Heart Disease and Stroke Statistics—2013 Update

A Report From the American Heart Association

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Summary

Each year, the American Heart Association (AHA), in conjunction with the Centers for Disease Control and Prevention, the National Institutes of Health, and other government agencies, brings together the most up-to-date statistics on heart disease, stroke, other vascular diseases, and their risk factors and presents them in its Heart Disease and Stroke Statistical Update. The Statistical Update is a valuable resource for researchers, clinicians, healthcare policy makers, media professionals, the lay public, and many others who seek the best national data available on heart disease, stroke, and other cardiovascular disease–related morbidity and mortality and the risks, quality of care, medical procedures and operations, and costs associated with the management of these diseases in a single document. Indeed, since 1999, the Statistical Update has been cited >10 500 times in the literature, based on citations of all annual versions. In 2011 alone, the various Statistical Updates were cited ≈1500 times (data from ISI Web of Science). In recent years, the Statistical Update has undergone some major changes with the addition of new chapters and major updates across multiple areas, as well as increasing the number of ways to access and use the information assembled.

For this year's edition, the Statistics Committee, which produces the document for the AHA, updated all of the current chapters with the most recent nationally representative data and inclusion of relevant articles from the literature over the past year. This year's edition also implements a new chapter organization to reflect the spectrum of cardiovascular health behaviors and health factors and risks, as well as subsequent complicating conditions, disease states, and outcomes. Also, the 2013 Statistical Update contains new data on the monitoring and benefits of cardiovascular health in the population, with additional new focus on evidence-based approaches to changing behaviors, implementation strategies, and implications of the AHA's 2020 Impact Goals. Below are a few highlights from this year's Update.

The 2013 Update Expands Data Coverage of the Epidemic of Poor Cardiovascular Health Behaviors and Their Antecedents and Consequences

- Adjusted population attributable fractions for cardiovascular disease (CVD) mortality were as follows[1]: 40.6% (95% confidence interval [CI], 24.5–54.6) for high blood pressure; 13.7% (95% CI, 4.8–22.3) for smoking; 13.2% (95% CI, 3.5–29.2) for poor diet; 11.9% (95% CI, 1.3–22.3) for insufficient physical activity; and 8.8% (95% CI, 2.1–15.4) for abnormal glucose levels.
- Despite 4 decades of progress, in 2011, among Americans ≥18 years of age, 21.3% of men and 16.7% of women continued to be cigarette smokers. In 2011, 18.1% of students in grades 9 through 12 reported current cigarette use.
- The percentage of the nonsmoking population with detectable serum cotinine (indicating exposure to secondhand smoke) declined from 52.5% in 1999 to 2000 to 40.1% in 2007 to 2008, with declines higher for those 3 to 11 years of age (–53.6%) and those 12 to 19 years of age (–46.5%) than for those 20 years of age and older (–36.7%).
- The proportion of youth (≤18 years of age) who report engaging in no regular physical activity is high, and the proportion increases with age. In 2011, among adolescents in grades 9 through 12, 17.7% of girls and 10.0% of boys reported that they had not engaged in ≥60 minutes of moderate-to-vigorous physical activity, defined as any activity that increased heart rate or breathing rate, even once in the previous 7 days, despite recommendations that children engage in such activity 7 days per week.
- Thirty two percent of adults reported engaging in no aerobic leisure-time physical activity.
- Data from the National Health and Nutrition Examination Survey (NHANES) indicate that between 1971 and 2004, average total energy consumption among US adults increased by 22% in women (from 1542 to 1886 kcal/d) and by 10% in men (from 2450 to 2693 kcal/d).
- The increases in calories consumed during this time period are attributable primarily to greater average carbohydrate intake, in particular, of starches, refined grains, and sugars. Other specific changes related to increased caloric intake in the United States include larger portion sizes, greater food quantity and calories per meal, and increased consumption of sugar-sweetened beverages, snacks, commercially prepared (especially fast food) meals, and higher energy-density foods.
- The estimated prevalence of overweight and obesity in US adults (≥20 years of age) is 154.7 million, which represents 68.2% of this group in 2010. Fully 34.6% of US adults are obese (body mass index ≥30 kg/m^2). Men and women of all race/ethnic groups in the population are affected by the epidemic of overweight and obesity.
- Among children 2 to 19 years of age, 31.8% are overweight and obese (which represents 23.9 million children) and 16.9% are obese (12.7 million children). Mexican American boys and girls and African American girls are disproportionately affected. Over the past 3 decades, the prevalence of obesity in children 6 to 11 years of age has increased from ≈4% to >20%.
- Obesity (body mass index ≥30 kg/m^2) is associated with marked excess mortality in the US population. Even more notable is the excess morbidity associated with overweight and obesity in terms of risk factor development and incidence of diabetes mellitus, CVD end points (including coronary heart disease, stroke, and heart failure), and numerous other health conditions, including asthma, cancer, end-stage renal disease, degenerative joint disease, and many others.

Prevalence and Control of Cardiovascular Health Factors and Risks Remains an Issue for Many Americans

- An estimated 31.9 million adults ≥20 years of age have total serum cholesterol levels ≥240 mg/dL, with a prevalence of 13.8%.

- Based on 2007 to 2010 data, 33.0% of US adults ≥20 years of age have hypertension. This represents 78 million US adults with hypertension. The prevalence of hypertension is nearly equal between men and women. African American adults have among the highest prevalence of hypertension (44%) in the world.
- Among hypertensive adults, ≈82% are aware of their condition and 75% are using antihypertensive medication, but only 53% of those with documented hypertension have their condition controlled to target levels.
- In 2010, an estimated 19.7 million Americans had diagnosed diabetes mellitus, representing 8.3% of the adult population. An additional 8.2 million had undiagnosed diabetes mellitus, and 38.2% had prediabetes, with abnormal fasting glucose levels. African Americans, Mexican Americans, Hispanic/Latino individuals, and other ethnic minorities bear a strikingly disproportionate burden of diabetes mellitus in the United States.
- The prevalence of diabetes mellitus is increasing dramatically over time, in parallel with the increases in prevalence of overweight and obesity.
- On the basis of NHANES 2003–2006 data, the age-adjusted prevalence of metabolic syndrome, a cluster of major cardiovascular risk factors related to overweight/obesity and insulin resistance, is ≈34% (35.1% among men and 32.6% among women).

Rates of Death Attributable to CVD Have Declined, but the Burden of Disease Remains High

- The 2009 overall rate of death attributable to CVD (*International Classification of Diseases, 10th Revision*, codes I00–I99) was 236.1 per 100 000. The rates were 281.4 per 100 000 for white males, 387.0 per 100 000 for black males, 190.4 per 100 000 for white females, and 267.9 per 100 000 for black females.
- From 1999 to 2009, the relative rate of death attributable to CVD declined by 32.7%. Yet in 2009, CVD (I00–I99; Q20–Q28) still accounted for 32.3% (787 931) of all 2 437 163 deaths, or 1 of every 3 deaths in the United States.
- On the basis of 2009 death rate data, >2150 Americans die of CVD each day, an average of 1 death every 40 seconds. About 153 000 Americans who died of CVD (I00–I99) in 2009 were <65 years of age. In 2009, 34% of deaths attributable to CVD occurred before the age of 75 years, which is well before the average life expectancy of 78.5 years.
- Coronary heart disease alone caused ≈1 of every 6 deaths in the United States in 2009. In 2009, 386 324 Americans died of coronary heart disease. Each year, an estimated ≈635 000 Americans have a new coronary attack (defined as first hospitalized myocardial infarction or coronary heart disease death) and ≈280 000 have a recurrent attack. It is estimated that an additional 150 000 silent first myocardial infarctions occur each year. Approximately every 34 seconds, 1 American has a coronary event, and approximately every 1 minute, an American will die of one.
- From 1999 to 2009, the relative rate of stroke death fell by 36.9% and the actual number of stroke deaths declined by 23.0%. Yet each year, ≈795 000 people continue to experience a new or recurrent stroke (ischemic or hemorrhagic). Approximately 610 000 of these are first attacks, and 185 000 are recurrent attacks. In 2009, stroke caused ≈1 of every 19 deaths in the United States. On average, every 40 seconds, someone in the United States has a stroke and dies of one approximately every 4 minutes.
- In 2009, 1 in 9 death certificates (274 601 deaths) in the United States mentioned heart failure. Heart failure was the underlying cause in 56 410 of those deaths in 2009. The number of any-mention deaths attributable to heart failure was approximately as high in 1995 (287 000) as it was in 2009 (275 000). Additionally, hospital discharges for heart failure remained essentially unchanged from 2000 to 2010, with first-listed discharges of 1 008 000 and 1 023 000, respectively.

The 2013 Update Provides Critical Data About Cardiovascular Quality of Care, Procedure Utilization, and Costs

In light of the current national focus on healthcare utilization, costs, and quality, it is critical to monitor and understand the magnitude of healthcare delivery and costs, as well as the quality of healthcare delivery, related to CVD risk factors and conditions. The Statistical Update provides these critical data in several sections.

Quality-of-Care Metrics for CVDs

Quality data are available from the AHA's "Get With The Guidelines" programs for coronary artery disease and heart failure and from the American Stroke Association/AHA's "Get With The Guidelines" program for acute stroke. Similar data from the Veterans Healthcare Administration, national Medicare and Medicaid data, and Acute Coronary Treatment and Intervention Outcomes Network (ACTION)–"Get With The Guidelines" Registry data are also reviewed. These data show impressive adherence to guideline recommendations for many, but not all, metrics of quality of care for these hospitalized patients. Data are also reviewed on screening for CVD risk factor levels and control.

Cardiovascular Procedure Use and Costs

- The total number of inpatient cardiovascular operations and procedures increased 28%, from 5 939 000 in 2000 to 7 588 000 in 2010 (National Heart, Lung, and Blood Institute computation based on National Center for Health Statistics annual data).
- The total direct and indirect cost of CVD and stroke in the United States for 2009 is estimated to be $312.6 billion. This figure includes health expenditures (direct costs, which include the cost of physicians and other professionals, hospital services, prescribed medications, home health care, and other medical durables) and lost productivity that results from morbidity and premature mortality (indirect costs).
- By comparison, in 2008, the estimated cost of all cancer and benign neoplasms was $228 billion ($93 billion in direct costs, $19 billion in morbidity indirect costs, and $116 billion in mortality indirect costs). CVD costs more than any other diagnostic group.

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The AHA, through its Statistics Committee, continuously monitors and evaluates sources of data on heart disease and stroke in the United States to provide the most current data available in the Statistics Update.

Finally, it must be noted that this annual Statistical Update is the product of an entire year's worth of effort by dedicated professionals, volunteer physicians and scientists, and outstanding AHA staff members, without whom publication of this valuable resource would be impossible. Their contributions are gratefully acknowledged.

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On behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee

Note: Population data used in the compilation of NHANES prevalence estimates are for the latest year of the NHANES survey being used. Extrapolations for NHANES prevalence estimates are based on the census resident population for 2010 because this is the most recent year of NHANES data used in the Statistical Update.

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*Modest.

†Significant.

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1. About These Statistics

The American Heart Association (AHA) works with the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics (NCHS); the National Heart, Lung, and Blood Institute (NHLBI); the National Institute of Neurological Disorders and Stroke (NINDS); and other government agencies to derive the annual statistics in this Heart Disease and Stroke Statistical Update. This chapter describes the most important sources and the types of data we use from them. For more details, see Chapter 25 of this document, the Glossary.

The surveys used are:

- Behavioral Risk Factor Surveillance System (BRFSS)—ongoing telephone health survey system
- Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS)—stroke incidence rates and outcomes within a biracial population
- Medical Expenditure Panel Survey (MEPS)—data on specific health services that Americans use, how frequently they use them, the cost of these services, and how the costs are paid
- National Health and Nutrition Examination Survey (NHANES)—disease and risk factor prevalence and nutrition statistics
- National Health Interview Survey (NHIS)—disease and risk factor prevalence
- National Hospital Discharge Survey (NHDS)—hospital inpatient discharges and procedures (discharged alive, dead, or status unknown)
- National Ambulatory Medical Care Survey (NAMCS)—physician office visits
- National Home and Hospice Care Survey (NHHCS)—staff, services, and patients of home health and hospice agencies
- National Hospital Ambulatory Medical Care Survey (NHAMCS)—hospital outpatient and emergency department (ED) visits
- Nationwide Inpatient Sample of the Agency for Healthcare Research and Quality (AHRQ)—hospital inpatient discharges, procedures, and charges
- National Nursing Home Survey (NNHS)—nursing home residents
- National Vital Statistics System—national and state mortality data
- World Health Organization (WHO)—mortality rates by country
- Youth Risk Behavior Surveillance System (YRBSS)—health-risk behaviors in youth and young adults

Disease Prevalence

Prevalence is an estimate of how many people have a disease at a given point or period in time. The NCHS conducts health examination and health interview surveys that provide estimates of the prevalence of diseases and risk factors. In this Update, the health interview part of the NHANES is used for the prevalence of cardiovascular diseases (CVDs). NHANES is used more than the NHIS because in NHANES, angina pectoris (AP) is based on the Rose Questionnaire; estimates are made regularly for heart failure (HF); hypertension is based on blood pressure (BP) measurements and interviews; and an estimate can be made for total CVD, including myocardial infarction (MI), AP, HF, stroke, and hypertension.

Abbreviations Used in Chapter 1

AHA	American Heart Association
AHRQ	Agency for Healthcare Research and Quality
AP	angina pectoris
ARIC	Atherosclerosis Risk in Communities Study
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHS	Cardiovascular Health Study
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
FHS	Framingham Heart Study
GCNKSS	Greater Cincinnati/Northern Kentucky Stroke Study
HD	heart disease
HF	heart failure
ICD	International Classification of Diseases
ICD-9-CM	*International Classification of Diseases, Clinical Modification, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHHCS	National Home and Hospice Care Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institute of Neurological Disorders and Stroke
NNHS	National Nursing Home Survey
PAD	peripheral artery disease
WHO	World Health Organization
YRBSS	Youth Risk Behavior Surveillance System

See Glossary (Chapter 25) for explanation of terms.

A major emphasis of this Statistical Update is to present the latest estimates of the number of people in the United States who have specific conditions to provide a realistic estimate of burden. Most estimates based on NHANES prevalence rates are based on data collected from 2007 to 2010 (in most cases, these are the latest published figures). These are applied to census population estimates for 2010. Differences in population estimates based on extrapolations of rates beyond the data collection period by use of more recent census population estimates cannot be used to evaluate possible trends in prevalence. Trends can only be evaluated by comparing prevalence rates estimated from surveys conducted in different years.

Risk Factor Prevalence

The NHANES 2007– 2010 data are used in this Update to present estimates of the percentage of people with high lipid values, diabetes mellitus (DM), overweight, and obesity. The

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NHIS is used for the prevalence of cigarette smoking and physical inactivity. Data for students in grades 9 through 12 are obtained from the YRBSS.

Incidence and Recurrent Attacks

An incidence rate refers to the number of new cases of a disease that develop in a population per unit of time. The unit of time for incidence is not necessarily 1 year, although we often discuss incidence in terms of 1 year. For some statistics, new and recurrent attacks or cases are combined. Our national incidence estimates for the various types of CVD are extrapolations to the US population from the Framingham Heart Study (FHS), the Atherosclerosis Risk in Communities (ARIC) study, and the Cardiovascular Health Study (CHS), all conducted by the NHLBI, as well as the GCNKSS, which is funded by the NINDS. The rates change only when new data are available; they are not computed annually. Do not compare the incidence or the rates with those in past editions of the Heart Disease and Stroke Statistics Update (also known as the Heart and Stroke Statistical Update for editions before 2005). Doing so can lead to serious misinterpretation of time trends.

Mortality

Mortality data are presented according to the underlying cause of death. "Any-mention" mortality means that the condition was nominally selected as the underlying cause or was otherwise mentioned on the death certificate. For many deaths classified as attributable to CVD, selection of the single most likely underlying cause can be difficult when several major comorbidities are present, as is often the case in the elderly population. It is useful, therefore, to know the extent of mortality attributable to a given cause regardless of whether it is the underlying cause or a contributing cause (ie, its "any-mention" status). The number of deaths in 2009 with any mention of specific causes of death was tabulated by the NHLBI from the NCHS public-use electronic files on mortality.

The first set of statistics for each disease in this Update includes the number of deaths for which the disease is the underlying cause. Two exceptions are Chapter 9 (High Blood Pressure) and Chapter 19 (Cardiomyopathy and Heart Failure). High BP, or hypertension, increases the mortality risks of CVD and other diseases, and HF should be selected as an underlying cause only when the true underlying cause is not known. In this Update, hypertension and HF death rates are presented in 2 ways: (1) As nominally classified as the underlying cause and (2) as any-mention mortality.

National and state mortality data presented according to the underlying cause of death were computed from the mortality tables of the NCHS World Wide Web site, the Health Data Interactive data system of the NCHS, or the CDC compressed mortality file. Any-mention numbers of deaths were tabulated from the electronic mortality files of the NCHS World Wide Web site and from Health Data Interactive.

Population Estimates

In this publication, we have used national population estimates from the US Census Bureau for 2010 in the computation of morbidity data. NCHS population estimates for 2009 were used in the computation of death rate data. The Census Bureau World Wide Web site[1] contains these data, as well as information on the file layout.

Hospital Discharges and Ambulatory Care Visits

Estimates of the numbers of hospital discharges and numbers of procedures performed are for inpatients discharged from short-stay hospitals. Discharges include those discharged alive, dead, or with unknown status. Unless otherwise specified, discharges are listed according to the first-listed (primary) diagnosis, and procedures are listed according to all listed procedures (primary plus secondary). These estimates are from the NHDS of the NCHS unless otherwise noted. Ambulatory care visit data include patient visits to physician offices and hospital outpatient departments and EDs. Ambulatory care visit data reflect the first-listed (primary) diagnosis. These estimates are from NAMCS and NHAMCS of the NCHS.

International Classification of Diseases

Morbidity (illness) and mortality (death) data in the United States have a standard classification system: the International Classification of Diseases (ICD). Approximately every 10 to 20 years, the ICD codes are revised to reflect changes over time in medical technology, diagnosis, or terminology. Where necessary for comparability of mortality trends across the 9th and 10th ICD revisions, comparability ratios computed by the NCHS are applied as noted.[2] Effective with mortality data for 1999, we are using the 10th revision (ICD-10). It will be a few more years before the 10th revision is used for hospital discharge data and ambulatory care visit data, which are based on the *International Classification of Diseases, Clinical Modification, 9th Revision* (ICD-9-CM).[3]

Age Adjustment

Prevalence and mortality estimates for the United States or individual states comparing demographic groups or estimates over time either are age specific or are age adjusted to the 2000 standard population by the direct method.[4] International mortality data are age adjusted to the European standard.[5] Unless otherwise stated, all death rates in this publication are age adjusted and are deaths per 100 000 population.

Data Years for National Estimates

In this Update, we estimate the annual number of new (incidence) and recurrent cases of a disease in the United States by extrapolating to the US population in 2010 from rates reported in a community- or hospital-based study or multiple studies. Age-adjusted *incidence* rates by sex and race are also given in this report as observed in the study or studies. For US *mortality*, most numbers and rates are for 2009. For disease and risk factor *prevalence*, most rates in this report are calculated from the 2007–2010 NHANES. Because NHANES is conducted only in the noninstitutionalized population, we extrapolated the rates to the total US population in 2008, recognizing that this probably underestimates the total prevalence, given the relatively high prevalence in the institutionalized population. The numbers and rates of *hospital inpatient discharges* for the United States are for 2010. Numbers of visits to *physician offices*, *hospital EDs*, and *hospital outpatient departments* are for 2010. Except as noted, *economic cost* estimates are for 2009.

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For data on hospitalizations, physician office visits, and mortality, CVD is defined according to ICD codes given in Chapter 25 of the present document. This definition includes all diseases of the circulatory system, as well as congenital CVD. Unless so specified, an estimate for total CVD does not include congenital CVD. Prevalence of CVD includes people with hypertension, heart disease (HD), stroke, peripheral artery disease (PAD), and diseases of the veins.

Race

Data published by governmental agencies for some racial groups are considered unreliable because of the small sample size in the studies. Because we try to provide data for as many racial groups as possible, we show these data for informational and comparative purposes.

Contacts

If you have questions about statistics or any points made in this Update, please contact the AHA National Center, Office of Science & Medicine at statistics@heart.org. Direct all media inquiries to News Media Relations at inquiries@heart.org or 214-706-1173.

We do our utmost to ensure that this Update is error free. If we discover errors after publication, we will provide corrections at our World Wide Web site, http://www.heart.org/statistics, and in the journal *Circulation*.

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2. American Heart Association's 2020 Impact Goals

See Tables 2-1 through 2-8 and Charts 2-1 through 2-12.

After achieving its major Impact Goals for 2010, the AHA created a new set of Impact Goals for the current decade.[1] Specifically, the AHA committed to the following central organizational goals:

> *By 2020, to improve the cardiovascular health of all Americans by 20%, while reducing deaths from CVDs and stroke by 20%.*[1]

These goals introduce a new concept, *cardiovascular health*, which is characterized by 7 health metrics. *Ideal cardiovascular health* is defined by the absence of clinically manifest CVD together with the simultaneous presence of optimal levels of all 7 metrics, including 4 health behaviors (not smoking and having sufficient physical activity [PA], a healthy diet pattern, and appropriate energy balance as represented by normal body weight) and 3 health factors (optimal total cholesterol, BP, and fasting blood glucose, in the absence of drug treatment; Table 2-1). Because a spectrum of cardiovascular health can also be envisioned and the ideal cardiovascular health profile is known to be rare in the US population, a broader spectrum of cardiovascular health can also be represented as being "ideal," "intermediate," or "poor" for each of the health behaviors and health factors.[1] Table 2-1 provides the specific definitions for ideal, intermediate, and poor cardiovascular health for each of the 7 metrics, both for adults (≥20 years of age) and for children (age ranges for each metric depending on data availability).

