Estimating Clinical Morbidity Due to Ischemic Heart Disease and Congestive Heart Failure: The Future Rise of Heart Failure

ABSTRACT

Objectives. Many developed countries have seen declining mortality rates for heart disease, together with an alleged decline in incidence and a seemingly paradoxical increase in health care demands. This paper presents a model for forecasting the plausible evolution of heart disease morbidity.

Methods. The simulation model combines data from different sources. It generates acute coronary event and mortality rates from published data on incidences, recurrences, and lethalities of different heart disease conditions and interventions. Forecasts are based on plausible scenarios for declining incidence and increasing survival.

Results. Mortality is postponed more than incidence. Prevalence rates of morbidity will decrease among the young and middle-aged but increase among the elderly. As the milder disease states act as risk factors for the more severe states, effects will culminate in the most severe disease states with a disproportionate increase in older people.

Conclusions. Increasing health care needs in the face of declining mortality rates are no contradiction, but reflect a tradeoff of mortality for morbidity. The aging of the population will accentuate this morbidity increase. (*Am J Public Health.* 1994;84: 20–28)

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Introduction

Heart disease, comprising ischemic heart disease and congestive heart failure, is not only the leading cause of death and lost life expectancy in most Western countries but also one of the most important causes of morbidity and health care costs.[1] After a period of increasing mortality during the 1950s and 1960s, Western countries have witnessed a dramatic decline of ischemic heart disease mortality (age-adjusted), with the United States taking the lead.[2] Though the dynamics are not completely understood, time trends in known risk factors for ischemic heart disease (hypertension, cholesterol, smoking) in Western countries indicate a clear decline.[3–5] Therefore it is reasonable to attribute at least part of this decrease in mortality to a decrease in incidence.

The observed sharp decline in mortality, together with the probable decline in incidence, leads one to expect a concomitant decrease in health care utilization. In reality, however, health care providers all over the Western world are facing increases in demand. After the introduction of percutaneous transluminal coronary angioplasties, utilization of these procedures increased almost exponentially, while the growth in utilization of coronary artery bypass graft surgery merely slowed.[6] Viewed superficially, the observed increases in demand are incongruent with the supposed decrease in need.

But is need truly decreasing? Basic epidemiologic theory teaches us that if mortality slows faster than incidence, prevalence will increase. Although the Framingham Heart Study has shown a small decline in incidence, a strong increase in prevalence has been observed, indicative of a survival gain.[3] This improved prognosis has been caused partly by improved risk factor distributions, but medical therapy has also contributed.[7,8] Recently, beta-blocking agents, platelet aggregation inhibitors, coronary artery bypass graft surgery, and angiotensine converting enzyme inhibitors have been applied in chronic therapy and thrombolytic therapy has been applied in emergencies. All have been proven effective in randomized controlled trials meeting high standards.[9–11] If trends in risk factors and survival remain favorable, the further decrease in incidence and mortality of ischemic heart disease is likely to be accompanied by a continuing increase in prevalence. In Western countries, the increased prevalence will be accentuated by the aging of the baby boom generation born after World War II. This cohort will soon reach middle age with its concomitant heart disease risks: the increase in age-specific prevalence will be accompanied by an increase in sheer size of the older age groups.

To make a quantitative analysis of the dynamics of the heart disease epidemic, we developed a simulation model for the most important manifestations of ischemic heart disease and congestive heart failure. Available information from the medical literature was combined with data obtained from mortality and morbid-

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ity registers. We explored the future of heart disease morbidity in the Netherlands in different scenarios.

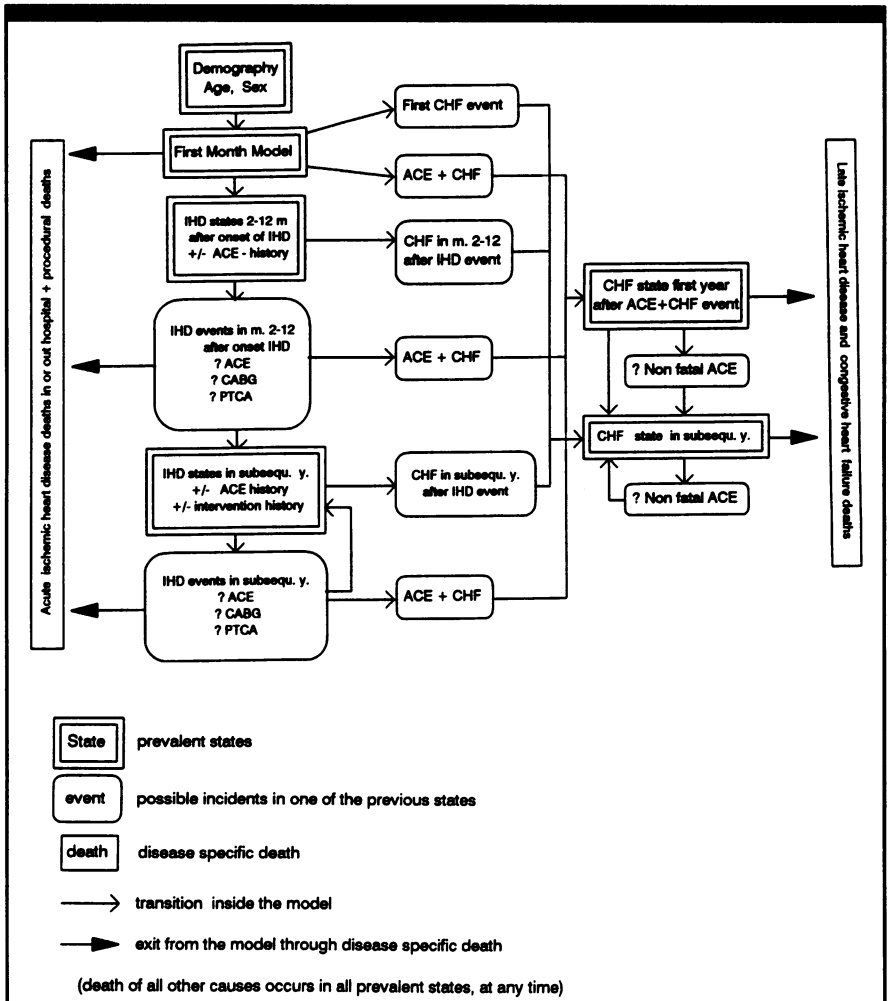
Methods

Model Structure

The deterministic state transition model used divides the population under study (here the entire Dutch population) into homogeneous subpopulations called states, defined by age, sex, and disease history. Each year, the population "ages" by 1 year and the states are updated. Transitions from one state to another are governed by transition probabilities, extracted from registries and literature. Transition probabilities are independent from the preceding states and depend only on the current state (Markov chain assumption).[12] The structure of the model resembles the Coronary Heart Disease Policy Model of Weinstein et al., but we have excluded cardiac arrest as a prevalent state and included congestive heart failure.[13]

