Compression of morbidity in the elderly

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Abstract

The Compression of morbidity paradigm envisions reduction in cumulative lifetime morbidity through primary prevention by postponing the age of onset of morbidity to a greater amount than life expectancy is increased, largely by reducing the lifestyle health risks which cause morbidity and disability. Recent data document slowly improving age-specific health status for seniors, indicate that postponement of the onset of disability by at least 10 years is feasible, and prove effectiveness of some lifestyle interventions by randomized controlled trials. Human aging is increasingly represented by frailty, with declining reserve function of many organ systems, including the immune system. Enhancement of immune function in this setting raises medical, ethical, and social issues which are sometimes in conflict. © 2000 Elsevier Science Ltd. All rights reserved.

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The health of persons over 65 years of age is a medical problem in the developed nations. Over 80% of all illness, morbidity, mortality, and medical costs are concentrated in the years after age 65. The illness burden is mostly made up of chronic illness, complicated by the frailty of increasing age. In this century we have seen a transfer of the illness burden from the acute infectious diseases prevalent in 1900 to chronic disease by mid-century, followed by a decline in major chronic illnesses, and now with a slow transition from chronic disease per se to the associated problems of aging [1].

1. An overview of the aging process

1.1. Mortality changes over time

Changes in survival curves in developed nations over this century are instructive, since they lead us beyond a simplistic, if popular, notion of ever-increasing life expectancy. Life expectancy from birth is affected most strongly by changes in infant mortality rates and in deaths in early life. Successive survival curves (Fig. 1) have become more rectangular, as marked reduction in deaths at early ages flatten the initial part of the curve. There are now few deaths prior to age 50 or 60. At the same time, the downslope of the curves has become steeper, although the insertion point has changed little [2]. A natural barrier to biological longevity may be visualized through these successive curves. In the United States, life expectancy from birth has increased from 47 to 75 years. However, life expectancy from advanced ages has changed relatively little. Since 1980, life expectancy for females from age 65 has increased only by 0.5 years. Almost unnoticed has been a striking occurrence; life expectancy for both sexes from age 85, 6.1 years, has not changed significantly during the past 20 years [3]. With the increasing size of successive birth cohorts and with increased survival to age 65 or age 85 the absolute numbers of senior citizens will increase markedly in coming years, while life expectancy for the average senior will increase little.

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1.2. Declines in organ reserve function with age

Data from longitudinal studies of aging show a consistent decline in the maximum function of the various organs with age, the decline being essentially linear at a rate of 1.5% per year after age 30 (Fig. 2). Data on maximal performance, such as world record marathon times, similarly show a nearly linear decline with age at the same rate from age 30 to age 80 [2].

Physiologic normal values, however, as represented by the central tube in Fig. 3, remain constant with increasing age. Normal pH, blood chemistry, white cell count, and other homeostatic values do not vary over the lifespan, representing the internal physiologic environment essential for cellular function. However, with the linear decrease in organ reserve in multiple organs the ability to respond physiologically to a perturbation decreases exponentially, represented by the decreasing area of the polygon in the figure. As a result, mortality rates increase exponentially, with a doubling of mortality rates each 8 years after age 30 [2].

With these realities of human aging come some paradoxes, particularly in the context of emerging scientific advances, some of which are more fully elaborated in this volume. Decline in organ reserve is inevitable, yet we can increase organ reserve quite readily, at almost any age. For example, an increase in exercise can increase cardiopulmonary reserve very substantially, even at advanced ages [4,5].

