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Chapter Author: David M. Cutler, Mary Beth Landrum, Kate A. Stewart

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Intensive Medical Care and Cardiovascular Disease Disability Reductions

David M. Cutler, Mary Beth Landrum, and Kate A. Stewart

Disability among the elderly has declined markedly in the United States in the past two decades. In 1984, 25 percent of the elderly population reported difficulty with activities associated with independent living.¹ By 2004 and 2005, the share had fallen to below 20 percent, a decline of one fifth.

Although these basic facts are well known, the interpretation of these facts is not clear. Is the reduction in disability a result of improved medical care, individual behavioral changes, or environmental modifications that allow the elderly to better function by themselves? Will the trend continue, or is it time limited? What does the reduction in disability mean for years of healthy life and labor force participation? We explore these issues in this chapter.

To make progress, we focus on disability caused by a specific set of medical conditions—cardiovascular disease. Focusing on one condition is helpful because it allows us to analyze health shocks and their sequelae in some detail. Cardiovascular disease (CVD) is a natural condition to pick because it is the most common cause of death in the U.S. (and most other developed

David M. Cutler is the Otto Eckstein Professor of Applied Economics and Dean for the Social Sciences at Harvard University and an affiliate of the National Bureau of Economic Research. Mary Beth Landrum is an associate professor of Biostatistics in the Department of Health Care Policy at Harvard Medical School. Kate A. Stewart is a researcher at Mathematica Policy Research, Inc., and was a Ph.D. candidate in Health Policy at Harvard Medical School when this research was completed.

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1. The data are from the National Long-Term Care Survey, a survey we describe later and use in this chapter.

countries), and more is spent on cardiovascular disease than any other condition. Thus, this is a case where medical care could really matter.

Our analysis has three parts. In the first part, we examine basic trends in disability associated with cardiovascular disease. We show that reduced disability for people with cardiovascular disease incidents is a major part of reductions in overall disability, accounting for between one fifth and one third of the total reduction in disability. The second part of the chapter considers the role of advances in medical care in reducing disability from cardiovascular disease. We show that medical technology in the treatment of cardiovascular disease is a major factor in reduced disability. We estimate that use of recommended treatments for heart attacks, including prescriptions of beta-blockers, aspirin, and ace-inhibitors at discharge, as well as use of reperfusion and other surgical procedures may have increased the probability that elderly patients survive an acute cardiovascular event in a nondisabled state by up to 50 percent between 1984 and 1994. The third part of the chapter considers the long-run health and financial impacts of improved care for people with cardiovascular disease.

6.1 Background on Cardiovascular Disease

Cardiovascular diseases are diseases of the heart and blood vessels, which carry oxygen to the body's major organs. Ischemic heart disease is the most common manifestation of cardiovascular disease. When the arteries supplying blood to the heart become occluded, the heart does not get enough oxygen. Like any muscle, oxygen is essential for the heart's performance. Constriction of the coronary arteries will result in chest pain on exertion, or perhaps at rest. A person with such constriction might be unable to engage in activities such as walking for a prolonged period (for example, to get to a grocery store) or engaging in light or heavy housework (cleaning, cooking, etc.)

A blockage of the arteries to some or all of the heart is termed a *my*ocardial infarction, or heart attack. The equivalent in other extremities, especially the legs, is termed peripheral vascular disease. Heart attacks can be fatal and can lead to substantial disability if survived. A person who survives a heart attack might be unable to shop or cook, might have difficulty walking up stairs or entering a raised bathtub, and might have difficulty keeping house. Peripheral vascular disease can lead to the same types of impairments.

Medical advances have made tremendous strides in preventing and treating coronary events. Several risk factors for heart disease are well known. Traditional risk factors include smoking, hypertension or high blood pressure, high cholesterol, obesity, family history, age, and diabetes. Since the early 1970s, standard recommendations for people at risk have been behavioral changes (stop smoking, reduce weight, cut back on fat intake, and exercise) combined with medical therapy (antihypertensive medication, and more recently, cholesterol-reducing medications). Cutler and Kadiyala (2003) show significant reductions in the incidence of heart disease over time attributable to reductions in these risk factors, especially reduced smoking and better blood pressure control.

There have also been technological changes in the treatment available for people with severe heart disease. Bed rest was once standard therapy for people with heart attacks. Today, therapy for a heart attack—and often heart disease in earlier stages of progression—generally starts with drugs such as aspirin, which help dissolve clots and restore blood flow to the heart. Beta-blockers are also given to reduce the workload of the heart and thus reduce the demand for oxygen. In addition, ace-inhibitors are prescribed to help reduce the workload of the heart by lowering blood pressure. Statins are prescribed to help process and break down cholesterol in the arteries (American Heart Association 2006).

Finally, there have been significant advances in acute care and invasive surgical procedures for treating coronary blockage. Thrombolytics are a class of drugs that may be used to help dissolve the clot. Percutaneous coronary intervention (PCI) is used to clear out blockages of the coronary arteries. These procedures are now frequently accompanied by use of a stent to keep the occluded artery open. A more invasive option is coronary artery bypass grafting (abbreviated CABG, and pronounced like the vegetable).

Each of these technologies has been shown to increase survival after a heart attack among patients without contraindications for treatment (Krumholz et al. 1995; Hennekens et al. 1996; Krumholz et al. 1996; Soumerai et al. 1997; Gottlieb, McCarter, and Vogel 1998; Krumholz et al. 1998; Freemantle et al. 1999; Shlipak et al. 2001; Braunwald et al. 2002; Antman et al. 2004; Vitagliano et al. 2004; Stukel, Lucas, and Wennberg 2005). They have an ambiguous effect on disability, however, with the increase in survival among those with serious heart damage possibly offsetting the improved health among traditional survivors (Crimmins, Saito, and Ingegneri 1989; Crimmins, Hayward, and Saito 1994; Waidmann, Bound, and Schoenbaum 1995).

Cerebrovascular disease, or stroke, is the second major form of cardiovascular disease. Ischemic strokes are the most common type of stroke and are similar to heart disease: an artery in the brain becomes blocked, and a part of the brain is denied oxygen. Disability is quite common after a stroke, particularly among the elderly (Pohjasvaara et al. 1997; Prencipe et al. 1997; Zhu et al. 1998). Recent studies report that 39 percent to 54 percent of stroke survivors are disabled three months after the stroke (Henon et al. 1995; Zhu et al. 1998; Glader et al. 2003). The high level of disability can persist among survivors. One study found that 37 percent of stroke survivors were disabled one year after the event (Appelros, Nydevik, and Viitanen 2003). In addition, stroke is associated with increased odds of cognitive impairment, both with and without dementia (Pohjasvaara et al. 1997; Prencipe et al. 1997; Zhu et al. 1998).

Thrombolytic medication may be given after a stroke, but the benefit is far less certain than in heart disease. Clinical trials show that thrombolytics are effective only if given in the first three hours after an acute event (National Institute of Neurological Disorders, 1995; Clark et al. 1999; Adams et al. 2005). Revascularization procedures such as carotid endarterectomy may be performed in patients with certain types of stroke—including transient ischemic attacks—after the patients have recovered from the acute phase of the stroke. A small share of strokes are hemorrhagic strokes, where a blood vessel bursts and there is bleeding in the skull. Little therapy is generally available in such cases, and death is common.

Heart failure and arrhythmias are other types of cardiovascular disease that cause substantial morbidity and mortality among the elderly. Heart failure occurs when the heart's ability to pump blood is impaired. Patients may experience breathlessness and fatigue that makes it difficult to keep up usual activities, fluid retention and edema, coughing, memory loss, and heart palpitations. An arrhythmia is an irregular heartbeat that can cause the heart to pump blood less effectively. Patients with heart failure and/or arrhythmias may also be at substantial risk for stroke and other complications (American Heart Association 2006). Treatment for heart failure includes ace-inhibitors or angiotensin II receptor blockers, beta-blockers, and diuretics. Appropriate patients may also undergo valve replacement surgery or other revascularization. Patients with arrhythmias often receive pacemakers and occasionally receive implantable cardioverter defibrillators along with antiarrhythmic drugs and blood thinners.

Other cardiovascular diseases, generally with smaller prevalence, include rheumatic heart disease, aneurysms, acute pulmonary heart disease, other diseases of the endocardium, capillary diseases, and problems with veins (e.g., varicose veins).

