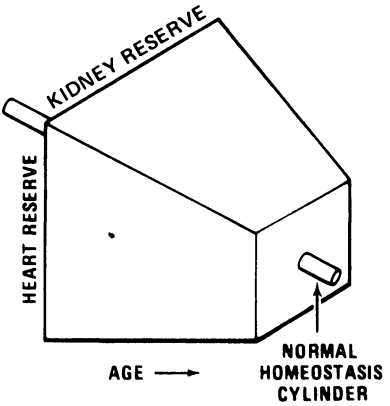

FIG. 2. Linear decline in multiple organs exponentially decreases the area within which perturbations may be restored as shown here for two organs. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

(figure 3). The rise has been reasonably constant throughout the century, with some periods of plateau and some periods of acceleration. This striking advance is not as apparent when one considers life expectancy from age 20, which has increased only some 13 years; life expectancy from age 40, which has increased eight years; life expectancy from age 60, which has increased 5 years; from age 80, 2.5 years; or from age 100, 0.7 years (Faber 1982). The greater slope of the curve representing life expectancy from birth reflects the great improvement in infant mortality over this period. In contrast, improvement in chronic disease control will result in a more nearly parallel slope to all lines, since these benefits accrue to individuals later in life. We are beginning to see, in terms of rate of change, some of these effects. To avoid misinterpretation of these "rate of change" data as indicative of galloping longevity, it is essential to look at absolute changes in life expectancy at the same time. Absolute changes in life expectancy, as above, show a progressive decline at higher ages.

Mortality statistics graphed as the percent surviving versus age are perhaps the most dramatic and decisive way to view the mortality rate events of this century (figure 4). In 1900 mortality occurred at a relatively steady rate throughout the life span. In successive decades, the curves have begun to bend upwards and to the right, each considerably different from the last. The form of the curve is increasingly rectangular,

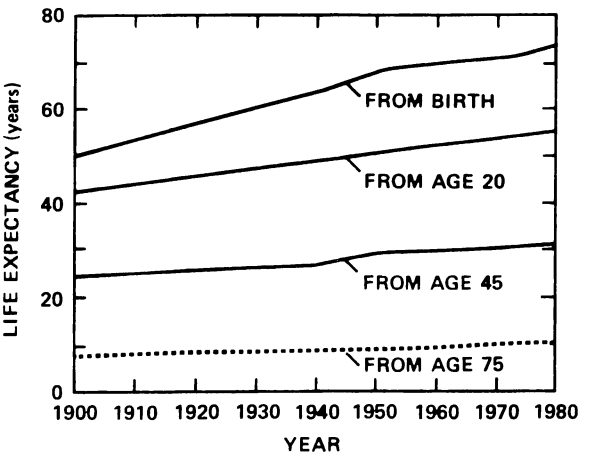

FIG. 3. Changes in life expectancy from different ages in the twentieth century. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

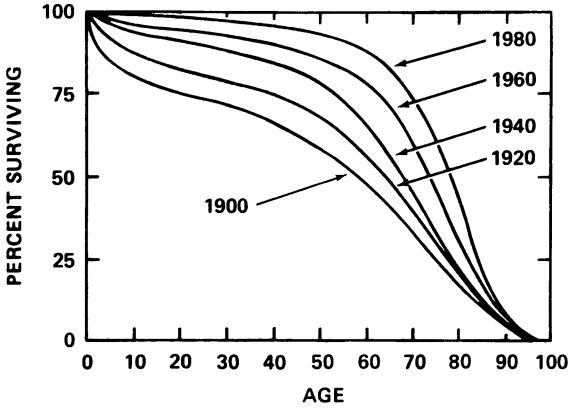

FIG. 4. Changes in survivorship curves in the United States in the twentieth century. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

having an increasingly flat top and an increasingly sharp downslope. This observation is frequently referred to as "rectangularization of the survival curve." The point on the age axis at which the curves insert has remained approximately the same, the differences lying within the width of the lines used to plot such curves. The progressive shape of these curves allows visual prediction of future trends. The ideal curve must lie in its initial 60 years within a very narrow flat zone, and then must plunge quite precipitously if it is to meet the historical lines at the bottom of the graph (figure 5). The increasingly rectangular survival curve, with the clear convergence of curves from different decades, demonstrates visually the limits of the human life span.