This concept of cardiovascular health represents a new focus for the AHA. Three novel emphases are central to the AHA 2020 Impact Goals:

- An expanded focus on CVD prevention and promotion of positive "cardiovascular health," rather than primarily the treatment of established CVD.
- Efforts to promote healthy behaviors (healthy diet pattern, appropriate energy intake, PA, and nonsmoking) and healthy biomarker levels (optimal blood lipids, BP, glucose levels) throughout the lifespan.
- A population-level health promotion strategy that shifts the majority of the public towards greater cardiovascular health in addition to targeting those individuals at greatest CVD risk, since healthy lifestyles in all domains are uncommon throughout the US population.

Beginning in 2011, and recognizing the time lag in the nationally representative US data sets, this chapter in the annual Statistical Update evaluates and publishes metrics and information to provide insights into both progress toward meeting the 2020 AHA goals and areas that require greater attention to meet these goals.

Cardiovascular Health: Current Prevalence

- The most up-to-date data on national prevalence of ideal, intermediate, and poor levels of each of the 7 cardiovascular health metrics are shown for children (adolescents and teens 12–19 years of age) in Chart 2-1 and for adults (≥20 years of age) in Chart 2-2.
- For most metrics, the prevalence of ideal levels of health behaviors and health factors is much higher in US children than in US adults. The major exceptions are diet and PA, for which the prevalence of ideal levels in children is similar (for PA) or worse (for diet) than in adults.
- Among children (Chart 2-1), the prevalence (unadjusted) of ideal levels of cardiovascular health behaviors and factors currently varies from 0% for the healthy diet pattern (ie, essentially no children meet at least 4 of the 5 dietary components) to >80% for the smoking, BP, and fasting glucose metrics. More than 90% of US children meet 0 or only 1 of the 5 healthy dietary components.
- Among US adults (Chart 2-2), the age-standardized prevalence of ideal levels of cardiovascular health behaviors and factors currently varies from 0.3% for having at least 4 of 5 components of the healthy diet pattern to up to 76% for never having smoked or being a former smoker who has quit for >12 months.

Abbreviations Used in Chapter 2

AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities Study
BMI	body mass index
BP	blood pressure
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHF	congestive heart failure
CI	confidence interval
CVD	cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	diastolic blood pressure
DM	diabetes mellitus
FDA	Food and Drug Administration
HbA_{1c}	hemoglobin A_{1c}
HBP	high blood pressure
HD	heart disease
HF	heart failure
HR	hazard ratio
IMT	intima-media thickness
LDL	low-density lipoprotein
MI	myocardial infarction
NHANES	National Health and Nutrition Examination Survey
NOMAS	Northern Manhattan Study
OR	odds ratio
PA	physical activity
PE	physical education
SBP	systolic blood pressure
SE	standard error
UN	United Nations
WHO	World Health Organization

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- Age-standardized and age-specific prevalence estimates for ideal cardiovascular health and for ideal levels of each of its components for 2007 to 2008 (baseline) and 2009 to 2010 are shown in Table 2-2.
  - —In 2009 to 2010, the prevalence of ideal levels across 6 available health factors and health behaviors (ie, excluding diet, for which 2009–2010 data were not available at the time of this analysis) decreases dramatically from younger to older age groups. The same trend was seen in 2007-2008.
- Chart 2-3 displays the prevalence estimates for the population of US children (12–19 years of age) meeting different numbers of criteria for ideal cardiovascular health (out of 7 possible) in 2007 to 2008.
  - —Very few US children (<5%) meet only 1 or 2 criteria for ideal cardiovascular health.
  - —About half of US children meet 3 or 4 fewer criteria for ideal cardiovascular health, and about half meet 5 or 6 criteria.
  - —Virtually no children meet all 7 criteria for ideal cardiovascular health.
  - —Overall distributions were similar in boys and girls.
- Charts 2-4 and 2-5 display the age-standardized prevalence estimates of US adults meeting different numbers of criteria for ideal cardiovascular health (out of 7 possible) in 2007 to 2008, overall and stratified by age, sex, and race.
  - —Approximately 2.5% of US adults have 0 of the 7 criteria at ideal levels, and another 15% meet only 1 of 7 criteria. This is much worse than among children.
  - —Most US adults (≈60%) have 2, 3, or 4 criteria at ideal cardiovascular health, with approximately 1 in 5 adults within each of these categories.
  - —Approximately 12% meet 5 metrics, 4% meet 6 metrics, and 0% meet 7 metrics at ideal levels.
  - —Presence of ideal cardiovascular health is strongly age related (Chart 2-4). Younger adults are more likely to meet greater numbers of ideal metrics, and older adults are much less likely. More than 60% of those >60 years of age have only 2 or fewer metrics at ideal levels (Chart 2-4).
  - —Women tend to have more metrics at ideal levels than do men (Chart 2-4).
  - —Blacks and Mexican Americans tend to have fewer metrics at ideal levels than whites or other races (Chart 2-5). Approximately 6 in 10 white adults and 7 in 10 black or Mexican American adults have no more than 3 of 7 metrics at ideal levels (Chart 2-5).
- Chart 2-6 displays the age-standardized percentages of US adults and percentages of children who have 5 or more of the metrics (out of 7 possible) at ideal levels.
  - —Approximately 40% of US children 12 to 19 years of age have 5 or more metrics at ideal levels, with lower levels in boys (36%) than in girls (45%).
  - —In comparison, only 16% of US adults have 5 or more metrics with ideal levels, with lower prevalence in men (12%) than in women (21%).
  - —Whites had nearly twice the percentage of adults with 5 or more metrics with ideal levels (18%) as Mexican Americans (9.5%) or blacks (11%).
- Chart 2-7 displays the age-standardized percentages of US adults meeting different numbers of criteria for both poor and ideal cardiovascular health. Meeting the AHA 2020 Strategic Impact Goals is predicated on reducing the relative percentage of those with poor levels while increasing the relative percentage of those with ideal levels for each of the 7 metrics.
  - —Approximately 94% of US adults have at least 1 metric at poor levels.
  - —Approximately 38% of US adults have at least 3 metrics at poor levels.
  - —Few US adults (<3%) have 5 or more metrics at poor levels.
  - —More US adults have 4 to 6 ideal metrics than 4 to 6 poor metrics.
- The prevalence of poor health behaviors and health factors and their awareness, treatment, and control are displayed in Table 2-3 separately for those with and without self-reported CVD.
  - —Americans with CVD are much more likely to be current or former smokers than Americans without CVD.
  - —Approximately 20% of US adults are current smokers or have quit recently (<12 months ago).
  - —As measured by self-reported data, Americans with CVD are very likely to have intermediate or poor levels of PA (74.1%), whereas Americans without CVD still commonly have such levels (58.4%). Furthermore, 64.5% of those with CVD and 47.3% of those without CVD report engaging in no moderate or vigorous activity at all.
  - —79% of US adults meet 0 or only 1 of the 5 healthy diet metrics.
  - —Two thirds of US adults are overweight, with little difference by prevalent CVD. Half of all US adults with CVD and one third without CVD are obese.
  - —Hypertension is present in 28.5% of US adults without CVD and 51.0% of US adults with CVD. Of these, nearly all with CVD are aware of their hypertension (98.6%) and are receiving treatment (97.4%), but a much smaller proportion of those without CVD are aware (70.6%) or receiving treatment (61.4%).
  - —Both presence of hypercholesterolemia (total cholesterol ≥240 mg/dL or receiving medication) and DM (fasting glucose ≥125 mg/dL or receiving medications) and awareness and treatment of these conditions are similarly higher among those with CVD than among those without CVD.

Cardiovascular Health: Trends Over Time

- The trends over the last decade in each of the 7 cardiovascular health metrics (for diet, trends from 2005–2006 to 2007–2008) are shown in Chart 2-8 (for children

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12–19 years of age) and Chart 2-9 (for adults ≥20 years of age).

—Among children, there appears to be a negative trend for meeting the body mass index (BMI) metric and positive trends for meeting the smoking and total cholesterol metrics. Other metrics do not show consistent trends.

—Among adults, there appears to be a positive trend for meeting the smoking metric and negative trends for meeting the BMI and glucose metrics. Trends for other metrics appear fairly flat.

- Huffman et al[2] made projections in cardiovascular health metrics to 2020 based on NHANES data from 1988–2008 (Chart 2-10). If current trends continue, estimated cardiovascular health will improve by 6%, short of the AHA's goal of 20%. On the basis of current trends among individual metrics, anticipated declines in prevalence of smoking, high cholesterol, and high BP (in men) would be offset by substantial increases in the prevalence of obesity and DM and small expected changes in ideal dietary patterns or PA.[2]

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- In 2009, the age-standardized death rate attributable to all CVDs was 237.1 per 100 000 (Chart 2-11), down 6% from 252.4 per 100 000 in 2007 (baseline data for the 2020 Impact Goals on CVD and stroke mortality).

  —Death rates in 2009 attributable to stroke, HDs, and other cardiovascular causes were 38.9, 116.1, and 81.0 per 100 000, respectively.

- Data from NHANES 2009–2010 reveal that overall, 7.2% of Americans self-reported having some type of CVD (Table 2-3), including 3.2% with coronary heart disease (CHD), 2.7% with stroke, and 2.0% with congestive heart failure (CHF) (some individuals reported more than 1 condition).

Relevance of Ideal Cardiovascular Health

Since the AHA announced its 2020 Impact Goals, several investigations have confirmed the importance of these metrics of cardiovascular health. Overall, these data demonstrate the relevance of the concept of cardiovascular health to risk of future risk factors, disease, and mortality, with strong inverse, stepwise association with all-cause, CVD, and ischemic HD mortality.

- Bambs et al[3] and Folsom et al[4] both described the low prevalence (<1%) of ideal cardiovascular health, defined as being in the ideal category of all 7 AHA metrics in the Heart Strategies Concentrating on Risk Evaluation and ARIC cohorts, respectively.
- In ARIC, a stepwise, inverse association was present between the number of ideal health metrics and incident CVD events (including CHD death, nonfatal MI, stroke, and HF) during 20 years of follow-up. For participants with 0, 1, 2, 3, 4, 5, 6, and 7 metrics at ideal levels, the age-, sex-, and race-adjusted rates of incident CVD incidence were 3.21, 2.19, 1.60, 1.20, 0.86, 0.64, 0.39, and 0 per 100 person-years, respectively.[4]
- Importantly, both ideal health behaviors and ideal health factors were independently associated with lower CVD risk in a stepwise fashion (Chart 2-12). Thus, across any levels of health behaviors, health factors were still associated with incident CVD; and across any levels of health factors, health behaviors were still associated with incident CVD.
- Dong et al[5] demonstrated a similar strong graded, inverse relationship between the number of ideal cardiovascular health metrics and risk of CVD in white, black, and Hispanic participants of the Northern Manhattan Study (NOMAS) after 11 years of follow-up.
- On the basis of data from NHANES (1988–2010), a similarly low prevalence of ideal cardiovascular health is present across the United States: 2.0% (95% confidence interval [CI], 1.5%–2.5%) in 1988–1994 and 1.2% (95% CI, 0.8%–1.9%) in 2005–2010.[6]
- Furthermore, a stepwise association is present between the number of ideal cardiovascular health metrics and risk of all-cause mortality, CVD mortality, and ischemic HD mortality after 14.5 years of follow-up in the United States.[6] The adjusted hazard ratios (HRs) for individuals with 6 or 7 ideal health metrics compared with individuals with 0 ideal health metrics were 0.49 (95% CI, 0.33–0.74) for all-cause mortality, 0.24 (95% CI, 0.13–0.47) for CVD mortality, and 0.30 (95% CI, 0.13–0.68) for ischemic HD mortality.[6] Ford et al[7] demonstrated similar relationships.
- Adjusted population attributable fractions for CVD mortality were as follows[6]:

  —40.6% (95% CI, 24.5%–54.6%) for HBP
  —13.7% (95% CI, 4.8%–22.3%) for smoking
  —13.2% (95% CI, 3.5%–29.2%) for poor diet
  —11.9% (95% CI, 1.3%–22.3%) for insufficient PA
  —8.8% (95% CI, 2.1%–15.4%) for abnormal glucose levels

- Adjusted population attributable fractions for ischemic HD mortality were as follows[6]:

  —34.7% (95% CI, 6.6%–57.7%) for HBP
  —16.7% (95% CI, 6.4%–26.6%) for smoking
  —20.6% (95% CI, 1.2%–38.6%) for poor diet
  —7.8% (95% CI, 0%–22.2%) for insufficient PA
  —7.5% (95% CI, 3.0%–14.7%) for abnormal glucose levels

- Interestingly, based on NHANES 1999–2002, only modest intercorrelations are present between different cardiovascular health metrics. For example, these ranged from a correlation of −0.12 between PA and hemoglobin A_{1c} (HbA_{1c}) to a correlation of 0.29 between BMI and HbA_{1c}. Thus, the 7 AHA cardiovascular health metrics appear interrelated, but only modestly, with substantial independent variation in each.[7]
- The AHA cardiovascular health metrics predict future cardiometabolic risk when assessed in youth. In the Young Finns Study, a stepwise, inverse association was found between the number of ideal cardiovascular health metrics in adolescence (12–18 years of age) and risk of developing hypertension, dyslipidemia, or high carotid

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intima-media thickness (IMT) in adulthood after 21 years of follow-up (1986–2007).[8] For every 1 additional ideal cardiovascular health metric present in adolescence, compared with either poor or intermediate levels, the age- and sex-adjusted odds ratios (ORs) for adult cardiometabolic outcomes were

—Hypertension: OR=0.66 (95% CI, 0.54–0.80)
—High low-density lipoprotein (LDL) cholesterol (>160 mg/dL): OR=0.66 (95% CI, 0.52–0.85)
—High-risk IMT (90th percentile or plaque present): OR=0.75 (95% CI, 0.60–0.94)

Achieving the 2020 Impact Goals

- Taken together, these data continue to demonstrate both the tremendous relevance of the AHA 2020 Impact Goals for cardiovascular health and the substantial progress that will be needed to achieve these goals over the next decade.
- A range of complementary strategies and approaches can lead to improvements in cardiovascular health. These include each of the following:

  —Individual-focused approaches, which target lifestyle and treatments at the individual level (Table 2-4).
  —Health-care systems approaches, which encourage, facilitate, and reward efforts by providers to improve health behaviors and health factors (Table 2-5).
  —Population approaches, which target lifestyle and treatments in schools or workplaces, local communities, and states, as well as throughout the nation (Table 2-6).

- Such approaches can focus on both (1) improving cardiovascular health among those who currently have less than optimal levels and (2) preserving cardiovascular health among those who currently have ideal levels (in particular, children, adolescents, and young adults) as they age.
- The metrics with the greatest potential for improvement are health behaviors, including diet quality, PA, and body weight. However, each of the cardiovascular health metrics can be improved and deserves major focus, including smoking, which remains the leading cause of preventable death in the United States.
- Continued emphasis is also needed on the treatment of acute CVD events and secondary prevention through treatment and control of health behaviors and risk factors.
- For each cardiovascular health metric, modest shifts in the population distribution toward improved health would produce relatively large increases in the proportion of Americans in both ideal and intermediate categories. For example, on the basis of NHANES 2009–2010, the current prevalence of ideal levels of BP among US adults is 44.3%. To achieve the 2020 goals, a 20% relative improvement would require an increase in this proportion to 53.1% by 2020 (44.3% × 1.20). On the basis of NHANES data, a reduction in population mean BP of just 2 mm Hg would result in 56.1% of US adults having ideal levels of BP, which represents a 26.8% relative improvement in this metric (Table 2-7). Larger population reductions in BP would lead to even larger numbers of people with ideal levels. Such small reductions in population BP could result from small health behavior changes at a population level, such as increased PA, increased fruit and vegetable consumption, decreased sodium intake, decreased adiposity, or some combination of these and other lifestyle changes, with resulting substantial projected decreases in CVD rates in US adults.[9]
- The AHA has a broad range of policy initiatives to improve cardiovascular health and meet the 2020 Strategic Impact Goals (Table 2-8). Future Statistical Updates will update these initiatives and track progress toward the 2020 Impact Goals.

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Table 2-1. Definitions of Poor, Intermediate, and Ideal Cardiovascular Health for Each Metric in the AHA 2020 Goals

	Level of Cardiovascular Health for Each Metric		
	Poor	Intermediate	Ideal
Current smoking			
Adults ≥20 y of age	Yes	Former ≤12 mo	Never or quit >12 mo
Children 12–19 y of age	Tried during the prior 30 d	…	Never tried; never smoked whole cigarette
BMI*			
Adults ≥20 y of age	≥30 kg/m^2	25–29.9 kg/m^2	<25 kg/m^2
Children 2–19 y of age	>95th percentile	85th-95th percentile	<85th percentile
PA			
Adults ≥20 y of age	None	1–149 min/wk moderate or 1–74 min/wk vigorous or 1–149 min/wk moderate + 2×vigorous	≥150 min/wk moderate or ≥75 min/wk vigorous or ≥150 min/wk moderate + 2×vigorous
Children 12–19 y of age	None	>0 and <60 min of moderate or vigorous every day	≥60 min of moderate or vigorous every day
Healthy diet pattern, No. of components†			
Adults ≥20 y of age	0–1	2–3	4–5
Children 5–19 y of age	0–1	2–3	4–5
Total cholesterol, mg/dL			
Adults ≥20 y of age	≥240	200–239 or treated to goal	<200
Children 6–19 y of age	≥200	170–199	<170
Blood pressure			
Adults ≥20 y of age	SBP ≥140 mm Hg or DBP ≥90 mm Hg	SBP120–139 mm Hg or DBP 80–89 mm Hg or treated to goal	<120 mm Hg/<80 mm Hg
Children 8–19 y of age	>95th percentile	90th–95th percentile or SBP ≥120 mm Hg or DBP ≥80 mm Hg	<90th percentile
Fasting plasma glucose, mg/dL			
Adults ≥20 y of age	≥126	100–125 or treated to goal	<100
Children 12–19 y of age	≥126	100–125	<100

AHA indicates American Heart Association; BMI, body mass index; PA, physical activity; SBP, systolic blood pressure; and DBP, diastolic blood pressure.

*Represents appropriate energy balance, ie, appropriate dietary quantity and PA to maintain normal body weight.

†In the context of a healthy dietary pattern that is consistent with a Dietary Approaches to Stop Hypertension [DASH]–type eating pattern, to consume ≥4.5 cups/d of fruits and vegetables, ≥2 servings/wk of fish, and ≥3 servings/d of whole grains and no more than 36 oz/wk of sugar-sweetened beverages and 1500 mg/d of sodium.

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Table 2-2. Prevalence of US Population With Ideal Cardiovascular Health and With Components of Ideal Cardiovascular Health, Overall and in Selected Age Strata From NHANES 2007–2008 and 2009–2010

	Prevalence, %				
	Ages 12–19 y	Ages ≥20 y*	Ages 20–39 y	Ages 40–59 y	Ages ≥60 y
2007–2008 (baseline)					
Ideal CV health profile (composite–all 7)	0.0	0.0	0.0	0.0	0.0
≥6 Ideal CV health composite score	8.2	3.6	7.1	2.1	0.1
≥5 Ideal CV health composite score	39.8	15.8	29.7	9.7	2.9
Ideal health factors index (composite–all 4)	35.5	13.9	27.7	7.3	1.0
Individual components					
Total cholesterol <200 mg/dL (untreated)	69.6	46.3	64.1	37.1	29.9
SBP <120 mm Hg and DBP <80 mm Hg (untreated)	82.3	43.8	63.8	36.9	14.6
Not current smoker (never or quit ≥12 mo)	83.7	72.9	66.4	72.9	86.1
Fasting blood glucose <100 mg/dL	76.2	52.0	67.4	45.6	31.9
Ideal health behaviors index (composite–all 4)	0.0	0.1	0.1	0.0	0.0
Individual components					
PA at goal	39.0	39.5	45.6	36.4	33.7
Not current smoker (never or quit ≥12 mo)	83.7	72.9	66.4	72.9	86.1
BMI <25 kg/m^2	62.5	31.9	39.1	28.0	25.3
4–5 Diet goals met†	0.0	0.3	0.3	0.1	0.5
Fruits and vegetables ≥4.5 cups/d	7.9	12.3	11.7	11.4	15.8
Fish ≥2 3.5-oz servings/wk (preferably oily fish)	9.2	18.3	16.8	19.7	19.4
Sodium <1500 mg/d	0.0	0.6	0.6	0.8	0.3
Sugar-sweetened beverages ≤450 kcal/wk	32.0	51.9	41.0	54.6	71.2
Whole grains (1.1 g fiber/10 g carb) ≥3 1-oz equivalents/d	3.2	7.3	7.0	7.1	8.4
Other dietary measures					
Nuts, legumes, seeds ≥4 servings/wk	8.7	21.7	19.6	22.5	24.7
Processed meats ≤2 servings/wk	56.3	57.6	54.0	59.7	61.1
Saturated fat <7% of total energy intake (kcal)	4.5	8.7	9.3	8.0	9.0
2009–2010					
Ideal CV health profile (composite–all 6)‡	17.4	4.4	8.1	2.6	0.6
≥5 Ideal CV health composite score	47.2	17.2	29.8	10.7	5.8
Ideal health factors index (composite–all 4)	48.4	15.9	29.7	9.2	2.4
Individual components					
Total cholesterol <200 mg/dL (untreated)	69.1	47.3	67.8	36.8	29.4
SBP <120 mm Hg and DBP <80 mm Hg (untreated)	85.8	44.3	64.3	40.7	15.8
Not current smoker (never or quit ≥12 mo)	85.2	76.2	69.5	75.5	88.3
Fasting blood glucose <100 mg/dL	88.2	57.4	73.5	54.0	35.7
Ideal health behaviors index (composite–all 3)‡	42.1	17.8	21.4	15.8	14.9
Individual components					
PA at goal	36.1	41.1	46.5	40.8	33.3
Not current smoker (never or quit ≥12 mo)	85.2	76.2	69.5	75.5	88.3
BMI <25 kg/m^2	64.2	31.3	38.6	27.5	25.4

NHANES indicates National Health and Nutrition Examination Survey; CV, cardiovascular; SBP, systolic blood pressure; DBP, diastolic blood pressure; PA, physical activity; and BMI, body mass index.