The morbidity model consists of three main sectors: the first-month model, the ischemic heart disease model, and the congestive heart failure model. Persons developing heart disease, the rate of which is dependent on age and sex, move into the first-month model, which characterizes the initial event and its immediate sequelae during the first 30 days following the onset of disease. Persons in whom congestive heart failure forms the first heart disease event move directly into the appropriate model. Survivors of the first 30 days enter the ischemic heart disease model or the congestive heart failure model that describes the remaining 11 months of the first year and all subsequent years. Persons with ischemic heart disease can move into the congestive heart failure model, but not vice versa. Death from heart disease is modeled through the specific transition probabilities of the model. In all prevalent states, persons are subjected to an additional risk of death from all other causes, corrected for heart disease, that is based on national life tables.[14]

The first-month model covers the month immediately after the initial cardiac disease event (Figure 1). This event may be angina pectoris, an acute coronary event (an acute myocardial infarction or cardiac arrest), or congestive heart failure. Persons suffering a first acute coronary event may die before reaching a health care facility (deaths before admission).



Note. CHF = congestive heart failure; ACE = acute coronary event; IHD = ischemic heart disease; CABG = coronary artery bypass graft; PTCA = percutaneous transluminal coronary angioplasty.

FIGURE 1—The heart disease model.

Once taken into care, they are subjected to a further death risk (deaths in hospital). If the person survives the coronary event, the pump function of the heart may be permanently impaired. Patients with heart failure after an acute myocardial infarction enter the congestive heart failure model; the others move into the ischemic heart disease model. Persons with a first diagnosis of uncomplicated angina pectoris or congestive heart failure proceed to the ischemic heart disease and congestive heart failure models, respectively.

On entry, the ischemic heart disease model distinguishes between angina pectoris and acute coronary event states. Conditional on these states, patients face possible new events in the 2 to 12 months after the first incident. These events range from acute coronary events to coronary artery bypass grafts to percutaneous transluminal coronary angioplasties to all possible combinations of these. Each combination of events in a state has a probability of sudden death from ischemic heart disease dependent on the current event, the current state (and therefore the ischemic heart disease history), and the patient's age and sex. The patients also risk heart failure as a sequel of an acute coronary event ("ACE + CHF" in the figure) or as a consequence of chronic ischemic damage; these patients move into the congestive heart failure model. If none of the above occurs, patients will remain in the ischemic heart disease model.

After this first year, patients remaining in the ischemic heart disease model are reclassified into four new states, conditional on previous disease history. These states are described by the presence or absence of an acute coronary event or an intervention. These patients face the same events as in the previous year. At the end of each subsequent model year, persons

TABLE 1—Transition Probabilities for the Age Group 60 through 64 Years

	Men, %	Women, %
First incidence in the total population		
ACE	0.80	0.26
AP	0.47	0.30
CHF	0.20	0.09
Yearly probability of an ACE in the diseased population		
In the population with a history of ACE		
Before no intervention in y 1	5.3	5.3
Before PTCA in y 1	7.1	7.1
Before CABG in y 1	10.7	10.7
After CHF after ACE in y 1	20.5	19.4
After no intervention in y > 1	2.8	2.8
After any intervention in y > 1	4.3	4.3
After CHF in y > 1	7.3	6.2
In the population with a history of uncomplicated AP		
Before no intervention in y 1	1.5	0.6
Before PTCA in y 1	3.7	3.7
Before CABG in y 1	5.5	5.5
After no intervention in y > 1	1.5	0.7
After any intervention in y > 1	2.2	2.2
Probability of CHF in the diseased population		
After ACE in mo 1	8.5	11.1
After ACE in mo > 1	2.5	2.5
After AP	1.0	0.5
Intervention rates		
CABG		
In y 1 after ACE	11.3	5.9
In y > 1 after ACE	2.3	1.2
In y 1 after AP	15.9	8.8
In y > 1 after AP	3.2	0.6
PTCA		
In y 1 after ACE	2.6	1.9
In y > 1 after ACE	0.5	0.4
In y 1 after AP	3.9	2.9
In y > 1 after AP	0.8	0.8
Mortality rates		
Heart disease		
After ACE in mo 1		
First ACE, before admission	17.4	18.0
First ACE, inside hospital	8.4	11.0
Recurrent ACE, before admission	24.0	24.8
Recurrent ACE, inside hospital	12.7	16.5
Recurrent ACE after CHF, before admission	47.1	44.0
Recurrent ACE after CHF, inside hospital	18.9	22.0
Operative mortality after first CABG	1.4	3.0
Operative mortality after recurrent CABG	3.4	3.4
Procedural mortality after PTCA	0.7	0.7
After CHF after ACE in mo 2–11	19.9	17.9
After CHF in y > 1	13.0	11.0
All other causes of death in all states	1.14	0.64

Note. ACE = acute coronary event (including acute myocardial infarction, unstable angina pectoris, and cardiac arrest); AP = uncomplicated angina pectoris; CHF = congestive heart failure; CABG = coronary artery bypass grafting; PTCA = percutaneous transluminal coronary angiography; in y > 1 = in the years subsequent to the first year after an event.

"age" 1 year and are reassigned to states that reflect their updated history.

Persons with heart failure after an acute myocardial infarction face a high risk of dying during the first year.[15] If they survive the first year, they move to the congestive heart failure state describing the subsequent years, where they will stay until death; this state is called "chronic" to distinguish it from the first critical year following such a complicated infarction. In these "chronic" years, the model assumes that mortality rates are lower. All other patients developing heart failure immediately enter the chronic congestive heart failure state.

Although nonfatal acute coronary events are possible in the congestive heart failure state, such events no longer effect any change in the assigned state. Patients in the congestive heart failure state face an annual probability of death due to cardiovascular causes that may be classified as acute myocardial infarction (ICD 410–411), chronic ischemic heart disease (ICD 412–414), congestive heart failure (ICD 428–429), or stroke (ICD 430–438).[16] We have incorporated stroke as a specific cause of death because the Framingham Heart Study showed that, because of common risk factors (hypertension and diabetes), congestive heart failure is associated with increased rates of stroke and death from stroke.[17]

Incidence and Recurrence Rates

Reliable and recent incidence data on heart disease are not available in the Netherlands. Incidence figures have been estimated by an iterative nonlinear procedure from observed numbers of events (acute myocardial infarctions, ischemic heart disease deaths, interventions) and externally estimated transition probabilities.[18] In this section we give a brief account of how they were derived. The transition probability matrix for a typical age group (60 through 64 years) is furnished in Table 1. The complete transition matrices and a more detailed account of references are available from the authors on request.