1.3. Enhanced personal autonomy and modifiable determinism

In philosophic writings, a classical conflict has been between the advocates of free will and those of determinism, and this conflict now exists in modern dress. Determinism is represented by molecular genetics, with the notion that your health over a lifespan is ultimately determined only by your genes. Free will is represented by the advocates of health promotion, seeking voluntary changes in behavioral risk factors, such as lack of exercise, cigarette smoking, obesity, and dietary fat, which can enhance organ reserve, preserve function, and extend life. In this view, health requires that you take care of yourself. The tension between these two paradigms is complicated by the new science, where the role of telomerase, apoptosis, the growth of pluri-potential stem cell lines, and other developments remains to be determined.
2. The Compression of Morbidity

2.1. The hypothesis and the paradigm

The Compression of Morbidity hypothesis was introduced in 1980 [1] and has become the dominant paradigm underlying health improvement programs and policies directed at more successful aging [6,7]. Most morbidity results from chronic processes and is concentrated in the years prior to death. The goal is the compression of morbidity between its time of onset, currently averaging 55 years, and the age of death, currently averaging 75 years, as diagrammed in Fig. 4. A crescendo of increasing morbidity occurs, on average, over this period. With social and scientific progress it is likely that both the age of onset of infirmity and the age at death will increase over time. At issue is the relative movement of the two points. If mortality decreases predominate there may be more morbidity in a typical life, the so-called “failure of success” [8,9], as shown in the second line of the figure. If postponement of the age of onset of morbidity predominates, then morbidity is compressed and the average illness burden is less, the period of adult vigor is prolonged, life quality is improved, and the need for medical care and associated costs may be reduced.

For many, the ideal life is one vigorous and vital over the life span, with a terminal collapse of physical and mental function limited to a few months or years immediately preceding death [2]. A central issue, when considering the eventual impact of new scientific discoveries, is whether they may add health or add illness to the average life. If morbidity is compressed by a new advance then the advantage is clear; if the process of dying is prolonged then major ethical issues arise [10].

2.2. Trends in morbidity compression

Over the past 20 years there has been some compression of morbidity in the elderly, even without health policies directed at morbidity compression [11,12]. The major chronic illnesses, including heart disease, cancer, and stroke, are about 70% preventable. The true causes of death are not the chronic diseases but the underlying causes of these diseases, which are led by cigarette smoking, sedentary living, obesity, and diets high in saturated fat and low in complex carbohydrates [13]. The potential for postponement of morbidity is now much better understood.

Freedman and Martin [14], among others, have shown significant age-specific functional improvement in seniors over a recent seven-year period. Cross-sectional studies have suggested compression of morbidity in favored groups, such as those with higher levels of education [15], higher socio-economic class [16], and those with fewer lifestyle health risks. Daviglus et al. [17] showed substantial decreases in Medicare costs for those with few health risk factors in mid-life. Reed et al. [18] related healthy aging to prospectively determined health risks. Clearly, behavioral health risks makes a very major contribution to both morbidity and mortality. Selected medical advances such as total joint replacement and cataract extraction, or any treatment which makes a major contribution to life quality, also contributes to the compression of morbidity. The central measure of success of the compression of morbidity is reduction in the average cumulative lifetime morbidity [19].

3. How long may the onset of morbidity be postponed?

3.1. Estimating postponement of morbidity

Our research group has been prospectively following substantial numbers of aging seniors in two cohorts longitudinally over the past 16 years to identify the factors which postpone onset of morbidity, the magni-
tude of the postponement, and the effects of lifestyle health risks upon cumulative lifetime disability. We use disability as a definable and measurable surrogate for morbidity and cumulative disability over the elder years as a surrogate for cumulative lifetime disability [5,19].

3.2. The University of Pennsylvania Study

In the University of Pennsylvania Study we follow 1741 university attendees studied in 1939 and 1940, surveyed again in 1962, and followed annually since 1986. Health risk strata were developed for persons at high, moderate, or low risk, based upon cigarette smoking, body mass index, and lack of exercise, and assigned by risk status in 1962 (average age 43 years). Cumulative disability from 1986 to 1994 (average age 75 years) served as the surrogate for lifetime disability. Persons with high health risks in 1962 or in 1986 had twice the cumulative disability of those in the low risk strata. Deceased low risk subjects had only one-half the cumulative lifetime disability of high risk subjects and also had only one-half the amount of disability in the last one or two years of life. The same results obtained in males and in females, and in those without disability in 1962 and 1986. This 100% decrease in disability rates was offset only partially by a 50% decrease in mortality rates in the high risk strata, demonstrating compression of morbidity. Onset of disability (Fig. 5) was postponed by 7.75 years in the low risk stratum as compared with the high risk stratum [19].

