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"Epidemiologic Observations on the Compression of Morbidity: Evidence From the Alameda County Study"

by George A. Kaplan, PhD

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2455 Teller Road
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Epidemiologic Observations on the Compression of Morbidity

Evidence From the Alameda County Study

GEORGE A. KAPLAN, PhD
California Department of Health Services

Data on the health experience of two cohorts of Alameda County, California adults selected in 1965 and 1974 are used to examine evidence for a compression of morbidity. These data, based on two representative cohorts that have been prospectively followed, indicate major declines in age-specific mortality rates between the two cohorts. Accompanying these declines in risk of death is an increase in the age-specific prevalence of a number of chronic conditions and symptoms and a decrease in the association between these conditions and symptoms and risk of death. This increase in morbidity is accompanied, in turn, by an increase in the amount of disability associated with specific chronic conditions and symptoms. The overall picture is one of increased survival accompanied by increased morbidity and disability. These data do not provide evidence in support of a compression of morbidity. A further consideration of the methodologic and conceptual problems that are raised by the compression of morbidity hypothesis suggests that it is not a testable hypothesis and that further efforts should concentrate, instead, on primary and secondary prevention efforts to lessen morbidity burdens and improve quality of life for older persons.

The compression of morbidity hypothesis (Fries, 1980) is an appealing scenario for the future. It is so appealing that it is hard to take issue with. Although it is possible to disagree about the exact limit of the

AUTHOR'S NOTE: Reprint requests should be addressed to George A. Kaplan, PhD, Chief, Human Population Laboratory, California Department of Health Services, 2151 Berkeley Way, Annex 2, Suite 300, Berkeley, CA 94704-9980.

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human life span, there surely must be one. Therefore, at some point in time, which is widely disputed, the compression of morbidity scenario will become possible. The debate over if and when that occurs has been characterized as generating a lot of "heat and little light" (Fries, 1987). Hopefully, the continued discussion of this topic will generate more of the latter and less of the former.

This discussion will focus on two areas. First, data will be presented from studies conducted over the last 25 years in Alameda County, California, that bear on the compression of morbidity debate. Although these data provide little support for a trend toward compression of morbidity in the recent past, they do not, of course, indicate that it will not occur in the future.

Secondly, the discussion will be broadened to a more general treatment of the type of epidemiologic information that is necessary to comprehensively address this compelling hypothesis. Based on this perspective, previous debates over the compression of morbidity appear to have been characterized by a lack of biologic specificity and detail, a lack of appreciation for the complexity of human health, its manifestations and its determinants, and a lack of data bases adequate to test competing hypotheses. In such a situation, much heat and little light is to be expected.

The Human Population Laboratory Studies

In 1965 and 1974, independent cohorts representative of the Alameda County, California population (n = 6,928 and 3,119, respectively) were selected based on strict probability samples of Alameda County housing units (Berkman & Breslow, 1983; Hochstim, 1970). All noninstitutionalized adults were eligible, and the oldest persons were 94 and 98 years old in the 1965 and 1974 cohorts, respectively. The response rates were high, 86% and 80%, and the loss to follow-up over the following years has been on the order of 0.5% per year.

In analyses of the health experience of the 1965 cohort, a variety of behavioral, social, psychological, demographic, and socioenvironmental risk factors were strongly associated with survival, even when the analyses were restricted to those who were 70 to 94 years at baseline and who were followed for 17 years (Kaplan, Seeman, Cohen, Knudsen, & Guralnik, 1987). For example, current smokers who were 70 to 94 years old had 1.43 (95% confidence interval = 1.08-1.89) times the mortality risk of those who had never smoked over the 17 year follow-up. Smoking cessation was also, prospectively, associated with declines in risk (Kaplan & Haan, 1989).

Examination of the mortality experience of the 1965 and 1974 cohorts indicates that there were major declines in age-specific mortality in the 9-year period between these two cohorts (Figure 1). In every age group, the age-specific 9-year risk of death was lower in the later cohort. The differences are striking. The 9-year risks for men were 30% to 45% lower in the 1974 cohort, with the declines increasing with increasing age. For women, those under 80 years of age showed a 22% to 40% decline, and for those 80 to 89, there was a 12% decline. Thus, in a relatively short time period, there were substantial changes in age-specific mortality.