Total numbers of admissions for acute myocardial infarction (ICD 410), total numbers of hospital deaths, and total numbers of interventions by age and sex are available from the nationwide hospital register.[19–22] Patients with symptomatic infarctions who are not admitted to hospital (6%; in the Netherlands, this group is limited to the oldest age groups) are added.[23] A constant ratio for the probability of dying before hospital admission versus dying inside the hospital from an acute coronary event is assumed.[20,21,24,25]

Reported probabilities of recurrent acute coronary events vary widely, from 2% to 13% and more per year, depending on age, case mix, and selection criteria.[13,26–30] We have assumed that ischemic heart disease patients without congestive heart failure have a first annual probability of a recurrent acute coronary event of 0.062. This probability is consistent with the estimate of Weinstein et al. of 0.078, in which patients with congestive heart failure are included.[13,31] Recurrence rates are strongly age dependent. We assume the age distribution to be identical to the known age distribution of a first acute coronary event in the reference population. First acute coronary events after a history

of angina pectoris were modeled on the basis of data derived from the Framingham study, which showed that male patients with angina pectoris, who have suffered no previous acute myocardial infarction have a relative risk of 0.54 compared with male patients with a previous acute myocardial infarction.[32] Female patients with angina pectoris have a relative risk of 0.45 compared with male patients.[32]

Patients eligible for coronary artery bypass graft surgery run a risk for recurrent events that is twice as high as that for patients who are not eligible.[31,33-35] Coronary artery bypass graft surgery reduces hazard rates by 20%.[36,37] Lacking data on the effectiveness of percutaneous transluminal coronary angioplasty, we assumed the relative risk for recurrent acute coronary events to be 1.5, with a rate reduction of 10%.

Intervention Rates

Intervention rates are known to vary widely from country to country and even from hospital to hospital.[38,39] We have used age- and sex-specific nationwide output data and assumed constant proportional relations between states to estimate the numbers of interventions in each state. The risk of an intervention after uncomplicated angina pectoris vs the risk of an intervention after an acute coronary event is 1.4: 35% of patients with a history of an acute coronary event are potentially eligible for an intervention, compared with 50% in uncomplicated angina.[40,41] The risk of undergoing an intervention in the first year after an ischemic heart disease event vs the risk in subsequent years was taken to be 5.[42] The annual probabilities of having a subsequent intervention after a first intervention are 1.0% (coronary artery bypass graft) and 0.5% (percutaneous transluminal coronary angioplasty).[43] For every 10 successful percutaneous transluminal coronary angioplasties, another 4 are considered to be unsuccessful because of restenosis.[44] If both a coronary artery bypass graft and percutaneous transluminal coronary angioplasty occur in the same year in the same patient, the percutaneous transluminal coronary angioplasty is ignored.

Heart Failure

Heart failure due to ischemic heart disease is caused by acute events or by chronic ischemic damage. Eighteen percent of ischemic heart disease patients survive an acute myocardial infarction with irreversible heart failure as a sequel.[33,34] The distribution by age, sex, and previous ischemic heart disease history is assumed to be the same as the distribution of in-hospital mortality by age, sex, and previous ischemic heart disease history after an acute myocardial infarction. The annual probabilities of developing congestive heart failure, given a history of ischemic heart disease, were estimated from the Framingham Heart Study data.[17] We obtained the published annual event rates of 1.5% given an uncomplicated angina pectoris history and 3.0% given a history of an acute coronary event and adjusted to allow for the number of acute cases caused by an acute coronary event. The age distribution was calculated by applying relative risks of 3.0 (angina pectoris) and 5.0 (acute myocardial infarction) to the age-specific incidence data taken from the Framingham study.[17]

The incidence rates of congestive heart failure for persons without a history of ischemic heart disease are calculated by assuming that persons in whom congestive heart failure is the certified cause of death survived their first diagnosis of congestive heart failure for 5 (males) to 7 (females) years.[11,12,15,17]

Heart Disease Mortality

The total age- and sex-specific in-hospital mortality rates following acute coronary events and interventions are known in the Netherlands.[20,22] The relative risk of dying after a subsequent vs a first acute coronary event was taken to be 1.5.[13,28,45] Publications are available in which the figures on mortality due to a subsequent vs a first coronary artery bypass graft operation are given.[46] The relative risk of death due to percutaneous transluminal coronary angioplasty vs coronary artery bypass graft surgery is 0.5.[47,48] Procedural mortality due to a subsequent or a first percutaneous transluminal coronary angioplasty is taken to be the same.

The risk of death during the first year after acute myocardial infarction followed by congestive heart failure was assumed to be 25%; the distribution by age was constructed by exponential interpolation and extrapolation of data taken from Ahnve et al.[34] The annual mortality risk of heart failure during the subsequent years is assumed to be constant: 10% for females and 13% for males.[15,17,33,34] Of all deaths caused directly or indirectly by heart failure, 25% (female) and 37% (male) are attributable to recurring ischemic heart events and another 25% (males) and 35% (females) are attributable to stroke (and hence to noncardiac disease).[17] The figures for nonfatal acute coronary events in persons with heart failure are estimated by assuming a case fatality of 60%.

All persons in all states are subject to the risk of dying of other diseases, adjusted to allow for the causes of death under study and based on Dutch life tables.