*Standardized to the age distribution of the 2000 US Standard population.

†Scaled for 2000 kcal/d and in the context of intake with appropriate energy balance and a DASH (Dietary Approaches to Stop Hypertension)–like eating plan.

‡Dietary data for the 2009–2010 cycle of NHANES were not available at the time of this analysis.

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Table 2-3. Selected Secondary Metrics for Monitoring CVD, NHANES 2009–2010*

	In the Presence of CVD		In the Absence of CVD	
	N†	% (SE)‡	N†	% (SE)‡
Total	16 209 474	7.2 (0.4)	199 590 596	92.8 (0.4)
CHD	6 916 012	3.2 (0.3)		
Stroke	5 717 759	2.7 (0.2)		
CHF	4 320 227	2.0 (0.3)		
Acute MI	6 929 905	3.2 (0.3)		
Health behaviors				
Smoking				
Current smoker or smokers who quit <12 mo ago	3 127 273	37.2 (4.9)	40 760 066	20.1 (0.9)
PA				
PA: intermediate or poor§	11 813 011	74.1 (5.1)	115 561 988	58.4 (1.5)
PA: none	10 598 908	64.5 (5.5)	93 459 556	47.3 (1.2)
Diet, no. of metrics*				
Total diet score 0–3 of 5	12 665 860	100.00 (0.00)	161 370 154	99.71 (0.11)
Total diet score 0–1 of 5	9 540 532	70.06 (4.69)	127 156 293	78.84 (1.42)
Overweight/obesity				
Overweight or obese (BMI ≥25.0 kg/m2)	12 621 701	69.4 (4.1)	134 879 713	68.1 (1.3)
Obese (BMI ≥30.0 kg/m2)	7 763 611	49.0 (5.2)	68 655 702	34.7 (1.0)
Health factors				
Hypertension				
Prevalence of BP ≥140/90 mm Hg or taking medications	10 591 170	51.0 (5.0)	53 523 895	28.5 (0.9)
Awareness among those with hypertension	10 071 343	98.6 (0.3)	42 436 782	70.6 (3.2)
Treatment those with hypertension	9 819 244	97.4 (0.4)	39 194 948	61.4 (2.9)
BP control to <140/<90 mm Hg among treated	6 886 176	64.2 (9.5)	27 323 649	72.4 (2.4)
BP control to <140/<90 mm Hg among hypertensive	6 886 176	62.3 (9.4)	27 323 649	43.3 (2.8)
Hypercholesterolemia				
Prevalence of total cholesterol ≥240 mg/dL or taking medications	8 201 829	37.1 (4.2)	48 701 198	25.7 (0.7)
Awareness among those with hypercholesterolemia	7 742 127	84.6 (8.0)	35 174 931	59.9 (2.6)
Treatment among those with hypercholesterolemia	7 219 078	79.3 (8.5)	25 405 334	38.7 (2.4)
Cholesterol control to <200 mg/dL among treated	6 659 732	95.0 (1.4)	22 804 724	90.4 (1.2)
Cholesterol control to <200 mg/dL among hypercholesterolemia	6 659 732	75.0 (8.9)	22 804 724	34.7 (2.4)
Diabetes mellitus				
Prevalence of fasting glucose ≥125 mg/dL or taking medications	4 769 759	15.2 (2.2)	21 078 443	10.3 (1.1)
Awareness among diabetics	4 006 153	90.4 (2.3)	14 242 760	64.3 (4.6)
Treatment among diabetics	3 935 446	87.1 (3.2)	13 391 291	58.4 (5.3)
Blood glucose control among treated	1 527 151	32.6 (9.9)	5 878 676	45.0 (8.0)
Blood glucose control among diabetics	1 527 151	27.2 (8.7)	5 878 676	25.5 (5.9)

CVD indicates cardiovascular disease; NHANES, National Health and Nutrition Examination Survey; SE, standard error; CHD, coronary heart disease; CHF, congestive heart failure; MI, myocardial infarction; PA, physical activity; BMI, body mass index; and BP, blood pressure.

*Dietary data are based on 2007–2008, the most recent data available at the time of this analysis.

†Weighted sample size.

‡Standardized to the age distribution of the 2000 US Standard population.

§Moderate <150 min/wk AND Vigorous <75 min/wk AND Combined <150 min/wk.

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Table 2-4. Evidence-Based Individual Approaches for Improving Health Behaviors and Health Factors in the Clinic Setting

- Set specific goals (Class IA). Set specific, proximal goals with the patient, including a personalized plan to achieve the goals (eg, over the next 3 mo, increase fish by 1 serving/wk, reduce smoking by half a pack per day, or walk 30 min 3 times per week).
- Establish self-monitoring (Class IA). Develop a strategy for self-monitoring, such as a dietary or physical activity diary or Web-based or mobile applications.
- Schedule follow-up (Class IA). Schedule regular follow-up (in-person, telephone, written, and/or electronic), with clear frequency and duration of contacts, to assess success, reinforce progress, and set new goals as necessary.
- Provide feedback (Class IA). Provide feedback on progress toward goals, including using in person, telephone, and/or electronic feedback.
- Increase self-efficacy (Class IA). Increase the patient's perception that they can successfully change their behavior.*
- Use motivational interviewing† (Class IA). Use motivational interviewing when patients are resistant or ambivalent about behavior change.
- Provide long-term support (Class IB). Arrange long-term support from family, friends, or peers for behavior change, such as in other workplace, school, or community-based programs.
- Use a multicomponent approach (Class IA). Combine 2 or more of the above strategies into the behavior change efforts.

*Examples of approaches include mastery experiences (set a reasonable, proximal goal that the person can successfully achieve); vicarious experiences (have the person see someone with similar capabilities performing the behavior, such as walking on a treadmill or preparing a healthy meal); physiological feedback (explain to the patient when a change in their symptoms is related to worse or improved behaviors); and verbal persuasion (persuade the person that you believe in their capability to perform the behavior).

†Motivational interviewing represents use of individual counseling to explore and resolve ambivalence toward changing behavior. Major principles include fostering the person's own awareness and resolution of their ambivalence, as well as their own self-motivation to change, in a partnership with the counselor or provider.

Table adapted from Artinian et al.[10]

Table 2-5. Evidence-Based Healthcare Systems Approaches to Support and Facilitate Improvements in Health Behaviors and Health Factors[11-15]

- Electronic systems for scheduling and tracking initial visits and regular follow-up contacts for behavior change and treatments.
- Electronic medical records systems to help assess, track, and report on specific health behaviors (diet, PA, tobacco, body weight) and health factors (BP, cholesterol, glucose), as well as to provide feedback and the latest guidelines to providers.
- Practical paper or electronic toolkits for assessment of key health behaviors and health factors, including during, before, and after provider visits.
- Electronic systems to facilitate provision of feedback to patients on their progress during behavior change and other treatment efforts.
- Education and ongoing training for providers on evidence-based behavior change strategies, as well as the most relevant behavioral targets, including training on relevant ethnic and cultural issues.
- Integrated systems to provide coordinated care by multidisciplinary teams of providers, including physicians, nurse practitioners, dieticians, PA specialists, and social workers.
- Reimbursement guidelines and incentives that reward efforts to change health behaviors and health factors.Restructuring of practice goals and quality benchmarks to incorporate health behavior (diet, PA, tobacco, body weight) and health factor (BP, cholesterol, glucose) interventions and targets for both primary and secondary prevention.

BP indicates blood pressure; PA, physical activity.

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Table 2-6. Summary of Evidence-Based Population Approaches for Improving Diet, Increasing Physical Activity, and ReducingTobacco Use*

Diet	
Media and education	Sustained, focused media and educational campaigns, using multiple modes, for increasing consumption of specific healthful foods or reducing consumption of specific less healthful foods or beverages, either alone (IIa B) or as part of multicomponent strategies (I B)†‡§
	On-site supermarket and grocery store educational programs to support the purchase of healthier foods (IIa B)†
Labeling and information	Mandated nutrition facts panels or front-of-pack labels/icons as a means to influence industry behavior and product formulations (IIa B)†
Economic incentives	Subsidy strategies to lower prices of more healthful foods and beverages (I A)†
	Tax strategies to increase prices of less healthful foods and beverages (IIa B)†
	Changes in both agricultural subsidies and other related policies to create an infrastructure that facilitates production, transportation, and marketing of healthier foods, sustained over several decades (IIa B)†
Schools	Multicomponent interventions focused on improving both diet and physical activity, including specialized educational curricula, trained teachers, supportive school policies, a formal PE program, healthy food and beverage options, and a parental/family component (I A)†
	School garden programs, including nutrition and gardening education and hands-on gardening experiences (IIa A)† Fresh fruit and vegetable programs that provide free fruits and vegetables to students during the school day (IIa A)†
Workplaces	Comprehensive worksite wellness programs with nutrition, physical activity, and tobacco cessation/prevention components (IIa A)†
	Increased availability of healthier food/beverage options and/or strong nutrition standards for foods and beverages served, in combination with vending machine prompts, labels, or icons to make healthier choices (IIa B)†
Local environment	Increased availability of supermarkets near homes (IIa B)†‡

(*Continued*)

Table 2-6. (Continued)

Restrictions and mandates	Restrictions on television advertisements for less healthful foods or beverages advertised to children (I B)†
	Restrictions on advertising and marketing of less healthful foods or beverages near schools and public places frequented by youths (IIa B)†
	General nutrition standards for foods and beverages marketed and advertised to children in any fashion, including on-package promotion (IIa B)†
	Regulatory policies to reduce specific nutrients in foods (eg, *trans* fats, salt, certain fats) (I B)†§
Physical activity	
Labeling and information	Point-of-decision prompts to encourage use of stairs (IIa A)†
Economic incentives	Increased gasoline taxes to increase active transport/commuting (IIa B)†
Schools	Multicomponent interventions focused on improving both diet and physical activity, including specialized educational curricula, trained teachers, supportive school policies, a formal PE program, serving of healthy food and beverage options, and a parental/family component (IIa A)†
	Increased availability and types of school playground spaces and equipment (I B)†
	Increased number of PE classes, revised PE curricula to increase time in at least moderate activity, and trained PE teachers at schools (IIa A/IIb A¶)†
	Regular classroom physical activity breaks during academic lessons (IIa A)†§
Workplaces	Comprehensive worksite wellness programs with nutrition, physical activity, and tobacco cessation/prevention components (IIa A)†
	Structured worksite programs that encourage activity and also provide a set time for physical activity during work hours (IIa B)†
	Improving stairway access and appeal, potentially in combination with "skip-stop" elevators that skip some floors (IIa B)†
	Adding new or updating worksite fitness centers (IIa B)†
Local environment	Improved accessibility of recreation and exercise spaces and facilities (eg, building of parks and playgrounds, increasing operating hours, use of school facilities during nonschool hours) (IIa B)†
	Improved land-use design (eg, integration and interrelationships of residential, school, work, retail, and public spaces) (IIa B)†
	Improved sidewalk and street design to increase active commuting (walking or bicycling) to school by children (IIa B)†
	Improved traffic safety (IIa B)†
	Improved neighborhood aesthetics (to increase activity in adults) (IIa B)†
	Improved walkability, a composite indicator that incorporates aspects of land-use mix, street connectivity, pedestrian infrastructure, aesthetics, traffic safety, and/or crime safety (IIa B)†
Smoking	
Media and education	Sustained, focused media and educational campaigns to reduce smoking, either alone (IIa B) or as part of larger multicomponent population-level strategies (I A)†
Labeling and information	Cigarette package warnings, especially those that are graphic and health related (I B)†‡§
Economic incentives	Higher taxes on tobacco products to reduce use and fund tobacco control programs (I A)†‡§
Schools and workplaces	Comprehensive worksite wellness programs with nutrition, physical activity, and tobacco cessation/prevention components (IIa A)†
Local environment	Reduced density of retail tobacco outlets around homes and schools (I B)†
	Development of community telephone lines for cessation counseling and support services (I A)†
Restrictions and mandates	Community (city, state, or federal) restrictions on smoking in public places (I A)†
	Local workplace-specific restrictions on smoking (I A)†‡§
	Stronger enforcement of local school-specific restrictions on smoking (IIa B)†
	Local residence-specific restrictions on smoking (IIa B)†§
	Partial or complete restrictions on advertising and promotion of tobacco products (I B)†

PE indicates physical education.

*The specific population interventions listed here are either a Class I or IIa recommendation with an evidence grade of either A or B. The American Heart Association evidence grading system for class of recommendation and level of evidence is summarized in Table 2. Because implementation of population-level strategies does not require perfect evidence but rather consideration of risks versus benefits, associated costs, and alternate approaches, the absence of any specific strategy herein does not mean it should not also be considered for implementation. See the more detailed tables and text below for further information on the evidence for each of these interventions, as well as other strategies that were reviewed.

†At least some evidence from studies conducted in high-income Western regions and countries (eg, North America, Europe, Australia, New Zealand).

‡At least some evidence from studies conducted in high-income non-Western regions and countries (eg, Japan, Hong Kong, South Korea, Singapore).

§At least some evidence from studies conducted in low- or middle-income regions and countries (eg, Africa, China, Pakistan, India).

||Based on cross-sectional studies only; only 2 longitudinal studies have been performed, with no significant relations seen.

¶Evidence IIa A for improving physical activity; evidence IIb B for reducing adiposity.

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Table 2-7. Reduction in BP Required to Increase Prevalence of Ideal BP Among Adults ≥20 y of Age; NHANES 2009–2010

	%
Percent BP ideal among adults, 2009–2010	44.26
20% Relative increase	53.11
Percent of US adults whose BP would be ideal if population mean BP were lowered by*	
2 mm Hg	56.13
3 mm Hg	59.49
4 mm Hg	61.59
5 mm Hg	65.31

Standardized to the age distribution of the 2000 US standard population.

BP indicates blood pressure; NHANES, National Health and Nutrition Examination Survey.

*Reduction in BP=(observed average systolic−X mm Hg) AND (observed average diastolic−X mm Hg).

Table 2-8. AHA Advocacy and Policy Strategies Related to the 2020 Impact Goals for Ideal Cardiovascular Health

Measure of Cardiovascular Health	Advocacy/Policy Solutions
Smoking Status Ideal for cardiovascular health: Adults: Never smoked or quit more than a year ago Children: Never tried or never smoked a whole cigarette	Federal • Support the full, authorized funding level for the FDA's Center for Tobacco Products and advocate for comprehensive implementation of FDA regulation of tobacco. • Implement clinical guidance and monitor health claims concerning smokeless tobacco and other "harm reduction" products. • Support the Tobacco Tax Equity Act that closes tax loopholes to ensure that all tobacco products are taxed at levels similar to the current tax rate for cigarettes. • Continue to advocate for ratification of WHO's Framework Convention of Tobacco Control as part of the UN Political Declaration on Non-Communicable Diseases for implementation by all countries who are a party to the treaty. State • Establish, strengthen, and protect smoke-free air laws in compliance with the Fundamentals of Smokefree Workplace Laws guidelines. • Support tobacco-free secondary school, college, university, and hospital campuses. • Support significant increases in tobacco excise taxes on all tobacco products. • Establish and protect sustainable funding for tobacco prevention and cessation programs to levels that meet or exceed the CDC recommendations. • Provide comprehensive tobacco cessation benefits in Medicaid, Medicare, and private health insurance plans. • Eliminate tobacco sales in pharmacies and other health-related institutions.
Physical Activity Ideal for cardiovascular health: Adults: At least 150 min of moderate or 75 min of vigorous PA each week Children: >60 min of moderate-vigorous PA per day	Federal • Preserve funding for Safe Routes to School and Complete Streets in Transportation Reauthorization. • Include PA in nutrition education funding for the Farm Bill Supplemental Nutrition Assistance Program. • Incorporate PA into electronic medical records. • Support implementation of the National Physical Activity Plan. • Increase the quality of physical education in schools and advocate for Physical Education for Progress grants to increase funding to schools to improve their PE programs. • Advocate for regular revision and update of the Physical Activity Guidelines for Americans. State • Implement shared use of school facilities within the community and support the construction of school fitness facilities. • Increase sports, recreational opportunities, parks, and green spaces in the community. • Support efforts to design workplaces, communities, and schools around active living and integrate PA opportunities throughout the day. • Provide safe routes to schools and school sites that offer walking/biking options for more students. • Support the creation of complete streets. • Support the use of zoning policy to increase access to safe places for recreation. • Create and maintain comprehensive worksite wellness programs. • Support the creation and implementation, through legislation and regulation (including licensing), of PA standards for preschool, day care, and other out-of-school care programs. • Require quality, more frequent physical education in schools. • Promote efforts within the school environment that will lead to increased PA.

(Continued)

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Table 2-8. Continued

Measure of Cardiovascular Health	Advocacy/Policy Solutions
BMI Ideal for cardiovascular health: Adults: between 18.5 and 25 kg/m^2 Children: between the 15th and 85th percentile Go to www.americanheart.org/obesitypolicy for additional policy resources	Federal • Provide obesity counseling and treatment coverage in the healthcare environment. • Provide robust surveillance and monitoring of obesity, diet, PA, and tobacco use. State • Provide robust coverage for guidelines-based prevention, diagnosis, and treatment of overweight and obesity in the healthcare environment. • Implement and monitor strong local wellness policies in all schools. • Ensure adequate funding and implementation of coordinated school health programs. • Establish comprehensive obesity prevention strategies in early childhood and day care programs. • Advocate for continued funding for obesity prevention research and work to ensure a strong evaluation component is a part of implementation of new laws and programs.
Healthy Diet Ideal for cardiovascular health: In the context of a DASH-type dietary pattern, adults and children should achieve at least 4 of the 5 following key components of a healthy diet: • Fruits and vegetables: >4.5 cups/d • Fish: More than two 3.5-oz servings/wk (preferably oily fish) • Fiber-rich whole grains (>1.1 g of fiber per 10 g of carbohydrates): three 1-oz-equivalent servings per day • Sodium: <1500 mg/d • Sugar-sweetened beverages: <450 kcal (36 oz) per week Go to www.americanheart.org/obesitypolicy for more specific policy resources	Federal • Work to eliminate food deserts and improve access and affordability of healthy foods. • Strengthen nutrition standards in schools for meals and competitive foods and in all government nutrition assistance or feeding programs. • Improve food labeling to make the labels easier to read and convey more accurately the content of added sugars, *trans* fats, sodium, and whole grains in foods. • Implement menu labeling in restaurants. • Continue to support and monitor the removal of industrially produced trans fats from the food supply and ensure the use of healthy replacement oils. • Restrict the marketing and advertising of unhealthy food to children. • Support robust implementation of nutrition education and promotion in schools. • Reduce added sugar and sodium in the food supply. • Support the implementation and dissemination of procurement standards across federal agencies. • Ensure that diet counseling is a covered benefit in Medicare. State • Support the implementation of the reauthorization of the Federal Childhood Nutrition Act and new regulations concerning competitive foods and beverages and use all available techniques, including legislation, to encourage schools to take advantage of opportunities to provide even healthier options for children. • Support improvements in the school food environment just outside of school property, including corner stores and food trucks • Support the creation and implementation of nutrition standards, through legislation and regulation (including licensing), for preschool and day care and other out-of-school care program meals. • Support opportunities for greater nutrition education in schools. Support opportunities to expand the availability of fruits, vegetables, and water, including policies that support expansion of school gardens and farm-to-school programs. • Support strategies that reduce sodium in the food supply. • Reduce *trans* fats in packaged foods, baked goods, restaurant meals, and school meal programs. • Support the elimination of food deserts through policies such as Healthy Food Financing that increase the availability of fruits, vegetables, and water in underserved neighborhoods. • Support the establishment of food procurement policies that meet the AHA or federal guidelines for government offices. • Support policies identified to reduce children's exposure to marketing and advertising for unhealthy food. • Support policies that change relative prices of healthy versus unhealthy food items. • Support on a pilot basis the taxation of sugar-sweetened beverages to assess impact on health and consumer behavior, including 6 minimum criteria (at least a portion of the money is dedicated for HD and stroke prevention and/or obesity prevention, the tax is structured so as to result in an increase in price for sugar-sweetened beverages, tax is at least 1 cent/oz, there is money dedicated for evaluation with guidance that ensures rigorous evaluation including health outcomes, there is a standard definition of "sugar-sweetened beverage," and there is no sunset). • Support policies designed to encourage retailers to increase access to healthy foods while decreasing access to unhealthy foods. • Expand state participation in the Department of Defense Fresh Fruit and Vegetable program.
Total Cholesterol Ideal for cardiovascular health: Adults: Total cholesterol <200 mg/dL Children: <170 mg/dL	Federal and State • Partner with Department of Health and Human Services to promote the Million Hearts Campaign through increased public awareness and partnership engagement, science and evaluation, clinical care improvement, patient outreach, and public policy. • Ensure adequate healthcare coverage for prevention and treatment of dyslipidemia. • Secure and protect dedicated state appropriations aligned with HD and stroke priorities and work to support appropriate program implementation. Support other public health initiatives and evaluation targeted at HD, stroke, and related risk factors, as well as the disparities that exist in these areas.

(Continued)

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Table 2-8. Continued

Measure of Cardiovascular Health	Advocacy/Policy Solutions
Blood Pressure Ideal for cardiovascular health: Adults: <120/80 mm Hg Children: <90th percentile	Federal • Partner with the Department of Health and Human Services to promote the Million Hearts Campaign, as above. • Implement the Institute of Medicine's recommendations to reduce sodium in the food supply. • Improve food labeling to increase consumer understanding of sodium levels in packaged foods. • Advocate for robust sodium limits in procurement standards, nutrition standards in schools, and other government feeding programs. State • Promote public funding for Heart Disease and Stroke Prevention Programs. • Ensure the availability of essential CVD preventive benefits in private insurance and public health programs.
Fasting Plasma Glucose Ideal for cardiovascular health: Children and Adults: Fasting blood glucose <100 mg/dL	Federal and State • Ensure adequate healthcare coverage for early treatment and prevention of diabetes mellitus.