6.2 The Importance of Cardiovascular Disease for Reductions in Disability

Like every multidimensional concept, there is no perfect measure of disability. We follow the lead of most researchers in measuring disability as the presence of impairments in Activities of Daily Living (ADLs) and Instrumental Activities of Daily Living (IADLs). Our data source, the National Long-Term Care Survey of 1984–1999 (NLTCS) includes information on six ADL measures: eating, getting in or out of bed, walking around inside, dressing, bathing, and getting to the toilet or using the toilet. Questions are also asked about eight IADL measures: doing light housework, laundry, preparing meals, shopping for groceries, getting around outside, managing money, taking medications, and making telephone calls. The NLTCS is a nationally representative longitudinal survey of the health and disability profile population aged sixty-five and over. The first NLTCS survey wave was conducted in 1982 and subsequent surveys were administered in 1984, 1989, 1994, and 1999. Each survey wave began with a screener that collected information on whether the respondent reported inability to conduct the six ADLs and eight IADLs without help (i.e., help from another person or special equipment), and whether these limitations had lasted or was expected to last at least three months. The screener also collected demographic information on marital status, race, and age.

Respondents who reported inability to perform any ADLs or IADLs for at least three months on the screener were asked to complete a detailed survey. Disability status was determined by responses to questions about use of help and inability to conduct the ADLs and IADLs on the detailed survey. Sampling and weighting issues are described fully elsewhere (Manton, Corder, and Stallard 1993; Manton, Corder, and Stallard 1997; Manton, Stallard, and Corder 1997; Singer and Manton 1998; Manton and Gu 2001).

We obtained Medicare-linked data for all NLTCS participants, including data on date of death from the denominator files. We used inpatient claims for all analyses because Medicare claims files for Part B and other nonhospital services were incomplete prior to 1991. We also obtained data on zip code of residence at the most recent interview for all NLTCS survey respondents.

Basic data on disability among the elderly population is shown in figure 6.1. For reasons that will become clear shortly, we report disability for the population that is aged seventy and older in each of three years: 1989, 1994, and 1999. The share of the elderly population that is disabled declined



Fig. 6.1 Share of population 70+ who are disabled

Note: Disability is defined as any difficulty conducting Activities of Daily Living or Instrumental Activities of Daily Living without help.

markedly in the 1990s. The cumulative decline was 6.3 percentage points, or 2.1 percent per year.

To examine the role of cardiovascular disease in explaining this reduction in disability, we form a population sample likely affected by the condition. We start by looking at the population aged seventy and older in the 1989, 1994, and 1999 surveys. For each of these cohorts, we group all hospitalizations over the preceding five years into one of thirty-two categories (the five-year look back is the reason for the restriction to people over seventy). These categories were designed to pick up relatively homogenous clinical conditions that would be predictive of disability. The set of thirtytwo categories is shown in table 6.1, along with the rate of disability for people hospitalized with each condition from the 1989 survey cohort. The relevant categories for cardiovascular disease are stroke, hypertension, ischemic heart disease, heart failure and arrhythmia, peripheral vascular disease, and other circulatory diseases.²

Figure 6.2 reports the share of people with a hospitalization for any of these conditions. Twenty-two percent of people were admitted to a hospital with some cardiovascular disease. Ischemic heart disease is the most common admission. Stroke and heart failure are also common, as are other circulatory diseases. Admissions for peripheral vascular disease and hypertension are much less common.

A person who had a hospital admission for cardiovascular disease and is disabled may or may not have been disabled because of that condition. The NLTCS does not reliably determine the precise condition that leads to each disability. We make two alternative assumptions about the probability of being disabled by cardiovascular disease. The first assumption, a less restrictive assumption, labels someone as disabled from cardiovascular disease if he or she was admitted to a hospital with cardiovascular disease in the previous five years. The more restrictive assumption subsets this group to those for whom the most disabling condition was cardiovascular disease,³ where the list of conditions by disability status is reported in table 6.1. Thus, a person who had a stroke and hip fracture would be termed disabled because of cardiovascular disease by the first measure, but not by the second measure. Fortunately, our results are very similar regardless of the definition used.

Figure 6.3 shows the probability of being disabled by cardiovascular dis-

3. Note even this more restrictive assumption may underestimate the importance of disabling diseases, such as arthritis or dementia, that are not common causes of hospitalizations.

^{2.} ICD-9 codes are as follows: stroke: 362.34, 430, 431, 432.9, 433–436; hypertension: 401–402, 405, 437.0, 437.9; ischemic heart disease; 4.10–4.14, 429.5–429.7, excluding 414.11 and 414.19; heart failure and arrhythmia: 425, 427.1, 427.3–427.5, 428, 429.1, 429.3; peripheral vascular disease: 440, 442, 443.0–443.1, 443.8–443.9, 444, 46, 447.0–447.5, 447.8–447.9, 451, 453.1; circulatory disease: 391–400, 406–409, 414.11, 414.19, 415–424, 426, 427.2, 427.6, 427.8, 427.9, 429.2, 429.4, 429.8, 429.9, 432.1–432.8, 437.1–437.8, 439, 441, 443.2, 445, 447.6, 448–450, 452–453.0, 453.2–459, 786.5, V717.

Condition	% disabled	Rank
Chronic renal failure	88.9	1
Dementia and organic brain diseases	83.1	2
Paralysis, Parkinson's, etc.	82.5	3
Hip and pelvic fracture	80.2	4
Acute renal failure and insufficiency	68.1	5
Other metabolic and immunity disorders	67.0	6
Other blood diseases	66.5	7
Respiratory failure and insufficiency	66.5	8
Anemia	65.9	9
Diabetes	64.1	10
Thyroid disorders	59.9	11
Stroke	59.5	12
Infectious diseases	59.2	13
Respiratory diseases	56.8	14
Depression	56.2	15
Peripheral vascular disease	55.4	16
Composite category	54.8	17
Musculoskeletal disorders	53.2	18
Heart failure and arrhythmia	51.5	19
Chronic obstructive pulmonary diseases and related diseases	51.0	20
Other mental disorders	49.9	21
Glaucoma and cataract	48.8	22
Hypertension	48.4	23
Gastrointestinal disease	45.2	24
Arthritis and arthropathy	45.0	25
Circulatory diseases	42.0	26
Colorectal and lung cancer	40.0	27
Back/neck pain	39.2	28
Ischemic heart disease	38.7	29
Genitourinary diseases	38.7	30
Other cancers	37.6	31
Breast and prostate cancer	28.9	32

Table 6.1	Most disabling o	conditions
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Notes: Analyses conducted using 1989 cohort, and based on primary hospitalization diagnosis codes in the proceeding five years. Composite category includes hospitalizations for any diagnoses other than the thirty-one specific categories above.

ease. Using the less restrictive measure, the decline in disability is 1.4 percentage points, or 22 percent of the 6.3 percentage point total reduction in disability. Using the more restrictive measure, the decline is 0.9 percentage points, or 14 percent of the total decline. In each case, cardiovascular disease is a substantial share of the total decline.

As previously noted, the conclusion that cardiovascular disease is a substantial share of disability decline contradicts an earlier literature that suggested that marginal survivors contribute to an increase in disability. If more people survive strokes, the argument went, the share of the elderly









Fig. 6.3 Probability of being disabled because of cardiovascular disease

Note: Cross-sectional analyses based on hospitalizations in the five years prior to the survey.

with disabilities would rise. The finding of a reduction in disability suggests, in contrast, two other hypotheses: either fewer people are suffering cardiovascular disease events, or those who have always survived such events are less disabled now than they were formerly (i.e., the incidence of disability among cardiovascular disease patients is falling). These possible effects are demonstrated in equation (1):

(1) Pr (Disabled from CVD) = Pr (Had CVD Incident)
 × Pr (Survive | Had Event)
 × Pr (Disabled | Had Event, Survival)

The first term on the right hand side is the incidence of events. The second term is the survival rate, and the third term is the health effect among sur-

vivors. The change in disability rates is arithmetically related to the change in one or more of these factors.⁴ The marginal survivors theory focuses on the second term: the change in the probability of survival after an acute event (i.e., as more patients survive acute events, the pool of people at risk of disability expands). The other theories focus on the first and third terms (i.e., either reduced incidence of disease or better health among survivors).