In addition to differences in mortality rates, these two cohorts differ in many other ways (Kaplan, Cohn, Cohen, & Guralnik, 1988). For example, the 1974 cohort had higher rates of leisure time physical activity up to age 80. Using an index of leisure time physical activity that sums the frequency of activity in a variety of domains (swimming, walking, physical exercising, active sports, and so on) and that is related to mortality risk (Kaplan et al., 1987), 50 to 79 year olds had 6% to 12% higher activity levels in 1974 than in 1965, whereas those 80 or more years old had identical levels.

More positive risk factor profiles were also seen when other risk factors were considered. For example, there were higher levels of social contacts in the 1974 cohort, and, for men, there were lower levels of smoking. Although it would be tempting to assume that more favorable risk factor levels in the later cohort must be accompanied by more favorable health status, the situation appears to be just the opposite. Figure 3 presents the age and sex-specific prevalence of two self-reported measures of physical functioning, adjusted for age within categories and sex. For limitations in both self-care (trouble feeding, dressing, or moving around for at least 6 months) and mobility (trouble climbing stairs or getting outdoors for at least 6 months), the rates are higher in the later cohort for every sex and age group. For those over 80 years of age, the rates of self-care limitations are more than twice

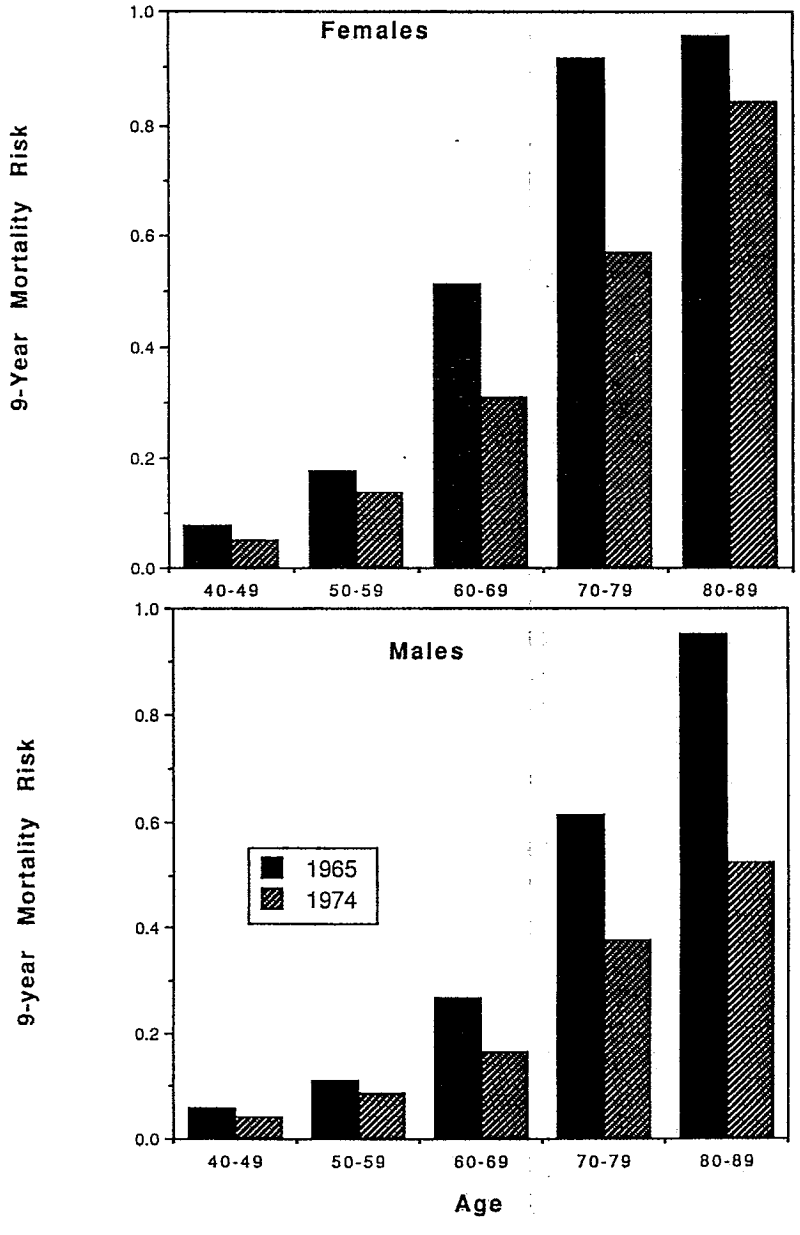

Figure 1. Nine-year mortality risk, by age, Alameda County, 1965 and 1974.