For AHA advocacy resources, including fact sheets, policy briefs, published papers, and position statements, go to http://www.heart.org/HEARTORG/Advocate/PolicyResources/Policy-Resources_UCM_001135_SubHomePage.jsp.

AHA indicates American Heart Association; FDA, Food and Drug Administration; WHO, World Health Organization; UN, United Nations; CDC, Centers for Disease Control and Prevention; PA, physical activity; PE, physical education; DASH, Dietary Approaches to Stop Hypertension; HD, heart disease; and CVD, cardiovascular disease.

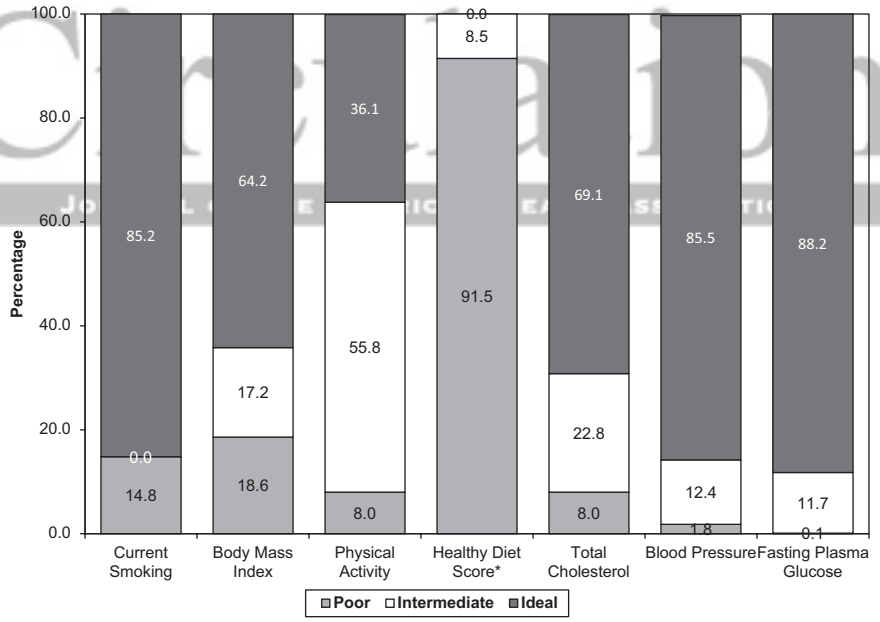

Chart 2-1. Prevalence (unadjusted) estimates for poor, intermediate, and ideal cardiovascular health for each of the 7 metrics of cardiovascular health in the American Heart Association 2020 goals, US children aged 12 to 19 years, National Health and Nutrition Examination Survey (NHANES) 2009-2010* (available data as of June 1, 2012). *Healthy Diet Score reflects 2007-2008 NHANES data.

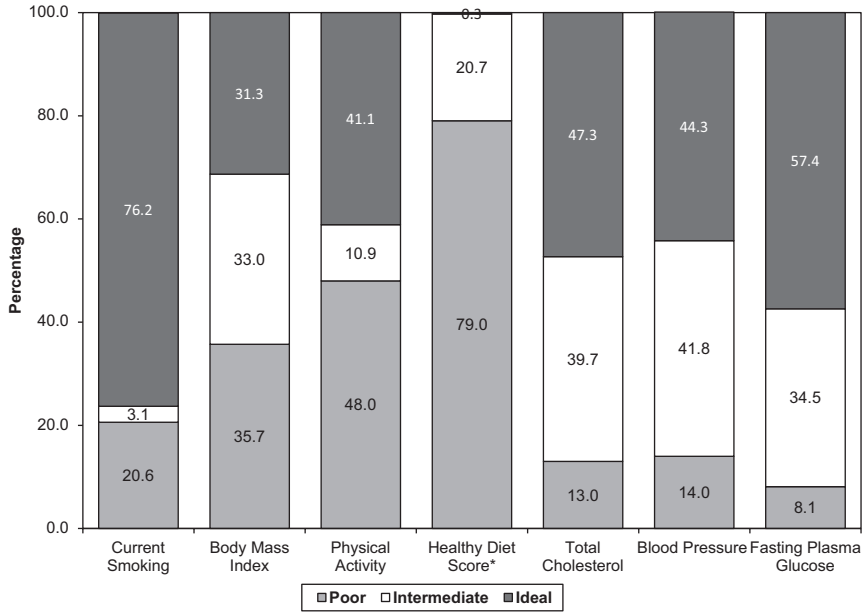

Chart 2-2. Age-standardized prevalence estimates for poor, intermediate, and ideal cardiovascular health for each of the 7 metrics of cardiovascular health in the American Heart Association 2020 goals, among US adults aged ≥20 years, National Health and Nutrition Examination Survey (NHANES) 2009-2010* (available data as of June 1, 2012). *Healthy Diet Score reflects 2007-2008 NHANES data

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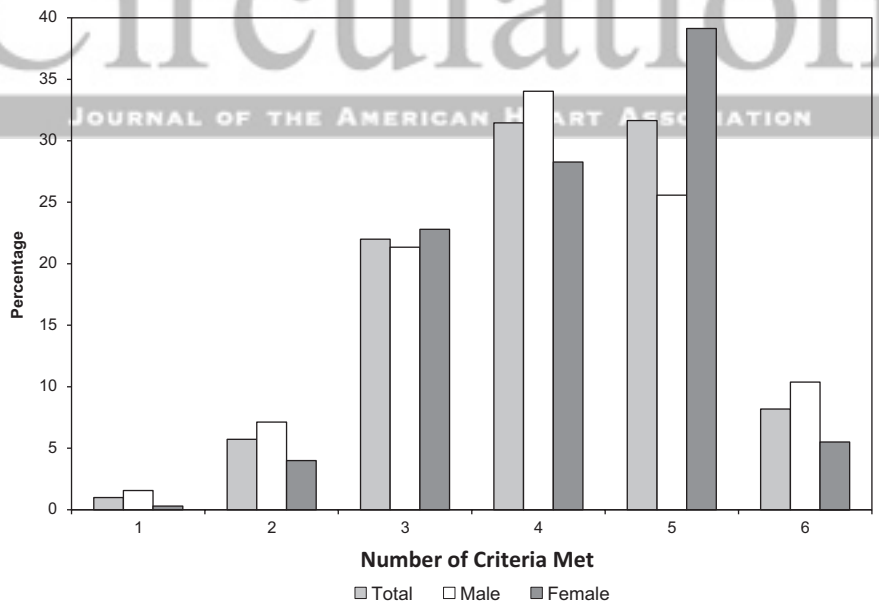

Chart 2-3. Proportion (unadjusted) of US children aged 12 to 19 years meeting different numbers of criteria for Ideal Cardiovascular Health, overall and by sex, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2012).

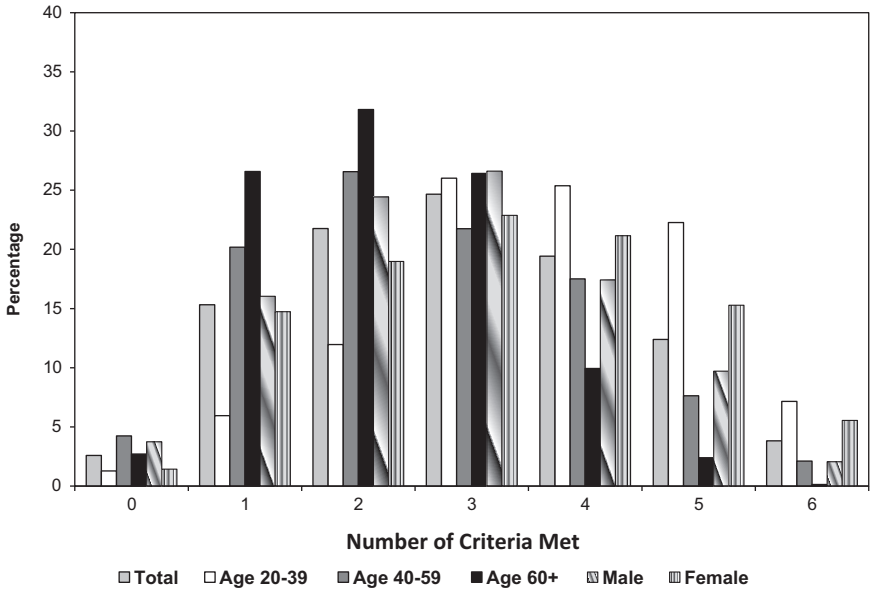

Chart 2-4. Age-standardized prevalence estimates of US adults aged ≥20 years meeting different numbers of criteria for Ideal Cardiovascular Health, overall and by age and sex subgroups, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2012).

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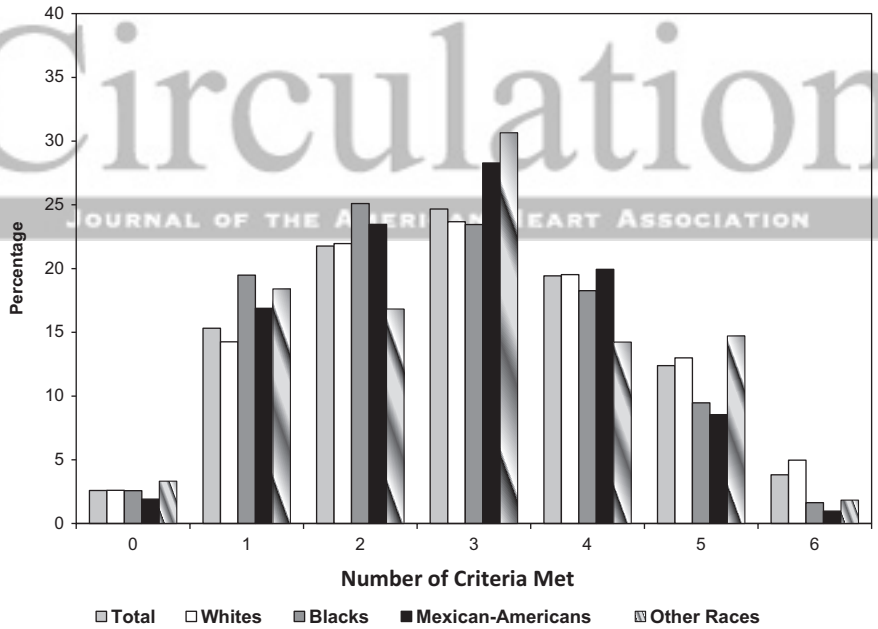

Chart 2-5. Age-standardized prevalence estimates of US adults aged ≥20 years meeting different numbers of criteria for Ideal Cardiovascular Health, overall and in selected race subgroups from National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2012).

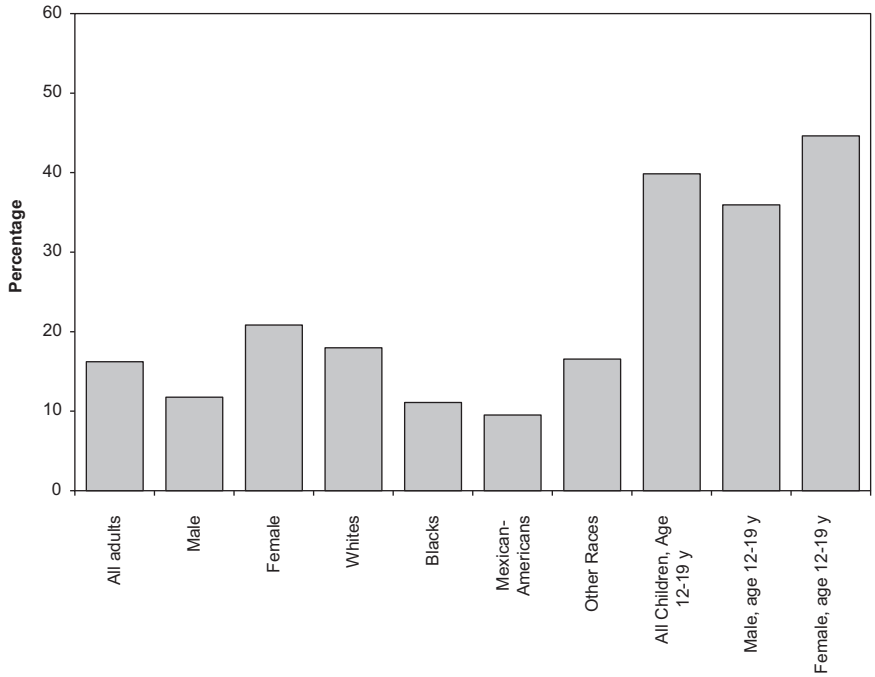

Chart 2-6. Prevalence estimates of meeting at least 5 criteria for Ideal Cardiovascular Health, US adults aged ≥20 years (age-standardized), overall and by sex and race, and US children aged 12 to 19 years (unadjusted), by sex, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2012).

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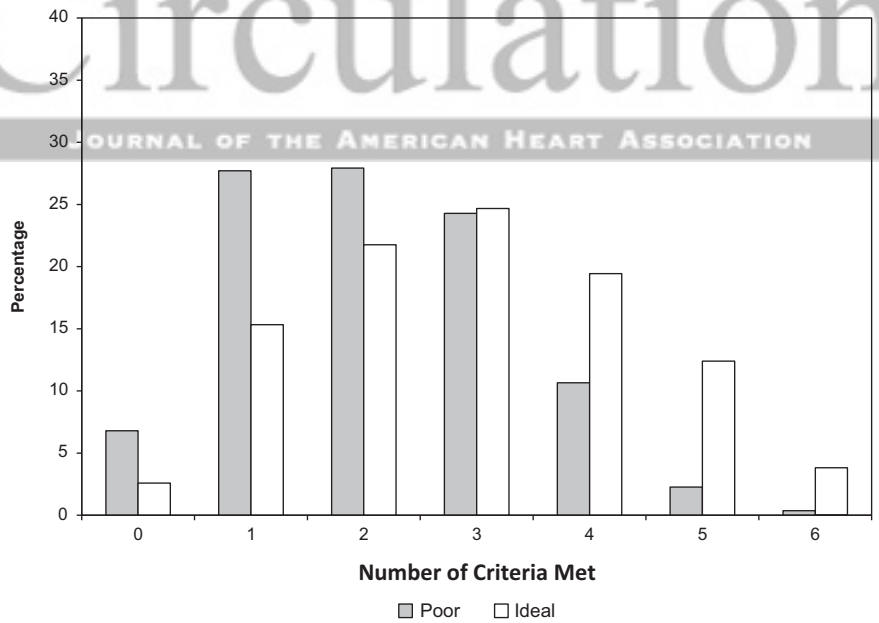

Chart 2-7. Age-standardized prevalence estimates of US adults meeting different numbers of criteria for Ideal and Poor Cardiovascular Health, for each of the 7 metrics of cardiovascular health in the American Heart Association 2020 goals, among US adults aged ≥20 years, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2012).

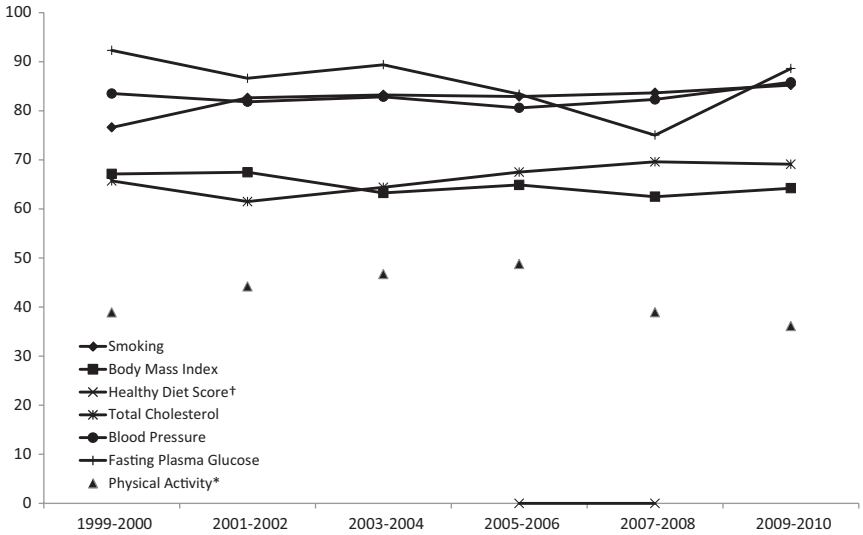

Chart 2-8. Trends in prevalence (unadjusted) of meeting criteria for Ideal Cardiovascular Health, for each of the 7 metrics of cardiovascular health in the American Heart Association 2020 goals, among US children aged 12 to 19 years, National Health and Nutrition Examination Survey (NHANES) 1999-2000 through 2009-2010*† (available data as of June 1, 2012). *Due to changes in the physical activity questionnaire between different cycles of the NHANES survey, trends over time for this indicator should be interpreted with caution and statistical comparisons should not be attempted. †Data for the Healthy Diet Score, based on a 2-day average intake, was only available for the 2003-2004, 2005-2006, and 2007-2008 NHANES cycles at the time of this analysis.

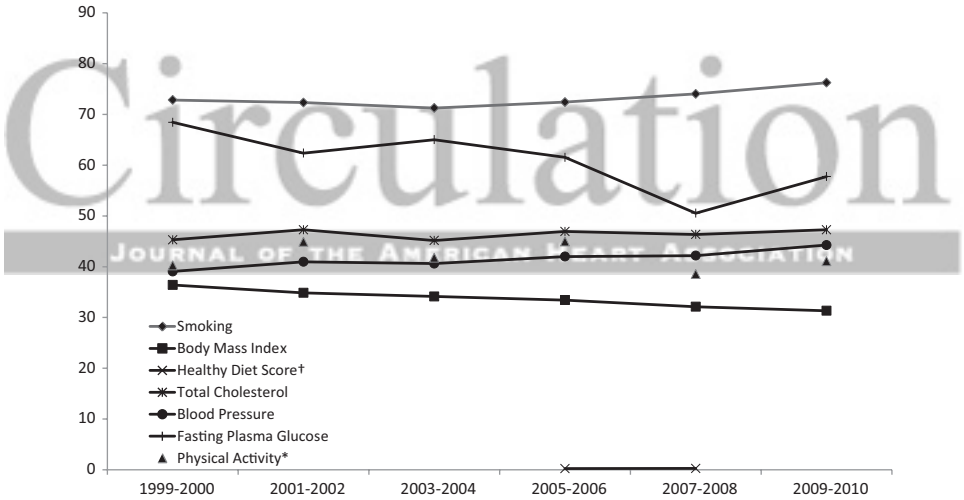

Chart 2-9. Age-standardized trends in prevalence of meeting criteria for Ideal Cardiovascular Health, for each of the 7 metrics of cardiovascular health in the American Heart Association 2020 goals, among US adults aged ≥20 years, National Health and Nutrition Examination Survey (NHANES) 1999-2000 through 2009-2010 (available data as of June 1, 2012). *Due to changes in the physical activity questionnaire between different cycles of the NHANES survey, trends over time for this indicator should be interpreted with caution and statistical comparisons should not be attempted. †Data for the Healthy Diet Score, based on a 2-day average intake, was only available for the 2003-2004, 2005-2006, and 2007-2008 NHANES cycles at the time of this analysis.

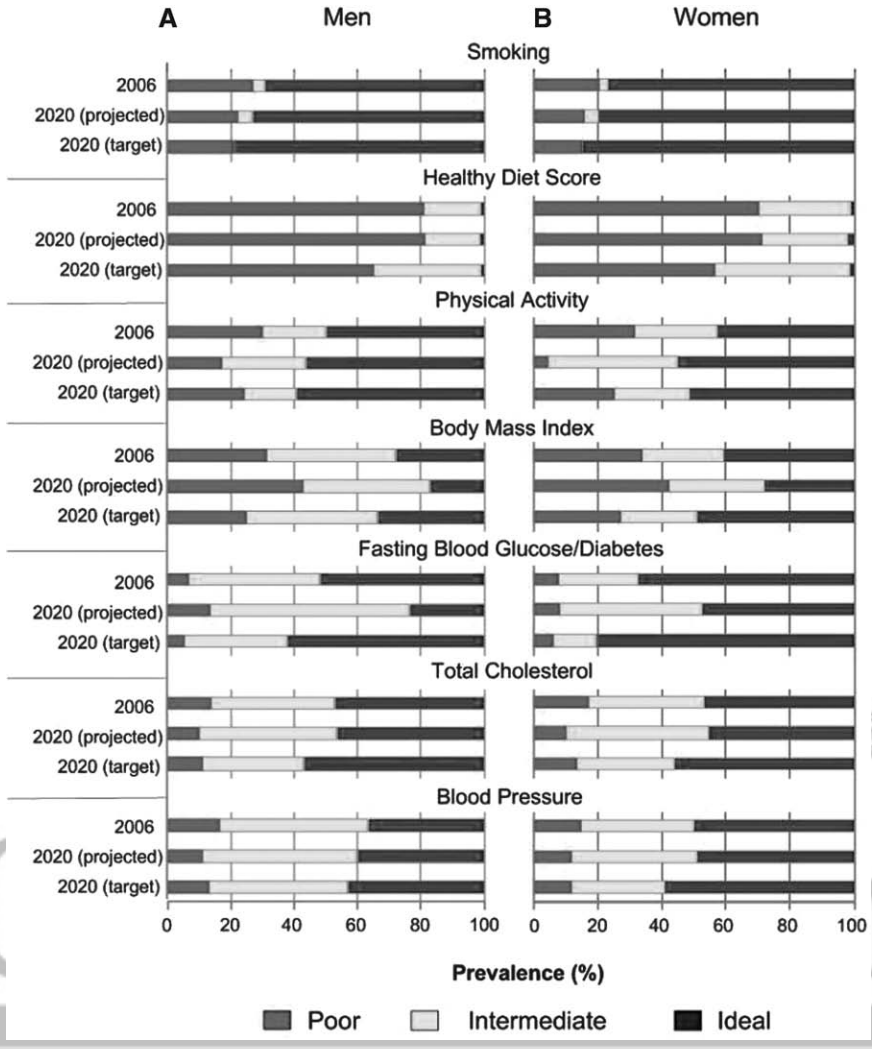

Chart 2-10. Prevalence of ideal, intermediate, and poor CV health metrics in 2006 (AHA 2020 Impact Goals baseline year) and 2020 projections assuming current trends continue. 2020 targets for each CV health metric, assuming a 20% relative increase in ideal CV health prevalence metrics and a 20% relative decrease in poor CV health prevalence metrics for men and women. Reprinted from Huffman et al[2] with permission. Copyright © 2012, American Heart Association.

