

How Change in Age-specific Mortality Affects Life Expectancy*

J. W. VAUPEL†

INTRODUCTION

Suppose the goal is to increase the life expectancy of a population or, equivalently, to save as many years of life as possible. If 100 deaths could be averted during any decade of life – say 0 to 10 or 42 to 52 – which decade would be best? The answer is simple – the first decade of life, because children lose most years of life expectancy. Suppose, however, deaths could be reduced by one per cent during any decade of life. Which decade would then be best?

It may seem reasonable, at first thought, to guess 0 to 10, or 17 to 27, or some other decade at young ages. From the life table for Swedish men for 1982, however, the correct answer is 67 to 77. And from the life table for Swedish women for 1982 the answer is 74 to 84.

In the Swedish life table for females, 653 infants die before their first birthday, each losing about 79.2 years of life expectancy, or about 52,000 years in total. But only 189 additional girls die between their first and tenth birthdays; the total loss of life expectancy between birth and age 10 is about 66,000 years. Compared with these 842 deaths, nearly 32,000 women die between the ages of 74 and 84. They lose 8.3 years of life expectancy each, or about 260,000 years in total. Thus four times as many years of expected life are lost between ages 74 and 84 as in the first ten years of life.

This does not imply that life-saving efforts should be focused on the elderly. For various reasons discussed at the end of this article, including considerations of quality of life, priority might be given to averting early deaths. Nonetheless, the potential for saving years of life at different ages is worth examining, if only to gain a deeper demographic understanding of the linkage between age-specific mortality and life expectancy.

HOW REDUCTIONS IN MORTALITY INCREASE LIFE EXPECTANCY

Let $\mu(a, t)$ be the force of mortality and $p(a, t)$ be the period survivorship at age a and time t , and let $e(a, t)$ represent period life expectancy. How does change in the function of μ affect e ? Demographers have used two basic approaches in answering this question. The first, exemplified by Pollard¹ and in a United Nations study,² is focused on how the difference between two alternative trajectories μ_1 and μ_2 , say – translates into the difference

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† Research Scholar, Population Programme, International Institute for Applied Systems Analysis, Laxenburg, Austria, and Professor of Public Affairs and Planning, University of Minnesota, USA.

¹ J. H. Pollard, 'The expectation of life and its relationship to mortality', *Journal of the Institute of Actuaries* 109, 2 (1982), pp. 225–240.

² United Nations, *Levels and Trends of Mortality Since 1950* (New York: United Nations, 1982).

between the resulting life expectancies, e_1 and e_2 . The second approach, pioneered by Keyfitz³ and extended here, is focused on how the rate or intensity of progress in μ , given by

$$\rho(a, t) = \frac{\partial \mu(a, t) / \partial t}{\mu(a, t)}, \quad (1)$$

translates into the rate of progress in life expectancy, given by

$$\pi(t) = \frac{\partial e(0, t) / \partial t}{e(0, t)}. \quad (2)$$

As Keyfitz has shown,³ if equal progress is achieved against mortality at all ages at time t , then

$$\pi(t) = \rho(t) H(t), \quad (3)$$

where

$$H(t) = \frac{\int_0^\omega p(a, t) \cdot \int_0^a \mu(x, t) dx da - \int_0^\omega p(a, t) \ln p(a, t) da}{\int_0^\omega p(a, t) da} = \frac{-\int_0^\omega p(a, t) \ln p(a, t) da}{\int_0^\omega p(a, t) da}. \quad (4)$$

As Demetrius⁴ and Keyfitz³ have shown, H , which is a variant of the measure known as entropy or information in other contexts, can be interpreted as a measure of the heterogeneity of a population with respect to mortality at different ages; if everyone dies at the same age, $H = 0$; if the force of mortality is equal at all ages, $H = 1$. As indicated by (3), H gives the percentage change in life expectancy produced by a reduction of one per cent in the force of mortality at all ages: if $H = 0.2$, say, a uniform decrease of one per cent in the force of mortality would increase life expectancy at birth by 0.2 per cent.