Table 6.2 shows cardiovascular disease event probabilities, survival rates, and conditional disability rates for each of our three time periods. To measure the cardiovascular disease event rate, we consider the population surveyed at the beginning of the five year interval, and look at events in those five years. For example, the cardiovascular disease event rate for the 1989 cohort is the share of the population aged sixty-five and older in 1984 that had a cardiovascular disease hospital admission in the subsequent five years.⁵

The first row shows that the share of people who had a hospitalization for cardiovascular disease was relatively constant over the time period, at about 26 percent. This is somewhat surprising given the reduction in event rates noted in other surveys such as the Framingham Heart Study (Sytkowski et al. 1996) and the Minnesota Heart Survey (McGovern et al. 1996; McGovern et al. 2001). It may be that some of the admissions among the later cohorts in our study were done explicitly to perform surgical operations such as angioplasty or bypass surgery, and thus contribute to an increased reporting of cardiovascular disease. However, another recent U.S. study of subjects aged thirty-five to seventy-four reported little change in the incidence of first myocardial infarction between 1987 and 1994 (Rosamond et al. 1998). Alternatively, it may be that less severe cases of these conditions are being diagnosed over time.⁶

In addition, the Framingham and Minnesota studies included patients younger than sixty-five, and results from these studies may have been driven by a decline in heart disease among the younger population. A Finnish study of coronary heart disease between 1978–1980 and 2000–2001 reported decreased prevalence of coronary heart disease among men and women aged forty-five to sixty-four, no change in prevalence among men and women aged sixty-five to seventy-four, and increased incidence

6. Clinical trials published in 1996 showed that a blood test for troponin, a protein released from damaged heart tissue, can be used to diagnose heart attacks. This likely led to greater diagnosis of smaller heart attacks and may also have led to increased hospitalizations, depending on how frequently these patients were previously admitted.

^{4.} There are covariance terms as well, but these are generally small.

^{5.} Were the NLTCS a fixed panel survey, the sample of people at the starting year (e.g., 1984) would be only those for whom the disability status is known five years later plus those who died in the interim. The NLTCS did not interview the entire sample every year, however, so some people are lost to follow-up. To generate a nationally representative sample, we analyzed only those people whose health and mortality status were known at follow-up for each survey year, and reweighted the sample weights to reflect the age-sex distribution of all respondents to the 1999 survey.

		Cl		
	1984–1989	1989–1994	1994–1999	to 1994–99 (%)
Share with CVD event	26.5	29.0	26.3	-0.2
Share with an event who survive Share of survivors who are disabled	57.2 47.6	58.4 43.7	61.5 39.4	4.3 -8.2

Table 6.2 Decomposing changes in cardiovascular disease disability

Source: Authors tabulations from the National Long-Term Care Survey.

Note: Data are based on respondents with a CVD hospitalization in between survey waves, and known health status at the second survey. Weights were adjusted to the age and sex distribution of the 1999 NLTCS survey population, and account for unknown follow-up status at the second survey.

among people aged seventy-five and over (Kattainen et al. 2004). Our results may reflect similar trends in age-related incidence of heart disease.

The second row of the table shows a significant increase in the survival rate to the next survey for people admitted to a hospital with cardiovascular disease. The survival rate increased by 4.3 percentage points, or about 7.5 percent. By itself, this would have led to an increase in disability from cardiovascular disease. This effect is overwhelmed, however, by the substantial reduction in disability among survivors, shown in the third row of the table. The share of CVD survivors who are disabled fell from 48 percent in 1989 to 39 percent in 1999, a 19 percent reduction. It is this massive reduction in event-specific disability that needs to be explained.

6.3 Medical Care and CVD-Related Disability

Before estimating formal statistical models to address the role of medical care in reduced CVD-related disability, we consider a less structural analysis of the role of medical care. Specifically, we look at how disability changed in the period shortly after the cardiovascular disease event relative to the period several years later. If the reduction in disability followed immediately after the cardiovascular event, it strongly suggests that medical treatment of the acute event was the major factor responsible for the reduction in disability. A future disability reduction might be attributable to medical intervention, but other factors, such as better coping with limitations due to improved environmental factors, could be important as well.

Figure 6.4 shows the change in disability rates for people whose cardiovascular disease event happened within six months of the survey, by type of event. The rate of disability declined from 1984–1989 cohort to the 1994–1999 cohort for people with hospitalizations for ischemic heart disease, heart failure and arrhythmia, stroke, and other cardiovascular disease. For heart failure and arrhythmia patients as well as other cardiovascular diseases, there were increases in the disability rate from the



Fig. 6.4 Likelihood of reporting disability among respondents with specific cardiovascular disease events in the six months prior to survey

1984–1989 to 1989–1994 cohorts that need further explanation.⁷ Overall, though, it seems that medical advances could have some role in this decline in disability between 1984–1989 and 1994–1999.

6.3.1 Empirical Methodology

To examine the role of medical technology changes in event-related reductions in disability more formally, we estimate regression models for the health of patients who have been admitted to a hospital with cardiovascular disease. Our sample is formed from each of the three cohorts. We select people who were admitted to a hospital with cardiovascular disease in 1984–1989, 1989–1994, and 1994–1999. In each case, the sample includes all people for whom we know health status at the beginning and end of the five-year period. There are three possible health states at the end of each period, that is, at the time of follow-up survey: dead, alive and nondisabled, and alive and disabled. We denote these possible outcomes with the subscript k.

Consider for the moment a single measure of medical treatments, which we wish to relate to the change in disability. For concreteness, assume that the variable is the share of people in an area who receive a surgical proce-

^{7.} For example, the average severity of heart failure hospitalizations may have increased over time if physicians and patients were better able to manage heart failure in the outpatient setting.

dure that has been shown to be effective in improving health for people with that condition. The treatment rate for area *j* in time period *t* is denoted $T_{j,t}$. We model the probability that person *i* in period *t* will be in health state *k* using a multinomial logit formulation:

(2) Pr (health state k)_{*i,j,t*} =
$$\frac{\exp(\mathbf{X}_{i,t}\beta_k + \gamma_k \mathbf{T}_{j,t} + \Delta_k \operatorname{cohort}_t)}{(1 + \exp(\Sigma \mathbf{X}_{i,t}\beta_1 + \gamma_1 \mathbf{T}_{j,t} + \Delta_1 \operatorname{cohort}_t)}$$

where $\mathbf{X}_{i,t}$ is a set of demographic and baseline health variables, and γ_k s are the coefficient of interest. Our control variables include: age/sex (five-year age groups differentiated by gender); disability status at the baseline interview; dummy variables for other cardiovascular disease hospitalizations (with the exception of the all-CVD model, which does not require indicator variables for other CVD diagnoses); a modified Charlson index (i.e., without cardiovascular disease diagnoses) (Deyo, Cherkin, and Ciol 1992); marital status at the beginning of the five-year window (married, widowed, divorced/separated, or missing marital status); race (white and nonwhite); and zip code-level education and poverty measures (respondents' education and income were only available from the detailed interview).

One issue that comes up in any estimation involving an equation like (2) is the issue of causality. If treatments are not randomly assigned, estimates of γ will be biased. We address this issue in several ways. The most important is to use area-level variation in treatments, rather than individual-level variation. Whether any individual receives a treatment is dependent on the physician's perception of that patient's underlying health. If the underlying severity of disease is relatively constant across areas and over time, however, variations in treatment at the area level will be good markers for exogenous changes in the use of medical care. As is standard in the literature (O'Connor et al. 1999; Fisher et al. 2003a; Fisher et al. 2003b; Stukel, Lucas, and Wennberg 2005), we group individuals into areas based on the Hospital Referral Region (HRR) they live in. Hospital Referral Regions are groups of zip codes where the bulk of patients go to the same set of hospitals and include at least one hospital with a tertiary cardiovascular or neurological surgical center. For example, the HRR for Chicago includes zip codes 60601–60712; within this area, the vast majority of people who are hospitalized get admitted to a hospital in that region.

6.3.2 Measures of Medical Treatment

We use several measures of medical care to predict disability. The first variable is the share of people who receive surgical interventions. To define relevant procedures, we identify treatments for each specific diagnosis that the medical literature has identified as being efficacious (generally in reducing mortality) for at least some subsets of patients with that diagnosis. These procedures are detailed in table 6.3. For hypertension, there are no

Condition	Appropriate procedures (CPT-4 Code)		
Stroke	Incision, excision, and occlusion of vessels (38)		
Hypertension	_		
Ischemic heart disease	Operations on vessels of heart (36)		
	Other operations on heart and pericardium (37)		
Heart failure and arrhythmia	Other operations on heart and pericardium (37)		
	Conversion of cardiac rhythm (99)		
Peripheral vascular disease	Incision, excision, and occlusion of vessels (38)		
	Operations on vessels (39)		
	Other procedures on musculoskeletal system (84)		
Other circulatory diseases	Operations on valves and septa of heart (35)		
	Other operations on heart and pericardium (37)		
	Incision, excision, and occlusion of vessels (38)		

1 able 6.3 Relevant procedures for cardiovascular disease admissio	Table 6.3	Relevant pr	ocedures for	cardiovascular	disease	admission
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generally accepted surgical therapies. There are one or more therapies for the other conditions, of which the most common appropriate procedures are "other operations on heart and pericardium" (CPT code 37), which includes PCI (angioplasty), heart replacement procedures and insertion of pacemakers, and "incision, excision and occlusion of vessels" (CPT code 38), which includes endarterectomies. CABG procedures for ischemic heart disease patients are coded under CPT code 36, "operations on vessels of heart."