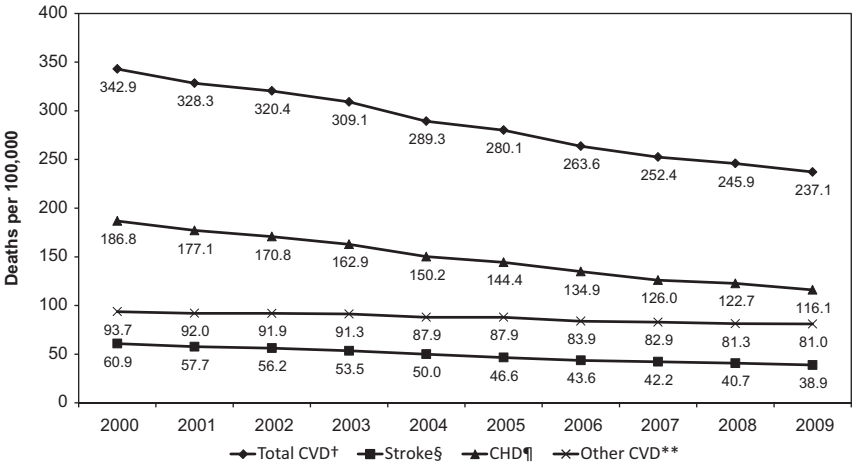

Chart 2-11. US age-standardized death rates* from cardiovascular diseases, 2000-2009. *Directly standardized to the age distribution of the 2000 US standard population. †Total CVD (Cardiovascular Disease): ICD-10 I00-I99, Q20-Q28. §Stroke (All cerebrovascular disease): ICD-10 I60-I69. ¶CHD (Coronary Heart Disease): ICD-10 I20-I25. **Other CVD: ICD-10 I00 –I15, I26 –I51, I70 –I78, I80 –I89, I95–I99. Source: Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999-2009 on CDC WONDER Online Database, released 2012. Data for year 2009 are compiled from the Multiple Cause of Death File 2009, Series 20 No. 2O, 2012, data for year 2008 are compiled from the Multiple Cause of Death File 2008, Series 20 No. 2N, 2011, data for year 2007 are compiled from the Multiple Cause of Death File 2007, Series 20 No. 2M, 2010, data for years 2005-2006 data are compiled from Multiple Cause of Death File 2005-2006, Series 20, No. 2L, 2009, and data for years 1999-2004 are compiled from the Multiple Cause of Death File 1999-2004, Series 20, No. 2J, 2007. http://wonder.cdc.gov/mcd-icd10.html. Accessed October 9, 2012.

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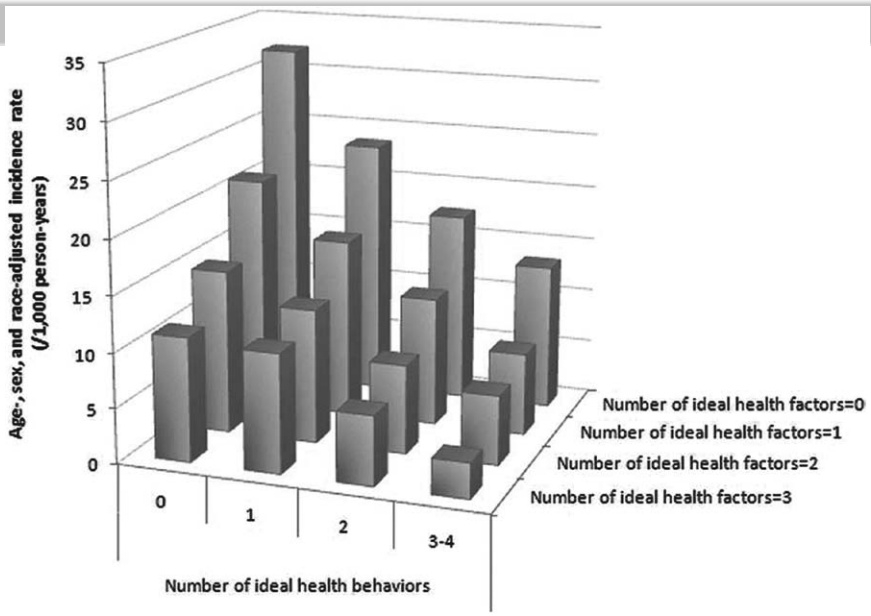

Chart 2-12. Incidence of cardiovascular disease according to the number of ideal health behaviors and health factors. Reprinted with permission from Folsom et al.[4]

3. Smoking/Tobacco Use

See Table 3-1 and Charts 3-1 and 3-2.

Prevalence

Youth

(See Chart 3-1.)

- In 2011, in grades 9 through 12:
  - — 18.1% of students reported current cigarette use (on at least 1 day during the 30 days before the survey), 13.1% of students reported current cigar use, and 7.7% of students reported current smokeless tobacco use. Overall, 23.4% of students reported any current tobacco use (Youth Risk Behavior Survey [YRBS]; Chart 3-1).[1]
  - — Male students were more likely than female students to report current cigarette use (19.9% compared with 16.1%). Male students were also more likely than female students to report current cigar use (17.8% compared with 8.0%) and current smokeless tobacco use (12.8% compared with 2.2%; YRBS).[1]
  - — Non-Hispanic white students were more likely than Hispanic or non-Hispanic black students to report any current tobacco use, which includes cigarettes, cigars, or smokeless tobacco (26.5% compared with 20.5% for Hispanic students and 15.4% for non-Hispanic black students; YRBS).[1]
- Among youths 12 to 17 years of age in 2010, 2.6 million (10.7%) used a tobacco product (cigarettes, cigars, or smokeless tobacco) in the past month, and 2.0 million (8.3%) used cigarettes. Cigarette use in the past month in this age group declined significantly from 13.0% in 2002 to 8.3% in 2010 (National Survey on Drug Use and Health [NSDUH]).[2]
- Data from the YRBS[3] for students in grades 9 to 12 indicated the following:
  - — The percentage of students who reported ever trying cigarettes remained stable from 1991 to 1999 and then declined from 70.4% in 1999 to 44.7% in 2011.
  - — The percentage who reported current cigarette use (on at least 1 day in the 30 days before the survey) increased between 1991 and 1997 and then declined from 36.4% in 1997 to 18.1% in 2011.
  - — The percentage who reported current frequent cigarette use (smoked on ≥20 of the 30 days before the survey) increased from 1991 to 1999 and then declined from 16.8% in 1999 to 6.4% in 2011.
- In 2011, 49.9% of students in grades 9 to 12 who currently smoked cigarettes had tried to quit smoking cigarettes during the previous 12 months. The prevalence of this behavior was higher among female student smokers (53.9%) than among male student smokers (47.0%) and among white females (54.0%) and Hispanic females (55.9%) than among white males (46.3%) and Hispanic males (44.7%; YRBS).[1]

Adults

(See Table 3-1 and Chart 3-2.)

- In 2011, among adults ≥18 years of age:
  - — 21.3% of men and 16.7% of women were current cigarette smokers (NHIS).[5]
  - — The percentage of current cigarette smokers (19.0%) declined 21% since 1998 (24.1%).[5–7]
  - — The states with the highest percentage of current cigarette smokers were Kentucky (29.0%), West Virginia (28.6%), and Arkansas (27.0%). Utah has the lowest percentage of smokers (11.8%) (BRFSS).[4]
- In 2010, an estimated 69.6 million Americans ≥12 years of age were current (past month) users of a tobacco product (cigarettes, cigars, smokeless tobacco, or tobacco in pipes). The rate of current use of any tobacco product in this age range declined from 2007 to 2010 (from 28.6% to 27.4%; NSDUH).[2]
- From 1998 to 2007, cigarette smoking prevalence among adults ≥18 years of age decreased in 44 states and the District of Columbia. Six states had no substantial changes in prevalence after controlling for age, sex, and race/ethnicity (BRFSS).[8]
- In 2008 to 2010, among people ≥65 years of age, 9.4% of men and 9.1% of women were current smokers. In this age group, men were more likely than women to be former smokers (54.0% compared with 30.0%) on the basis of age-adjusted estimates (NHIS).[9]
- In 2008 to 2010, among adults ≥18 years of age, Asian men (15.2%) and Hispanic men (17.3%) were less likely to be current cigarette smokers than non-Hispanic black men (23.7%), non-Hispanic white men (23.9%), and American Indian or Alaska Native men (24.6%) on the basis of age-adjusted estimates (NHIS). Similarly, in 2008 to 2010, Asian women (5.5%) and Hispanic women (9.6%) were less likely to be current cigarette smokers than non-Hispanic black women (17.6%), non-Hispanic white women (20.9%), and American Indian or Alaska Native women (20.7%; NHIS).[9]
- In 2004 to 2006 data, adult cigarette smoking varied among Asian subgroups. Most Asian adults had never smoked, with rates ranging from 65% of Korean adults to 84% of Chinese adults. Korean adults (22%) were approximately 2 to 3 times as likely to be current smokers as Japanese (12%), Asian Indian (7%), or Chinese (7%) adults on the basis of age-adjusted estimates (NHIS).[10]
- In 2009 to 2010, among women 15 to 44 years of age, past-month cigarette use was lower for those who were pregnant (16.3%) than among those who were not pregnant

Abbreviations Used in Chapter 3

AMI	acute myocardial infarction
BRFSS	Behavioral Risk Factor Surveillance System
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
MEPS	Medical Expenditure Panel Survey
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NSDUH	National Survey on Drug Use and Health
RR	relative risk
YRBS	Youth Risk Behavior Survey

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(26.7%). This pattern was found for women 18 to 25 years of age (22.7% versus 31.2% for pregnant and nonpregnant women, respectively) and for women 26 to 44 years of age (11.8% versus 27.0%, respectively). Among adolescents 15 to 17 years of age, past-month cigarette use was higher for those who were pregnant (22.7%) than for those who were not pregnant (13.4%; NSDUH).[2]

Incidence

- In 2010:
  - ≈2.4 million people ≥12 years of age smoked cigarettes for the first time within the past 12 months, which was similar to the estimate in 2009 (2.5 million). The 2010 estimate averages out to ≈6500 new cigarette smokers every day. Most new smokers (58.8%) in 2010 were <18 years of age when they first smoked cigarettes (NSDUH).[2]
  - The number of new smokers <18 years of age (1.4 million) is similar to that in 2002 (1.3 million); however, new smokers ≥18 years of age increased from ≈600 000 in 2002 to 1 million in 2010 (NSDUH).[2]
  - Among people ages 12 to 49 years of age who had started smoking within the past 12 months, the average age of first cigarette use was 17.3 years, similar to the average in 2009 (17.5 years).[2]
- Data from 2002 to 2004 suggest that ≈1 in 5 nonsmokers 12 to 17 years of age is likely to start smoking. Youths in the Mexican subpopulations were significantly more susceptible (28.8%) to start smoking than those in non-Hispanic white (20.8%), non-Hispanic black (23.0%), Cuban (16.4%), Asian Indian (15.4%), Chinese (15.3%), and Vietnamese (13.8%) subpopulations. There was no significant difference in susceptibility to start smoking between boys and girls in any of the major populations or subpopulations (NSDUH).[11]

Morbidity

A 2010 report of the US Surgeon General on how tobacco causes disease summarizes an extensive body of literature on smoking and CVD and the mechanisms through which smoking is thought to cause CVD. Among its conclusions are the following:

- There is a sharp increase in CVD risk with low levels of exposure to cigarette smoke, including secondhand smoke, and a less rapid further increase in risk as the number of cigarettes per day increases.
- A meta-analysis comparing pooled data of ≈2.4 million smokers and nonsmokers found the relative risk (RR) ratio of smokers to nonsmokers for developing CHD was 25% higher in women than in men (95% CI, 1.12–1.39).[12]
- Current smokers have a 2 to 4 times increased risk of stroke compared with nonsmokers or those who have quit for >10 years.[13,14]

Mortality

- In 2005, tobacco smoking was the cause of ≈467 000 adult deaths (19.1%) in the United States. Approximately one third of these deaths were related to CVD.[15]
- During 2000 to 2004, ≈49 000 (11.1%) of cigarette smoking–related deaths were attributable to secondhand smoke.[16]
- Each year from 2000 to 2004, smoking caused 3.1 million years of potential life lost for males and 2.0 million years for females, excluding deaths attributable to smoking-attributable residential fires and adult deaths attributable to secondhand smoke.[16]
- From 2000 to 2004, smoking during pregnancy resulted in an estimated 776 infant deaths annually.[16]
- During 2000 to 2004, cigarette smoking resulted in an estimated 269 655 deaths annually among males and 173 940 deaths annually among females.[16]
- On average, male smokers die 13.2 years earlier than male nonsmokers, and female smokers die 14.5 years earlier than female nonsmokers.[17]

Smoking Cessation

- Smoking cessation reduces the risk of cardiovascular morbidity and mortality for smokers with and without CHD.
  - There is no evidence to date that reducing the amount smoked by smoking fewer cigarettes per day reduces the risk of CVD.[18]
- In 2008, 64.5% of adult current smokers ≥18 years of age with a checkup during the preceding year reported that they had been advised to quit, which was not significantly different from 2002 (63.1%). Smokers between 18 and 44 years of age were less likely to be advised to quit than those at older ages. Women were more likely than men to receive advice to quit smoking (MEPS).[19]

Secondhand Smoke

- Data from a 2006 report of the US Surgeon General on the consequences of involuntary exposure to tobacco smoke[20] indicate the following:
  - Nonsmokers who are exposed to secondhand smoke at home or at work increase their risk of developing CHD by 25% to 30%.
  - Short exposures to secondhand smoke can cause blood platelets to become stickier, damage the lining of blood vessels, and decrease coronary flow velocity reserves, potentially increasing the risk of an acute MI (AMI).
- In 2008, data from 11 states showed that the majority of people surveyed in each state reported having smoke-free home rules, ranging from 68.8% in West Virginia to 85.6% in Arizona (BRFSS).[21]
- As of December 31, 2010, 25 states and the District of Columbia had laws that prohibited smoking in indoor areas of worksites, restaurants, and bars; no states had such laws in 2000. As of December 31, 2010, an additional 10 states had laws that prohibited smoking in 1 or 2 but not all 3 venues.[22]

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- Pooled data from 17 studies in North America, Europe, and Australasia suggest that smoke-free legislation can reduce the incidence of acute coronary events by 10%.[23]
- The percentage of the US nonsmoking population with detectable serum cotinine declined from 52.5% in 1999 to 2000 to 40.1% in 2007 to 2008, with declines occurring for children and adults. During 2007 to 2008, the percentage of nonsmokers with detectable serum cotinine was higher for those 3 to 11 years of age (53.6%) and those 12 to 19 years of age (46.5%) than for those ≥20 years of age (36.7%); the percentage was also higher for non-Hispanic blacks (55.9%) than for non-Hispanic whites (40.1%) and Mexican Americans (28.5%; NHANES).[24]

Cost

- Direct medical costs ($96 billion) and lost productivity costs ($97 billion) associated with smoking totaled an estimated $193 billion per year between 2000 and 2004.[16]
- In 2008, $9.94 billion was spent on marketing cigarettes in the United States.[25]
- Cigarette prices have increased 283% between the early 1980s and 2011, resulting in decreased sales from ≈30 million packs sold in 1982 to ≈14 million packs sold in 2011.[25]

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Table 3-1. Cigarette Smoking

Population Group	Prevalence, 2011 Age ≥18 y*[5]	Cost[16]
Both sexes	43 821 000 (19.0%)	$193 Billion per year
Males	24 138 000 (21.3%)	…
Females	19 683 000 (16.7%)	…
NH white males	22.8%	…
NH white females	19.7%	…
NH black males	23.3%	…
NH black females	15.1%	…
Hispanic or Latino males	16.2%	…
Hispanic or Latino females	8.3%	…
Asian only (both sexes)	9.6%	…
American Indian/Alaska Native only (both sexes)	26.7%	…

Percentages are age adjusted. Estimates for Asian only and American Indian/Alaska Native only include non-Hispanic and Hispanic persons.

Ellipses (. . .) indicate data not available; NH, non-Hispanic.

*Rounded to the nearest thousand.

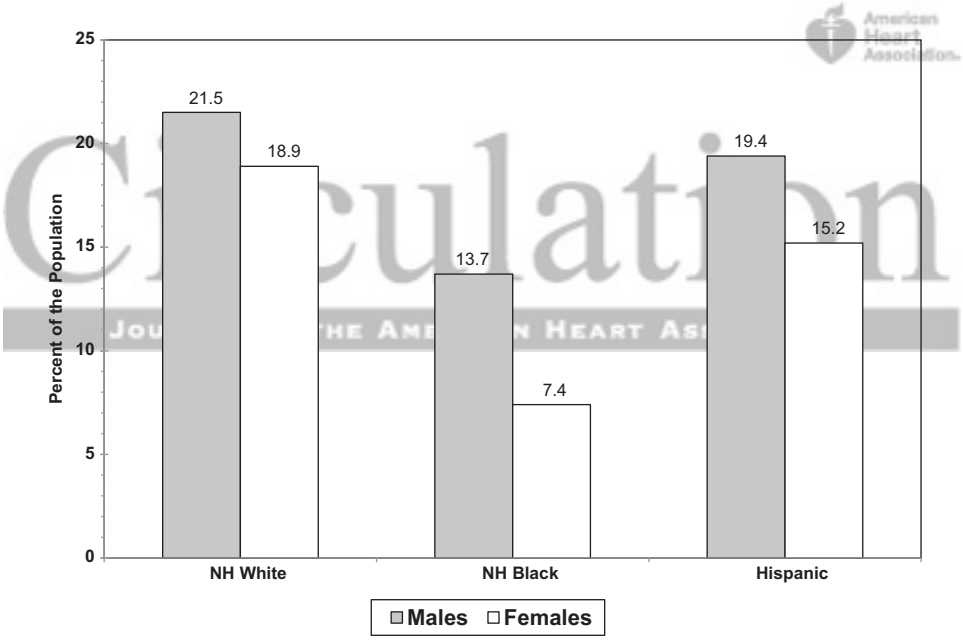

Chart 3-1. Prevalence (%) of students in grades 9 to 12 reporting current cigarette use by sex and race/ethnicity (Youth Risk Behavior Surveillance System, 2011). NH indicates non-Hispanic. Data derived from *MMWR: Morbidity and Mortality Weekly Report.*[1]

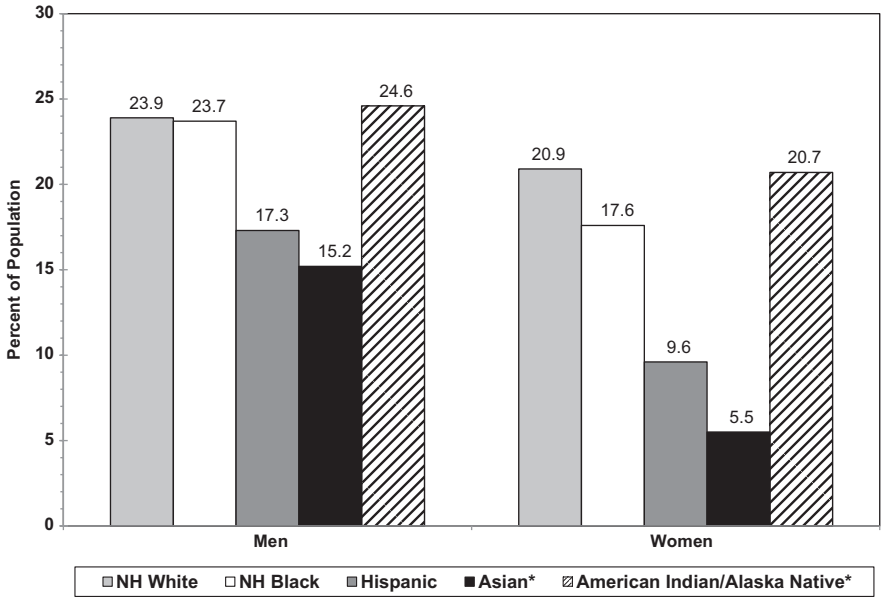

Chart 3-2. Prevalence (%) of current smoking for adults >18 years of age by race/ethnicity and sex (National Health Interview Survey: 2008–2010). All percentages are age-adjusted. NH indicates non-Hispanic. *Includes both Hispanics and non-Hispanics. Data derived from Centers for Disease Control and Prevention/National Center for Health Statistics, Health Data Interactive.[9]

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4. Physical Inactivity

See Table 4-1 and Charts 4-1 through 4-5.

Prevalence

Youth

Inactivity
(See Chart 4-1.)

- The proportion of adolescents (grades 9–12) who report engaging in no regular PA is high and varies by sex and race.[1]
- Nationwide, 13.8% of adolescents were inactive during the previous 7 days, as indicated by their response that they did not participate in ≥60 minutes of any kind of PA that increased their heart rate and made them breathe hard on any 1 of the previous 7 days.[1]
- Girls were more likely than boys to report inactivity (17.7% versus 10.0%).[1]
- The prevalence of inactivity was highest among black (26.7%) and Hispanic (21.3%) girls, followed by white girls (13.7%), black boys (12.3%), Hispanic boys (10.7%), and white boys (8.5%).[1]
- A study of 3068 youths between 14 and 24 years of age from 1999 to 2006 found that the prevalence of inactivity went up with age for both boys and girls. Across ages, girls had a higher prevalence of physical inactivity than boys.[2]
- In a study of 12 812 youth 9 to 18 years of age, the PA level in boys and girls declined starting at the age of 13, with a significantly greater decline in activity among girls.[3]

Television/Video/Computers
(See Chart 4-2.)