An alternative expression of H is revealing. It follows from (4) that

$$\begin{aligned} H(t) &= \frac{\int_0^\omega \int_0^a p(a, t) \mu(x, t) dx da}{e(0, t)} = \frac{\int_0^\omega \int_x^\omega p(a, t) \mu(x, t) da dx}{e(0, t)} \\ &= \frac{\int_0^\omega \mu(x, t) \cdot \int_x^\omega p(a, t) da dx}{e(0, t)} = \frac{\int_0^\omega \mu(x, t) p(x, t) e(x, t)}{e(0, t)}. \end{aligned} \quad (5)$$

Because the product of μ and p gives the density of deaths at age x , this formula helps to show why H is a measure of the heterogeneity of a population with regard to age at death (or lifespan). Furthermore, this formula facilitates understanding of why H measures the percentage increase in life expectancy generated by a decrease in mortality rates of one per cent. If a death is averted at age x , then $e(x)$ years of life are gained. The numerator of the last expression in (5) measures the total effect of reducing deaths at all ages; the denominator converts the absolute effect in a relative effect. As suggested to me by my colleague Anatoli I. Yashin, this implies that $H(t)$ gives the proportional increase in life expectancy at birth if everyone's *first* death were averted. The assumption is that each individual is saved at the hour of death and given the life expectancy of individuals surviving at that age. Thus, if $H(t) = 0.15$, staying the hand of death once would increase life expectancy by 15 per cent.

³ N. Keyfitz, *Applied Mathematical Demography* (New York: Wiley 1977). Also see W. B. Arthur, 'The analysis of linkages in demographic theory', *Demography* 21, 1, (1984), pp. 109-128.

⁴ L. Demetrius, 'Demographic parameters and natural selection', *Proceedings of the National Academy of Sciences* 71, 12, (1974), pp. 4645-4647. *Idem*, 'Relations between demographic parameters', *Demography* 2, 16 (1979), pp. 329-338.

⁵ *Op. cit.* in footnote 3.

The expression in (5) for H can be usefully decomposed. Let

$$\eta(x, t) = \mu(x, t)p(x, t)e(x, t)/e(0, t), \quad (6)$$

so that

$$H(t) = \int_0^{\omega} \eta(x, t) dx. \quad (7)$$

If progress against mortality only occurs at a single instantaneous age a , then

$$\pi(t) = \rho(a, t)\eta(a, t). \quad (8)$$

Thus, $\eta(a)$ is a measure of the potential for increasing life expectancy (or, equivalently, saving life years) by reducing mortality at age a . If uniform progress against mortality at rate $\rho(t)$ is made between ages α and β , then

$$\pi(t) = \rho(t) \int_{\alpha}^{\beta} \eta(a, t) da. \quad (9)$$

This formula was used to answer the question posed at the beginning of this article. More generally,

$$\pi(t) = \int_0^{\omega} \eta(a, t)\rho(a, t) da. \quad (10)$$

THE POTENTIAL FOR SAVING YEARS OF LIFE

In Table 1 values are presented of $\int_x^{x+5} \eta(a) da$ for Swedish males and females in 1982.⁶

After infancy, the maximum value of η for the men occurs at age 72.5; for women, it occurs at age 80.0. A reduction of one per cent in the force of mortality between ages 75 and 80 would increase men's life expectancy by 0.036 per cent and women's by 0.31 per cent. A reduction in the force of mortality by one per cent at all ages would increase men's life expectancy by about 0.15 per cent and women's by about 0.13 per cent.

In which conventional five-year period (e.g. 25–30 or 60–65), not counting early childhood from 0 to 5, is the potential for saving life years greatest? In Table 2 the answer is presented for an assortment of countries at different times with varying life expectancies. The rule of thumb is that the optimal five-year period is near the life expectancy of the population: the rule holds particularly well for populations with life expectancies of 65 years or more.