Thus, nine general lines of evidence confirm the existence of a finite human life span:

1) There are no exceptions to the declining numbers of individuals present at successive ages.

2) Gompertz's law appears to hold in all populations and assures an exponentially increasing mortality rate and, therefore, death for the entire population within a decade or two past the age of 100.

3) There has been no historical change over several centuries of observation with regard to maximum life potential, as underscored by studies of centenarians; this observation has been repeatedly made in the United States with good data since 1939 or earlier (Bowerman 1939; Faber 1982). Life expectancy at age 100 has changed at most 0.7 years over 80 years (Faber 1982), and much of this improvement

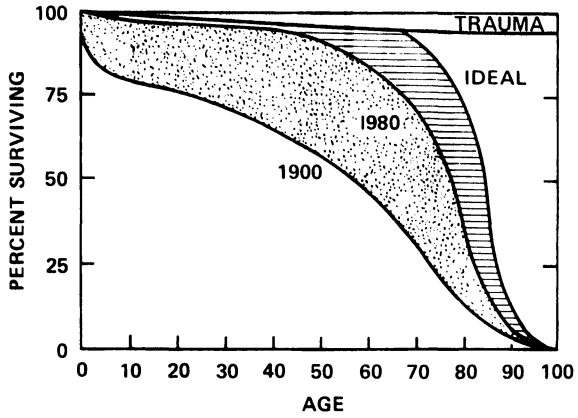

FIG. 5. Ideal survivorship curve. Trauma plays a large and potentially reversible role. Chronic disease accounts for almost all of the approximately ten-year-wide area of premature death remaining over ages 60–90. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

must have been due to reduction of premature death, not change in life span.

4) There is no biological reason to assume that any change in genetic longevity characteristics should have occurred merely because we have improved infant mortality, cleaned up water supplies, or invented penicillin.

5) The difference in species life span among animals is a commonplace daily observation.

6) Anthropological analyses (Cutler 1979) suggest a formula by which mammalian life spans may be predicted by the brain size/body weight ratio; such models suggest an approximately constant life span for the human species for the past 100,000 years.

7) The linear decline in organ reserve, repeatedly the subject of physiologic observation, mandates a point at which function must be inadequate to support life, that point apparently being when organ reserve is reduced to approximately 20 percent over that function required for the maintenance of basic life processes; reserve of this magnitude is required for daily functions outside of bed.

8) The increasingly rectangular curve demonstrates the barrier to immortality.

9) We have the important phenomenon of a priori aging, the daily evidence of our senses. People do grow older, with changes which

are apparent to all of us, as we age. And these changes—from hair color to hearing—are not the result of disease as we usually define it. A new group of Americans has become a subject of increasing concern, the "frail elderly." This term is a new one; it refers to individuals, often without demonstrable disease, who have manifestly limited organ reserve and increased frailty to external perturbation.

There are several methods of estimating the human life span. One may use the anthropological formulas, reconstruct an ideal survival curve from the tail of the present curve using the assumption that these individuals have been essentially free of disease, make extrapolations from the rectangularizing survival curve, or use estimates based on observed decline in organ reserve. All suggest an average life span of approximately 85 years, with a distribution which includes 99 percent of individuals between the ages of 70 and 100 (figure 5). It is not clear whether this distribution is "normal," based on the Gompertz function (which gives a slightly sharper drop-off) or some other distribution. For policy purposes, these distinctions are minor.