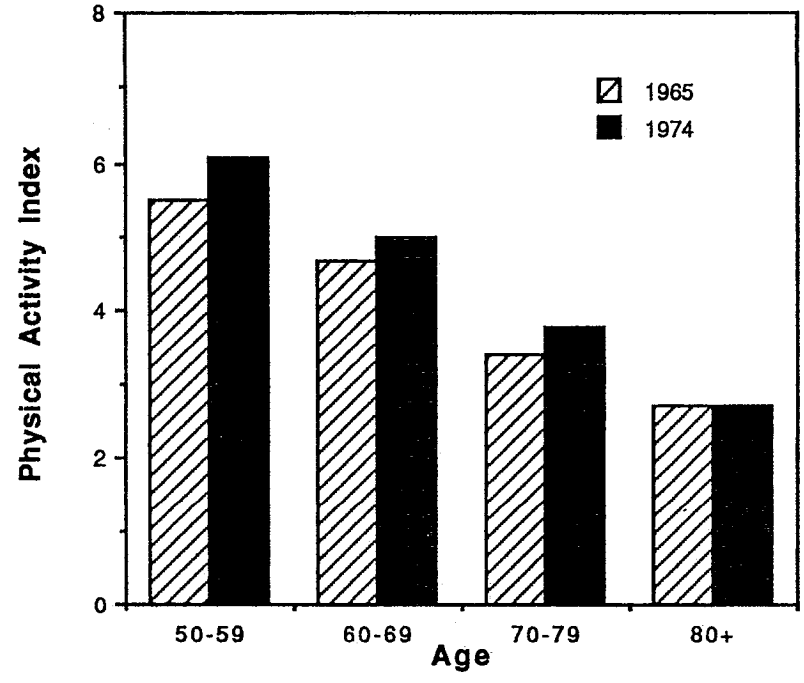

Figure 2. **Physical activity in two cohorts, Alameda County Study, 1965 and 1974.**

as high in the later cohort, and mobility limitations are almost 40% higher.

Thus, for these two cohorts, separated by 9 years, there is a substantial decline in the age-specific, 9-year mortality risk, a more positive profile for some risk factors, but substantial increases in the age-specific prevalence of two types of functional limitations. To borrow a phrase, older persons in Alameda County are living longer but doing worse.

To investigate this in more detail, the prevalence of specific chronic conditions, the mortality associated with these conditions, and the disability associated with these conditions were examined. As has been emphasized by a number of authors, it is important to study these linkages between morbidity, mortality, and disability.

Table 1 presents the prevalence of fifteen chronic conditions and symptoms for males and females in the 1965 and 1974 cohorts who