Table 6.4 shows the average rate of procedure use over time across hospital referral regions for all cardiovascular disease patients and by specific conditions. In the 1984–1989 cohort, the average procedure rate was only 21 percent across regions. The average procedure rate was highest for other circulatory diseases (30 percent), followed by ischemic heart disease (23 percent), stroke (13 percent) and heart failure and arrhythmia (11.3 percent). By 1994–1999, the average procedure rate for all patients across all regions increased to 34 percent. The average procedure rate for ischemic heart disease patients jumped to 48 percent. Average procedure rates increased to 43 percent for other circulatory diseases, 25 percent for stroke, and 14.2 percent for heart failure and arrhythmia. These increases reflect the greater belief among physicians about the efficacy of therapy, and advances in the therapy itself.⁸

Our other measures of medical technology involve use of pharmaceuticals for patients with acute myocardial infarction. As noted in the previous section, these pharmaceuticals have been shown to improve survival, although the overall effect of pharmaceutical treatment on both improved survival and disability in the elderly has not been well established. Ran-

^{8.} For example, catheters used in surgery have improved, and stents were developed for use in angioplasty in the mid-1990s.

include of interaction of the second se	in the second		
Measure	1984–1989	1989–1994	1994–1999
Share of people receiving relevant			
procedure, % (SD)	21.1 (0.4)	26.1 (0.5)	34.2 (0.6)
By specific conditions			
Ischemic Heart Disease	22.6 (0.9)	33.2 (1.0)	47.9 (1.3)
Stroke	13.0 (0.8)	17.1 (0.9)	24.8 (1.0)
Heart Failure	11.3 (0.6)	11.9 (0.8)	14.2 (0.9)
Other Circulatory Diseases	29.7 (1.1)	32.5 (1.2)	43.0 (1.3)
Beta-blockers		50.7 (1.0)	_
Reperfusion		67.1 (0.8)	
Aspirin		76.4 (0.6)	
Ace-inhibitors		60.0 (0.6)	

Average rates of procedures and pharmaceuticals

Table 64

Note: Based on respondents with a CVD hospitalization between survey waves. The procedure figures are the averages across HRRs, consistent with the CCP data.

domized studies comparing various treatments for ischemic heart disease on functional status and quality of life reported improvements for most outcome measures for both medical and surgical therapies (Rogers et al. 1990; Strauss et al. 1995; Hlatky et al. 1997; Pocock et al. 2000; Borkon et al. 2002; Pfisterer et al. 2003). However, most of these studies included patients under age sixty-five who may be more likely to improve than elderly patients (Rogers et al. 1990; Strauss et al. 1995; Hlatky et al. 1997; Pocock et al. 2000; Borkon et al. 2002). A recent trial comparing medical and surgical management of elderly patients with coronary artery disease reported improved quality of life at one year for both treatment arms (Pfisterer et al. 2003), suggesting that treatment likely reduces disability in the elderly population. However, no studies to date have estimated the effect of increased use of appropriate pharmaceutical treatments over time on disability rates in the elderly population.

Pharmaceutical use is not captured in Medicare claims. Thus, we do not have time series data on the use of pharmaceuticals by area. We do have a snapshot of data on pharmaceutical use, taken from a survey of medical records in the mid-1990s. The Cooperative Cardiovascular Project (CCP) abstracted medical record data on 186,800 Medicare patients hospitalized for an AMI between February 1994 and July 1995, including data on appropriateness for and receipt of guideline-recommended treatments (Marciniak et al. 1998).

Use among patients most suited for treatment ranged from 51 percent for beta-blockers in the immediate postmyocardial infarction treatment to 76 percent for aspirin. Average utilization rates for the mid-1990s are shown in table 6.4. Researchers at Dartmouth have calculated the average use rate of each of these pharmaceuticals at the HRR level, which we employ in our analysis (O'Connor et al. 1999). While not known at the area level, use of these pharmaceuticals did increase during our study time frame. Reported use of aspirin for heart attack patients in 1985 and between 1998 and 2000 was 30 percent and 85 percent, respectively, and over the same time, use of beta-blockers was 48 percent and 72 percent, thrombolytics was 9 percent and approximately 80 percent, and ace-inhibitors was 0 percent and 71 percent, respectively (Jencks et al. 2000; Heidenreich and McClellan 2001; Vaccarino et al. 2005). The change in ace-inhibitors use from this study was based only on changes in the Worcester, Massachusetts area and may not reflect changes in use nationally.

The lack of time series data on pharmaceutical use at the area level requires us to modify our analyses. Equation (2) assumes that we have timevarying data on procedures and pharmaceuticals. Since we can only assign patients to true area levels for pharmacological treatments in 1994–1995, we estimated a model with all CCP variables on the 1994–1999 cohort only. We estimate a separate model using the panel data and time-varying procedures variables based on equation (2).

From our models, we estimate the likelihood of being disabled, dead, alive, and nondisabled at follow-up if all respondents lived in HRRs that provided relevant procedures and pharmaceuticals from the 10th to the 90th percentiles of care, holding all other covariates at their observed levels. We further estimate how much of the change in disability and death over time may be explained by increased use of appropriate treatments that were significantly associated with lower disability and death, based on average use of procedures during each cohort period and estimates from the literature of average use in 1985 and 1999 (i.e., between 1998 and 2000). We defined appropriate treatments for these analyses as those treatments with class IA recommendations from recent guidelines. These include the relevant procedures for Ischemic Heart Disease (IHD) and heart failure as well as all of the pharmacological treatments for IHD, reperfusion and aspirin for stroke, and beta-blockers and ace-inhibitors for heart failure and arrhythmia (Braunwald et al. 2002; Antman et al. 2004; Hunt et al. 2005; Adams et al. 2007). We did not estimate a separate model for other circulatory diseases, because this category includes multiple diagnoses and has no guidelines.

6.3.3 Estimation Results

Table 6.5 shows demographic characteristics of our cohort by year of baseline survey. The proportion of respondents disabled at baseline declined over time, from 32 percent in 1984 to 25.9 percent in 1994. This 6 percentage point decline in disability at baseline is consistent with other analyses using the NLTCS (Manton and Gu 2001). In addition, there was a slight increase in the mean modified Charlson index score from 0.94 in 1984 to 1.07 in 1994.

Table 0.5 Demograph	Demographic characteristics of CVD conort, by year of baseline survey				
	1984 N = 4,146 wN = 6,403,326	1989 N = 3,671 wN = 7,935,884	1994 N = 3,676 wN = 7,723,454		
Age groups, %					
65–69	20.0	21.6	21.0		
70–74	24.8	24.3	22.7		
75–79	23.2	23.4	24.0		
80-84	17.2	16.7	17.1		
85+	14.8	14.1	15.2		
Male, %	45.0	45.7	45.4		
Nonwhite, %	8.0	7.9	8.8		
Marital status, %					
Married	51.3	51.1	51.3		
Widowed	39.6	37.8	36.7		
Divorced, separated, or single	8.1	10.0	9.5		
Unknown marital status	1.1	1.1	2.4		
Disabled at baseline survey, %	32.2	30.0	25.9		
Modified Charlson comorbidy index ^a , mean (SD)	0.94 (0.02)	1.04 (0.02)	1.07 (0.02)		

stariation of CVD schort, by year of baseline an

Note: Estimates adjusted to the age and sex distribution of the 1999 population of Medicare beneficiaries.

^aHospitalizations for cardiovascular disease events were excluded from the Charlson index.

Disability and death at follow-up declined over time for patients with any of the CVD conditions, except for heart failure and arrhythmia patients who had slightly increased probability of disability over time (table 6.6). The share of patients alive and nondisabled at follow-up increased over time for all CVD conditions, including heart failure and arrhythmia; the increase ranged from 8 percent among circulatory disease patients to 32 percent for stroke patients.

Coefficients and standard errors from our estimation results are shown in tables 6.7–6.10 for models with all CVD patients, ischemic heart disease, stroke, and heart failure patients. Each table includes a panel data model with covariates for baseline survey year and area-level relevant procedures as well as a model on the 1994–1999 cohort with covariates for area-level appropriate pharmaceutical use and relevant procedures, with the exception of the stroke table, which only includes the model on the 1994–1999 cohort.