There is some controversy about whether the life span is totally fixed and about the precise projection of life span, but these disagreements fall within a narrow range. Advocates of a slowly increasing life span tend to cite continued gains by white females (Manton 1982), and note increased percentage gains past age 50 in recent years; these observations are fully consistent with those discussed here. Present gains, accruing by postponement of chronic disease, are reflected in life expectancy increases at all ages; hence, the historically more slowly rising life expectancy from advanced ages will show a larger percentage, although smaller absolute, gain. The model presented here predicts that the male-female gap eventually may decrease, but does not anticipate dramatic change to occur in the next few years; such change requires that premature death in males decreases more rapidly than in females, and may occur if the present cardiovascular disease decline broadens, lung cancer rates follow anticipated trends, and traumatic deaths of males in early life decrease. The Hayflick (1980) phenomenon of cellular senescence is sometimes argued to be allegorical or even to represent a laboratory artifact (Manton 1982). This may well be true, and the discussions presented here are not based on the Hayflick phenomenon.

When we ask medical students to draw a set of curves which

represent changes in life expectancy in this century, they usually draw curves closely similar to a set of advancing sigmoid curves, equidistant along their entire length. Such are the curves that would be represented if the life span, as well as the life expectancy, were increasing (figure 6). As noted (figure 4), the actual curves show an increasingly rectangular character (Fries and Crapo 1981). One does not have to be sophisticated in interpretation of mortality curves to have a reasonable feeling for where future progress may be made, and it is curious when demographic projections (Faber 1982; Manton 1982) show characteristics not present in the historical record. A problem with elaborate demographic projections may arise if a faulty model is used, a model which has built into it the shape of the equidistant curves of figure 6. The competing risk model, used without a hazard function representing natural death, leads to such a result. This model assumes that if there were no disease then there would be no death, and, thus, underestimates future mortality at the higher ages by not accounting for the mortal effects of physiologic frailty. A similar error can arise if recent "rates of change," rather than absolute change, are used in projections. Since we have moved from an acute disease era to a chronic disease one, premature death now occurs at higher ages, and percentage gains with further improvement will be accordingly greater at higher ages.

Clinical observation suggests that a significant number of deaths,

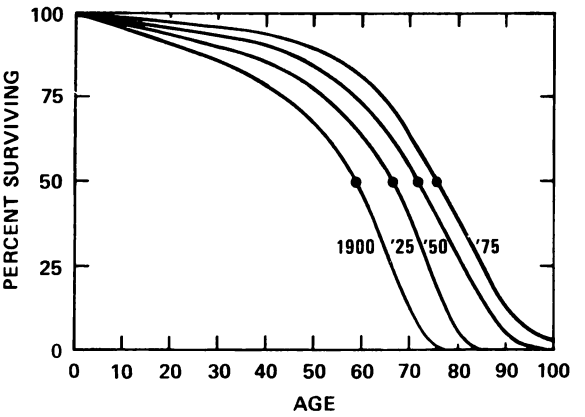

FIG. 6. The curves that are not. These survival curves would suggest an increasing life span, but only the median values and the 1950 curve are the true ones. The actual data are shown in figure 4. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

perhaps as many as one-fourth, presently occur in individuals with minimal organ reserve, and hence are essentially natural deaths, occurring within a few months of ultimate physiologic limits. The elderly individual who gradually begins to "fail," the quiet death at home, or the terminal "multiple catastrophe" hospital course characterized by failure of several organs are examples of nearly natural death; this phenomenon is obscured by our social customs, which prevent tabulation of natural death. "Natural" deaths are hidden in the statistics for bronchopneumonia, heart failure, generalized atherosclerosis, and other categories, since there is no death certificate category for natural death, and everyone must be assigned. When the number of natural deaths is relatively low, projections ignoring these classification errors are reasonably accurate, but as the frequency increases, such models increasingly underestimate future mortality at advanced ages. If there is a major force for mortality, natural death, with a hazard function increasing rapidly from essentially zero at age 70 to nearly 99 percent at age 100, and the demographic model does not include this term (Faber 1982), then the projections will be wrong.

However, quibbling about the precise numbers obscures the much greater area of agreement in all projections. Even the most optimistic calculations (Faber 1982) project that life expectancy at age 85 will increase by only two years by the year 2020. Life expectancy at age 100 is projected to increase from 2.45 years in 1980 to only 3.01 years in 2000, and 3.35 years in 2020. Reduction of premature death, as opposed to change in life span, must account for at least part of such projected change. But such an increase of eleven months over the next forty years, even if due entirely to a change in the genetic life span, does not distort the policy implications of the "compression of morbidity" (Fries 1980).