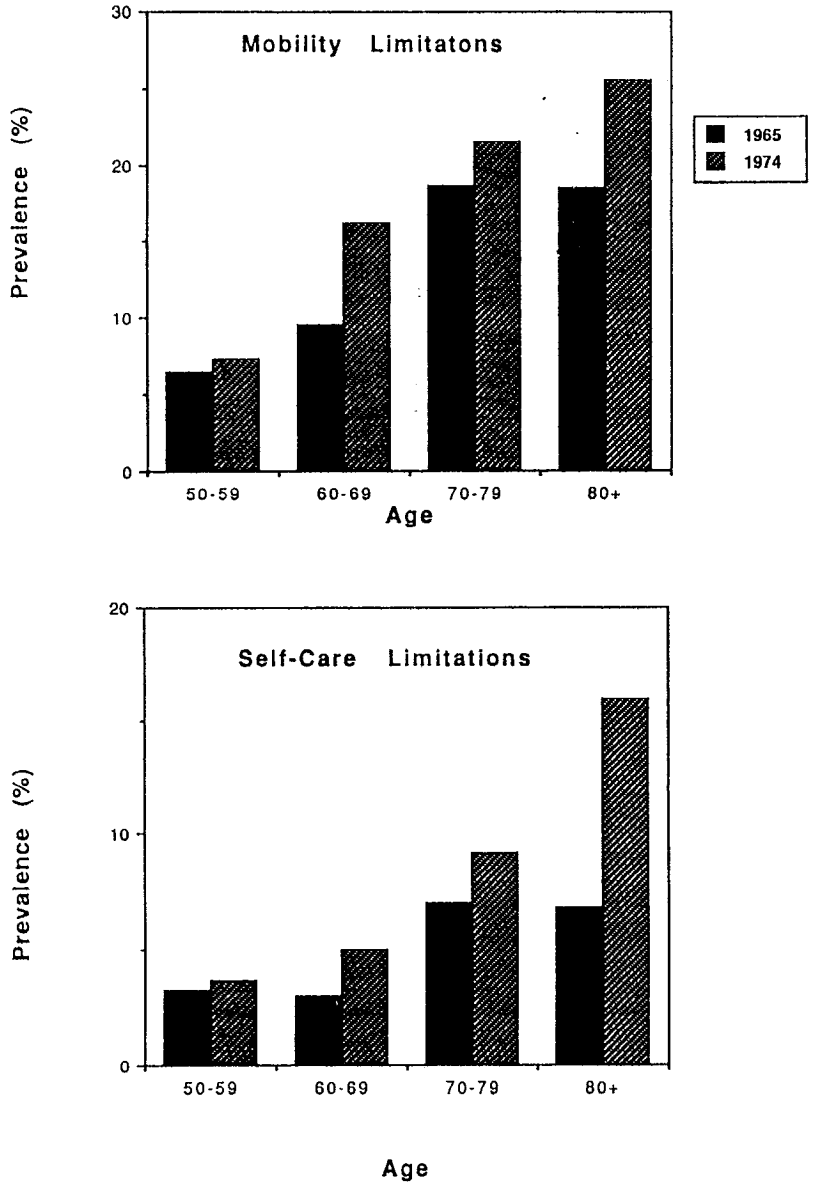

Figure 3. **Prevalence (%) of limitations, Alameda County, 1965 and 1974.**

Table 1
Age-Adjusted Prevalence of Chronic Conditions and Symptoms in 1965 and 1974 Cohorts, by Age and Sex, Alameda County Study (%)

	Males				Females			
	50-59	60-69	70-79	80+	50-59	60-69	70-79	80+
Stroke								
1965	0.60	2.20	3.10	5.30	0.20	2.60	3.90	0.00
1974	4.60	4.20	6.10	7.70	0.80	2.30	5.40	15.6
Heart trouble								
1965	5.00	12.0	17.0	13.6	3.50	12.8	18.2	17.7
1974	17.6	15.3	9.10	15.4	6.80	27.0	21.4	28.1
Arthritis								
1965	19.8	27.6	31.0	43.1	26.5	45.9	48.3	44.7
1974	21.3	33.3	42.4	38.5	32.3	47.2	58.9	68.8
Diabetes								
1965	3.60	5.50	7.50	5.10	3.70	5.70	3.90	4.70
1974	3.70	8.30	9.10	15.4	9.90	11.5	12.5	3.10
Chest pain								
1965	11.0	13.5	15.1	7.00	11.6	13.1	16.2	11.9
1974	19.6	15.3	9.10	0.00	12.9	21.6	25.0	15.6
Shortness of breath								
1965	12.1	18.9	16.9	10.3	15.8	14.1	20.3	20.0
1974	16.7	19.4	21.9	23.1	15.2	23.0	30.4	31.3
Back pain								
1965	23.1	23.5	22.8	25.0	26.7	33.0	32.6	29.4
1974	25.9	30.6	21.2	30.8	30.8	34.1	41.1	40.6
Stiff joints								
1965	21.6	20.6	20.4	31.0	29.3	34.0	37.1	44.1
1974	22.4	22.2	18.8	30.8	31.3	36.8	41.1	53.1
Asthma								
1965	3.80	3.10	3.10	8.60	2.10	3.40	3.50	1.30
1974	3.70	5.60	6.10	0.00	5.30	5.70	8.90	3.60
High blood pressure								
1965	12.8	13.9	17.7	25.4	16.2	32.6	35.5	25.6
1974	13.9	22.2	27.3	27.3	24.8	34.5	33.9	44.4
Bronchitis								
1965	3.60	5.20	4.40	6.80	4.40	4.40	4.30	3.80
1974	1.90	12.5	3.00	0.00	5.30	8.10	10.7	3.60
Ulcer								
1965	6.80	8.30	4.40	1.70	4.40	5.20	2.70	8.90
1974	8.30	12.7	15.2	9.10	6.80	8.00	10.7	3.60
Leg cramps								
1965	16.9	21.9	27.0	32.2	24.5	30.0	31.8	36.7
1974	17.6	13.9	24.2	18.2	26.7	28.1	28.6	32.1
Tire easily								
1965	13.4	21.1	30.8	36.8	21.0	24.9	33.2	41.8
1974	15.0	20.8	37.5	45.5	22.9	30.7	41.1	42.9
Sleep trouble								
1965	6.40	8.10	8.20	20.3	11.4	16.0	17.8	18.0
1974	8.30	15.3	6.50	18.2	19.6	20.5	26.8	21.4