All-CVD

Table 6 5

Area-level use of relevant procedures was not significantly associated with lower disability or death in the model with panel data nor in the model

	1984–1989	1989–1994	1994–1999	% change, 1984–1989— 1994–1999				
	Disabled at t	follow-up %						
All CVD	26 1	25 3	24.0	-8.1				
IHD	23.3	23.3	21.5	-8.0				
Stroke	31.0	31.7	29.5	-4.8				
Heart failure and arrhythmia	21.8	23.5	22.9	5 5				
Other circulatory disease	28.6	30.2	26.0	-9.1				
Dead at follow-up. %								
All CVD	41.7	42.2	39.3	-5.8				
IHD	39.2	36.8	32.2	-17.9				
Stroke	47.4	46.0	42.1	-11.2				
Heart failure and arrhythmia	57.0	55.0	51.3	-9.9				
Other circulatory disease	31.0	33.2	30.3	-2.2				
Aliv	e and nondisab	led at follow-up,	%					
All CVD	32.2	32.5	36.7	14.0				
IHD	37.4	41.0	46.3	23.8				
Stroke	21.6	22.4	28.4	31.6				
Heart failure and arrhythmia	21.3	21.6	25.7	20.8				
Other circulatory disease	40.4	36.6	43.7	8.1				

Health outcomes at follow-up over time, by CVD condition

Table 6.7 All CVD: Multinomial regression models for health status outcome five years after baseline survey

	Model 1: Panel data		Model 2: 1994 cohort only	
Coefficients (SEs)	Disability	Death	Disability	Death
Relevant procedures	-0.000 (0.004)	-0.006 (0.003)	-0.001 (0.005)	-0.006 (0.005)
Beta-blockers			-0.012 (0.006)**	-0.017 (0.005)***
Aspirin			0.002 (0.011)	0.001 (0.010)
Reperfusion			-0.019 (0.007)***	-0.006 (0.007)
Ace-inhibitors	_	_	0.012 (0.006)**	0.013 (0.006)**
		Model Statistics		
Ν	11,491		3,	576
F-test	56.26		14.78	
P-value	P < 0	.0001	<i>P</i> < 0.0001	

Note: Models also adjust for age and sex interactions, disability status at the baseline interview, marital status, race (white versus other), Charlson comorbidity score, and zip code-level measures of education and poverty.

*** Significant at or below the 1 percent level.

Table 6.6

** Significant at or below the 5 percent level.

	Model 1:	Model 1: Panel data Model 2: 199		94 cohort only	
Coefficients (SEs)	Disability	Death	Disability	Death	
Relevant procedures	-0.005 (0.004)	013 (.003)***	-0.007 (0.005)	-0.013 (0.005)**	
Beta-blockers			-0.020 (0.010)**	-0.020 (0.008)**	
Aspirin	_	_	-0.009 (0.018)	-0.010 (0.018)	
Reperfusion		_	-0.014 (0.012)	-0.010 (0.011)	
Ace-inhibitors	_	_	0.008 (0.012)	0.008 (0.011)	
		Model statistics			
Ν	3,842		1,2	202	
F-test	15.39		5.	99	
P-value	P < 0.0001		P < 0	0.0001	

Table 6.8 Ischemic heart disease: Multinomial regression models for health status outcome five years after baseline survey

Note: Models also adjust for age and sex interactions, disability status at the baseline interview, marital status, race (white versus other), zip code-level measures of education and poverty, Charlson comorbidity score, and hospitalizations for stroke, hypertension, heart failure, peripheral vascular disease, and circulatory diseases.

*** Significant at or below the 1 percent level.

** Significant at or below the 5 percent level.

Table 6.9	Stroke: Multinomial regression models for health s years after baseline survey	status outcome five		
Coefficients (SEs)	Model 2: 1994 cc	ohort only		
	Disability	Death		
Relevant procedures	_	_		
Beta-blockers		_		
Aspirin	0.009 (0.023)	-0.019 (0.021)		
Reperfusion	$-0.040 (0.014)^{**}$	-0.021 (0.016)		
Ace-inhibitors	_			
	Model Statistics			
Ν	1,066			
F-test	3.94	3.94		
P-value	P < 0.00	P < 0.0001		

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Note: Model also adjusts for age and sex interactions, disability status at the baseline interview, marital status, race (white versus other), zip code-level measures of education and poverty, Charlson comorbidity score, and hospitalizations for ischemic heart disease, hypertension, heart failure, peripheral vascular disease, and circulatory diseases.

** Significant at or below the 5 percent level.

	Model 1: P	Model 1: Panel Data		Model 2: 1994 cohort only	
Coefficients (SEs)	Disability	Death	Disability	Death	
Relevant procedures	-0.001 (0.006)	0.002 (0.005)	-0.003 (0.010)	0.005 (0.008)	
Beta-blockers	_	_	-0.023 (0.009) **	-0.019 (0.008)**	
Aspirin	_	_	_	_	
Reperfusion	_	_	_	_	
Ace-inhibitors	_	_	0.027 (0.012)**	0.024 (0.011)**	
		Model Statistics			
N	3,7:	52	1,2	.28	
F-test	16.61		5.45		
<i>P</i> -value	P < 0.	0001	<i>P</i> < 0.0001		

Table 6.10 Heart failure and arrhythmia: Multinomial regression models for health status outcome five years after baseline survey

Note: Models also adjust for age and sex interactions, disability status at the baseline interview, marital status, race (white versus other), zip code-level measures of education and poverty, Charlson comorbidity score, and hospitalizations for stroke, hypertension, ischemic heart disease, peripheral vascular disease, and circulatory diseases.

**Significant at or below the 5 percent level.

on the 1994–1999 cohort, after adjusting for baseline survey year, demographics, and health characteristics. In the 1994–1999 cohort model, betablockers were significantly associated with lower disability and mortality, and reperfusion was associated with significantly lower disability. Aceinhibitors were significantly associated with worse outcomes in this model, including increased disability and death.

Predicted event rates by percentiles of beta-blocker and reperfusion use for all CVD patients are shown in figure 6.5. We estimate that disability and death at follow-up would decline 5 percent and 19 percent, respectively, if all patients moved from areas providing beta-blockers at the 10th percentile level to areas providing beta-blockers at the 90th percentile level. Based on average use in 1984 (48 percent) and 1999 (72 percent), we estimate that increased use of beta-blockers may have led to a 12 percent decline in mortality (from 39.2 percent to 34.5 percent) and a 3 percent decline in disability associated with increased beta-blocker use represent approximately 194 percent and 38 percent of the observed declines in mortality and disability, respectively, between 1984 and 1999.

The effect of reperfusion on disability across the percentiles of care is quite large. The probability of disability at follow-up declines approximately 22 percent from 27 percent to 21 percent if all patients moved from 10th percentile areas for reperfusion to 90th percentile areas. Based on average use over the study time frame, we estimate that increased use of reperfusion may have led to a 51.5 percent decline in disability, from 41.8 per-

5a. Beta-Blockers



Fig. 6.5 All CVD: Adjusted probability of death and disability at follow-up by percentiles of beta-blocker and reperfusion use

cent to 20.3 percent. The decline in disability associated with reperfusion is substantially greater than the observed decline in disability. However, because of the substantial increase in the use of reperfusion over this time frame, these calculations involve out-of-sample projections and should be viewed cautiously.

Ischemic Heart Disease

In both the panel data model and the model on the 1994–1999 cohort, relevant procedures were associated with significantly lower mortality. The coefficient on the relevant procedures variable in both models was approximately the same (-0.013), suggesting the effect of the procedures on death

may not be confounded by inclusion or exclusion of the pharmacological treatments in the two models. In the model on the 1994–1999 cohort, betablockers were significantly associated with lower disability and death.

Figure 6.6 shows predicted event rates based on percentiles of relevant procedures and beta-blocker use. If all patients lived in 10th percentile areas (0 percent procedure use), approximately 40 percent would die by follow-up compared to only 28 percent if all patients lived in 90th percentile regions (70 percent procedure use). If all patients were treated at the

6a. Relevant Procedures*





*Adjusted probabilities based on panel data model

Fig. 6.6 IHD: Adjusted probability of death and disability at follow-up by percentiles of invasive procedures and beta-blocker use

average level in 1984 (23 percent) and in 1999 (48 percent), the percentage dead at follow-up would fall from 35.4 percent to 31.1 percent, which accounts for approximately 61 percent of the decline in IHD mortality over time. For beta-blockers, the share disabled and dead at follow-up would decline by approximately 23 percent and 20 percent, respectively, if all patients moved from 10th percentile to 90th percentile treatment areas (29.2 percent versus 70.8 percent). If all patients were treated at the average levels in 1984 (48 percent) and 1999 (72 percent), disability would decline from 21.8 percent at the 1984 level to 18.6 percent at the 1994 level. Mortality would also fall from 33.5 percent to 29.1 percent. These figures represent more than 100 percent of the observed disability decline and 63 percent of the decline in mortality.