A simple model and some elementary calculus sheds some light on this finding. If the

⁶ A number of different life tables, from different sources, were used to make the calculation in this paper. The life tables for Sweden from 1780 to 1950 are from Keyfitz and Flieger (see end of this note); Swedish life tables after 1950, except for 1970 and 1982, are from the annual Swedish *Statistical Yearbook*. These life tables are based on five years of data centered on the year given: the 1910 table, for example, is based on data from 1908 to 1912. The Swedish life tables for 1970 and 1982 were supplied by Professor Ingvar Holmberg of the University of Göteborg: these tables relate to a single year. The U.S. life table for 1979 is based on the advance report of final mortality statistics in the *Monthly Vital Statistics Report* (September 1982); the figures were adjusted by the correction factors given in that Report so that they are consistent with population estimates based on the Census of 1980. The U.S. life table for 1970 is the decennial table based on data from 1969 to 1971, as published in *United States Life Tables: 1969–71* (National Center for Health Statistics, May 1975). The U.S. life table for 1900 is taken from S.H. Preston, N. Keyfitz and M. Schoen, *Causes of Death: Life Tables for National Populations* (New York: Seminar Press, 1972). The U.S. life tables for 1980 and 2000 are from *Life Tables in the United States: 1900–2050*, Actuarial Study No. 87, U.S. Dept. of Health and Human Services (September 1982). All the remaining life tables which relate to years between 1974 and 1979 are from the United Nations *Demographic Yearbook* (1980). All other life tables are from N. Keyfitz and W. Flieger, *World Population: An Analysis of Vital Data* (Chicago: University of Chicago Press, 1968).

Table 1. Values of $\int_x^{x+5} \eta(a) da$ for Swedish males and females in 1982

Age period	Males	Females
0-5	0.00853	0.00763
5-10	0.00085	0.00068
10-15	0.00060	0.00060
15-20	0.00226	0.00079
20-25	0.00289	0.00120
25-30	0.00344	0.00140
30-35	0.00341	0.00172
35-40	0.00434	0.00243
40-45	0.00530	0.00313
45-50	0.00736	0.00447
50-55	0.00942	0.00591
55-60	0.01258	0.00779
60-65	0.01555	0.00936
65-70	0.01788	0.01202
70-75	0.01869	0.01464
75-80	0.01719	0.01679
80-85	0.01282	0.01650
85-90	0.00675	0.01201
90-95	0.00231	0.00545
95-100	0.00055	0.00142
<i>H</i> (i.e. total for all ages)	0.15270	0.12622

force of mortality follows a Gompertz curve with exponential rate of increase β , then setting the derivative of $\eta(a)$ with respect to a equal to zero yields the result that the maximum value of $\eta(a)$ occurs at the age at which life expectancy equals the reciprocal of β . For instance, if $\beta = 0.1$, the potential for saving years of life is greatest at the age at which remaining life expectancy is ten years. For a Gompertz curve of mortality, this age turns out to be roughly equal to life expectancy at birth.

A reduction in the force of mortality by one per cent at all ages would produce a much lower increase in life expectancy to-day than would have been the case 50 years or a century ago. This decline is, in large measure, a price of the progress in reducing deaths in infancy – the age at which the highest number of years of life expectancy are lost. Another result of this progress is a shift in the ages when further progress against mortality would be most effective in increasing life expectancy. Before 1900, most of the potential for saving years of life was concentrated in the first five years of childhood; to-day, in developed countries, most is in old age. In Table 3 the decline in H and the shift in the profile of η is shown by presenting data based on Swedish life tables from 1800 to 1980. Keyfitz⁷ shows the decline in H for males and females in the U.S. from 1920 to 1960. As H falls, the value of η at most ages must also fall. Thus, as life expectancy increases progress in reducing age-specific mortality translates into less and less progress in further increasing life expectancy. More generally, differing profiles of η lead to Pollard's paradox:⁸ more rapid progress against mortality may be occurring at all ages in one population compared with another, but, nonetheless, life expectancy may be increasing less rapidly.