were 50+ years old. The prevalence rates are age-adjusted within the four age groups. Overall, there is a marked tendency for the prevalence to be higher in the later cohort. When all age-sex condition/symptom strata are considered, the rates are higher in 78% of the cases. Although there is some variation, for 14 of the conditions, the prevalence rate is higher for the 1974 cohort for 75% or more of the comparisons. The only exception is the generally higher prevalence of leg cramps in the 1965 cohort.

For each condition or symptom the 1965 prevalence was compared with the 1974 prevalence across the eight age-sex specific groups. The Wilcoxon Signed Ranks test (Siegal, 1956) was used to test the hypothesis that no difference in rates existed between the two time periods versus the one-sided alternative that the rates were higher in 1974. For 11 of the 15 conditions or symptoms, the 1974 rates were significantly higher with p values ranging from $p = .008$ to $p = .04$ (average $p = .02$). For three conditions, asthma, chest pain, and bronchitis, the rates were not significantly higher in 1974, although they were generally higher in 1974 for the first two. The prevalence of leg cramps was higher in the 1965 cohort ($p = .03$).

The interpretation of increased rates of chronic disease and symptoms in the later cohort is not simple. Increased age-specific prevalence could be interpreted in a number of ways. Increases in prevalence are consistent with increased incidence, decreased case fatality rates, or improvements in the detection of the conditions resulting in a greater number of milder cases being diagnosed.

To analyze some aspects of this, the age- and sex-adjusted association between seven chronic conditions and symptoms and 9-year risk of death from all causes was examined in the two cohorts (Table 2). For six of seven conditions or symptoms there was a weaker association with risk of death in the later cohort. The association was 17% to 55% weaker in the later cohort, providing some evidence for earlier detection and better treatment of these conditions. For only one condition, high blood pressure, was the association higher, but the odds ratio only increased from 1.7 to 1.8.

The picture thus far is somewhat encouraging. Increased prevalence of chronic conditions in the later cohort may indicate that persons with these conditions who might have previously died are

Table 2
Age and Sex-Adjusted 9-Year Risk of Death for Those 50+ Years in 1965 and 1974 Cohorts, Alameda County Study

	1965 cohort		1974 cohort	
	Odds ratio	95% confidence interval	Odds ratio	95% confidence interval
Stroke	7.3	3.3-16.1	3.9	2.0-7.7
Heart trouble	2.3	1.7-3.20	1.9	1.3-2.8
Arthritis	1.1	0.9-1.30	1.2	0.8-1.6
Diabetes	1.9	1.2-3.00	1.8	1.1-3.2
Chest pain	1.9	1.4-2.50	1.5	1.0-2.3
Shortness of breath	2.3	1.8-3.00	1.8	1.2-2.6
Back pain	1.1	0.9-1.40	1.1	0.8-1.6
Stiff joints	1.1	0.9-1.40	1.6	1.1-2.2
Asthma	1.7	1.0-2.90	1.3	0.5-3.2
High blood pressure	1.7	1.3-2.20	1.8	1.1-2.9
Bronchitis	1.5	0.9-2.40	2.3	1.1-5.1
Ulcer	1.2	0.7-1.80	1.4	0.7-2.9
Leg cramps	1.1	0.8-1.40	1.2	0.7-2.0
Tire easily	1.9	1.5-2.40	2.1	1.3-3.4
Sleep trouble	1.6	1.2-2.20	1.1	0.7-2.0

being kept alive or that there is earlier detection of health problems. The decreased association between these conditions and risk of death in the later cohort is consistent with both earlier detection and better treatment. However, the public health impact of this possible earlier detection and treatment needs to be measured against the impact of these diseases on the quality of life of those who survive with them. To examine this issue in the Alameda County Study cohorts, the association between the presence of 15 chronic conditions and symptoms and the presence of two functional limitations was studied (Table 3).