In 2011:

- Nationwide, 31.1% of adolescents used a computer for activities other than school work (eg, videogames or other computer games) for ≥3 hours per day on an average school day.[1]
- The prevalence of using computers or watching television ≥3 hours per day was highest among black (41.1%) and Hispanic boys (36.3%), followed by white boys (33.3%), black girls (35.2%), Hispanic girls (28.3%), and white girls (22.6%).[1]
- 32.4% of adolescents watched television for ≥3 hours per day.[1]
- The prevalence of watching television ≥3 hours per day was highest among black girls (54.9%) and boys (54.4%), followed by Hispanic boys (38.4%) and girls (37.2%) and white boys (27.3%) and girls (23.9%).[1]
- Increased television time has significant nutritional associations with weight gain (refer to Chapter 5, Nutrition).

Activity Recommendations
(See Charts 4-3 and 4-4.)

- In 2011, the proportion of students who met activity recommendations of at least 60 minutes of PA on 7 days of the week was 28.7% nationwide and declined from 9th (30.7%) to 12th (25.1%) grades. At each grade level, the proportion was higher in boys than in girls.[1]
- In 2011, more high school boys (38.3%) than girls (18.5%) self-reported having been physically active at least 60 minutes per day on all 7 days; self-reported rates of activity were higher in white (30.4%) than in black (26.0%) or Hispanic (26.5%) adolescents.[1]
- The 2010 National Youth Physical Activity and Nutrition Study showed that a total of 15.3% of high school students met the recommendations for aerobic activity, 51.0% met the recommendations for muscle-strengthening activity, and 12.2% met the recommendations for both aerobic and muscle-strengthening activities.[4]
- There was a marked discrepancy between the proportion of youth (ages 6–11 years) who reported engaging in ≥60 minutes of moderate-to-vigorous PA on most days of the week and those who actually engaged in moderate-to-vigorous PA for ≥60 minutes when activity was measured objectively with accelerometers (ie, portable motion sensors that record and quantify the duration and intensity of movements) in the NHANES 2003–2004 survey.[5]
- On the basis of accelerometer counts per minute >2020, 42% of 6- to 11-year-olds accumulated ≥260 minutes of moderate-to-vigorous PA on ≥5 days per week, whereas only 8% of 12-to 15-year-olds and 7.6% of 16- to 19-year-olds achieved similar counts.[5]
- More boys than girls met PA recommendations (≥60 minutes of moderate-to-vigorous activity on most days of the week) as measured by accelerometry.[5]

Abbreviations Used in Chapter 4

BP	blood pressure
CARDIA	Coronary Artery Risk Development in Young Adults
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
EF	ejection fraction
FMD	flow-mediated dilation
HbA_{1c}	hemoglobin A_{1c}
HBP	high blood pressure
HDL	high-density lipoprotein
HF	heart failure
HR	hazard ratio
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
PA	physical activity
PAD	peripheral artery disease
RR	relative risk
SBP	systolic blood pressure
WHO	World Health Organization

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Structured Activity Participation

- Despite recommendations from the National Association for Sport and Physical Education that schools should require daily physical education for students in kindergarten through 12th grade,[6] only 51.8% of students attended physical education classes in school daily (56.7% of boys and 46.7% of girls).[1]
- Physical education class participation declined from the 9th through the 12th grades among boys and girls.[1]
- Little more than half (58.4%) of high school students played on at least 1 school or community sports team in the previous year; however, the prevalence declined with increasing grade level, from 61.4% in the 9th grade to 52.5% in the 12th grade.[1]

Adults

Inactivity

According to 2011 data from the NHIS, in adults ≥18 years of age:

- Thirty-two percent do not engage in leisure-time PA ("no leisure-time PA/inactivity" refers to no sessions of light/moderate or vigorous PA of at least 10 minutes duration per day).[7]
- Inactivity was higher among women than men (33.2% versus 29.9%, age-adjusted) and increased with age from 26.1% to 33.4%, 40.0%, and 52.4% among adults 18 to 44, 45 to 64, 65 to 74, and ≥75 years of age, respectively.[7]
- Non-Hispanic black and Hispanic adults were more likely to be inactive (41.1% and 42.2%, respectively) than were non-Hispanic white adults (27.7%) on the basis of age-adjusted estimates.[7]

Activity Recommendations

(See Table 4-1 and Chart 4-5.)

According to 2011 data from the NHIS, in adults ≥18 years of age:

- 21.0% met the 2008 federal PA guidelines for both aerobic and strengthening activity, an important component of overall physical fitness.[7]
- The age-adjusted proportion who reported engaging in moderate or vigorous PA that met the 2008 aerobic Physical Activity Guidelines for Americans (at least 150 minutes of moderate PA or 75 minutes of vigorous PA or an equivalent combination each week) was 48.9%; 52.7% of men and 45.5% of women met the recommendations. Age-adjusted prevalence was 52.5% for non-Hispanic whites, 41.2% for non-Hispanic blacks, and 40.1% for Hispanics.[7]
- The proportion of respondents who did not meet the federal PA guidelines increased with age from 44.1% of 18- to 44-year-olds to 72.6% of adults ≥75 years of age.[7]
- Hispanic/Latino adults (59.8%) and non-Hispanic black adults (58.8%) were more likely to not meet the federal PA guidelines than non-Hispanic white (47.4%) adults, according to age-adjusted estimates.[7]
- The percentage of adults ≥25 years of age not meeting the full (aerobic and muscle-strengthening) federal PA guidelines was inversely associated with education; participants with no high school diploma (68.4%), a high school diploma (59.0%), some college (48.2%), or a bachelor's degree or higher (34.0%), respectively, did not meet the full federal PA guidelines.[7]
- The proportion of adults ≥25 years of age who met the 2008 federal PA guidelines for aerobic activity was positively associated with education level: 62.5% of those with a college degree or higher met the PA guidelines compared with 28.6% of adults with less than a high school diploma.[7]
- The proportion of adults reporting levels of PA consistent with the 2008 Physical Activity Guidelines for Americans remains low and decreases with age.[8,9] Thirty-three percent of respondents in a study examining awareness of current US PA guidelines had direct knowledge of the recommended dosage of PA (ie, frequency/duration).[10]
- The percentage of adults reporting at least 150 minutes of moderate PA or 75 minutes of vigorous PA or an equivalent combination weekly decreased with age from 55.8% for adults 18 to 44 years of age to 27.4% for those ≥75 years of age, on the basis of the 2011 NHIS.[9]
- The percentage of men who engaged in both leisure-time aerobic and strengthening activities decreased with age, from 39.8% at age 18 to 24 years to 11.1% at ≥75 years of age. The percentage of women who engaged in both leisure-time aerobic and strengthening activities also decreased with age, from 20.7% at age 18 to 24 years to 5.3% at ≥75 years of age, on the basis of the 2011 NHIS.[9]
- Using PA recommendations that existed at the time of the survey, adherence to PA recommendations was much lower when based on PA measured by accelerometer in NHANES 2003–2004[5]:
  - —Among adults 20 to 59 years of age, 3.8% of men and 3.2% of women met recommendations to engage in moderate-to-vigorous PA (accelerometer counts >2020/min) for 30 minutes (in sessions of ≥10 minutes) on ≥5 of 7 days.
  - —Among those ≥60 years of age, adherence was 2.5% in men and 2.3% in women.
- Accelerometry data from NHANES 2003–2006 showed that men engaged in 35 minutes of moderate activity per day, whereas for women, it was 21 minutes. More than 75% of moderate activity was accumulated in 1-minute bouts. Levels of activity declined sharply after the age of 50 years in all groups.[11]
- In a review examining self-reported versus actual measured PA (eg, accelerometers, pedometers, indirect calorimetry, double-labeled water, heart rate monitor), 60% of respondents self-reported higher values of activity than what was measured by use of direct methods.[12]
- Among men, self-reported PA was 44% greater than actual measured values; among women, self-reported activity was 138% greater than actual measured PA.[12]

Trends

Youth

In 2011:

- Among adolescents, there was a significant decrease in the prevalence of watching television ≥3 hours per day, from 42.8% in 1999 to 32.4%, although there was no significant decrease from the 2009 prevalence of 32.8%.[1]
- Among students nationwide, there was a significant increase in the prevalence of having participated in muscle-strengthening activities on ≥3 days per week, from 47.8% in 1991 to 55.6%.[1]

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- Nationwide, the prevalence of adolescents using computers ≥3 hours per day increased from 21.1% in 2005 to 24.9% in 2009 and 31.1%.[1]
- Among adolescents nationwide, the prevalence of attending physical education classes at least once per week did not increase significantly, from 48.9% in 1991 to 51.8%.[1]
- The prevalence of adolescents playing at least 1 team sport in the past year increased from 55.1% in 1999 to 58.4%.[1]

Adults

- Between NHANES III (1988–1994) and NHANES 2001–2006, the non–age-adjusted proportion of adults who engaged in >12 bouts of PA per month declined from 57.0% to 43.3% in men and from 49.0% to 43.3% in women.[13]
- The proportion of US adults who meet criteria for muscle strength has improved between 1998 and 2011. Annual estimates of the percentage of US adults who met the muscle-strengthening criteria increased from 17.7% in 1998 to 24.5% in 2011, and estimates of the percentage who met both the muscle-strengthening and aerobic criteria increased from 14.4% in 1998 to 21.0% in 2011.[7,8]
- A 2.3% decline in physical inactivity between 1980 and 2000 prevented or postponed ≈17 445 deaths (≈5%) attributable to CHD in the United States.[14]

CVD and Metabolic Risk Factors

Youth

- More girls (67.9%) than boys (55.7%) reported having exercised to lose weight or to keep from gaining weight.[1]
- White girls (72.2%) were more likely than black (54.2%) and Hispanic (66.3%) girls to report exercising to lose weight or to keep from gaining weight.[1]
- Total and vigorous PA are inversely correlated with body fat and the prevalence of obesity.[15]
- Among children 4 to 18 years of age, increased time in moderate-to-vigorous PA was associated with improvements in waist circumference, systolic BP (SBP), fasting triglycerides, high-density lipoprotein (HDL) cholesterol, and insulin. These findings were significant regardless of the amount of the children's sedentary time.[16]

Adults

- Participants in the Diabetes Prevention Project randomized trial who met the PA goal of 150 minutes of PA per week were 44% less likely to develop DM after 3.2 years of follow-up, even if they did not meet the weight-loss target.[17]
- Exercise for weight loss, without dietary interventions, was associated with significant reductions in diastolic BP (DBP; –2 mm Hg; 95% CI, –4 to –1 mm Hg), triglycerides (–0.2 mmol/L; 95% CI, –0.3 to –0.1 mmol/L), and fasting glucose (–0.2 mmol/L; 95% CI, –0.3 to –0.1 mmol/L).[18]
- A total of 120 to 150 minutes per week of moderate-intensity activity, compared with none, can reduce the risk of developing metabolic syndrome.[19]
- In Coronary Artery Risk Development in Young Adults (CARDIA), women who maintained high activity through young adulthood gained 6.1 fewer kilograms of weight and 3.8 fewer centimeters in waist circumference in middle age than those with lower activity. Highly active men gained 2.6 fewer kilograms and 3.1 fewer centimeters than their lower-activity counterparts.[20]
- Self-reported low lifetime recreational activity has been associated with increased PAD.[21]
- In 3 US cohort studies, men and women who increased their PA over time gained less weight in the long-term, whereas those who decreased their PA over time gained more weight and those who maintained their current PA had intermediate weight gain.[22]
- Among US men and women, every hour per day of increased television watching was associated with 0.3 pounds of greater weight gain every 4 years, whereas every hour per day of decreased television watching was associated with a similar amount of relative weight loss.[22]

Morbidity and Mortality

- Physical inactivity is responsible for 12.2% of the global burden of MI after accounting for other CVD risk factors such as cigarette smoking, DM, hypertension, abdominal obesity, lipid profile, no alcohol intake, and psychosocial factors.[23]
- In a meta-analysis of longitudinal studies among women, RRs of incident CHD were 0.83 (95% CI, 0.69–0.99), 0.77 (95% CI, 0.64–0.92), 0.72 (95% CI, 0.59–0.87), and 0.57 (95% CI, 0.41–0.79) across increasing quintiles of PA compared with the lowest quintile.[28]
- A 2003 meta-analysis of 23 studies on the association of PA with stroke indicated that compared with low levels of activity, high (RR, 0.79; 95% CI, 0.69–0.91) and moderate (RR, 0.91; 95% CI, 0.80–1.05) levels of activity were inversely associated with the likelihood of developing total stroke (ischemic and hemorrhagic).[24]
- With television watching as a sedentary activity, 2 hours of television per day is associated with an RR for type 2 DM of 1.20 (95% CI, 1.14–1.27), an RR for fatal or nonfatal CVD of 1.15 (95% CI, 1.06–1.23), and an RR for all-cause mortality of 1.13 (95% CI, 1.07–1.18). The risk for all-cause mortality further increases with >3 hours of television daily.[25]
- Longitudinal studies commonly report a graded, inverse association of PA amount and duration (ie, dose) with incident CHD and stroke.[26]
- The PA guidelines for adults cite evidence that ≈150 minutes per week of moderate-intensity aerobic activity, compared with none, can reduce the risk of CVD.[27]
- Adherence to PA guidelines for both aerobic and muscle-strengthening activities is associated with 27% lower all-cause mortality among adults without existing chronic conditions such as DM, cancer, MI, angina, CVD, stroke, or respiratory diseases and with 46% lower mortality among people with chronic comorbidities.[28]
- In the Health Professionals Follow-Up Study, for every 3-hour-per-week increase in vigorous-intensity activity, the multivariate RR of MI was 0.78 (95% CI, 0.61–0.98) for men. This 22% reduction of risk can be explained in part by beneficial effects of PA on HDL cholesterol, vitamin D, apolipoprotein B, and HbA_{1c}.[29]
- In a 20-year study of older male veterans, an inverse, graded, and independent association between impaired

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exercise capacity and all-cause mortality risk was found. For each increase of 1 metabolic equivalent tasks in exercise capacity, mortality risk was 12% lower (HR 0.88; 95% CI, 0.86–0.90). Unfit individuals who improved their fitness status had a 35% lower mortality risk (HR 0.65; 95% CI, 0.46–0.93) than those who remained unfit.[30]

Secondary Prevention

- PA improves inflammatory markers in people with existing stable CHD. After a 6-week training session, C-reactive protein levels declined by 23.7% ($P<0.001$), and plasma vascular cell adhesion molecule-1 levels declined by 10.23% ($P<0.05$); there was no difference in leukocyte count or levels of intercellular adhesion molecule-1.[31]
- In a randomized trial of patients with PAD, supervised treadmill exercise training and lower-extremity resistance training were each associated with significant improvements in functional performance and quality of life compared with a usual-care control group. Exercise training was additionally associated with improved brachial artery flow-mediated dilation (FMD), whereas resistance training was associated with better stair-climbing ability versus control.[32]
- On the basis of a meta-analysis of 34 randomized controlled trials, exercise-based cardiac rehabilitation after MI was associated with lower rates of reinfarction, cardiac mortality, and overall mortality.[33]
- The benefit of intense exercise training for cardiac rehabilitation in people with HF was tested in a trial of 27 patients with stable, medically treated HF. Intense activity (an aerobic interval-training program 3 times per week for 12 weeks) was associated with a significant 35% improvement in left ventricular ejection fraction (EF) and decreases in pro-brain natriuretic peptide (40%), left ventricular end-diastolic volume (18%), and left ventricular end-systolic volume (25%) compared with control and endurance-training groups.[34]

Costs

- The economic consequences of physical inactivity are substantial. In a summary of WHO data sources, the economic costs of physical inactivity were estimated to account for 1.5% to 3.0% of total direct healthcare expenditures in developed countries such as the United States.[35]
- The 1996 MEPS was linked to self-reported activity in the 1995 NHIS. On the basis of a self-reported prevalence of inactivity of 47.5% and a prevalence of CVD of 21.5%, direct expenditures for CVD associated with inactivity were estimated to be $23.7 billion in 2001.[36]

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Table 4-1. Met 2008 Federal PA Guidelines for Adults

Population Group	Prevalence, 2011 (Age ≥18 y), %
Both sexes	21.0
Males	24.9
Females	17.1
NH white only	23.0
NH black only	18.0
Hispanic or Latino	15.4
American Indian/Alaska Native only	17.0
Asian only	16.7

"Met 2008 Federal PA Guidelines for Adults" is defined as engaging in at least 150 minutes of moderate or 75 minutes of vigorous aerobic leisure-time PA per week (or an equivalent combination) and engaging in leisure-time strengthening PA at least twice a week.

Data are age-adjusted for adults ≥18 y of age.

PA indicates physical activity; NH, non-Hispanic.

Source: National Health Interview Survey 2011 (National Center for Health Statistics).[7]

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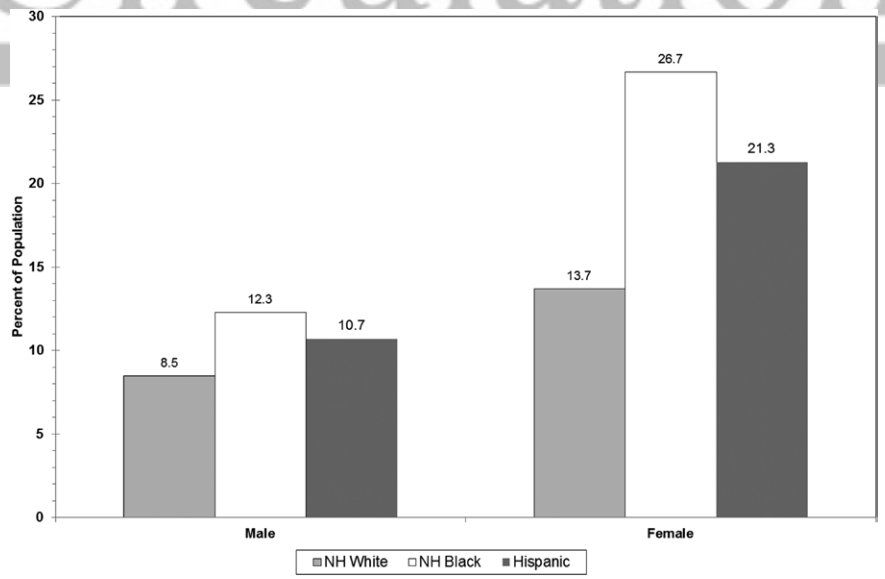

Chart 4-1. Prevalence of students in grades 9 to 12 who did not participate in at least 60 minutes of physical activity on any day by race/ethnicity and sex (Youth Risk Behavior Surveillance: 2011). NH indicates non-Hispanic. Data derived from *MMWR Surveillance Summaries.*[1]

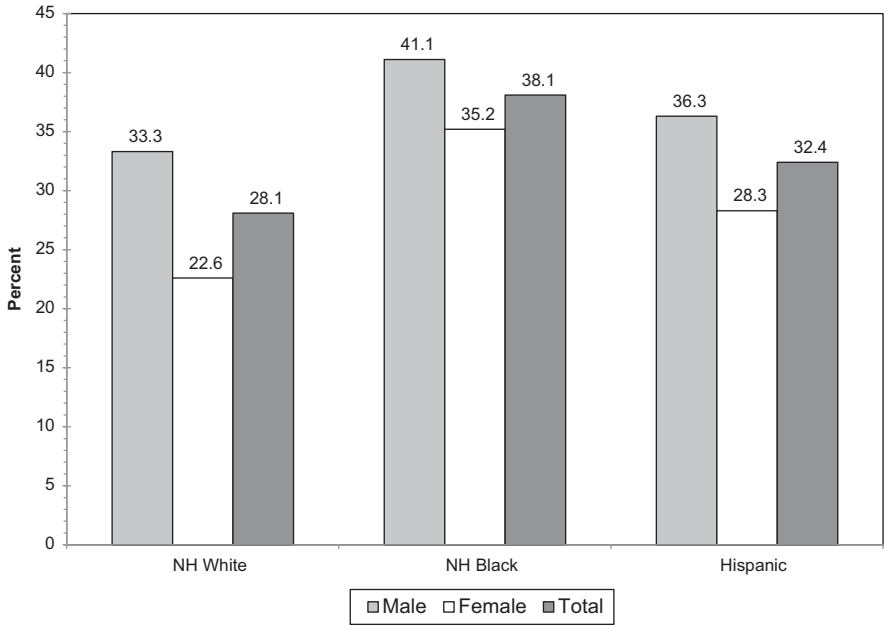

Chart 4-2. Percentage of students in grades 9 to 12 who used a computer for ≥3 hours a day by race/ethnicity and sex (Youth Risk Behavior Surveillance: 2011). NH indicates non-Hispanic. Data derived from *MMWR Surveillance Summaries.*[1]

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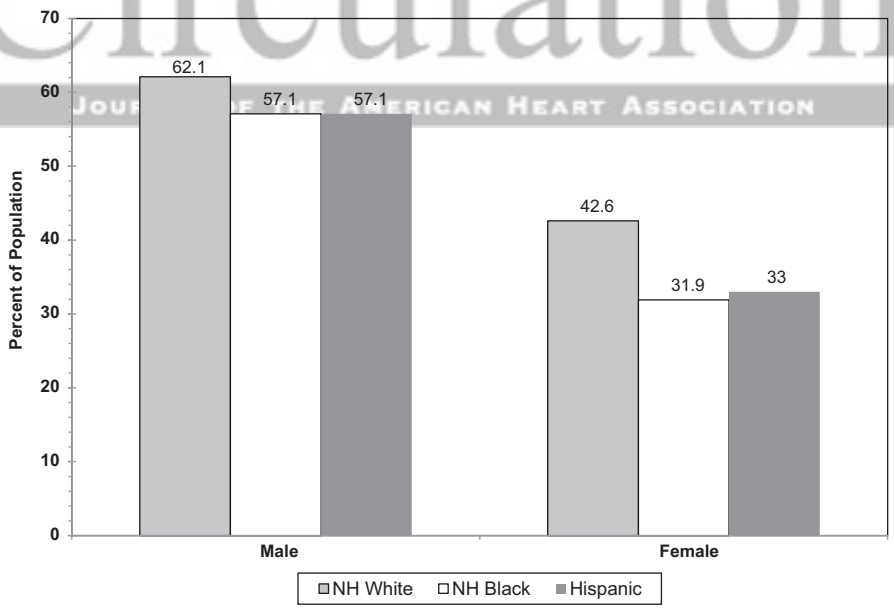