The Compression of Morbidity

The compression of morbidity occurs if the age at first appearance of aging manifestations and chronic disease symptoms can increase more rapidly than life expectancy. This statement of the thesis recognizes that increases in life expectancy, whether or not associated with minor changes in the life span, are likely over the next 25 years. The question of whether the period of morbidity may be shortened depends upon

whether the average onset age of a marker of morbidity (first heart attack, first dyspnea from emphysema, first disability from osteoarthritis, first memory loss of a certain magnitude) can increase more rapidly than does life expectancy from the same age. If it does, then the period between that marker and the end of life is shortened. Absolute compression of morbidity occurs if age-specific morbidity rates decrease more rapidly than age-specific mortality rates. Relative compression of morbidity occurs if the amount of life after first chronic morbidity decreases as a percentage of life expectancy.

The Characteristics of Chronic Disease

The acute infectious diseases have ceased to be statistically major causes of mortality in the United States. Tuberculosis, small pox, diphtheria, tetanus, polio, typhoid fever, and others have declined by 99 percent to 100 percent in this century (Fries and Ehrlich 1980). In turn, the major medical problems are now well-known to be chronic illnesses: atherosclerosis in all of its guises, cancer in its many forms, emphysema, diabetes, cirrhosis, osteoarthritis. These illnesses are not well conceptualized under the medical model of diseases with single causes and specific cures. These present health problems are characterized by "risk factors" which accelerate their course or which increase the probability of their occurrence. Their "cause" is thus multifactorial, and no single cause is essential. Even more importantly, these illnesses have other characteristics which are not those of the acute diseases. They are, to one degree or another, universal. Every individual has, to a greater or lesser degree, the potential for increasing atherosclerosis, an increasing statistical possibility of malignant change, and slow degeneration of the articular cartilage. Moreover, the chronic illnesses have their onset early in life; signs of such problems may be found in autopsy studies of individuals in their twenties. The severity of the conditions increases progressively with age (figure 7).

Thus, our presently most important illnesses are universal, have early onset, are progressive, are generally characterized by a symptom threshold at which time they become clinically obvious, and are multifactorial in cause (Fries and Crapo 1981). The differences between individuals are manifested not as much by the presence or absence of the condition as by the rate at which the condition progresses. This

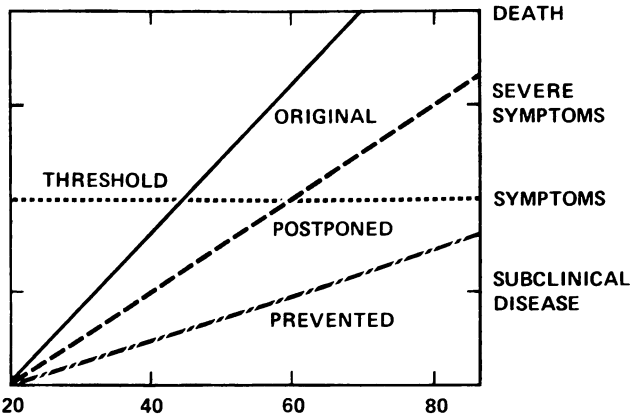

FIG. 7. An incremental model of chronic disease. The model is characterized by early age of onset, progression at various rates, and passage of a symptomatic threshold at which time a clinical diagnosis may be made. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

rate may be very low (as in atherosclerosis in native Japanese on native diets) or higher (as in Japanese on American diets).

As a caveat, there are a number of major chronic diseases, less important statistically, which do not have these characteristics, and which are not the subject of this discussion. Such illnesses include rheumatoid arthritis, Hodgkin's disease, systemic lupus, ulcerative colitis, and multiple sclerosis. They are not universal, not age-related; "risk factors" are few or none; and they have a definite onset point. These illnesses ultimately may fit the traditional medical model quite closely. Alzheimer's disease, a major problem currently increasing in prevalence, is difficult to classify, and clearly deserves both preventive and curative study. This condition is heavily age dependent, and has a specific pathology (Beck et al. 1982; Blass and Weksler 1983).