In these analyses, the age- and sex-adjusted association between the presence of the 15 conditions and symptoms and prevalent functional limitations was separately assessed for the 1965 and 1974 cohorts. The first functional limitation assessed was a report of greater than 6 months trouble feeding, dressing, or moving around. Not surprisingly, there were strong and significant associations between prevalent health problems and these limitations in both cohorts. For 10 of 15 health problems, there were stronger associations with

Table 3
Odds Ratios for Disability, by Presence of Chronic Conditions Among Age 50+, Adjusted for Age and Sex, 1965 and 1974 Cohorts

Condition	Trouble dressing, feeding				Trouble moving around, climbing stairs			
	1965 cohort		1974 cohort		1965 cohort		1974 cohort	
	Odds ratio	95% confidence interval	Odds ratio	95% confidence interval	Odds ratio	95% confidence interval	Odds ratio	95% confidence interval
Stroke	6.10	2.80- 13.40	7.20	2.70- 19.10	7.00	3.60- 13.60	9.50	3.60- 25.10
Heart trouble	2.50	1.50- 4.10	1.40	0.70- 3.00	6.50	4.70- 9.00	7.30	4.30- 12.40
Arthritis	3.80	2.40- 5.80	8.70	3.80- 20.30	3.00	2.30- 4.00	3.50	2.10- 5.60
Diabetes	1.80	0.90- 3.90	5.10	2.40- 11.10	2.70	1.70- 4.40	6.10	3.20- 11.60
Chest pain	2.00	1.20- 3.20	2.20	1.10- 4.60	6.10	4.50- 8.40	6.20	3.70- 10.00
Shortness of breath	3.90	2.50- 5.90	3.70	1.90- 7.10	7.40	5.50- 9.90	9.50	5.70- 15.70
Back pain	3.20	2.10- 4.90	3.70	1.90- 7.00	3.00	2.30- 3.90	3.20	2.00- 5.00
Stiff joints	4.20	2.80- 6.50	14.90	6.30- 34.90	4.00	3.00- 5.30	5.00	3.10- 8.10
Asthma	2.34	1.03- 5.29	5.70	2.26- 14.30	3.75	2.18- 6.42	5.89	2.58- 13.47
High blood pressure	2.28	1.48- 3.49	2.40	1.26- 4.56	2.31	1.74- 3.08	3.29	2.05- 5.27
Bronchitis	2.60	1.30- 5.20	2.23	0.80- 6.22	3.21	1.98- 5.19	4.47	2.12- 9.46
Ulcer	1.43	0.64- 3.16	1.38	0.51- 3.77	1.44	0.84- 2.49	2.05	1.03- 4.08
Leg cramps	3.58	2.36- 5.42	5.87	3.04- 11.33	2.59	1.96- 3.42	2.96	1.83- 4.79
Tire easily	9.34	5.85- 14.93	10.03	4.74- 21.17	9.26	6.87- 12.47	16.20	9.46- 27.75
Sleep trouble	5.04	3.26- 7.80	4.13	2.10- 8.12	3.83	2.79 -5.26	6.40	3.79 -10.80

disability in the later cohort. The picture is even more striking when trouble of more than 6 months duration in either climbing stairs or getting outdoors was considered. For every one of the 15 conditions and symptoms there was a stronger association with functional limitations in the 1974 cohort than in the 1965 cohort.

Probably, more than one conclusion can be drawn from these data, but they do not suggest a pattern that is consistent with a compression of morbidity occurring during the last 2 decades. Indeed, the pattern of increases in the prevalence of conditions and symptoms and in the disability associated with them, coupled with decreased mortality associated with these conditions suggests that there has been an increase in the ability to detect diseases and their manifestations and to keep people with these diseases alive. However, there seems to be increased disability associated with this increased survival. Because earlier detection of disease and better treatment are both desirable goals, further attention will need to be focused on the reasons for greater disability in those persons who have survived and who are being treated for the conditions and symptoms examined in these analyses.

Perspectives on the Compression of Morbidity

Although it is important to continue to gather data that bear on the compression of morbidity hypothesis, there are a series of epidemiologic, biologic, and aging issues that will be important to address before coming to any firm conclusion about the current or future state of a compression of morbidity.