Results

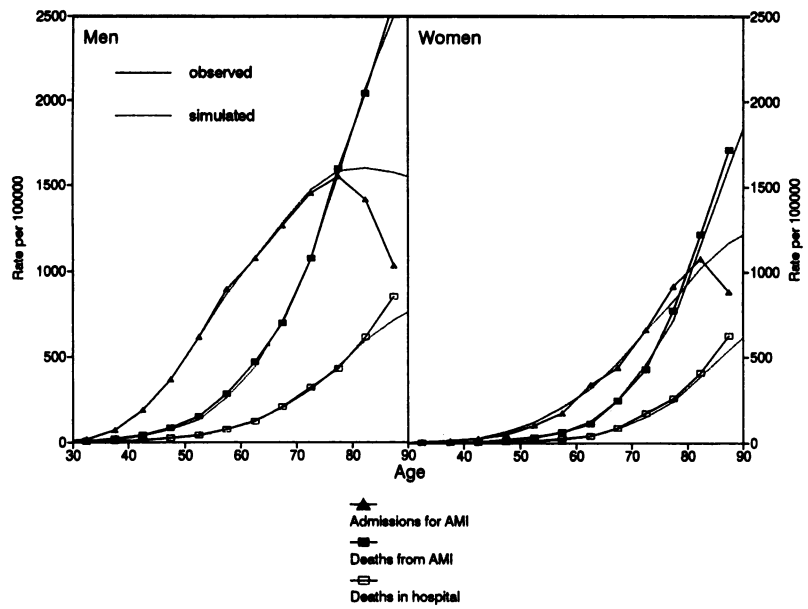
Calculated Estimates for 1985

Figure 2 shows the observed and simulated events of acute myocardial infarction: total admissions and deaths in and outside the hospital, by age and sex. The model closely reproduces the observed data. The gap between simulated and observed rates in the older age groups represents persons with recognized acute myocardial infarctions who are not admitted to a hospital. Calculated prevalences of heart disease by age and sex are shown in Figure 3. Heart disease is more concentrated in the oldest age groups in women, whereas the burden of morbidity in the male population is already high at middle age. The leveling off and decrease by age in the prevalence of clinical ischemic heart disease in men shown in the figure is caused by the steep rise in the incidence of congestive heart failure and sudden cardiac death, reducing clinical ischemic heart disease incidence. Prevalence of all heart disease reaches 25% in the age groups 70 through 74 years (men) and 80 through 84 years (women). Women suffer relatively more often than men from angina pectoris and congestive heart failure, but less often from severe ischemic heart disease. This pattern has been confirmed time and again in many surveys.[32,49,50]

Projections for 1985–2010

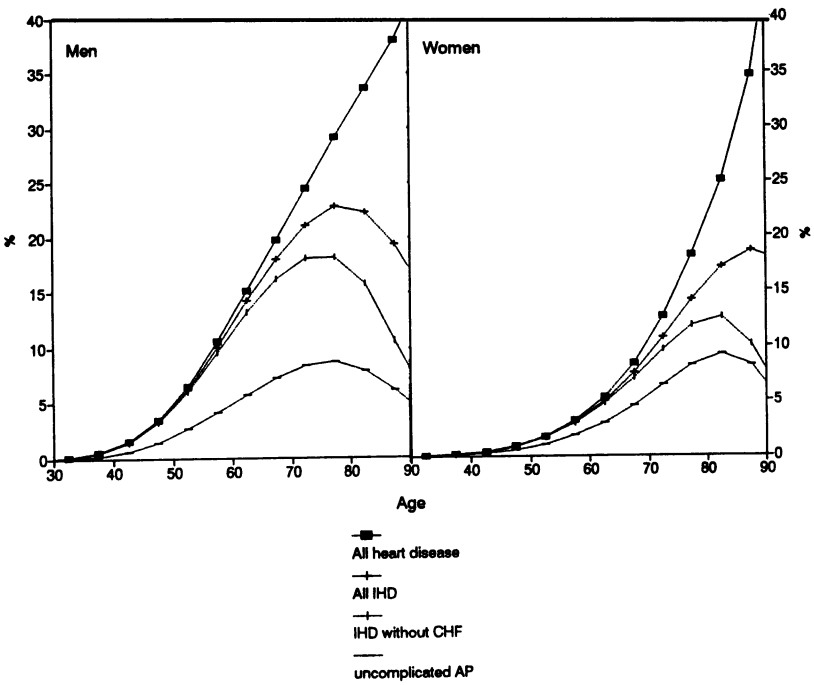
Disease-specific mortality is the direct result of incidence and survival. If two of these elements are known, the third can be calculated. Because the decline in ischemic heart disease mortality that has been observed since 1975 is indisputable, we postulated an ongoing decline of this mortality into the near future. We provide three plausible scenarios, based on varying assumptions of further decreasing incidence rates and improving prognoses (see Table 2).

The reference scenario, based on the Framingham Heart Study, supposes that one third of the mortality decline is explained by a decline in incidence (−1.0% per year) and that two thirds is explained



Note. "Admissions for AMI" are the rates of patients discharged with a primary diagnosis of acute myocardial infarction (ICD 410). "Deaths in hospital" are the rates of patients with the same diagnosis dying in hospital.[20] "Deaths from AMI" are the total rates of death from acute myocardial infarction (ICD 410).[21] The solid lines with symbols show observed numbers; the dotted lines represent the simulation by the heart disease model.

FIGURE 2—Observed and simulated events in 1985.



Note. "Uncomplicated AP" is defined as angina pectoris only, without a history of acute myocardial infarction. "IHD without CHF" describes all patients with a history of ischemic heart disease but without congestive heart failure. "All IHD" comprises all ischemic heart disease, including patients who developed congestive heart failure after ischemic heart disease. "All heart disease" summarizes all three conditions. The difference between all heart disease and all ischemic heart disease shows the prevalence of congestive heart failure.

FIGURE 3—Cumulative prevalences of heart disease, by age and sex, the Netherlands, 1985.

by an increase in survival.[3,4,8,9,26,51] In an extreme low-incidence-change scenario, we assume that the incidence decrease is even less: −0.5% per year. This is the magnitude of change predicted by a risk factor model, based on changes in risk factor prevalences in the Netherlands.[5] In an extreme high-incidence-change scenario, we assume a high decrease in incidence (−1.7% per year).

We have projected these trends over the period 1985 through 2010 to juxtapose the effects of demographic and epidemiologic change.

The results are summarized in Figures 4 through 6 and Table 3. Figure 4 shows how the decreasing incidence, together with the increase in survival, results in lower prevalence rates in the young and increasing prevalence rates in older age groups. In the youngest (30- to 39-year-old) age group, the decrease will be about 25%; in the oldest old (85 years and older), prevalence will increase by 20% for men and 10% for women. Superimposing the population projections on these figures results in a completely different picture, which is shown in the same figure. The movement of the postwar baby boom generation into middle age and the sharp increase in the number of the very elderly is clearly revealed. Together with the projected increase in prevalence rates, this demographic change will result in a steep climb in the number of heart disease patients in the oldest age groups. Hence, while epidemiological changes in cardiovascular disease will result in an increase of 10% to 20% in prevalence rates for the oldest old (85 years and older), the absolute number of patients will more than double.