The multiple risk factor, universal susceptibility model fits our prevalent health problems, with important implications. In this model, as risk factors are modified, the slope of the progression is decreased. As the slope decreases, the date of crossing the symptomatic threshold (figure 7) can be postponed; death due to the disease can be postponed or even prevented; and the severity of symptoms experienced can be decreased. If the slope is sufficiently reduced, the disease may be said to be "prevented," since the symptom threshold may not be passed during life.

A model of universal progressive disease with a symptom threshold allows one to divide life into a "firm" portion, occurring before the threshold is passed, and an "infirm" portion following passage of that threshold. As the slope is decreased, the "firm" period of adult vigor is prolonged and the "infirm" period of disease or senescence is compressed against the natural barrier at the end of life. Both the absolute amount and the percentage of life spent in less than good health may, thus, be decreased.

Another significant attribute of this model is that any reduction in the average slope of lines representing individuals in a population will result in a decrease in age-specific mortality rates. And, it will also result in an increase in the average age at which the first symptom is experienced. Thus, as improvement in the rate of accretion of chronic disease occurs, an effect on morbidity is linked to the effect on mortality. Importantly, this model may be used to describe the senescent changes of aging in multiple organ systems as well as the accelerated decrepitude in a particular organ associated with a chronic disease.

Consider two brothers (figure 8), one of whom smokes three packages of cigarettes daily while the other smokes one-half package a day. The top line represents the life of the heavy-smoking brother. Moving life expectancy toward the right along such a life history provides insights into many of the phenomena of contemporary medicine and its interactions with society. In 1900 perhaps this individual would have encountered pneumonia at age 30 and have died, after a life of 30 years and an illness of three days—premature death, to be sure, but inexpensive (at least in terms of direct medical costs), with relatively little illness burden upon the society, and with a high proportion of vigorous life to sickness. Now, with penicillin, this man survives to begin to develop a cough, wheezing, and shortness of breath at age 40. If he continues to smoke, he will be increasingly short of breath for the remainder of his life. In his fifties he has a heart attack; perhaps, prior to modern management, he might have died at this point. Now his arrythmia is controlled and he goes on to encounter a stroke a few years later, requiring intensive rehabilitation efforts. Throughout, he remains short of breath. Finally, a lung cancer develops and he dies, in a crescendo of chronic disease. Such patients appear to require, not surprisingly, up to four times the medical resources of the average individual. Moreover, the more that the life of such individuals is extended toward the right, the greater the illness burden they represent

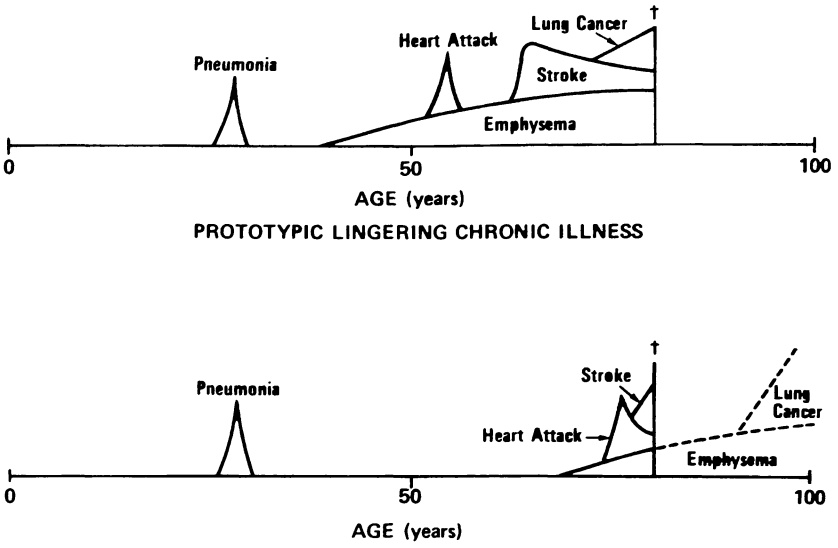

FIG. 8. The compression of morbidity. Two health-lives are diagrammed, the upper with poor health habits and the lower with better health habits. The period of adult vigor prior to infirmity is reduced in the lower example. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

to society and the greater the amount of their life which is spent in less than good health. Such individuals may "linger" for actually half or more of their lifetime, at enormous personal and social cost.