First, it is useful to review the compression of morbidity hypothesis. Fries (1987) postulated that a compression of morbidity will occur if there is (a) a limit to the life span, and (b) if the average time of onset of the first irreversible chronic disease, or a marker of that disease, occurs later and later in successive birth cohorts, and if (c) these delays in the onset of disease "catch up" with the limits to the adult life span. Thus, due to the later appearance of disease, accompanied by a fixed life span, a smaller portion of adult life will be experienced in a morbid state.

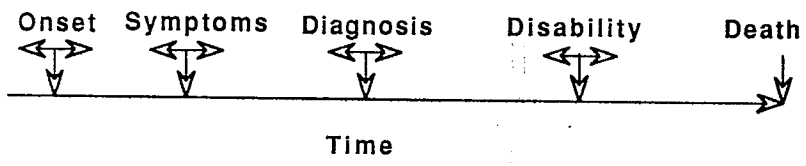

Figure 4. **Transition points in the natural history of disease that are relevant to the compression of morbidity.**

One cannot argue with the internal logic, or desirability, of such a formulation, but from an epidemiologic point of view, the simplicity of the formulation masks the complexity of the processes being discussed. Consider Figure 4. The figure schematically represents five points in time, all of which are relevant to the concepts involved in the compression of morbidity discussion. Each of these transition points can vary in time, and these variations will be critical in determining whether or not a compression of morbidity is experienced. A number of important issues are raised by a consideration of these transition points in the natural history of disease.

Can we identify the onset of disease? For the most part when the chronic, degenerative diseases of older age are being considered, the answer is most certainly no. These diseases are characterized by long acting pathophysiological processes and adaptations. Consider, for example, atherosclerotic heart disease. Is the onset of disease the first signs of wall thickening in the coronary arteries or the first appearance of significant calcification or the first significant stenosis? Given the indeterminancy of the onset of chronic disease, is it wise to frame an hypothesis in terms of changes in the time of onset of disease?

How are we to interpret changes in the time of onset of "markers" of disease? Two types of markers must be distinguished—those related to symptoms and those related to clinical or physiologic findings. If the appearance of symptoms is taken as a "marker" of disease, then there are significant barriers to the interpretation of changes in the time of onset of symptoms. For many chronic diseases, symptoms develop insidiously, and come and go. This intermittent and remittent pattern

coupled with the gradual appearance of many symptoms makes timing of onset problematic. But even more important, there are wide variations in the presentation of symptoms. These variations are related to age, sex, race, socioeconomic status, psychological state, and other factors. Further complications arise when we consider that the interpretation of symptoms and their linkage with medical care use varies widely according to the same factors. Presentation of symptoms for medical care may also vary with time period, as our public health efforts bring one or another set of symptoms to the public's attention. Thus, if determined by the time of presentation of symptoms for medical care, time of onset will be influenced by many factors unrelated to underlying pathophysiologic state.

There are also major problems when considering the onset of clinical or physiologic findings when using data that has been gathered from patient populations. All the factors just mentioned will bear on the point in time at which the patient appears in the medical setting. In such situations, dating the onset of the marker of disease by the time at which the patient appears to be examined will potentially result in flawed findings. Of course, such problems will not occur in studies based on periodic screenings or examinations.

There is an even more serious problem with the use of such markers. There are major secular drifts in the use of particular tests and the procedures used to observe such markers. Development of new technologies, changes in reimbursement practices, and, optimally, the results of outcome studies, all will have an impact on the ability to observe certain markers. Many of the new technologies will have a major impact on the ability to identify markers of disease earlier and earlier. For example, the development of noninvasive imaging technologies has led to a dramatic acceleration in the ability to detect underlying disease earlier. Thus, across cohorts, there will be a substantial noncomparability in the ability to detect the appearance of markers of the onset of disease. A further complication is that the use of new technologies is not randomly distributed; access to them will be available to some strata in society before others. Given all these difficulties in interpretation, it is not at all clear that any definitive answers can come from questions about the onset of either symptoms or markers of disease.