Figures 5 and 6 show the projected changes in age-specific prevalences for different types of ischemic heart disease; Figure 5 shows the relative changes in rates and Figure 6 shows the absolute changes in numbers. The change in age-specific rates for all ischemic heart disease combined is the same as in Figure 4.

Figure 5 shows a relative increase of severe ischemic heart disease. Milder stages of the disease act as a risk factor for the more severe stages. If survival increases in all disease stages, effects will culminate in the most serious stages: as more patients stay alive with ischemic heart disease, more will have the opportunity to develop congestive heart failure. Figure 6 shows the changes in absolute terms: the relative decrease of prevalence at younger ages has hardly any impact because of the low number of patients with

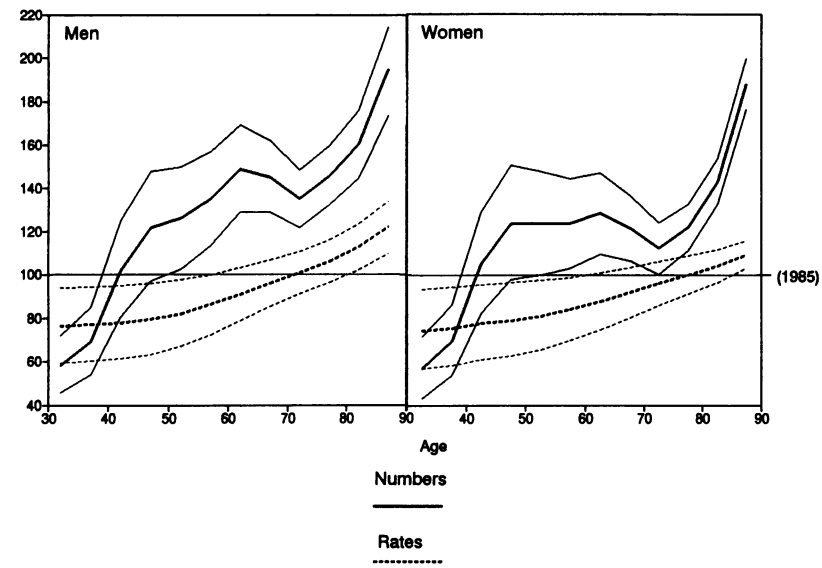
ischemic heart disease at early ages. However, the combination of aging and increasing survival of heart disease leads to a flood of middle-aged men with ischemic heart disease followed by a slower wave of elderly persons with heart failure.

Table 3 shows expected changes in age-standardized rates, using the European Standard Population as the standard, and in absolute numbers, combining the rates with the expected population dynamics in the Netherlands. The mortality decline (observed between 1975 and 1990 and projected over the next 20 years) is so strong that it will be evident even when expressed in absolute numbers. Although the assumed decline in incidence of first myocardial infarctions, which accounts for one third of the decline in mortality, is too small to counteract the aging of the Dutch population, the rate of all myocardial infarctions together will nevertheless decrease substantially. This decrease is the result of the combined decline in incidence and recurrence; the better survival, on the other hand, through which the number of patients at higher risk of recurrent infarctions is increased, is not strong enough to counteract this decline. Because more patients will stay alive and at risk for interventions and reinterventions and because of the aging of the population, the need for coronary artery bypass graft surgery and percutaneous transluminal coronary angioplasty will increase by 1% per year (range: 0.4%–1.6%). The age-standardized prevalence ratios for milder ischemic heart disease tend to decrease, but those for ischemic heart disease complicated by congestive heart failure increase strongly: in absolute numbers, a growth of more than 70% is to be expected.

TABLE 2—Annual Percentage Changes Postulated in Three Scenarios for the Period 1985 through 2010

	Men	Women
	Reference (Extremes)	Reference (Extremes)
Incidence	−1.1 (−1.7, −0.6)	−0.9 (−1.4, −0.5)
Mortality before admission	−1.2 (−0.9, −1.4)	−0.7 (−0.5, −0.8)
Mortality within first month after admission	−2.1 (−1.6, −2.5)	−1.3 (−1.0, −1.6)
Late mortality	−1.8 (−1.4, −2.1)	−1.8 (−1.4, −2.1)
Recurrence	−3.1 (−2.3, −4.7)	−1.9 (−1.4, −2.9)
Ischemic heart disease mortality	−2.4	−2.0

Note. It is assumed that the mortality decline observed between 1975 and 1990 remains equal in all scenarios.



Note. Dashed lines show the expected index of change in prevalence rates between 1985 and 2010 (prevalence in 2010 divided by prevalence in 1985 multiplied by 100). Solid lines indicate the change in absolute numbers, showing the force of demography. The heavier lines indicate the reference scenario; the thinner lines indicate the extreme scenarios.

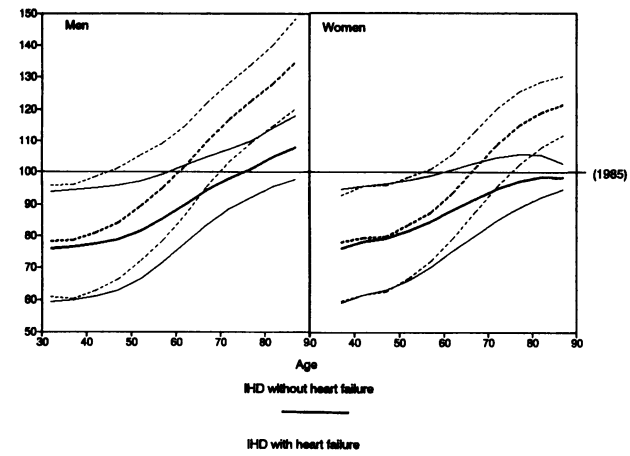
FIGURE 4—Expected changes in absolute and relative prevalence of patients with ischemic heart disease, the Netherlands, 2010 compared with 1985.

Discussion

Simulation modeling provides quantitative insight into epidemiological and public health dynamics and potential future developments. The information needed for these models can never be supplied by a single data source. Combining data from different sources is therefore appropriate, and the use of state-transition models provides a simple and comprehensible means of doing so. The methods used are relatively easy to understand. The parameters of the model are meaningful to physicians and the results are interpretable by health care providers, who are the principal users of these results.