⁷ *Op. cit.* in footnote 3.

⁸ *Loc. cit.* in footnote 1.

Table 2. *The five-year period following infancy for which the potential for saving years of life is greatest, for various male and female populations with different life expectancies, from different countries, at different periods*

Country	Period	Sex	Five-year period following infancy for which $\int_a^{a+5} \eta(x) dx$ is greatest	e_0
Italy	1881	M	20-25	33
Sweden	1780	M		36
USA	1900	M		46
Italy	1881	F	25-30	34
England and Wales	1861	F		43
USA	1900	F		48
Sweden	1780	F	30-35	39
England and Wales	1861	M		40
Japan	1899	F	35-40	44
Japan	1899	M	56-60	42
Czechoslovakia	1934	M		52
Australia	1911	M	60-65	58
Mexico	1975	M		63
Czechoslovakia	1934	F	65-70	56
Australia	1911	F		61
Poland	1960	M		65
Mexico	1975	F		67
Japan	1964	M		68
England and Wales	1976-8	M		70
USA	1980	M		70
USA	2000	M		73
Poland	1960	F	70-75	71
Japan	1964	F		73
Japan	1978	M		73
Sweden	1982	M		73
Iceland	1977-8	M		74
England and Wales	1976-8	F	75-80	76
USA	1980	F		78
Japan	1978	F		78
Sweden	1982	F		79
Iceland	1977-8	F	80-85	79
USA	2000	F		81

RATES OF PROGRESS AGAINST MORTALITY

The potential for saving years of life is measured by η ; progress against mortality is given by ρ . As indicated in (10), progress in increasing life expectancy, as measured by π , depends on the product of η and ρ . Thus, even if the potential for saving years of life is greatest in old age, if little progress is being made in reducing mortality at older ages then this potential will not translate into gains in life expectancy.

In Table 4 data are presented on η and ρ for Swedish females in 1982. Progress in reducing mortality is highest in infancy and childhood; afterwards, the annual rate of progress hovers between approximately one and two per cent at most ages. Because of the rapid rate of progress in childhood, almost one-sixth of the gains in life expectancy occur before age 20 even though less than one-tenth of the potential lies in these years. By age 55, however, potential and actual progress are in rough balance: 70 per cent of the potential for saving years of life occurs after age 55 and 70 per cent of the actual improvement in life expectancy can be attributed to progress made in reducing mortality after age 55.

Table 3. *The potential for saving years of life (H), the proportion of this potential below age 5 and above age 65, and life expectancy at birth for selected Swedish populations*

Period	H	$\int_0^5 \eta(x) dx/H$	$\int_{65}^{\omega} \eta(x) dx/H$	$e(0)$
Males				
1800	0.772	0.584	0.050	33.7
1820	0.722	0.534	0.056	35.4
1840	0.636	0.490	0.065	35.4
1860	0.587	4.482	0.096	43.2
1880	0.545	0.458	0.116	46.2
1900	0.432	0.398	0.130	52.0
1920	0.346	0.305	0.172	57.5
1940	0.238	0.233	0.273	65.0
1960	0.167	0.130	0.428	71.2
1980	0.157	0.060	0.476	72.7
Females				
1800	0.712	0.567	0.066	36.9
1820	0.663	0.507	0.075	39.0
1840	0.573	0.471	0.092	43.1
1860	0.538	0.461	0.119	46.7
1880	0.509	0.431	0.136	49.2
1900	0.403	0.366	0.152	54.6
1920	0.318	0.272	0.199	60.1
1940	0.211	0.198	0.324	67.7
1960	0.142	0.116	0.530	74.9
1980	0.126	0.058	0.613	79.0

Note. The life tables used before 1900 included no estimate of the force of mortality after age 85. For these tables I assumed $\int_{85}^{\omega} \eta(x) dx = \int_{80}^{85} \eta(x) dx$. This approximation is based on the life tables for which mortality rates are available after age 85.