In contrast, the light-smoking brother does not develop symptomatic emphysema until perhaps age 70. The heart attack is postponed a few years, as is the stroke. The lung cancer is postponed all the way out of his lifetime and does not occur. This individual is more vigorous, with a higher quality of life, for a longer period of time, and represents socially a much smaller burden on society. The change in the point of first breathlessness represents, in this commonplace example, as much as thirty years of improved quality of life for the individual without the heavy-smoking habit.

The Plasticity of Many Senescent Phenomena

The same linear senescence observed by Shock in cross-sectional studies of organ function with age is seen with human optimal performance

as, for example, with the world age-group records for men in the marathon (Fries 1980; Fries and Crapo 1981). World-class performance is optimal in the twenties and early thirties and then shows a linear decline (Fries 1980), up to the point where sample size is inadequate for estimation. Similar linear decline is evident in age-group records for other athletic endeavors. It is also present in longitudinal data of the same marathon runner (e.g., Clarence DeMar) over 50 years. However, the decrement associated with age is relatively small; in the marathon it approximates two minutes per year. Variation within individuals of the same age is much larger than this. The individual not performing at personal maximum levels may readily improve marathon times with age. This ability of the individual to swim against the current of senescence holds as a general truth when one considers the modifiability (or lack thereof) of the physical and psychological markers of aging. Training in a particular faculty results in improvement in performance in that faculty, at any age.

Some aspects of the aging process appear to be nonmodifiable. For the most part, these have in common the slow accumulation of fibrous tissue, replacing tissue which previously functioned. Thus, developing rigidity of the arterial wall, cataract formation, the graying of hair, the gradual loss of glomeruli in the kidney, the thinning of hair, and the loss of elasticity of skin. These capacities appear to be insidiously and irreversibly lost with age, according to present data (Fries and Crapo 1981). Given present knowledge, there is little reason to expect that lifestyle or therapeutic interventions will reduce decrements in these areas.

When questioned about fears of growing old, individuals over the age of 50 usually do not cite fear of death (Neugarten 1977; Baltes 1982). As more significant concerns, they first describe a dread of approaching chronic illness, pain, and inability to physically get around. Second, they report fears of approaching senility and loss of memory. And third, they describe a fear of total dependence upon others.

In these areas, there is good reason to expect improvement from preventive and lifestyle approaches. There are convincing studies which indicate that cardiac reserve, dental decay, glucose tolerance, intelligence test performance and memory, osteoporosis, physical endurance, physical strength, pulmonary reserve, reaction time, social ability, and blood pressure, among other variables, are modifiable by the individual at any age. This modifiability is sometimes termed the "plasticity of aging" (Baltes 1982). Modification consists in most instances of training

and practice in the specific faculty (Bortz 1982; Dehn 1972; Farquhar 1978; Labouvie-Vief and Blanchard-Fields 1982; Langer and Rodin 1976; Paffenbarger et al. 1978; Rodin and Langer 1977; Spirduso 1980; Valliant 1979; Baltes and Baltes 1980; Fries and Crapo 1981). In many instances, there is relatively little cross-over from training in one attribute to success at another.

The ability both to postpone chronic disease (Walker 1977) and to utilize the plasticity of aging affords an approach to the major fears of chronic disease, loss of intellect and memory, and total dependence upon others. The compression of morbidity and the plasticity of aging are related concepts, and are applicable both to the problems of chronic disease and to the problems of senescence.