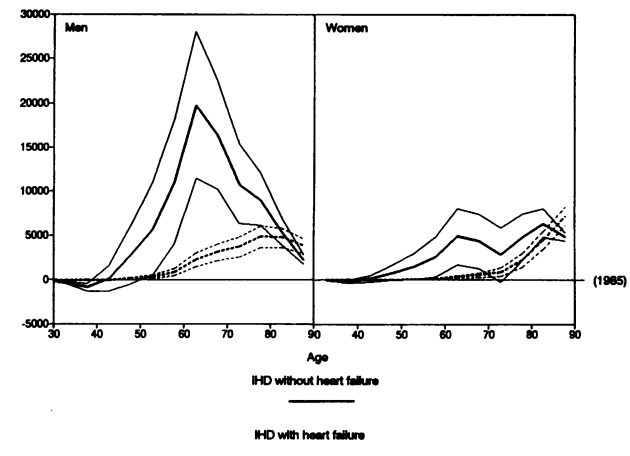
Nevertheless, to interpret results correctly, the limitations of this kind of modeling should be understood. A number of simplifying assumptions are always necessary to keep the model, and more particularly the number of parameters to be estimated, manageable. We included certain disease manifestations and excluded others. Unlike Weinstein et al., we excluded cardiac arrest as a prevalent state but included congestive heart failure. Cardiac arrest is highly lethal, and no major error is introduced by mixing the history of those surviving an acute myocardial infarction with that of a few survivors of cardiac arrest. On the other hand, the inclusion of congestive heart failure takes into account an important cause of chronic and severe morbidity. Modeling the dynamics of and the relations between these different types and levels of morbidity should result in a more realistic estimate of future health care needs.

We used registered admissions for acute myocardial infarction (ICD 410), deaths from ischemic heart disease (ICD 410–414) and congestive heart failure (ICD 428–429), and transition probabilities extracted from multiple sources to estimate incidences. This complicates the assessment of validity, which is the most important problem of this or any other model with such complexity. The model output reflects state-of-the-art clinical evidence, closely simulating present hospitalization, intervention, and mortality rates (see Fig-



Note. Solid lines indicate the relative changes in number of ischemic heart disease patients without heart failure; dashed lines indicate those with heart failure (age-specific prevalences in 2010 divided by the same prevalences in 1985 multiplied by 100). The heavier lines indicate the reference scenario; the thinner lines indicate the extreme scenarios.

FIGURE 5—Expected relative changes in prevalence rates of ischemic heart disease, the Netherlands, 2010 compared with 1985.



Note. Solid lines indicate the absolute changes in number of ischemic heart disease patients without heart failure; dashed lines indicate those with heart failure (age-specific numbers of patients in 2010 minus the same numbers in 1985). The heavier lines indicate the reference scenario; the thinner lines indicate the extreme scenarios.

FIGURE 6—Expected absolute changes in numbers of patients with ischemic heart disease, the Netherlands, 2010 compared with 1985.

TABLE 3—Age-Standardized Rates[a] and Absolute Numbers of Admissions, Interventions, Patients, and Deaths due to Ischemic Heart Disease (IHD) and Congestive Heart Failure (CHF) in the Netherlands, in 1985 and the Projected Index of Change (1985 = 100) in 2010

	1985		Index of Change in 2010 (1985 = 100)	
	Rate	Number	Rate	Number
Men				
ACE incidence	267	16 438	76 (65, 87)	114 (97, 130)
First admissions	214	13 150	80 (67, 93)	119 (100, 139)
Admissions for ACE	352	21 687	67 (60, 73)	100 (90, 108)
CABG	96	5 797	84 (72, 90)	128 (110, 147)
History of AP	1726	106 968	91 (79, 104)	136 (119, 156)
History of ACE	2087	129 149	90 (79, 102)	136 (118, 153)
CHF with history of IHD	575	35 761	118 (104, 131)	170 (149, 189)
CHF without history of IHD	697	41 323	94 (88, 126)	144 (133, 163)
Deaths coded AMI	194	12 045	54[b]	80[b]
Deaths coded chronic IHD or CHF	106	6 422	65[b]	96[b]
Women				
ACE incidence	109	9 507	80 (71, 89)	115 (105, 126)
First admissions	81	6 863	83 (73, 94)	118 (105, 131)
Admissions for ACE	122	10 529	73 (66, 78)	105 (96, 111)
CABG	18	1 302	84 (72, 97)	115 (98, 132)
History of AP	1088	94 635	89 (78, 99)	123 (110, 136)
History of ACE	547	45 195	94 (82, 106)	130 (114, 146)
CHF with history of IHD	284	29 251	115 (103, 125)	169 (153, 183)
CHF without history of IHD	509	50 729	84 (77, 104)	130 (120, 146)
Deaths coded AMI	82	7 889	63[b]	96[b]
Deaths coded chronic IHD or CHF	60	6 036	59[b]	90[b]

Note. The expected rates and numbers can be calculated by multiplying the figures for 1985 by the index of change in 2010 divided by 100. The upper and lower bounds (in parentheses) represent the results of scenarios assuming extremely high or low incidence changes but leading to the same mortality changes as a result of extremely low or high survival changes (see Table 2). ACE = acute coronary event; CABG = coronary artery bypass graft; AP = angina pectoris; AMI = acute myocardial infarction.

[a]Per 100 000; the European Standard Population is used as the standard.

[b]In the calculation of upper and lower bounds, the mortality decline is assumed to remain equal.

ure 2). Its estimated prevalences of clinical disease coincide closely with empirical observations.[49,50,52]

Our forecasts are based on mortality projections. For health care planning purposes, projections of incidence based on past risk factor prevalences are less reliable. First, prevalences of all known risk factors for heart disease are not available; prevalence numbers that are available are often highly inconsistent between studies. For example, estimates of the prevalence of hypertension in Dutchmen aged 50 through 59 years vary from 14.4% to 67%.[5,52] Second, not all risk factors are known or observable; projections based only on known risk factors are bound to underestimate future trends. The assumption that mortality will continue to decline is uncertain, but not implausible. It supposes a progressive change in risk factor distributions, a more widespread use of effective health care interventions in the future, and ongoing technological advances.[3,4,8,9] The level of ischemic heart disease mortality expected in 2010 is comparable to that of 1950 (making allowances for changes in codification). Given the absence of effective therapies in 1950, this is not an extreme result. The reference scenario supposes a decrease in incidence, explaining one third of the mortality decline, based on the Framingham study.[4] Other assumptions about incidence decreases and survival increases within extreme ranges are shown in the alternative

scenarios.[3,4,8] These assumptions influence the absolute magnitude of the expected changes, but not the direction.

Declining incidence rates mean that the onset of disease is postponed but not halted. The delay in occurrence causes a reduction of morbidity in the young, which, however, is canceled out with age. Mortality is deferred to an even greater extent, owing to a combination of the decrease in incidence and improved prognosis. This mortality decrease will be traded off for a steep increase of more severe morbidity in the old.