Chart 4-3. Prevalence of students in grades 9 to 12 who met currently recommended levels of physical activity during the past 7 days by race/ethnicity and sex (Youth Risk Behavior Surveillance: 2011). "Currently recommended levels" was defined as activity that increased their heart rate and made them breathe hard some of the time for a total of at least 60 minutes per day on 5 of the 7 days preceding the survey. NH indicates non-Hispanic. Data derived from *MMWR Surveillance Summaries.*[1]

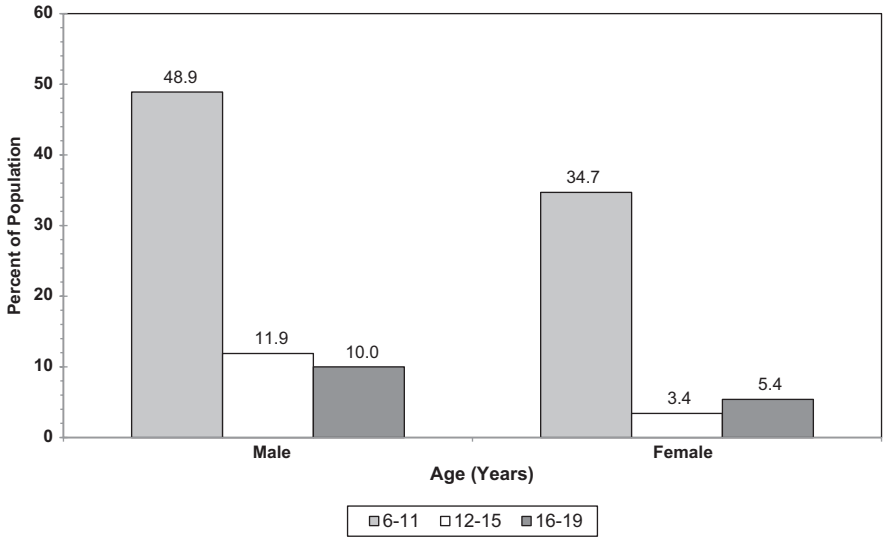

Chart 4-4. Prevalence of children 6 to 19 years of age who attained sufficient moderate-to-vigorous physical activity to meet public health recommendations (≥60 minutes per day on 5 or more of the 7 days preceding the survey), by sex and age (National Health and Nutrition Examination Survey: 2003–2004). Source: Troiano et al.[5]

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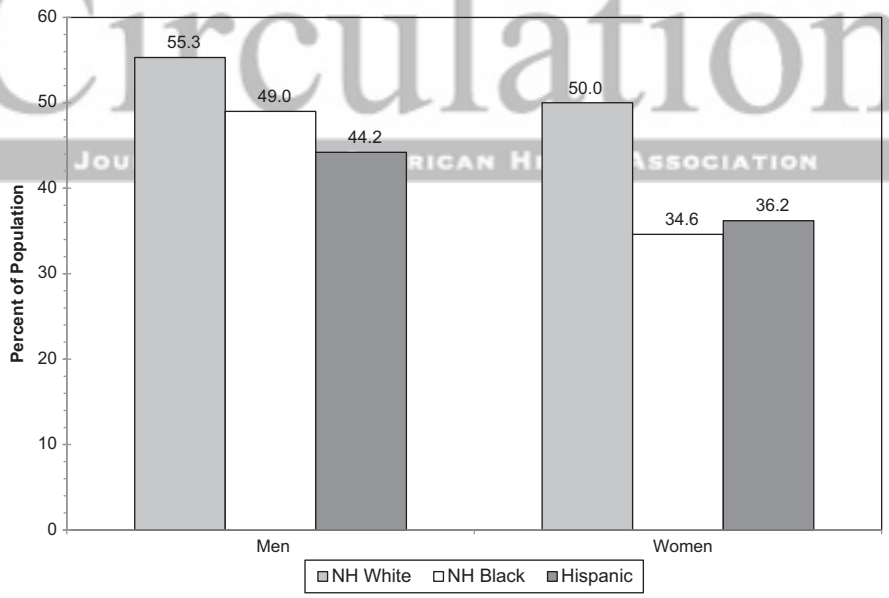

Chart 4-5. Prevalence of meeting the 2008 Federal Physical Activity Guidelines among adults ≥18 years of age by race/ethnicity and sex (National Health Interview Survey: 2010). NH indicates non-Hispanic. Percentages are age-adjusted. Meeting the 2008 Federal Physical Activity Guidelines is defined as engaging in moderate leisure-time physical activity for at least 150 minutes per week or vigorous activity at least 75 minutes per week or an equivalent combination. Source: Schiller et al.[7]

5. Nutrition

See Tables 5-1 and 5-2 and Charts 5-1 through 5-3.

This chapter of the update highlights national dietary consumption data, focusing on key foods, nutrients, dietary patterns, and other dietary factors related to cardiometabolic health. It is intended to examine current intakes, trends and changes in intakes, and estimated effects on disease to support and further stimulate efforts to monitor and improve dietary habits in relation to cardiovascular health.

Prevalence

Foods and Nutrients: Adults
See Table 5–1; NHANES 2005–2008).

The dietary consumption by US adults of selected foods and nutrients related to cardiometabolic health is detailed in Table 5-1 according to sex and race or ethnic subgroups:

Abbreviations Used in Chapter 5

ALA	α-linoleic acid
ARIC	Atherosclerosis Risk in Communities Study
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CHD	coronary heart disease
CHF	congestive heart failure
CI	confidence interval
CVD	cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	diastolic blood pressure
DHA	docosahexaenoic acid
DM	diabetes mellitus
EPA	eicosapentaenoic acid
GFR	glomerular filtration rate
GISSI	Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico
HD	heart disease
HDL	high-density lipoprotein
HEI	Healthy Eating Index
LDL	low-density lipoprotein
MI	myocardial infarction
n-6-PUFA	ω-6-polyunsaturated fatty acid
NA	not available
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
OR	odds ratio
PA	physical activity
PREMIER	Prospective Registry Evaluating Myocardial Infarction: Events and Recovery
RR	relative risk
SBP	systolic blood pressure
SD	standard deviation
WHI	Women's Health Initiative

- Average consumption of whole grains by white and black men and women was between 0.5 and 0.8 servings per day, with only between 3% and 5% of white and black adults meeting guidelines of ≥3 servings per day. Average whole grain consumption by Mexican Americans was ≈2 servings per day, with 21% to 27% consuming ≥3 servings per day.
- Average fruit consumption ranged from 1.1 to 1.8 servings per day in these sex and race or ethnic subgroups: 9% to 11% of whites, 6% to 7% of blacks, and 8% to 10% of Mexican Americans met guidelines of ≥2 cups per day. When 100% fruit juices were included, the number of servings consumed and the proportions of adults consuming ≥2 cups per day approximately doubled.
- Average vegetable consumption ranged from 1.3 to 2.2 servings per day; 6% to 7% of whites, 3% of blacks, and 3% of Mexican Americans consumed ≥2.5 cups per day. The inclusion of vegetable juices and sauces generally produced little change in these consumption patterns.
- Average consumption of fish and shellfish was lowest among white women (1.2 servings per week) and highest among black men and women (1.7 servings per week); ≈75% to 80% of all adults in each sex and race or ethnic subgroup consumed <2 servings per week. Approximately 10% to 13% of whites, 14% to 15% of blacks, and 12% of Mexican Americans consumed ≥250 mg of eicosapentaenoic acid and docosahexaenoic acid per day.
- Average consumption of nuts, legumes, and seeds was ≈2 to 3 servings per week among white and black men and women and 6 servings per week among Mexican American men and women. Approximately 20% of whites, 15% of blacks, and 40% of Mexican Americans met guidelines of ≥4 servings per week.
- Average consumption of processed meats was lowest among Mexican American women (1.8 servings per week) and highest among black men (3.6 servings per week). Between 36% (Mexican American women) and 66% (black men) of adults consumed ≥1 serving per week.
- Average consumption of sugar-sweetened beverages ranged from ≈7 servings per week among white women to 16 servings per week among Mexican American men. Approximately 50% and 33% of white men and women, 73% and 65% of black men and women, and 76% and 62% of Mexican American men and women, respectively, consumed >36 oz (4.5 8-oz servings) per week.
- Average consumption of sweets and bakery desserts ranged from ≈4 servings per day (Mexican American men) to 7 servings per day (white men). Approximately two thirds of white and black men and women and half of all Mexican American men and women consumed >2.5 servings per week.
- Between 33% and 50% of adults in each sex and race or ethnic subgroup consumed <10% of total calories from saturated fat, and between 58% and 70% consumed <300 mg of dietary cholesterol per day.
- Only 4% to 7% of whites, 2% to 4% of blacks, and 9% to 11% of Mexican Americans consumed ≥28 g of dietary fiber per day.
- Only 8% to 11% of whites, 9% to 11% of blacks, and 13% to 19% of Mexican Americans consumed <2.3 g of sodium per day. In 2005, the US Department of Health and Human Services and US Department of Agriculture recommended that adults in specific groups, including people

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with hypertension, all middle-aged and older adults, and all blacks, should consume ≤1.5 g of sodium per day. In 2005 to 2006, the majority (69.2%) of US adults belonged to ≥1 of these specific groups in whom sodium consumption should be <1.5 g/d.[1]

Foods and Nutrients: Children and Teenagers
See Table 5-2; NHANES 2005–2008).

The dietary consumption by US children and teenagers of selected foods and nutrients related to cardiometabolic health is detailed in Table 5-2:

- Average whole grain consumption was low, ranging from 0.4 to 0.6 servings per day, with <4% of all children in different age and sex subgroups meeting guidelines of ≥3 servings per day.
- Average fruit consumption was low and decreased with age: ≈1.5 servings per day in younger boys and girls (5–9 years of age), 1.3 servings per day in adolescent boys and girls (10–14 years of age), and 0.9 servings per day in teenage boys and girls (15–19 years of age). The proportion meeting guidelines of ≥2 cups per day was also low and decreased with age: ≈8% in those 5 to 9 years of age, 7% to 8% in those 10 to 14 years of age, and 4% in those 15 to 19 years of age. When 100% fruit juices were included, the number of servings consumed approximately doubled or tripled, and proportions consuming ≥2 cups per day were 29% to 36% of those 5 to 9 years of age, 22% to 26% of those 10 to 14 years of age, and 21% to 22% of those 15 to 19 years of age.
- Average vegetable consumption was low, ranging from 0.9 to 1.1 servings per day, with <2% of children in different age and sex subgroups meeting guidelines of ≥2.5 cups per day.
- Average consumption of fish and shellfish was low, ranging between 0.5 and 0.7 servings per week in all age and sex groups. Among all ages, only 10% to 13% of children and teenagers consumed ≥2 servings per week.
- Average consumption of nuts, legumes, and seeds ranged from 1.3 to 1.4 servings per week among 5- to 9-year-olds, 1.4 to 2.1 servings per week among 10- to 14-year-olds, and 0.8 to 1.1 servings per week among 15- to 19-year-olds. Only between 7% and 14% of children in different age and sex subgroups consumed ≥4 servings per week.
- Average consumption of processed meats ranged from 2.1 to 3.2 servings per week; was uniformly higher than the average consumption of nuts, legumes, and seeds; and was up to 6 times higher than the average consumption of fish and shellfish. Between 40% and 54% of children consumed ≥2 servings per week.
- Average consumption of sugar-sweetened beverages was higher in boys than in girls and was ≈8 servings per week in 5- to 9-year-olds, 11 to 13 servings per week in 10- to 14-year-olds, and 14 to 18 servings per week in 15- to 19-year-olds. This was generally considerably higher than the average consumption of whole grains, fruits, vegetables, fish and shellfish, or nuts, legumes, and seeds. Only between 17% (boys 15–19 years of age) and 42% (boys and girls 5–9 years of age) of children consumed <4.5 servings per week.
- Average consumption of sweets and bakery desserts was ≈8 to 10 servings per week in 5- to 9-year-olds and 10- to 14-year-olds and 6 to 8 servings per week in 15- to 19-year-olds. From 82% (girls 5–9 years of age) to 58% (boys 15–19 years of age) of youths consumed >2.5 servings per week.
- Average consumption of eicosapentaenoic acid and docosahexaenoic acid was low, ranging from ≈45 to 75 mg/d in boys and girls at all ages. Only between 3% and 7% of children and teenagers at all ages consumed ≥250 mg/d.
- Average consumption of saturated fat was between 11% and 12% of calories, and average consumption of dietary cholesterol was ≈230 mg/d. Approximately one fifth to one third of children consumed <10% energy from saturated fat, and ≈80% consumed <300 mg of dietary cholesterol per day.
- Average consumption of dietary fiber ranged from 12 to 14 g/d. Less than 2% of children in all different age and sex subgroups consumed ≥28 g/d.
- Average consumption of sodium ranged from 3.1 to 3.4 g/d. Between 7% and 12% of children in different age and sex subgroups consumed <2.3 g/d.

Energy Balance
Energy balance, or consumption of total calories appropriate for needs, is determined by the balance of average calories consumed versus expended, with this balance depending on multiple factors, including calories consumed, PA, body size, age, sex, and underlying basal metabolic rate. Thus, one individual may consume relatively high calories but have negative energy balance (as a result of even greater calories expended), whereas another individual may consume relatively few calories but have positive energy balance (because of low calories expended). Given such variation, the most practical and reasonable method to assess energy balance in populations is to assess changes in weight over time (Trends section).

- Average daily caloric intake in the United States is ≈2500 calories in adult men and 1800 calories in adult women (Table 5-1). In children and teenagers, average caloric intake is higher in boys than in girls and increases with age in boys (Table 5-2). Trends in energy balance are described below. The average US adult gains ≈1 pound per year. In an analysis of >120 000 US men and women in 3 separate US cohorts followed up for up to 20 years, changes in intakes of different foods and beverages were linked to long-term weight gain in different ways.[2] Foods and beverages most positively linked to weight gain included poor-quality carbohydrates, such as potatoes, refined grains (eg, white bread, white rice, low-fiber breakfast cereals), sweets/desserts, sugar-sweetened beverages, and red and processed meats. In contrast, increased consumption of several other foods, including nuts, whole grains, fruits, vegetables, and yogurt, was linked to less weight gain over time.[2]
- Other nutritional determinants of positive energy balance (more calories consumed than expended), as determined by adiposity or weight gain, include larger portion sizes[3,4] and greater consumption of fast food and commercially prepared meals.[5–9]
- Preferences for portion size are associated with BMI, socioeconomic status, eating in fast-food restaurants, and television watching.[10,11] Most portion sizes are larger at fast-food restaurants than at home or at other restaurants.[12]

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- Between 1999 and 2004, 53% of Americans consumed an average of 1 to 3 restaurant meals per week, and 23% consumed ≥4 restaurant meals per week.[13] Spending on food away from home, including restaurant meals, catered foods, and food eaten during out-of-town trips, increased from 26% of average annual food expenditures in 1970 to 42% in 2004.[13] Macronutrient composition of the overall diet or of specific foods, such as percent calories from total fat, does not appear to be strongly associated with energy balance as ascertained by weight gain or loss.[2,14–16] In contrast, dietary quality as characterized by higher or lower intakes of specific foods and beverages is associated with weight gain (see above).[2]
- Preliminary evidence suggests that consumption of *trans* fat may be associated with energy imbalance as assessed by changes in adiposity or weight, as well as more specific adverse effects on visceral adiposity, but such data are still emerging.[17–19]
- Other individual factors associated with positive energy balance (weight gain) include greater television watching (with evidence that effects are mediated by diet, rather than physical inactivity, including greater snacking in front of the television and the influence of advertising on poor food choices)[2,20–24] and lower average sleep duration.[2,25]
- Randomized controlled trials of weight loss in obese individuals generally show modestly greater weight loss with low-carbohydrate (high-fat) diets than with low-fat diets at 6 months, but at 1 year, such differences diminish, and a diet that focuses on dietary quality and whole foods may be most successful in the long-term.[26–29]
- On the basis of BRFSS data from 2003, among all American adults who are overweight or obese, a higher proportion are trying to lose weight if also diagnosed with hypertension (58% trying to lose weight), DM (60%), or both diseases (72%) than adults with neither condition (50%).[31]
- A 2007–2008 national survey of 1082 retail stores in 19 US cities found that energy-dense snack foods/beverages were present in 96% of pharmacies, 94% of gas stations, 22% of furniture stores, 16% of apparel stores, and 29% to 65% of other types of stores.[32]
- Societal and environmental factors independently associated with energy imbalance (weight gain), via either increased caloric consumption or decreased expenditure, include education, income, race/ethnicity, and local conditions such as availability of grocery stores, types of restaurants, safety, parks and open spaces, and walking or biking paths.[33–35] PA is covered in Chapter 4 of this update.

Dietary Patterns

In addition to individual foods and nutrients, overall dietary patterns can be used to assess more global dietary quality. Different dietary patterns have been defined, including the Healthy Eating Index (HEI), Alternative HEI, Western versus prudent dietary patterns, Mediterranean dietary pattern, and DASH (Dietary Approaches to Stop Hypertension)-type diet. The higher-monounsaturated-fat DASH–type diet is generally similar to a traditional Mediterranean dietary pattern.[36]

- In 1999 to 2004, only 19.4% of hypertensive US adults were following a DASH-type diet (based on intake of fiber, magnesium, calcium, sodium, potassium, protein, total fat, saturated fat, and cholesterol). This represented a decrease from 26.7% of hypertensive US adults in 1988 to 1994.[37]
- Among older US adults (≥60 years of age) in 1999 to 2002, 72% met guidelines for dietary cholesterol intake, but only between 18% and 32% met guidelines for the HEI food groups (meats, dairy, fruits, vegetables, and grains). On the basis of the HEI score, only 17% of older US adults consumed a good-quality diet. Higher HEI scores were seen in white adults and individuals with greater education; lower HEI scores were seen in black adults and smokers.[38]

Dietary Supplements

Use of dietary supplements is common in the United States among both adults and children:

- More than half (53%) of US adults in 2003 to 2006 used dietary supplements, with the most common supplement being multivitamins and multiminerals (40% of men and women reporting use).[39] It has been shown that most supplements are taken daily and for at least 2 years. Supplement use was associated with older age, higher education, greater PA, wine intake, lower BMI, and white race.[40]
- One third (32%) of US children (birth to 18 years of age) used dietary supplements in 1999 to 2002, with the highest use (48.5%) occurring among 4- to 8-year-olds. The most common supplements were multivitamins and multiminerals (58% of supplement users). The primary nutrients supplemented (either by multivitamins and/or individual vitamins) included vitamin C (29% of US children), vitamin A (26%), vitamin D (26%), calcium (21%), and iron (19%). Supplement use was associated with higher family income, a smoke-free home environment, lower child BMI, and less screen time (television, video games, or computers).[41]
- In a 2005 to 2006 telephone survey of US adults, 41.3% were making or had made in the past a serious weight-loss attempt. Of these, one third (33.9%) had used a dietary supplement for weight loss, with such use being more common in women (44.9%) than in men (19.8%) and in blacks (48.7%) or Hispanics (41.6%) than in whites (31.2%); in those with high school education or less (38.4%) than in those with some college or more (31.1%); and in those with household income less than $40 000 per year (41.8%) than in those with higher incomes (30.3%).[42]
- Multiple trials of most dietary supplements, including folate, vitamin C, and vitamin E, have generally shown no significant benefits for CVD risk, and even potential for harm.[36] For example, a multicenter randomized trial in patients with diabetic nephropathy found that B vitamin supplementation (folic acid 2.5 mg/d, vitamin B6 25 mg/d, and vitamin B12 1 mg/d) decreased glomerular filtration rate (GFR) and increased risk of MI and stroke compared with placebo.[43] Fish oil supplements at doses of 1 to 2 g/d have shown CVD benefits in 2 large randomized, open-label trials and 1 large randomized, placebo-controlled trial (Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico [GISSI]-Prevenzione, Japan Eicosapentaenoic Acid Lipid Intervention Study, and GISSI-HF),[44–46] but other smaller trials of fish oil have not shown significant effects on CVD risk.[47] A meta-analysis of placebo-controlled trials (ie, excluding the 2 large open-label

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trials) showed a modest benefit for CVD mortality but no statistically significant effects on other CVD end points.[48]

Trends

Energy Balance

(See Chart 5-1)

Energy balance, or consumption of total calories appropriate for needs, has been steadily worsening in the United States over the past several decades, as evidenced by the dramatic increases in overweight and obesity among both children and adults across broad cross sections of sex, race/ethnicity, geographic residence, and socioeconomic status.[49,50,50a]

- Although trends in total calories consumed are difficult to quantify exactly because of differing methods of serial national dietary surveys over time, multiple lines of evidence indicate that average total energy consumption has increased by at least 200 kcal/d per person in the past 3 decades.
- Data from NHANES indicate that between 1971 and 2004, average total energy consumption among US adults increased by 22% in women (from 1542 to 1886 kcal/d) and by 10% in men (from 2450 to 2693 kcal/d). These increases are supported by data from the Nationwide Food Consumption Survey (1977–1978) and the Continuing Surveys of Food Intake (1989–1998).[12]
- The increases in calories consumed during this time period are attributable primarily to greater average carbohydrate intake, particularly of starches, refined grains, and sugars (Foods and Nutrients section). Other specific changes related to increased caloric intake in the United States include larger portion sizes, greater food quantity and calories per meal, and increased consumption of sugar-sweetened beverages, snacks, commercially prepared (especially fast-food) meals, and higher-energy-density foods.[6,12,51–55]
- Between 1977 and 1978 and 1994 and 1996, the average portion sizes for nearly all foods increased at fast-food outlets, other restaurants, and home. These included a 33% increase in the average portion of Mexican food (from 408 to 541 calories), a 34% increase in the average portion of cheeseburgers (from 397 to 533 calories), a 36% increase in the average portion of French fries (from 188 to 256 calories), and a 70% increase in the average portion of salty snacks such as crackers, potato chips, pretzels, puffed rice cakes, and popcorn (from 132 to 225 calories).[12]
- Among US children 2 to 7 years of age, an estimated energy imbalance of only 110 to 165 kcal/d (the equivalent of one 12- to 16-oz bottle of soda/cola) was sufficient to account for the excess weight gain between 1988 and 1994 and 1999 and 2002.[56]
- In a quantitative analysis using various US surveys between 1977 and 2006, the relations of changes in energy density, portion sizes, and number of daily eating/drinking occasions to changes in total energy intake were assessed.[57] Decreases in energy density were actually linked to lower total energy intake over time, whereas increases in both portion size and number of eating occasions were linked to greater energy intake.
- Among US children 2 to 18 years of age, increases in energy intake between 1977 and 2006 (179 kcal/d) were entirely attributable to substantial increases in energy eaten away from home (255 kcal/d).[58] The percentage of energy eaten away from home increased from 23.4% to 33.9% during this time, with a shift toward energy from fast food as the largest contributor to foods away from home for all age groups.