Stroke

In the 1994–1999 model with stroke patients, reperfusion was associated with significantly lower disability at follow-up. Predicted event rates for stroke patients, based on percentiles of reperfusion use, are shown in figure 6.7. Increasing reperfusion use from 10th to 90th percentile levels would lower disability approximately 34 percent from 35 percent to 23 percent. Based on the increase in average use from 1984 to 1999, reperfusion explains more than 100 percent of the decline in disability among stroke patients.

Heart Failure and Arrhythmia

In the panel data model, heart failure and arrhythmia patients were significantly less likely to die in 1994–1999 cohort compared to 1984–1989



Fig. 6.7 Stroke: Adjusted probability of death and disability at follow-up by percentiles of reperfusion use

(table 6.10). Relevant procedures were not associated with disability or death in either the panel data model or the model on the 1994-1999 cohort. Beta-blockers were associated with lower disability and death, and ace-inhibitors were associated with increased mortality and disability. We estimate that moving all patients from 10th percentile to 90th percentile levels of beta-blocker treatment would lower disability by 21 percent and mortality by 9 percent (fig. 6.8). We also find that increased use of beta-blockers from average levels in 1984 to 1999 would have led to a decline in disability among heart failure patients, in contrast to the observed increase in disability. In addition, this would have led to a 6 percent decline in death over time from 50.6 percent to 47.6 percent, and would explain approximately 53 percent of the observed decline in mortality.

6.4 Interpreting the Results

Use of effective treatments contributed to the decline in disability and death among cardiovascular disease patients. With the exception of aceinhibitors in the heart failure and arrhythmia and all-CVD models, increased use of effective treatments was associated with improved health outcomes. In particular, increased use of beta-blockers explained more than 100 percent of the decline in disability among IHD and heart failure patients, as well as 63 percent to 53 percent of the decline in mortality over time. Stroke patients benefited from increased use of reperfusion between 1984 and 1999, which explained over 100 percent of the decline in disability. Invasive procedures were important for IHD patients and explained



Fig. 6.8 Heart failure and Arrhythmia: Adjusted probability of death and disability at follow-up by percentiles of beta-blocker use

approximately 60 percent of the decline in mortality over the study time frame. The role of beta-blockers in explaining more than 100 percent of the decline in disability for IHD and heart failure patients as well as reperfusion for disability among stroke patients may seem overstated. However, adverse trends in risk factors for cardiovascular disease morbidity and mortality not included in our models, such as diabetes and obesity, were increasing over this time frame (Cooper et al. 2000; Villareal et al. 2005; Cowie et al. 2006). Our results suggest that the excess reduction in disability attributable to improved treatments may be explained by increased risk for morbidity in the elderly population over time.

Improved medical treatment after an acute cardiovascular event resulted in improved survival and reductions in disability. It may also affect medical spending. While we cannot do a complete evaluation of the impact of these changes, we can provide some information. We begin with the change in quality-adjusted life expectancy. To consider how reductions in disability in one year translate into long-term changes in quality-adjusted life expectancy, we estimate regression models for future survival and disability status as a function of disability in a base year. For a cohort in year t, we estimate linear probability models of the form:

(3) Pr (Alive in year
$$t + k$$
)_i = $\mathbf{X}_{i,t} \boldsymbol{\beta} + \gamma$ Disability_{i,t} + $e_{i,t}$

The coefficient γ indicates how changes in disability in year *t* affect longterm health outcomes, and $\mathbf{X}_{i,t}$ is a set of demographic and health status variables. The identifying assumption in equation (3) is that people who are not disabled because of medical treatment are subsequently equivalent in their health to those who never had an incident. This may or may not be the case. If this is not true, and survivors of events are less healthy, conditional on disability status, we will overstate the benefits of reductions in disability. Thus, one should properly view these estimates as an upper bound on the impact of medical interventions to reduce disability.

Figure 6.9 shows the survival rate by year, conditional on disability status in the base survey year. We report the results for the ten years after the survey for the 1989 cohort, and the five years after the survey for the 1994 cohort due to data limitations on long-term follow-up. Not surprisingly, survival for the disabled is below that for the nondisabled by a large margin. The difference is about 20 percentage points, and remains at that level throughout the decade. To forecast mortality beyond the ten-year observation window, we assume that the mortality hazard estimated in 1997– 1999 prevails in all subsequent years. Making this assumption, we estimate that those not disabled in 1989 have an average life expectancy of 7.8 years while those disabled in 1989 were expected to live only another 5.1 years on average (table 6.11).

This calculation does not account for the difference in subsequent disability in the next decade. Table 6.12 shows the disability rate for the 1989



Fig. 6.9 Survival by disability and year

Table 6.11	The persistence of disability status across surveys
------------	-----------------------------------------------------

Cohort	Not disabled at baseline	Disabled at baseline	
1989 Cohort			
Pr [Disabled in 1994]	31.4	79.1	
Pr [Disabled in 1999]	36.9	72.1	
1994 Cohort Pr [Disabled in 1999]	25.2	83.9	

Note: Disability frequencies are conditional on being alive at follow-up.

Table 6.12 Life expectancy by disability status

Measure	Not disabled	Disabled	Difference
Life expectancy	7.8	5.1	2.7
Quality-adjusted life expectancy	6.7	3.0	3.7

Source: Details about the calculations are described in text.

cohort in the 1994 and 1999 surveys, and the disability rate for the 1994 cohort in 1999. In each case, we condition disability status on being alive at the end date. Seventy-nine percent of people who were disabled in 1989 and still alive in 1994 were also disabled in 1994, compared to 31 percent of those not disabled in 1989. This pattern repeats itself in 1999 and is true for the 1994 cohort as well.

To estimate quality-adjusted life expectancy, we interpolate disability rates between 1989 and 1994, and 1994 and 1999. In each case, we assume that disability rates change at a common rate each year of the interval. We

also extrapolate disability rates after 1999, using the annual change between 1994 and 1999. As a rough approximation, we assume that one year in a disabled state is equivalent to 0.5 quality-adjusted life years.

As the second row of table 6.12 shows, quality-adjusted life expectancy is lower in each case than is life expectancy, but the difference between the disabled and nondisabled is similar to the unadjusted estimates. The increase in quality-adjusted life expectancy associated with not being disabled is 3.7 years.

The value of this improvement in life expectancy depends on the value of a year of life. Following a substantial recent literature (Viscusi and Aldy 2003), we assume that a year of life in good health is worth \$100,000 (in 1992 dollars). We also assume that future values are discounted at a 3 percent real rate of interest. Using these assumptions, we estimate the value of disability prevention to be \$316,000.

These benefits need to be weighed against the cost of reducing disability. These costs have two parts. The first is the initial treatment cost that led to the reduction in disability. To measure these costs, we use data on hospitalization spending for the 1989 cohort in the year after the CVD admission.⁹ The one-year interval is relatively common in studying acute treatment for cardiovascular disease (Cutler and McClellan 2001; Skinner, Staiger, and Fisher 2006). The limitation to hospital costs is because only those data are reliable prior to 1991. We inflated all costs to a common year of 1992 using the implicit GDP Price Deflator (Economic Report of the President 1997). We estimate that in the year after the CVD event, hospital spending averages \$8,610 for patients who do not receive relevant procedures and \$16,332 for patients receiving relevant procedures.

Although treatment costs are approximately twice as high for respondents receiving appropriate treatments, these costs may be offset by lower yearly spending in subsequent years among survivors. A previous study of 1982 and 1984 NLTCS respondents found annual per capita spending by Medicare for respondents without any ADL or IADL limitations was approximately \$3,275, compared to \$7,400 for respondents with at least four ADL and five IADL impairments, and \$13,100 for institutionalized respondents. These data suggest that the costs of intensive medical treatments that prevent or delay disability may be offset by lower annual average spending among healthier beneficiaries.

More recent studies using the Medicare Current Beneficiary Survey found similar lifetime spending between nondisabled and disabled seventyyear-olds, but life expectancy was approximately 2.7 years longer among the nondisabled (Lubitz et al. 2003). This provides further evidence that av-

^{9.} By definition, all participants in the 1989 cohort had at least one CVD admissions between 1984 and 1989. We averaged costs across all CVD hospitalizations for respondents with multiple relevant hospitalizations.

erage annual spending may be lower among the nondisabled relative to the disabled. However, another recent study found spending on the nondisabled is growing faster than spending on the disabled (Chernew et al. 2005). Whether increased spending on intensive medical care treatments, such as those for cardiovascular disease, continues to increase life expectancy and reduce average annual yearly spending among the nondisabled relative to the disabled will require further investigation.