Changes in demography will prevent a decrease of new cases of heart disease.[13] However, while age-specific incidence rates show a decline, acute myocardial infarction rates will drop more steeply, owing to a reduction in both incidence and recurrence rates; the increasing prevalences are insufficient to cancel these reductions. The reference scenario predicts a decline in acute myocardial infarction rates that will be sufficiently strong to offset population aging; this development has been observed in the Netherlands since 1985.[19-21] But although disease incidence rates are declining, intervention rates will remain unchanged, even assuming that intervention rates per group of patients in a certain disease state remain constant. The reason is that more patients will survive and, on average, survival is longer, resulting in more people in disease states who are eligible for invasive coronary revascularization procedures.

The overall picture emerging from the heart disease scenarios is one of transition from an acute to a more chronic disease: event rates are declining steeply, but the postponed mortality is traded off for increased morbidity and severity of disease among the elderly. This pattern can be observed in hospital admission rates in the Netherlands, in data from the National Health Interview Survey in the United States, and in data from the National Health Survey of Japan.[19,53] All of these data show increasing prevalences of ischemic heart disease. As a consequence, overall health care needs will continue to rise, regardless of declining mortality and incidence.

These calculations, based on empirical observations, shed a new light on Fries' compression of morbidity theory.[54] The compression of morbidity theory hinges on the proposition that the incidence of disease will be postponed more rapidly than mortality. For heart disease, the evidence suggests that the decline of lethality expands the number of years spent with heart disease morbidity, regardless of the incidence postponement. Healthy life expectancy is extended, as expected by Fries and assumed by the scenarios, but contrary to his prediction, total life expectancy is extended even further. Among the middle-aged and elderly, acute heart disease lethality in the Netherlands is high, increasing sharply with age. Obviously, here would appear to be a serious potential for reduction. In 1985, for example, the model calculated that 41% of the 70-year-old men died within 4 weeks of their first acute coronary event.[20-24] In our scenarios 32% (range: 30%–34%) are predicted to die within the same period in the year 2010, which in our view remains a conservative estimate. Because heart disease is concentrated in the older age groups, even small differences in the rates of change in incidence and mortality can have major consequences, particularly now that pushing back mortality has been shown to be counterbalanced by an increase in the more severe stages of heart disease. With short-term case-fatality subsequent to an acute event as high as it is, lowering these rates will remain an important goal of health care intervention. Should this aim be successful, the resulting increase in the number of survivors will boost heart disease morbidity and lengthen life expectancy with disease. Only if health care interventions aimed at reducing mortality subsequent to the event should prove a waste of time and money would no expansion of morbidity be expected. □

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References

1. Koopmanschap MA, van Roijen L, Bonneux L. *Costs of Illness in the Netherlands* [in Dutch]. Rotterdam, the Netherlands: Department of Public Health and Social Medicine, Institute of Medical Technology Assessment; 1991. Report MGZ 91.03.
2. Uemura K, Pisa Z. Trends in cardiovascular disease mortality in industrialised countries. *World Health Stat Q.* 1988;41:155–168.
3. Sytkowsky PA, Kannel WB, D'Agostinho RB. Changes in risk factors and the decline in mortality from cardiovascular disease. *N Engl J Med.* 1990;322:1635–1641.
4. Sigfusson N, Sigvaldason H, Steingrimsdottir L, et al. Decline in ischemic heart disease in Iceland and change in risk factor levels. *BMJ.* 1991;79:366–371.
5. Gunning-Schepers L. The health benefits of prevention: a simulation approach. *Health Policy.* 1989;12/1–2 (special issue): 93–129.
6. Graboys TB, Biegelsen B, Lampert S, Blatt CM, Lown B. Results of a second-opinion trial among patients recommended for coronary angiography. *JAMA.* 1992; 268:2537–2540.
7. Mackenbach JP, Looman CWM, Kunst AE, Habbema JDF, van der Maas PJ. Post-1950 mortality trends and medical care: gains in life expectancy due to declines in mortality from conditions amenable to medical intervention in the Netherlands. *Soc Sci Med.* 1988;27:889–894.
8. Goldman L, Cook EF. The decline in ischemic heart disease mortality rates. *Ann Intern Med.* 1984;101:825–836.
9. Moss AJ, Benhorin J. Prognosis and management after a first myocardial infarction. *N Engl J Med.* 1990;322:743–753.
10. SOLVD Investigators. Effect of enalapril on survival in patients with reduced left ventricular ejection fractions and congestive heart failure. *N Engl J Med.* 1991;325: 293–302.
11. Cohn JN, Johnson G, Ziesche S, et al. A comparison of enalapril with hydralazine-isosorbide dinitrate in the treatment of chronic congestive heart failure. *N Engl J Med.* 1991;325:303–310.
12. Manton KG, Stallard E. *Chronic Disease Modelling.* London, England: Charles Griffins & Co; 1988:207–210.
13. Weinstein MC, Coxson PG, Williams LW, Pass TM, Stason WB, Goldman L. Forecasting coronary heart disease incidence, mortality and cost: the Coronary Heart Disease Policy Model. *Am J Public Health.* 1987;77:1417–1426.
14. *Monthly Population Statistics.* Voorburg, the Netherlands: Central Bureau of Statistics; 1986(3):20–28.
15. Smith WM. Epidemiology of congestive heart failure. *Am J Cardiol.* 1985;55:3A–8A.
16. *International Classification of Diseases, Injuries and Causes of Death.* 9th revision. Geneva, Switzerland: World Health Organization; 1977.
17. Kannel WB. Epidemiological aspects of heart failure. *Cardiol Clin.* 1989;7:1–9.
18. Press W, Flannery BP, Teukolsky SA, Vetterling T. *The Art of Scientific Computing.* Cambridge, England: Cambridge University Press; 1990.
19. Hoogendoorn DE. Atherosclerotic diseases of the heart in the hospital and in the vital statistics [in Dutch]. *Ned Tijdschr Geneeskd.* 1985;129:1827–1833.
20. Hoogendoorn D. Some notes on the current situation regarding the epidemic of acute myocardial infarction [in Dutch]. *Ned Tijdschr Geneeskd.* 1990;134:592–595.