Some emerging data suggest that the compression of morbidity may be a present, as well as a future, phenomenon. Conclusive data are difficult to come by because data on the incidence of markers of morbidity have not been systematically collected, nor do we have, even in cross-sectional studies, prevalence figures for the "quality of life." Indeed, it is not even clear what measures of morbidity should be used. If we accept as such measures the age at first heart attack or the age at development of identifiable lung cancer or the average age at admission to nursing homes, available data suggest that the onset age of these markers may be increasing more rapidly than is life expectancy from age 40 in the United States. For example, the average age at first heart attack for men appears to have increased approximately four years in the past sixteen, while life expectancy from age 40 increased but two years over the past 20. Despite the decreasing prevalence of heart attack over the period of 1968 to 1978, the percentage of hospital discharges for this condition over the age of 65 has risen from 47 to 52 percent of the total (National Center for Health Statistics 1965, 1968, 1974, 1978; Faber 1982; Elvebach, Connolly, and Kurland 1981; Connolly et al. 1981). The ratio of fatal and nonfatal heart attacks appears to have remained constant during this period, suggesting that morbidity and mortality from this cause have both been reduced (Paffenbarger 1979). The stroke data are more equivocal, but appear to show the same thing (Kramer, Diamond, and Lilienfeld 1982; Robins and Baum 1981). Lung cancer age-specific incidence rates appear to have similarly changed and the risk-factor model based heavily on cigarette smoking (pack/years) suggests that as smoking decreases the effect will first be postponement of

onset; that is, the requisite number of pack/years will be reached later in life. Lifetime medical costs of heavy smokers and/or heavy drinkers, inferred from studies of high-cost hospital users (Schroeder, Showstack, and Roberts 1980), appear to be as much as four times those of individuals with moderate habits, even though the duration of life is shorter. The rate of admission to nursing homes has remained essentially constant for those over 65 or over 85 (Neugarten and Havighurst 1977), despite empty beds and an increasing age as the population gradually drifts older.

Human potential may be conceptualized within the following paradigm. There is a level of optimal human performance in each faculty, approximated by world-class performance at each age. Athletic performance is only the most measurable faculty; optimal performance is possible for almost any endeavor, from shopping to playing chess. The decline in optimal performance is linear. The mean performance of a population, on the other hand, has also been measured to show a linear decline, at a level of performance markedly below that of the best human performance (figure 9).

By inference, each individual must have his or her own particular curve of optimum performance. That is, with maximum training and effort, there is a theoretically optimum performance for each individual, and this curve should be expected to have the same characteristics as

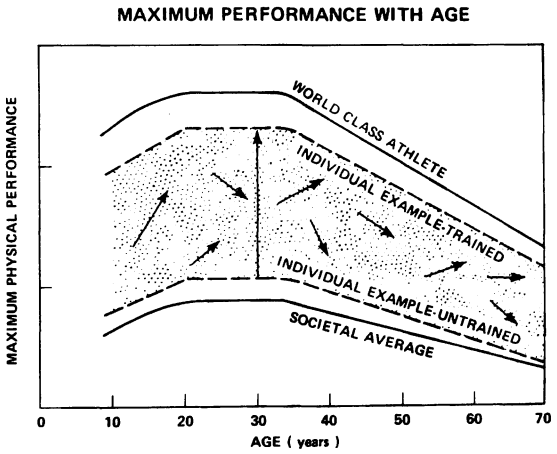

FIG. 9. The plasticity of aging. Within the biological potential of the organism are multiple possible pathways to improvement of performance with age bounded by present performance and maximum potential performance. Reprinted with permission from J.F. Fries and L.M. Crapo, *Vitality and Aging* (San Francisco: W.H. Freeman, 1981).

do the measurable curves. The same individual, untrained and expending less than maximal effort, will show a similar curve but at a lower level of performance. For each individual, the area between the line of present performance and that of potential performance enables the plasticity of aging. With time and age, one may improve, regress, or stay the same within surprisingly broad limits of performance; the limits are evidenced by the wide variation in interindividual performances, and by the increase in such variation with age (Dittman-Kohli and Baltes 1983). If one is not performing near an individually optimal level with regard to a particular faculty, then improvement, despite increasing age, remains possible.