It may seem surprising that progress in reducing mortality rates hovers around roughly the same level at all ages after childhood and that significant improvements are being made at older ages. In Table 5 data are presented on trends in mortality rates since 1780 for Swedish females and males, and since 1920 for U.S. females and males. In most cases, progress in reducing mortality after age 65 is comparable to that between ages 5 and 65. Except for Swedish males, progress since 1950 and especially since 1970 against mortality in old age has been substantial.⁹

Suppose progress against mortality were to continue. Will H decline much further? Will life expectancy level off as it becomes more and more difficult to increase life expectancy by decreasing mortality rates? Some insight into these questions can be gained by a simple model. Assume that the force of mortality can be described by a Gompertz curve; this is not an unreasonable assumption for our purposes here, given the low level

⁹ For a discussion of recent trends in mortality in the U.S.A. see E. M. Crimmins, 'The changing pattern of American mortality decline, 1940-1977, and its implications for the future', *Population and Development Review* 7, 2 (1981), pp. 229-254. K. G. Manton, 'Changing concepts of morbidity and mortality in the elderly population', *Milbank Memorial Fund Quarterly/Health and Society* 60, (1982), pp. 183-244. J. M. Owen and J. W. Vaupel, 'Anna's life expectancy', WP-85-11 (Laxenburg, Austria: International Institute for Applied Systems Analysis, 1985).

Table 4. Average values of η and ρ in various age groups, the cumulative percentage of η and of the product of $\eta\rho$ in these age groups, for Swedish females in 1982

Age group	$\bar{\eta}$	$\frac{\int_0^a \eta(x) dx}{\int_0^{\omega} \eta(x) dx}$ (%)	$\bar{\rho}$	$\frac{\int_0^a \eta(x) \rho(x) dx}{\int_0^{\omega} \eta(x) \rho(x) dx}$ (%)
0-5	0.00149	5.9	3.0	9.3
5-10	0.00014	6.4	7.3	11.4
10-15	0.00012	6.9	3.5	12.3
15-20	0.00021	7.7	4.4	14.3
20-25	0.00024	8.7	2.8	15.6
25-30	0.00028	9.8	1.6	16.6
30-35	0.00034	11.1	1.1	17.3
35-40	0.00049	13.1	1.7	19.1
40-45	0.00063	15.5	2.0	21.7
45-50	0.00090	19.1	1.4	24.4
50-55	0.00119	23.8	1.5	28.2
55-60	0.00156	30.0	0.7	30.5
60-65	0.00189	37.4	1.5	36.4
65-70	0.00243	46.9	1.5	44.0
70-75	0.00297	58.6	1.8	55.2
75-80	0.00340	72.0	2.0	69.5
80-85	0.00334	85.2	1.7	81.3
85-90	0.00242	94.7	2.0	91.5
90-95	0.00108	98.9	2.9	98.1
95-100	0.00027	100.0	3.4	100.0

Note. The rate of progress in reducing mortality, $\bar{\rho}$, is the average rate from 1970 to 1982. The formulae used to calculate $\bar{\eta}$ and $\bar{\rho}$ are:

$$\bar{\eta} = \frac{n d_x \cdot e_x + e_{x+n}}{2} / e_0 \quad (\text{where } x+n = a),$$

and

$$\bar{\rho} = (\ln(-\ln(1 - {}_n q'_x)) - \ln(-\ln(1 - {}_n q_x))) / t,$$

where q' is taken from the earlier life table and t is the number of years that have elapsed.

of mortality in infancy and childhood in developed countries. Then it can be shown¹⁰ that

$$\frac{de(0, t)}{dt} \approx \frac{\rho}{\beta} \quad (11)$$

and

$$H(t) \approx 1/(\beta e(0, t)), \quad (12)$$

where ρ is the steady rate of progress being made against mortality and β is the exponential rate of increase with age in the force of mortality. Hence, H does continue to decline but the absolute increase in life expectancy remains roughly constant. If $\rho = 0.01$ per year and $\beta = 0.1$, then a decade will be added to life expectancy every century.