6.5 Conclusions

Examining disability associated with cardiovascular disease leads to several important results. Reduced disability associated with cardiovascular disease accounts for a significant part of the total reduction in disability between 19 and 22 percent. The evidence suggests that improvements in medical care, including both increased use of relevant procedures and pharmaceuticals, led to a significant part of this decline.

While precise data on the implications of reduced disability are lacking, the possible impact of disability reductions is staggering. We estimate that preventing disability after an acute event can add as much as 3.7 years of quality-adjusted life expectancy, or perhaps \$316,000 of value. The cost of this change is much smaller. The initial treatment costs range from \$8,610 to \$16,332, depending on procedure use. Further, recent cost analyses report that annual Medicare spending was lower for the nondisabled compared to the disabled, which suggests that higher treatment costs may be offset by lower future spending among a more healthy population. By virtually any measure, therefore, medical technology after acute cardiovascular episodes is worth the cost.

The major issue raised by our results is whether these conclusions extend to other conditions. Disability reductions are complex, and will certainly involve medical as well as nonmedical factors. Sorting these out for other conditions is a high priority for future research.

References

- Adams, H., R. Adams, G. Del Zoppo, and L. B. Goldstein. 2005. Guidelines for the early management of patients with ischemic stroke. 2005 Guidelines Update: A scientific statement from the stroke council of the American Heart Association/ American Stroke Association. *Stroke* 36:916–21.
- Adams, H. P., Jr., G. Del Zoppo, M. J. Alberts, D. L. Bhatt, L. Brass, A. Furlan, R. L. Grubb, et al. 2007. Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral

Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. *Stroke* 38:1655–1711.

- American Heart Association. 2006. Medication information and drug classification http://www.americanheart.org/presenter.jhtml?identifier=70.
- Antman, E. M., D. T. Anbe, P. W. Armstrong, E. R. Bates, L. A. Green, M. Hand, J. S. Hochman, et al. 2004. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: Executive summary: A report of the ACC/AHA task force on practice guidelines (committee to revise the 1999 guidelines on the management of patients with acute myocardial infarction). *Journal* of the American College of Cardiology 44:671–719.
- Appelros, P., I. Nydevik, M. Viitanen. 2003. Poor outcome after first-ever stroke: Predictors for death, dependency, and recurrent stroke within the first year. *Stroke* 34:122–26.
- Borkon, A. M., G. F. Muehlebach, J. House, S. P. Marso, and J. A. Spertus. 2002. A comparison of the recovery of health status after percutaneous coronary intervention and coronary artery bypass. *Annals of Thoracic Surgery* 74:1526–30.
- Braunwald, E., E. M. Antman, J. W. Beasley, R. M. Califf, M. D. Cheitlin, J. S. Hochman, R. H. Jones, et al. 2002. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST segment elevation my-ocardial infarction: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee on the management of patients with unstable angina). Accessed at http://www.ncbi.nlrm.nih .gov/pubrmed/12383588
- Chernew, M. E., D. P. Goldman, F. Pan, and B. Shang. 2005. Disability and health care spending among Medicare beneficiaries. *Health Affairs* 24(2): W5-R42–W5-R51.
- Clark, W. M., S. Wissman, G. W. Albers, J. H. Jhamandas, K. P. Madden, and S. Hamilton. 1999. Recombinant tissue-type plasminogen activator (alteplase) for ischemic stroke 3 to 5 hours after symptom onset. *Journal of the American Medical Association* 282(21): 2019–26.
- Cooper, R., J. Cutler, P. Desvigne-Nickens, S. P. Fortmann, L. Friedman, R. Havlik, G. Hogelin et al. 2000. Trends and disparities in coronary heart disease, stroke and other cardiovascular diseases in the United States: Findings of the National Conference on Cardiovascular Disease Prevention. *Circulation* 102:3137–47.
- Cowie, C. C., K. F. Rust, D. D. Byrd-Holt, M. S. Eberhardt, K. M. Flegal, M. M. Engelgau, S. H. Sydah, D. E. Williams, L. S. Geiss, and E. W. Gregg. 2006. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population. *Diabetes Care* 29:1263–68.
- Crimmins, E. M., M. D. Hayward, and Y. Saito. 1994. Changing mortality and morbidity rates and the health status and life expectancy of the older population. *Demography* 31 (1): 159–75.
- Crimmins, E. M., Y. Saito, and D. Ingegneri. 1989. Changes in life expectancy and disability-free life expectancy in the United States. *Population and Development Review* 15 (2): 235–67.
- Cutler, D. M., and S. Kadiyala. 2003. The return to biomedical research: Treatment and behavioral effects. In *Measuring the gains from medical research: An economic approach*, ed. K. M. Murphy and R. H. Topel, 110–162. Chicago: University of Chicago Press.
- Cutler, D. M., and M. McClellan. 2001. Is technological change in medicine worth it? *Health Affairs* 20 (5): 11–29.
- Deyo, R. A., D. C. Cherkin, and M. A. Ciol. 1992. Adapting a clinical comorbid-

ity index for use with ICD-9-CM administrative databases. *Journal of Clinical Epidemiology* 45 (6): 613–19.

- Economic Report of the President. 1997. Accessed at http://www.gpoaccess.gov/ usbudget/fy98/pdf/erp.pdf Washington, D.C.: United States Government Printing Office.
- Fisher, E. S., D. E. Wennberg, T. A. Stukel, D. J. Gottlieb, F. L. Lucas, and E. L. Pinder. 2003a. The implications of regional variations in Medicare spending. Part 2: Health outcomes and satisfaction with care. *Annals of Internal Medicine* 138:288–98.
 - 2003b. The implications of regional variations in Medicare spending. Part
 1: The content, quality and accessibility of care. *Annals of Internal Medicine* 138:273–87.
- Freemantle, N., J. Cleland, P. Young, J. Mason, and J. Harrison. 1999. B blockade after myocardial infarction: Systematic review and meta regression analysis. *British Medical Journal* 318:1730–37.
- Glader, E.-L., B. Stegmayr, B. Norring, A. Terent, K. Hulter-Asberg, P.-O. Wester, and K. Asplund. 2003. Sex differences in management and outcome after stroke: A Swedish national perspective. *Stroke* 34:1970–75.
- Gottlieb, S. S., R. J. McCarter, and R. A. Vogel. 1998. Effect of beta blockade on mortality among high-risk and low-risk patients after myocardial infarction. *New England Journal of Medicine* 339 (8): 489–97.
- Heidenreich, P. A., and M. McClellan. 2001. Trends in treatment and outcomes for acute myocardial infarction: 1975–1995. American Journal of Medicine 110:165–74.
- Hennekens, C. H., C. M. Albert, S. L. Godfried, J. M. Gaziano, and J. E. Buring. 1996. Adjunctive drug therapy of acute myocardial infarction—evidence from clinical trials. *New England Journal of Medicine* 335 (22): 1660–67.
- Henon, H., O. Godefroy, D. Leys, F. Mounier-Vehier, C. Lucas, P. Rondepierre, A. Duhamel, and J. P. Pruvo. 1995. Early predictors of death and disability after acute cerebral ischemic event. *Stroke* 26:392–98.
- Hlatky, M. A., W. J. Rogers, I. Johnstone, and D. Boothroyd. 1997. Medical care costs and quality of life after randomization to coronary angioplasty or coronary bypass surgery. *New England Journal of Medicine* 336:92–99.
- Hunt, S. A., W. T. Abraham, M. H. Chin, A. M. Feldman, G. S. Francis, T. G. Ganiats, M. Jessup, et al. 2005. ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: Summary article: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (writing committee to update the 2001 guidelines for the evaluation and management of heart failure). *Circulation* 112: e154–e235.
- Jencks, S. F., T. Cuerdon, D. R. Burwen, B. Fleming, P. M. Houck, A. E. Kussmaul, D. S. Nilasena, D. L. Ordin, and D. R. Arday. 2000. Quality of medical care delivered to Medicare beneficiaries: A profile at state and national levels. *Journal of the American Medical Association* 284:1670–76.
- Kattainen, A., A. Reunanen, S. Koskinen, T. Martelin, P. Knekt, P. Sainio, T. Harkanen, and A. Aromaa. 2004. Secular changes in disability among middleaged and elderly Finns with and without coronary heart disease from 1978–1980 to 2000–2001. Annals of Epidemiology 14:479–85.
- Krumholz, H. M., M. J. Radford, E. F. Ellerbeck, J. Hennen, T. P. Meehan, M. Petrillo, Y. Wang, and S. F. Jencks. 1996. Aspirin for secondary prevention after acute myocardial infarction in the elderly: Prescribed use and outcomes. *Annals of Internal Medicine* 124:292–98.
- Krumholz, H. M., M. J. Radford, E. F. Ellerbeck, J. Hennen, T. P. Meehan, M. Petrillo, Y. Wang, T. F. Kresowik, and S. F. Jencks. 1995. Aspirin in the

treatment of acute myocardial infarction in elderly Medicare beneficiaries. *Circulation* 92:2841–7.