3.3. The Precursors of Arthritis Study

In the Precursors of Arthritis Study we have followed 537 senior runners and vigorous exercisers with 423 age-matched community controls. We controlled for self-selection bias by a longitudinal "intention to treat" study since 1984, by separate analyses for gender, by accounting for pre-study dropouts by including all who had ever begun an exercise program at any point in life, by separately analyzing those without initial disability, and by controlling for joint X-ray status, history of arthritis, and other factors [5, 20]. Present data show the exercising group to have less

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**Fig. 5.** Progression of disability over time in low, medium, and high risk groups from the University of Pennsylvania study. The average postponement of disability in the low risk group as compared with the high risk group is 7.75 years.
than one-half the cumulative disability of the sedentary controls and this major difference between groups actually increased over the 13 years of observation. Results held for men and women, those without initial disability, and for all of the other subgroups. The proportion of those disabled was also reduced by more than on-half in the exercise groups. The postponement of disability for the exercising group was 8.7 years for minimum disability and approx. 12 years for higher levels of disability.

4. Reduction in need and demand for medical service

Epidemiologic studies of associations, no matter how strong and consistent, ultimately require randomized study of interventions for proof of causality, and these are now available. Health promotion programs in senior populations directed at risk prevention, health improvement, and medical cost reduction have been studied in a number of large and well performed randomized trials, with positive results. The Bank of America randomized study of 4500 retired subjects reduced risks by 12% in the intervention groups in the first year compared with controls and reduced medical care and costs, as confirmed by a study of medical claims [22], by even a greater percentage. The California Public Employment Retirement System study [23], involved 57,000 subjects in a 1-year randomized trial with similarly dramatic results confirmed by claims data endpoints. Randomized studies of self-management programs for chronic diseases such as arthritis [24] and Parkinson’s Disease [25] have had similarly positive results. Carefully designed high quality health improvement programs have now been proven effective and cost-effective both preventing illness and reducing medical need [26].

5. Issues for contemplation

The human aging process, when not prematurely stopped by trauma or disease, moves towards multiple organ system frailty [6,7,9]. The immediate cause of death shifts from external towards intrinsic factors. The formally assigned “cause of death” becomes increasingly irrelevant compared with the underlying frailty, the inability of the aging organism to withstand even a minor perturbation. Frailty is like an old curtain rotted by the sun, where an attempt to repair a tear in one place is followed by a tear in another. In a similar context, William Osler considered pneumonia to be the “old mans’ friend”, terminating the infirmities of frailty. Influenza epidemics result in an increase in death rates, but they are followed by a 6-month period of death rates which are actually below expected baselines. Pneumovax and influenza vaccines can prevent a number of specific perturbations and associated attributable deaths, but only in the context of multi-organ frailty. The ultimate health benefits of successful interventions in the terminally frail may prove in substantial part illusory when so many competing risks remain.

Age-associated declines in the immune and inflammatory responses have been well defined. Similar declines occur in other organ systems. What would be the full range of effects if declines in the immune and inflammatory systems were prevented or reversed in the setting of declines in other organ systems with multi-organ failure and increased frailty? Is there an important signal in the apparently accelerated aging of Dolly, the telomerase-impaired cloned sheep? The central question: would enhanced immune responsiveness increase health or increase morbidity? Answers to such questions are clearly speculative, but it seems that one answer might carry a bit of a paradox with it; that improved immune responsiveness might be constructive in terms of decreasing morbidity if achieved prior to terminal multiple organ frailty, but might increase lifetime morbidity, albeit only slightly, if achieved only shortly before the time of natural death.

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References