¹⁰ J. W. Vaupel, 'How change in age-specific mortality affects life expectancy', WP-85-17. (Laxenburg, Austria: International Institute for Applied Systems Analysis, 1985).

Table 5. *The average annual rate of progress \bar{p} (per cent) in reducing the force of mortality for Swedish females and males from 1780 to 1982 and for U.S. females and males for 1920 to 1979 for various age groups*

Population	Period	Age group				
		0-5	5-25	25-45	45-65	65-85
Swedish F	1780-1870	0.4	0.4	0.3	0.1	0.1
	1870-1910	2.1	0.8	0.7	1.1	0.8
	1910-1950	3.9	4.9	3.5	1.1	0.2
	1950-1970	3.4	2.3	2.1	1.8	1.6
	1970-1982	3.0	4.3	1.7	1.3	1.8
Swedish M	1780-1870	0.4	0.5	0.2	0.0	-0.1
	1870-1910	2.0	1.0	1.0	1.3	0.9
	1910-1950	3.6	3.6	2.9	1.0	0.2
	1950-1970	3.2	1.7	0.5	0.3	0.4
	1970-1982	4.7	3.9	0.6	0.3	0.1
U.S. F	1920-1950	3.9	5.5	4.0	1.8	1.2
	1950-1970	2.5	1.5	1.4	1.3	1.2
	1970-1979	4.8	2.3	3.6	2.1	2.7
U.S. M	1920-1950	3.7	1.3	3.5	0.3	0.5
	1950-1970	2.6	-0.1	0.3	0.4	0.2
	1970-1979	5.0	1.7	2.3	2.4	1.9

Note: The values of \bar{p} were calculated using the formula given in Table 4.

THE IMPACT OF HETEROGENEITY ON THE LIFE EXPECTANCY OF THE DEAD

The assumption is questionable that those who die at some age would, if saved, have the same life expectancy as those who live. For instance, the victim of a serious heart attack or motor vehicle accident might, if death were averted, be prone to another heart attack or motor vehicle accident.¹¹ More generally, individuals of the same age may differ from each other in their 'frailty' or relative risk of death.¹² Let the life expectancy of those who are saved at age a (i.e. the average number of years, under current mortality conditions, that these individuals would live if death could be averted) be denoted by $e^*(a)$. In a homogeneous population, this life expectancy would equal $e(a)$; in a heterogeneous population it will probably be lower, although it could, conceivably, be higher. Then, (6) becomes

$$\eta(a) = \mu(a)p(a)e^*(a)/e(0). \quad (13)$$

As a simple example, suppose $e^* = \frac{1}{2}e$ at all ages. The values of η and the value of their integral, H , would be half as great as the assumption of homogeneity would indicate. The profile of the η s would be same – and hence the age at which there was the greatest potential for saving years of life would not change – but the impact of a reduction of one per cent cut in death rates on life expectancy would be cut in half.

More generally, let $\mu_a^*(x)$ represent the force of mortality at age x of those who would

¹¹ R. Zeckhauser and D. Shephard, 'Where now for saving lives?', *Law and Contemporary Problems* 40, 4 (1976), pp. 5-45.

¹² J. W. Vaupel, K. G. Manton and E. Stallard, 'The impact of heterogeneity in individual frailty on the dynamics of mortality', *Demography* 16, (1979), pp. 439-454. J. W. Vaupel and A. I. Yashin, 'The deviant dynamics of death in heterogeneous populations', RR-83-1 (Laxenburg, Austria: International Institute for Applied Systems Analysis, 1983). Abridged version in Nancy Tuma (ed.), *Sociological Methodology 1985* (San Francisco: Jossey-Bass). J. W. Vaupel and A. I. Yashin, 'Heterogeneity's ruses: some surprising effects of selection on population dynamics', *The American Statistician* (August 1985).