- Krumholz, H. M., M. J. Radford, Y. Wang, J. Chen, A. Heiat, and T. Marciniak. 1998. National use and effectiveness of β-blockers for the treatment of elderly patients after acute myocardial infarction: National cooperative cardiovascular project. *Journal of the American Medical Association* 280 (7): 623–29.
- Lubitz, J., L. Cai, E. Kramarow, and H. Lentzner. 2003. Health, life expectancy and health care spending among the elderly. *New England Journal of Medicine* 349:1048–55.
- Manton, K. G., L. S. Corder, and E. Stallard. 1993. Estimates of change in chronic disability and institutional incidence and prevalence rates in the U.S. elderly population from the 1982, 1984, and 1989 National Long Term Care Survey. *Journal* of Gerontology 48 (4): S153–66.
 - . 1997. Chronic disability trends in elderly United States populations: 1982– 1994. Proceedings of the National Academy of Sciences of the United States of America 94:2593–98.
- Manton, K. G., and X. Gu. 2001. Changes in the prevalence of chronic disability in the United States black and nonblack population above age 65 from 1982 to 1999. Proceedings of the National Academy of Sciences of the United States of America 98 (11): 6354–59.
- Manton, K. G., E. Stallard, and L. S. Corder. 1997. Changes in the age dependence of mortality and disability: Cohort and other determinants. *Demography* 34 (1): 135–57.
- Marciniak, T. A., E. F. Ellerbeck, M. J. Radford, T. F. Kresowik, J. A. Gold, H. M. Krumholz, C. I. Kiefe, R. M. Allman, R. A. Vogel, and S. F. Jencks. 1998. Improving the quality of care for Medicare patients with acute myocardial infarction: Results from the cooperative cardiovascular project. *Journal of the American Medical Association* 279 (17): 1351–57.
- McGovern, P. G., D. R. Jacobs, E. Shahar, D. K. Arnett, A. R. Folsom, H. Blackburn, and R. V. Luepker. 2001. Trends in acute coronary heart disease mortality, morbidity and medical care from 1985 through 1997: The Minnesota Heart Study. *Circulation* 104:19–24.
- McGovern, P. G., J. S. Pankow, E. Shahar, K. M. Doliszny, A. R. Folsom, H. Blackburn, and R. V. Luepker. 1996. Recent trends in acute coronary heart disease: Mortality, morbidity, medical care, and risk factors. *New England Journal of Medicine* 334:884–90.
- The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. 1995. Tissue plasminogen activator for acute ischemic stroke. *New England Journal of Medicine* 333 (24): 1581–87.
- O'Connor, G. T., H. B. Quinton, N. D. Traven, L. D. Ramunno, T. A. Dodds, T. A. Marciniak, and J. E. Wennberg. 1999. Geographic variation in the treatment of acute myocardial infarction: The Cooperative Cardiovascular Project. *Journal of the American Medical Association* 281 (7): 627–33.
- Pfisterer, M., P. Buser, S. Osswald, U. Allermann, W. Armann, W. Angehrn, E. Eeckhout, et al. 2003. Outcome of elderly patients with chronic symptomatic coronary artery disease with an invasive vs optimised medical treatment strategy: One year results of the randomized TIME trial. *Journal of the American Medical Association* 289:1117–23.
- Pocock, S. J., R. A. Henderson, T. Clayton, G. H. Lyman, and D. A. Chamberlain. 2000. Quality of life after coronary angioplasty or continued medical treatment for angina: Three-year follow-up in the RITA-2 trial. *Journal of the American College of Cardiology* 35:907–14.

- Pohjasvaara, T., T. Erkinjuntti, R. Vataja, and M. Kaste. 1997. Comparison of stroke features and disability in daily life in patients with ischemic stroke aged 55 to 70 and 71 to 85 years. *Stroke* 28:729–35.
- Prencipe, M., C. Ferretti, A. R. Casini, M. Santini, F. Giubilei, and F. Culasso. 1997. Stroke, disability, and dementia. *Stroke* 28:531–36.
- Rogers, W. J., J. Coggin, B. J. Gersh, L. D. Fisher, W. O. Myers, A. Oberman, and L. T. Sheffield. 1990. Ten-year follow-up of quality of life in patients randomized to receive medical therapy or coronary artery bypass graft surgery: The coronary artery surgery study (CASS). *Circulation* 82:1647–58.
- Rosamond, W. D., L. E. Chambless, A. R. Folsom, L. S. Cooper, D. E. Conwill, L. Clegg, C.-H. Wang, and G. Heiss. 1998. Trends in the incidence of myocardial infarction and in mortality due to coronary heart disease, 1987–1994. *New England Journal of Medicine* 339 (13): 861–67.
- Shlipak, M. G., W. S. Browner, H. Noguchi, B. Massie, C. D. Frances, and M. Mc-Clellan. 2001. Comparison of the effects of angiotensin converting-enzyme inhibitors and beta blockers on survival in elderly patients with reduced left ventricular function after myocardial infarction. *American Journal of Medicine* 110:425–33.
- Singer, B. H., and K. G. Manton. 1998. The effects of health changes on projections of health service needs for the elderly population of the United States. *Proceedings of the National Academy of Sciences of the United States of America* 95 (26): 15618–22.
- Skinner, J. S., D. O. Staiger, and E. S. Fisher. 2006. Is technological change in medicine always worth it? The case of acute myocardial infarction. *Health Affairs* 25 (2): W34–W47.
- Soumerai, S. B., T. J. McLaughlin, E. Speigelman, G. Hertzmark, G. Thibault, and L. Goldman. 1997. Adverse outcomes of underuse of beta-blockers in elderly survivors of acute myocardial infarction. *Journal of the American Medical Association* 277 (2): 115–21.
- Strauss, W. E., T. Fortin, P. Hartigan, E. D. Folland, and A. F. Parisi. 1995. A comparison of quality of life scores in patients with angina pectoris after angioplasty compared with after medical therapy. *Circulation* 92:1710–19.
- Stukel, T. A., F. L. Lucas, and D. E. Wennberg. 2005. Long-term outcomes of regional variations in intensity of invasive vs. medical management of Medicare patients with acute myocardial infarction. *Journal of the American Medical Association* 293:1329–37.
- Sytkowski, P. A., R. B. D'Agostino, A. Belanger, and W. B. Kannel. 1996. Sex and time trends in cardiovascular disease incidence and mortality: The Framingham Heart Study, 1950–1989. *American Journal of Epidemiology* 143(4): 338–50.
- Vaccarino, V., S. S. Rathore, N. K. Wenger, P. D. Frederick, J. L. Abramson, H. V. Barron, A. Manhapra, S. Mallik, and H. M. Krumholz. 2005. Sex and racial differences in the management of acute myocardial infarction, 1994 through 2002. *New England Journal of Medicine* 353 (7): 671–82.
- Villareal, D. T., C. M. Apovian, R. F. Kushner, and S. Klein. 2005. Obesity in older adults: Technical review and position statement of the American Society for Nutrition and NAASO, The Obesity Society. *American Journal of Clinical Nutrition* 82:923–34.
- Viscusi, W. K., and J. E. Aldy. 2003. The value of a statistical life: A critical review of market estimates throughout the world. *Journal of Risk and Uncertainty* 27 (1): 5–76.
- Vitagliano, G., J. P. Curtis, P. Jeptha, J. Concato, A. R. Feinstein, M. J. Radford, and H. M. Krumholz. 2004. Association between functional status and use and

effectiveness of beta-blocker prophylaxis in elderly survivors of acute myocardial infarction. *Journal of the American Geriatric Society* 52:496–501.

- Waidmann, T. A., J. Bound, and M. Schoenbaum. 1995. The illusion of failure: Trends in the self-reported health of the U.S. elderly. *The Milbank Quarterly* 73 (2): 253–87.
- Zhu, L., L. Fratiglioni, Z. Guo, H. Aguero-Torres, B. Winblad, and M. Viitanen. 1998. Association of stroke with dementia, cognitive impairment, and functional disability in the very old: A population-based study. *Stroke* 29: 2094–99.