What is the role of disability in the compression of morbidity discussion? Although the compression of morbidity hypothesis is primarily concerned with changes in the period from onset of disease to death, and the relationship of that period to the life span, the "one hoss shay" metaphor suggests that the intention is to prevent or delay disability. However, a number of interpretive problems arise when considering disability. The presumption is that most problems in functioning are consequences of disease processes. As can be seen in Table 3, this is a reasonable position. However, as Table 3 also indicates, the relationship between chronic conditions and disability is not fixed. In addition, this relationship can be influenced by the physical environment, social resources, economic resources, psychological state, comorbidities, medications, and so forth. Table 4, again using the Alameda County Study, shows the association between smoking status, presence of depression, social isolation, and socioeconomic position and the risk of incident problems in climbing stairs or getting outdoors in those with incident heart trouble, stroke, diabetes, or arthritis. There is substantial variation in the risk of being disabled depending on income, smoking status, social isolation, and level of depression. Thus the relationship between disease and disability is modulated by a number of factors.

In short, levels of functioning post-onset of disease are variable and determined by a multiplicity of factors. Because of this, it cannot automatically be assumed that delayed onset of disease is invariably associated with lower levels of disability.

Where does aging fit in to the compression of morbidity hypothesis? Age-related physiological changes may have an impact on the relationship between changes in the age of onset of diseases and their consequences. A relatively clear case of this would be found for infectious diseases. Declines in immune function with age suggest that delayed onset of an infectious disease may result in increased morbidity and mortality associated with that disease. Age-related changes in arterial stiffness, pulmonary function, or renal function may accelerate the impact of other disease processes. Thus assessment of the impact on morbidity and mortality of a delay in time of onset of a disease must take into consideration age-related changes in organ systems that may

Table 4
Predictors of 1965 to 1974 Incidence of Two Types of Functional Limitations Among Persons With 1965 to 1974 Incident Heart Trouble, Stroke, Diabetes, or Arthritis

	Trouble feeding, dressing, or moving around		Trouble climbing stairs, getting outdoors	
Predictors	Odds ratio	95% confidence interval	Odds ratio	95% confidence interval
1. Age	1.05	1.01-1.09	1.04	1.01-1.60
Sex (male)	0.61	0.28-1.30	0.42	0.25-0.72
Current smoker	2.31	1.06-5.03	2.02	1.21-3.38
2. Age	1.04	1.00-1.07	1.03	1.00-1.05
Sex (male)	0.70	0.33-1.46	0.49	0.29-0.82
Depressed	1.54	0.61-3.90	1.76	0.95-3.27
3. Age	1.04	1.00-1.07	1.03	1.00-1.05
Sex (male)	0.69	0.33-1.47	0.48	0.29-0.81
Socially isolated	1.02	0.30-3.48	1.95	0.97-3.91
4. Age	1.04	1.00-1.07	1.02	0.99-1.04
Sex (male)	0.71	0.33-1.52	0.54	0.32-0.92
Inadequate income	0.93	0.17-5.12	3.54	1.50-8.36
Marginal income	2.93	0.90-9.51	2.50	1.08-5.75
Adequate income (reference = very adequate)	2.51	0.90-6.96	2.45	1.24-4.85

potentiate the effects of that disease when it occurs later. Whether these age-related changes would, for diseases with associated mortality, decrease the time to death and thus lead to a compression of morbidity, or whether they would simply increase the average level of severity of disease over the same time period with no effect on survival, is not clear.

Similarly, cohort specific differences in factors, such as nutrition or childhood experiences, that might influence the rate of progression of age-related changes in functioning must be taken into account.

Summary

In summary, it is an interesting and, perhaps, valuable exercise to conduct analyses such as these. However, considerably more detailed information is needed. Although studies of elderly cohorts, such as those underway at Kaiser-Permanente in California or the Mayo Clinic

in Minnesota represent a good start in that direction, there do not appear to be any databases available that would allow us to truly grapple with the claims and predictions of the compression of morbidity model.

Unfortunately, from an epidemiologic perspective, the specification of the compression of morbidity model contains a number of unmeasurable or problematic concepts, and an incomplete identification of the complex paths that link pathophysiological processes, morbidity, disability, and death. In the face of such problems, and a major lack of data of the appropriate complexity, should the debate continue?

Although the wish to prolong life and reduce disability and suffering to a minimum is shared by all, projections for the future based on inadequate data of the past should not be the basis of policy. On the other hand, there is a growing body of epidemiologic evidence that suggests that primary and secondary prevention efforts may be effective among the elderly (Benfante & Reed, 1990; Kaplan & Haan, 1989; Stamler, 1988). Decreases in the burden of morbidity and improvements in quality of life that arise from such efforts, independent of life span considerations, represent a desirable and achievable goal, independent of the compression of morbidity controversy.

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