Table 6. *Life expectancies of those saved from death for various values of their relative risk, based on 1978 Swedish female life table*

Age	e^* when $\gamma =$					
	1.0	1.1	1.25	1.5	2	5
0	78.5	77.5	76.2	74.3	71.2	60.3
10	69.2	68.3	67.1	65.3	62.5	53.0
20	59.4	58.5	57.3	55.6	52.8	43.7
30	49.6	48.8	47.6	45.9	43.3	34.6
40	40.0	39.2	38.0	36.4	33.9	25.8
50	30.7	29.9	28.8	27.3	25.0	18.0
60	21.8	21.1	20.2	18.9	16.9	11.2
70	13.8	13.2	12.4	11.3	9.80	5.7
80	7.3	6.9	6.3	5.6	4.6	2.2
90	3.2	3.0	2.7	2.3	1.7	0.6
H	0.127	0.121	0.115	0.105	0.091	0.055
$\int_{65}^{\omega} \eta(x) dx / \bar{H}$	60%	60%	58%	56%	64%	43%

have died at age $a < x$, perhaps from some specified cause, but who were saved. Let the risk ratio be given by

$$\gamma_a(x) = \mu_a^*(x) / \mu(x), \quad (14)$$

where $\mu(x)$ is simply the force of mortality at age x (i.e. among those who would not have died). Then, letting $e_a^*(x)$ denote the life expectancy at age x of those who were saved from death at age a ,

$$e_a^*(x) = \int_a^{\omega} \exp\left(-\int_a^x \gamma_a(t) \mu(t) dt\right) dx. \quad (15)$$

Various special cases of this general result may be of interest. For instance, $\gamma_a(t)$ could be constant for all t , could gradually decline towards unity, or could be constant for a decade, say, and then fall to unity. Table 6 provides some illustrative results when γ_a is constant. For example, consider a group of 50-year-olds who would have died from a heart attack, but who were saved. Suppose this group face a force of mortality, for the rest of their lives, some five times greater than the normal force of mortality. Then the table indicates that their remaining life expectancy would be 18.0 years, rather than the normal 30.7 years.

The values of H given at the bottom of Table 6 were calculated on the assumption that $\gamma_a(x)$ was constant not only for all values of x but also for all values of a .¹³ As the relative risk of mortality for the resuscitated increases, H decreases. The proportion of the potential for saving lives that lies above age 65 also decreases somewhat but much less dramatically.

¹³ A simple mode of heterogeneity that leads to a constant value of γ is the model with gamma-distributed frailty proposed by Beard and developed by Vaupel *et al.* and Vaupel and Yashin. See R. E. Beard, 'A theory of mortality based on actuarial biological and medical considerations', *Proceedings of the International Population Conference* (New York, 1961), vol. 1, pp. 611-625; Vaupel, Manton and Stallard, *loc. cit.* in note 12. Vaupel and Yashin, 'The deviant dynamics of death in heterogeneous populations', *loc. cit.* in note 12. It can be shown that in this model $\gamma = 1 + \sigma^2$, where σ^2 is the variance, at birth, in the distribution of frailty. If, for instance, $\sigma^2 = 0.25$, then the resuscitated, at all ages and regardless of the age at which they were saved, would suffer a force of mortality 25 per cent higher than ordinary individuals. See Vaupel, *loc. cit.* in note 10.

POLICY IMPLICATIONS AND INSINUATIONS

As I have discussed elsewhere,¹⁴ nearly all statistics presented in policy-relevant studies are really vectors: they not only summarize a body of data, but also imply a policy thrust. Intellectual honesty requires some discussion of lurking insinuations that may appear to be simple facts. If mortality rates were reduced by one per cent, over 60 per cent of the years of life gained would be gained by averting deaths of persons over 65 years old. Does this imply that life-saving efforts should be directed toward the elderly population? Not necessarily, for several reasons. First, the figure of 60 per cent is based on the 1982 life table for Swedish females. For males and for other countries the figure is generally lower – for Swedish males in 1982 it is less than 50 per cent. Heterogeneity, as discussed above, would reduce this still further.