As an additional factor tending to reduce the effects of biological senescence, it should be observed that many psychologists favor a life span concept with regard to cognitive and social functions (Labouvie-Vief and Blanchard-Fields 1982). In these models, a variety of stages occur, one following the other. With regard to certain functions, such as the accumulation of wisdom and judgment, an individual may move from "immature" thought processes through successive stages of "maturity" as he or she ages. Studies showing declines in cognitive function with age are felt by those workers as due largely to the use of youth-oriented measurements of factual cognitive function which do not account for the more complex and less easily measurable manifestations of mature wisdom (Dittman-Kohli and Baltes 1983).

Policy Implications

Human variability, the compression of morbidity, and the recognition of untapped human potential at advanced age contain implications for research and public policies. They suggest need for development of some new research directions. First, a broad biopsychosocial model of health must be utilized, since many of the interventions important in affecting senescent phenomena are certain to be outside of a traditional biological model. Second, there is a lack of good data on the "quality of life," and efforts at systematic accumulation of such data must be accelerated. Initially, there need to be decisions on those specific measures of "quality of life" to be collected and cross-sectional data on these measures needs to be gathered. Third, longitudinal studies need to be initiated at the same time, and these need to include

biologic, psychological, and social variables in the same studies. Fourth, the means for affecting positive change in an aging population are to be found in the variability of the population, as well as in the average values. Studies need to identify and quantitate interindividual variation for specific marker variables at particular ages, identify the factors which predict the variability, and design rigorous prospective intervention trials based upon identification of associated "risk" factors.

On the public policy side, there are several implications. First, there should be no mandatory retirement age. Studies of plasticity suggest strongly the health and vitality benefits of continuing challenge, problem solving, perception of productivity, continued activity, and more money; for some, these features will be best obtained by continued employment. Second, creative vocational opportunities should be available, with multiple and flexible pathways toward optimal use of the later years. Third, health enhancement programs must begin early in adult life and be continued throughout. Aging programs directed only at the aged will have less impact than those addressing the same problems earlier. Fourth, to the extent possible, we should seek deinstitutionalization of long-term care programs, which in their most severe forms prevent individual initiative. Usually this will consist of seeking the most independent living alternative possible for the individual. It will require better development of home health care services, a redevelopment of the role of the family, and a more peripheral distribution of needed care away from large impersonal institutions. Fifth, programs should stimulate the independence of individuals. A false dichotomy is sometimes raised between "caring" and "curing," and there is some risk that our elderly may be smothered by good intentions. The elderly have a right to an independent life and to the execution of personal choices within the broadest possible framework. Finally, solutions are hampered by the existence of certain adverse incentives within our society. We subsidize bad health habits and we encourage disuse of the mental and physical faculties of older individuals. Our health insurance programs take money from those with good health habits and give it to those with poor health habits. We need to look closely at how our laws and our customs may affect the independent expression of vitality in the older individual.

The rectangular morbidity curve represents, in many ways, a social ideal—a long, vigorous life culminating, as in Oliver Wendell Holmes's One-Hoss Shay (1908), with a sudden terminal collapse; vitality until

the end, and death coming without fear or fury at the natural end of the individual life span. It will not happen this way, of course. Utopias may be envisioned, but not totally achieved. Increasing birth cohorts will continue to discharge ever larger numbers of individuals into the older age groups until equilibrium is reached after some 50 years, and the problems we have been experiencing will grow worse before they grow better. Clinical observation of our most vital older citizens suggests a usual terminal decline of months to even a few years, not an abrupt collapse as that of the one-hoss shay. But the compression of morbidity is an achievable phenomenon; it is already occurring in some areas, and it can be made to grow importantly. Projection of health needs under the scenario presented are more favorable than sometimes supposed. But the many problems of our increasingly elderly population continue to exist and require vigorous attack from many directions. The paradigm of increasing vitality and finite life, with the consequent compression of morbidity into a shorter period prior to the end of life, offers a framework within which to view these problems, and within which we may begin to develop some constructive solutions.

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Address correspondence to: James F. Fries, M.D., Associate Professor of Medicine, Department of Medicine—Division of Immunology, Stanford University School of Medicine, Stanford, CA 94305.