21. *Files from the National Medical Registration concerning Admissions and Interventions in 1988.* Utrecht, the Netherlands: LMR (Information Center for Health Care), 1990.
22. Hoogendoorn D. Frequencies and operative mortality of interventions on the heart and the large intrathoracic vessels, I: direct revascularisation of the coronary arteries [in Dutch]. *Ned Tijdschr Geneeskd.* 1989; 133:2445–2448.
23. Fracheboud J. *Coronary Care or Home Care? A Descriptive Study of Home Care for Patients with an Acute Myocardial Infarction* [in Dutch]. Utrecht, the Netherlands: Nederlands Instituut Voor onderzoek van de Eerste Lijnsgezondheidszorg (NIVEL); 1987.
24. Lubsen J, van der Does E, Pool J. Incidence of acute myocardial infarction and sudden death. *Hart Bull.* 1976;7:107–113.
25. Goldman L, Cook F, Hashimoto B, Stone P, Muller J, Loscalzo A. Evidence that hospital care for acute myocardial infarction has not contributed to the decline in coronary mortality between 1973–74 and 1978–79. *Circulation.* 1982;65:936–942.
26. Ericsson CG, Erhardt L, Rehnqvist N. Two-year survival after myocardial infarction. *J Intern Med.* 1990;227:195–199.
27. Marcus FI, Friday K, McCans J, et al. Age related prognosis after acute myocardial infarction (the Multicenter Diltiazem Postinfarction Trial Research Group). *Am J Cardiol.* 1990;65:559–566.
28. Benhorin J, Moss AJ, Oakes D, and the Multicenter Diltiazem Postinfarction Trial Research Group. Prognostic significance of nonfatal myocardial reinfarction. *J Am Coll Cardiol.* 1990;15:253–258.
29. Multicenter Diltiazem Postinfarction Trial Research Group. The effect of diltiazem on mortality and reinfarction after myocardial infarction. *N Engl J Med.* 1988;319:385–392.
30. Norwegian Multicenter Study Group. Timolol induced reduction in mortality and reinfarction in patients surviving acute myocardial infarction. *N Engl J Med.* 1981; 304:801–807.
31. DeBusk RF, Kraemer HC, Nash E, Berger WE III, Lew H. Stepwise risk stratification soon after acute myocardial infarction. *Am J Cardiol.* 1983;52:1161–1166.
32. Kannel WB, Feinleib M. Natural history of angina pectoris in the Framingham study. *Am J Cardiol.* 1972;29:154–163.
33. DeBusk RF, Blomqvist G, Kouchoukos NT, et al. Identification and treatment of low-risk patients after acute myocardial infarction and coronary artery bypass graft surgery. *N Engl J Med.* 1986;314:161–166.
34. Ahnve S, Gilpin E, Dittrich H, et al. First myocardial infarction: age and ejection fraction identify a low-risk group. *Am Heart J.* 1988;116:925–932.
35. Sanz G, Castañer A, Betriu A, et al. Determinants of prognosis in survivors of myocardial infarction. *N Engl J Med.* 1982; 306:1065–1070.
36. European Coronary Study Group. Long term results of a prospective randomised study of coronary artery bypass surgery in stable angina pectoris. *Lancet.* 1982;ii: 1173–1180.
37. Coronary Artery Surgery Study principal investigators and associates. Coronary Artery Surgery Study: a randomized trial of coronary artery bypass surgery. *Circulation.* 1983;68:939–950.
38. Brook RH, Kosecoff JB, Park RE, Chassin MR, Winslow CM, Hampton JR. Diagnosis and treatment of coronary artery disease: comparisons of doctors' attitudes in the USA and the UK. *Lancet.* 1988;i:750–753.
39. Winslow CM, Kosecoff JB, Chassin M, Kanouse DE, Brook RH. The appropriateness of performing coronary artery bypass surgery. *JAMA.* 1988;260:505–509.
40. Weintraub WS, Jones EL, King SB, et al. Changing use of coronary angioplasty and coronary bypass surgery in the treatment of chronic coronary artery disease. *Am J Cardiol.* 1990;65:183–188.
41. Kaul S, Lilly DR, Gascho JA, et al. Prognostic utility of the exercise thallium-201 test in ambulatory patients with chest pain: comparison with cardiac catheterization. *Circulation.* 1988;77:745–758.
42. Parisi AF, Peduzzi P, Detre K, et al. Characteristics and outcome of medical nonadherers in the Veterans Administration cooperative study of coronary artery surgery. *Am J Cardiol.* 1984;53:23–28.
43. Veldkamp RF, Baartman GJ, van Domburg R, Thijssen JGP, Bos E, Meeter K. Survival 11 years after an aortocoronary bypass operation [in Dutch]. *Ned Tijdschr Geneeskd.* 1991;135:1229–1233.
44. Glazier JJ, Variccicchione TR, Ryan TJ, et al. Outcome in patients with recurrent stenosis after percutaneous transluminal balloon angioplasty. *Br Heart J.* 1989;61:485–488.
45. Fioretti P, Tijssen JGP, Azar AJ, et al. Prognostic value of predischarge 12 lead ECG after myocardial infarction compared with other routine variables. *Br Heart J.* 1987;57:306–312.
46. Laird-Meeter K, van Domburg R, van den Brand MJBM, Lubsen J, Bos E, Hugenholtz PG. Repeat interventions after initial aorto-coronary bypass surgery and its results [in Dutch]. *Ned Tijdschr Geneeskd.* 1988;132:2316–2320.
47. Detre K, Holubkow R, Kelsey S, et al. One year follow-up results of the 1985–1986 National Heart, Lung, and Blood Institute's PTCA registry. *Circulation.* 1989;80:421–428.
48. Epstein SE, Palmeri ST, Patterson RE. Evaluation of patients after acute myocardial infarction. *N Engl J Med.* 1982;307: 1487–1492.
49. Anonymous. Epidemiology of chest pain and angina pectoris, with special references to treatment needs. *Acta Med Scand Suppl.* 1984;682:1–120.
50. Reunanen A, Aroma A, Pyörälä K, Punsar S, Maatela J, Knekt P. The social insurance institution's coronary heart disease study. *Acta Med Scand Suppl.* 1983;673:1–120.
51. de Cock CC, Visser FC, van Eenige MJ. Prognosis after an infarction: short- and long-term follow-up data [in Dutch]. *Ned Tijdsch Cardiol.* 1991;5:96–104.
52. *Morbidity Figures from General Practice.* Nijmegen, the Netherlands: Nijmeegs Universitair Huisartsen Instituut; 1985.
53. Uemura K. International trends in cardiovascular diseases in the elderly. *Eur Heart J.* 1988;9(suppl D):1–8.
54. Fries JF. Aging, natural death and the compression of morbidity. *N Engl J Med.* 1980; 303:130–135.