Secondly, the figure is based on a life table – i.e. on an hypothetical, stationary population – rather than on the actual distribution of a population by age. In most populations there are more young people than is implied by the life table. Consequently, the goal of increasing life expectancy is not completely congruent with the goal of saving as many years of life as possible, given the current population distribution. For instance, for the U.S. life table of 1979, about 50 per cent of the increase in life expectancy produced by a reduction in mortality rates of one per cent can be attributed to the reduction in mortality rates of persons over 65 years old. However, only about 36 per cent of the gain in years of life produced by a similar reduction in the actual number of deaths at all ages would be due to averting deaths above age 65.

Thirdly, the quality of life at advanced ages may tend to be lower than at younger ages. If the goal is to save as many quality-adjusted life years as possible,¹⁵ efforts to avert deaths at younger ages will appear more favourable. Other goals that might be proposed – e.g. maximize years of life saved before the biblical allotment of three score and ten, maximize economic production, minimize deaths of parents with young children, or minimize inequalities in lifespans – also favour efforts to reduce early deaths. I have examined several criteria and conclude that most of the losses due to death are due to deaths before age 65.¹⁶

Fourthly, it may be easier to avert deaths at ages below 65 than afterwards. As the data presented on the rate of progress against mortality show, progress against early death has generally tended to be somewhat more rapid than progress against death after age 65.

Offsetting these considerations are many others. As Vaupel and Yashin suggest,¹⁷ the true rate of progress in reducing mortality rates at advanced ages may be masked by the effects of heterogeneity. The quality of life of many of those who die before their 65th birthday may be relatively low, even if the quality of life at younger ages does tend to be higher than that after that age. Furthermore, those who die early may tend to be those whose life expectancies, if saved, would be relatively short. Finally, there are several appealing objectives that favour saving life at older ages. It is desirable to avert death *per se*, regardless of life expectancy, and most deaths occur in old age. It is desirable to have a society that is diverse in its age composition – and in its memories and experiences.

¹⁴ J. W. Vaupel, 'Statistical insinuation', *Journal of Policy Analysis and Management* 1,2 (1982), pp. 261–263.

¹⁵ Zeckhauser and Shepard, *loc. cit.* in footnote 11.

¹⁶ J. W. Vaupel, 'Early death: an American tragedy', *Law and Contemporary Problems* 40, 4 (1976), pp. 73–121. *idem*, 'The prospects for saving lives: a policy analysis', Working Paper 778, Institute for Policy Sciences and Public Affairs, Duke University, Durham, N.C. (1978). Reprinted in *Comparative Risk Analysis* (U.S. House Committee on Science and Technology, 1980).

¹⁷ *Loc. cit.* in footnote 13.

Suppose it were possible to save the lives of ten 80-year-olds, giving them, on average, seven additional years of life. And suppose the alternative was to save the lives of two 40-year-olds, giving each an expected additional lifespan of 35 years. In each case, 70 years of life are gained. Which alternative would be preferable? Recommendations concerning the focus of policies to save lives depend not only on statistical analyses but also on answers to such difficult value questions.

Beyond this, policy decisions are usually made concerning specific life-saving alternatives. Should an extra million dollars be devoted to research on influenza? Should passive restraint systems for automobiles be required? These decisions depend not only on broad value judgments, but also on the details of the specific proposal. How effective is it likely to be? How much will it cost? How many voters will like it?

Nonetheless, the methods and findings of this paper may be of some relevance to policy discussions. In particular, there is considerable potential for saving years of life and increasing life expectancy by reducing mortality in old age, more than is generally realized. Furthermore, because considerable progress is being made in reducing mortality among the elderly, this potential is being achieved. The result is a shift in the age composition of the population: progress in reducing mortality rates is adding relatively few years of life among the working-age population compared with the extra years added after age 65.