Foods and Nutrients

Several changes in foods and nutrients have occurred over time. Selected changes are highlighted:

Macronutrients

(See Chart 5-1)

- Starting in 1977 and continuing until the most recent dietary guidelines revision in 2010, a major focus of US dietary guidelines was reduction of dietary fats.[58a] During this time, average total fat consumption declined as a percent of calories from 36.9% to 33.4% in men and from 36.1% to 33.8% in women.[13]
- Dietary guidelines during this time also emphasized carbohydrate consumption as the base of one's dietary pattern,[59] and more recently specifies the importance of complex rather than refined carbohydrates[58a] (eg, as the base of the Food Guide Pyramid).[59] Total carbohydrate intake increased from 42.4% to 48.2% of calories in men and from 45.4% to 50.6% of calories in women.[13] Evaluated as absolute intakes, the increase in total calories consumed during this period was attributable primarily to the greater consumption of carbohydrates, both as foods (starches and grains) and as beverages.[60,61]

Sugar-Sweetened Beverages

(See Chart 5-2)

- Between 1965 and 2002, the average percentage of total calories consumed from beverages in the United States increased from 11.8% to 21.0% of energy, which represents an overall absolute increase of 222 cal/d per person.[54] This increase was largely caused by increased consumption of sugar-sweetened beverages and alcohol: Average consumption of fruit juices went from 20 to 39 kcal/d; of milk, from 125 to 94 kcal/d; of alcohol, from 26 to 99 kcal/d; of sweetened fruit drinks, from 13 to 38 kcal/d; and of soda/cola, from 35 to 143 kcal/d.[57]
- In addition to increased overall consumption, the average portion size of a single sugar-sweetened beverage increased by >50% between 1977 and 1996, from 13.1 to 19.9 fl oz.[12]
- Among children and teenagers (2–19 years of age), the largest increases in consumption of sugar-sweetened beverages between 1988 to 1994 and 1999 to 2004 were seen among black and Mexican American youths compared with white youths.[55]

Fruits and Vegetables

- Between 1994 and 2005, the average consumption of fruits and vegetables declined slightly, from a total of 3.4 to 3.2 servings per day. The proportions of men and women consuming combined fruits and vegetables ≥5 times per day were low (≈20% and 29%, respectively) and did not change during this period.[62]

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Morbidity and Mortality

Effects on Cardiovascular Risk Factors

Dietary habits affect multiple cardiovascular risk factors, including both established risk factors (SBP, DBP, LDL cholesterol levels, HDL cholesterol levels, glucose levels, and obesity/weight gain) and novel risk factors (eg, inflammation, cardiac arrhythmias, endothelial cell function, triglyceride levels, lipoprotein[a] levels, and heart rate):

- A DASH dietary pattern with low sodium reduced SBP by 7.1 mm Hg in adults without hypertension and by 11.5 mm Hg in adults with hypertension.[63]
- Compared with the low-fat DASH diet, DASH-type diets that increased consumption of either protein or unsaturated fat had similar or greater beneficial effects on CVD risk factors. Compared with a baseline usual diet, each of the DASH-type diets, which included various percentages (27%–37%) of total fat and focused on whole foods such as fruits, vegetables, whole grains, and fish, as well as potassium and other minerals and low sodium, reduced SBP by 8 to 10 mm Hg, DBP by 4 to 5 mm Hg, and LDL cholesterol by 12 to 14 mg/dL. The diets that had higher levels of protein and unsaturated fat also lowered triglyceride levels by 16 and 9 mg/dL, respectively.[64] The DASH-type diet higher in unsaturated fat also improved glucose-insulin homeostasis compared with the low-fat/high-carbohydrate DASH diet.[65]
- In a meta-analysis of randomized controlled trials, consumption of 1% of calories from *trans* fat in place of saturated fat, monounsaturated fat, or polyunsaturated fat increased the ratio of total to HDL cholesterol by 0.031, 0.054, and 0.67; increased apolipoprotein B levels by 3, 10, and 11 mg/L; decreased apolipoprotein A-1 levels by 7, 5, and 3 mg/L; and increased lipoprotein(a) levels by 3.8, 1.4, and 1.1 mg/L, respectively.[66]
- In meta-analyses of randomized controlled trials, consumption of eicosapentaenoic acid and docosahexaenoic acid for 212 weeks lowered SBP by 2.1 mm Hg[67] and lowered resting heart rate by 2.5 beats per minute.[68]
- In a pooled analysis of 25 randomized trials totaling 583 men and women both with and without hypercholesterolemia, nut consumption significantly improved blood lipid levels.[69] For a mean consumption of 67 g of nuts per day, total cholesterol was reduced by 10.9 mg/dL (5.1%), LDL cholesterol by 10.2 mg/dL (7.4%), and the ratio of total cholesterol to HDL cholesterol by 0.24 (5.6% change; $P<0.001$ for each). Triglyceride levels were also reduced by 20.6 mg/dL (10.2%) in subjects with high triglycerides (2150 mg/dL). Different types of nuts had similar effects.[69] A review of cross-sectional and prospective cohort studies suggests that higher intake of sugar-sweetened beverages is associated with greater visceral fat and higher risk of type 2 DM.[70] In the Prospective Registry Evaluating Myocardial Infarction: Events and Recovery (PREMIER) study, a prospective analysis of the 810 participants indicated that a reduction in sugar-sweetened beverages of 1 serving per day was associated with a reduction in SBP of 1.8 mm Hg (95% CI, 1.2–2.4 mm Hg) and a reduction in DBP of 1.1 mm Hg (95% CI, 0.7–1.4 mm Hg).[71]
- In a cross-sectional analysis among nearly 3000 adults from the United States and the United Kingdom, higher consumption of sugar-sweetened beverages was linked to higher BP (1.6 and 0.8 mm Hg higher SBP and DBP per serving per day, respectively, and 1.1 and 0.4 mm Hg higher SBP and DBP per serving per day after adjustment for weight and weight).[72]
- In a randomized controlled trial, compared with a low-fat diet, 2 Mediterranean dietary patterns that included either virgin olive oil or mixed nuts lowered SBP by 5.9 and 7.1 mm Hg, plasma glucose by 7.0 and 5.4 mg/dL, fasting insulin by 16.7 and 20.4 pmol/L, the homeostasis model assessment index by 0.9 and 1.1, and the ratio of total to HDL cholesterol by 0.38 and 0.26 and raised HDL cholesterol by 2.9 and 1.6 mg/dL, respectively. The Mediterranean dietary patterns also lowered levels of C-reactive protein, interleukin-6, intercellular adhesion molecule-1, and vascular cell adhesion molecule-1.[73]

Effects on Cardiovascular Outcomes

Because dietary habits affect a broad range of established and novel risk factors, estimation of the impact of nutritional factors on cardiovascular health by considering only a limited number of pathways (eg, only effects on lipids, BP, and obesity) will systematically underestimate or even misconstrue the actual total impact on cardiovascular health. Randomized controlled trials and prospective observational studies have been used to quantify the total effects of dietary habits on clinical outcomes:

Fats and Carbohydrates

- In the Women's Health Initiative (WHI) randomized clinical trial (n=48 835), reduction of total fat consumption from 37.8% energy (baseline) to 24.3% energy (at 1 year) and 28.8% energy (at 6 years) had no effect on incidence of CHD (RR, 0.98; 95% CI, 0.88–1.09), stroke (RR, 1.02; 95% CI, 0.90–1.15), or total CVD (RR, 0.98; 95% CI, 0.92–1.05) over a mean of 8.1 years.[74] This was consistent with null results of 4 prior randomized clinical trials and multiple large prospective cohort studies that indicated little effect of total fat consumption on CVD risk.[75]
- In 3 separate meta-analyses of prospective cohort studies, the largest of which included 21 studies with up to 2 decades of follow-up, saturated fat consumption overall had no significant association with incidence of CHD, stroke, or total CVD.[76–78] In comparison, in a pooled individual-level analysis of 11 prospective cohort studies, the specific exchange of polyunsaturated fat consumption in place of saturated fat was associated with lower CHD risk, with 13% lower risk for each 5% energy exchange (RR, 0.87; 95% CI, 0.70–0.97).[79] These findings are consistent with a meta-analysis of randomized controlled trials in which increased polyunsaturated fat consumption in place of saturated fat reduced CHD events, with 10% lower risk for each 5% energy exchange (RR, 0.90; 95% CI, 0.83–0.97).[80]
- In a pooled analysis of individual-level data from 11 prospective cohort studies in the United States, Europe, and Israel that included 344 696 participants, each 5% higher energy consumption of carbohydrate in place of saturated fat was associated with a 7% higher risk of CHD (RR, 1.07; 95% CI, 1.01–1.14).[79] Each 5% higher energy consumption of monounsaturated fat in place of saturated fat was not significantly associated with CHD risk.[79]

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- Together these findings suggest that reducing saturated fat without specifying the replacement may have minimal effects on CHD risk, whereas increasing polyunsaturated fats from vegetable oils will reduce CHD.[36]
- In a meta-analysis of prospective cohort studies, each 2% of calories from *trans* fat was associated with a 23% higher risk of CHD (RR, 1.23; 95% CI, 1.11–1.37).[81]
- In meta-analyses of prospective cohort studies, greater consumption of refined complex carbohydrates, starches, and sugars, as assessed by glycemic index or load, was associated with significantly higher risk of CHD and DM. When the highest category was compared with the lowest category, risk of CHD was 36% greater (glycemic load: RR, 1.36; 95% CI, 1.13–1.63), and risk of DM was 40% greater (glycemic index: RR, 1.40; 95% CI, 1.23–1.59).[82,83]

Foods and Beverages

- In meta-analyses of prospective cohort studies, each daily serving of fruits or vegetables was associated with a 4% lower risk of CHD (RR, 0.96; 95% CI, 0.93–0.99) and a 5% lower risk of stroke (RR, 0.95; 95% CI, 0.92–0.97).[84,85]
- In a meta-analysis of prospective cohort studies, greater whole grain intake (2.5 compared with 0.2 servings per day) was associated with a 21% lower risk of CVD events (RR, 0.79; 95% CI, 0.73–0.85), with similar estimates in men and women and for various outcomes (CHD, stroke, and fatal CVD). In contrast, refined grain intake was not associated with lower risk of CVD (RR, 1.07; 95% CI, 0.94–1.22).[86]
- In a meta-analysis of 16 prospective cohort studies that included 326 572 generally healthy individuals in Europe, the United States, China, and Japan, fish consumption was associated with significantly lower risk of CHD mortality.[87] Compared with no consumption, an estimated 250 mg of long-chain omega-3 fatty acids per day was associated with 35% lower risk of CHD death ($P<0.001$).
- In a meta-analysis of prospective cohort and case-control studies from multiple countries, consumption of unprocessed red meat was not significantly associated with incidence of CHD. In contrast, each 50-g serving per day of processed meats (eg, sausage, bacon, hot dogs, deli meats) was associated with a higher incidence of CHD (RR, 1.42; 95% CI, 1.07–1.89).[88]
- In a meta-analysis of prospective cohort studies that included 442 101 participants and 28 228 DM cases, unprocessed red meat consumption was associated with a higher risk of DM (RR, 1.19; 95% CI, 1.04–1.37, per 100 g/d). On a per g/d basis, risk of DM was nearly 7-fold higher for processed meat consumption (RR, 1.51; 95% CI, 1.25–1.83, per 50 g/d).[89]
- In a meta-analysis of 6 prospective observational studies, nut consumption was associated with significantly lower incidence of CHD (comparing higher to low intake: RR, 0.70; 95% CI, 0.57–0.82).[77]
- Higher consumption of dairy or milk products is associated with lower incidence of DM and trends toward lower risk of stroke.[77,90,91] Some limited evidence suggests that these associations are stronger for low-fat dairy or milk than for other dairy products. Dairy consumption is not significantly associated with higher or lower risk of CHD.[77,91]
- Among 88 520 generally healthy women in the Nurses' Health Study who were 34 to 59 years of age in 1980 and were followed up from 1980–2004, regular consumption of sugar-sweetened beverages was independently associated with higher incidence of CHD, with 23% and 35% higher risk with 1 and ≥2 servings per day, respectively, compared with <1 per month.[92] Among the 15 745 participants in the ARIC study, the OR for developing CHD was 2.59 for participants who had a serum uric acid level >9.0 mg/dL and who drank >1 sugar-sweetened soda per day.[93]

Sodium and Potassium

- Lower estimated consumption of dietary sodium was not associated with lower CVD mortality in NHANES,[94] although such findings may be limited by changes in behaviors that result from underlying risk (reverse causation). In a post hoc analysis of the Trials of Hypertension Prevention, participants randomized to low-sodium interventions had a 25% lower risk of CVD (RR, 0.75; 95% CI, 0.57–0.99) after 10 to 15 years of follow-up after the original trials.[95]
- In a meta-analysis of small randomized trials of sodium reduction of ≥6 months' duration, nonsignificant trends were seen toward fewer CVD events in subjects with normal BP (RR, 0.71; 95% CI, 0.42–1.20; n=200 events) or hypertension (RR, 0.84; 95% CI, 0.57–1.23; n=93 events), but the findings were not statistically significant, with relatively low statistical power because of the small numbers of events. Sodium restriction increased total mortality in trials of patients with CHF (RR, 2.59; 95% CI, 1.04–6.44), but these data were based on very few events (n=21 deaths).[96]
- In a meta-analysis of 13 prospective cohorts that included 177 025 participants and >11 000 vascular events, higher sodium consumption was associated with greater risk of stroke (pooled RR, 1.23; 95% CI, 1.06–1.43; $P=0.007$) and a trend toward higher risk of CVD (1.14, 0.99–1.32; $P=0.07$). These associations were greater with larger differences in sodium intake and longer follow-up.[97]
- In a meta-analysis of 15 prospective cohort samples that included 247 510 participants and 7066 strokes, 3058 CHD events, and 2497 total CVD events, each 1.64-g/d (42 mmol/d) higher potassium intake was associated with a 21% lower risk of stroke (RR, 0.79; 95% CI, 0.68–0.90) and trends toward lower risk of CHD and total CVD.[98]

Dietary Patterns

- In a cohort of 380 296 US men and women, greater versus lower adherence to a Mediterranean dietary pattern, characterized by higher intakes of vegetables, legumes, nuts, fruits, whole grains, fish, and unsaturated fat and lower intakes of red and processed meat, was associated with a 22% lower cardiovascular mortality (RR, 0.78; 95% CI, 0.69–0.87).[99] Similar findings have been seen for the Mediterranean dietary pattern and risk of incident CHD and stroke[100] and for the DASH-type dietary pattern.[101]
- In a cohort of 72 113 US female nurses, a dietary pattern characterized by higher intakes of vegetables, fruits, legumes, fish, poultry, and whole grains was associated with a 28% lower cardiovascular mortality (RR, 0.72; 95% CI, 0.60–0.87), whereas a dietary pattern characterized by

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higher intakes of processed meat, red meat, refined grains, French fries, and sweets/desserts was associated with a 22% higher cardiovascular mortality (RR, 1.22; 95% CI, 1.01–1.48).[102] Similar findings have been seen in other cohorts and for other outcomes, including development of DM and metabolic syndrome.[103–109]

Impact on US Mortality

- In one report that used consistent and comparable risk assessment methods and nationally representative data, the mortality effects in the United States of 12 modifiable dietary, lifestyle, and metabolic risk factors were assessed. High dietary salt consumption was estimated to be responsible for 102 000 annual deaths, low dietary omega-3 fatty acids for 84 000 annual deaths, high dietary *trans* fatty acids for 82 000 annual deaths, and low consumption of fruits and vegetables for 55 000 annual deaths.[110]

Cost

(See Chart 5-3)

The US Department of Agriculture forecast that the Consumer Price Index for all food would increase 3.0% to 4.0% in 2013 as retailers continued to pass on higher commodity and energy costs to consumers in the form of higher retail prices. The Consumer Price Index for food increased 3.7% in 2011. Prices for foods eaten at home increased 4.8% in 2011, whereas prices for foods eaten away from home increased by 1.9%.[111]

- The proportion of total US food expenditures for meals outside the home, as a share of total food dollars, increased from 27% in 1961 to 40% in 1981 to 49% in 2011.[59]
- The proportion of sales of meals and snacks from fast-food restaurants compared with total meals and snacks away from home increased from 5% in 1958 to 29% in 1982 to 36% in 2011.[111]
- As a proportion of income, food has become less expensive over time in the United States. As a share of personal disposable income, average (mean) total food expenditures by families and individuals have decreased from 22.3% (1949) to 18.1% (1961) to 14.9% (1981) to 11.3% (2011). For any given year, the share of disposable income spent on food is inversely proportional to absolute income. The share increases as absolute income levels decline.[111]
- Among 153 forms of fruits and vegetables priced with 2008 Nielsen Homescan data, price and calorie per portion of 20 fruits and vegetables were compared with 20 common snack foods such as cookies, chips, pastries, and crackers. Average price per portion of fruits and vegetables was 31 cents with an average of 57 calories per portion, compared with 33 cents and 183 calories per portion for snack foods.[111]
- An overview of the costs of various strategies for primary prevention of CVD determined that the estimated costs per year of life gained were between $9800 and $18 000 for statin therapy, ≥$1500 for nurse screening and lifestyle advice, $500 to $1250 for smoking cessation, and $20 to $900 for population-based healthy eating.[112]
- Each year, >$33 billion in medical costs and $9 billion in lost productivity resulting from HD, cancer, stroke, and DM are attributed to poor nutrition.[113–117]
- Two separate cost-effectiveness analyses estimated that population reductions in dietary salt would not only be cost-effective but actually cost-saving.[118,119] In 1 analysis, a 1.2 g/d reduction in dietary sodium was projected to reduce US annual cases of incident CHD by 60 000 to 120 000, stroke by 32 000 to 66 000, and total mortality by 44 000 to 92 000.[119] If accomplished through a regulatory intervention, estimated savings in healthcare costs would be $10 to $24 billion annually.[119] Such an intervention would be more cost-effective than using medications to lower BP in all people with hypertension.

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Table 5-1. Dietary Consumption in 2005–2008 Among US Adults ≥20 Years of Age of Selected Foods and Nutrients Related to Cardiometabolic Health[96–99]

	NH White Men		NH White Women		NH Black Men		NH Black Women		Mexican American Men		Mexican American Women	
	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*
Foods												
Whole grains, servings/d	0.7±0.7	4.6	0.8±0.7	5.3	0.5±0.5	3.1	0.6±0.6	4.1	2.1±1.6	27.4	1.7±1.4	21.0
Fruits, servings/d	1.3±1.3	8.9	1.6±1.4	10.7	1.1±1.4	6.2	1.2±1.3	7.0	1.4±1.3	8.0	1.8±1.5	10.4
Fruits including 100% juices, servings/d	2.4±2.3	23.3	2.5±2.1	24.9	2.8±0.9	26.6	2.9±1.2	26.7	2.8±2.3	23.9	3.2±2.6	28.7
Vegetables including starch, servings/d	1.9±1.1	6.3	2.2±1.1	7.2	1.6±0.9	3.2	1.8±1.0	3.6	1.3±0.7	2.6	1.6±0.7	2.5
Vegetables including starch and juices/ sauces, servings/d	2.2±1.2	8.8	2.5±1.3	9.6	1.7±0.9	3.9	1.9±1.1	4.9	1.7±0.8	3.9	1.9±0.7	4.8
Fish and shellfish, servings/wk	1.5±1.4	22.0	1.2±0.6	19.1	1.7±1.3	23.0	1.7±0.9	25.2	1.6±1.3	20.0	1.4±1.3	19.7
Nuts, legumes, and seeds, servings/wk	2.7±2.0	20.3	2.4±1.9	19.9	2.3±1.5	15.9	1.8±0.4	14.5	6.3±6.8	41.2	5.8±3.6	39.9
Processed meats, servings/wk	3.2±1.9	46.1	2.0±1.0	60.1	3.6±2.2	43.8	2.7±2.2	52.1	2.1±2.2	60.8	1.8±2.2	64.2
Sugar-sweetened beverages, servings/wk	9.9±11.6	50.0	6.6±10.9	66.7	13.8±9.0	27.2	11.8±8.9	34.8	15.6±10.3	24.2	10.0±8.8	37.9
Sweets and bakery desserts, servings/wk	6.5±4.8	35.4	7.4±4.5	32.2	6.0±4.1	42.0	6.8±3.5	38.6	3.7±2.9	55.1	5.5±0.9	48.4
Nutrients												
Total calories, kcal/d	2520±659	NA	1757±455	NA	2371±722	NA	1749±568	NA	2400±703	NA	1798±528	NA
EPA/DHA, g/d	0.129±0.138	13.0	0.109±0.138	10.2	0.146±0.131	15.2	0.146±0.102	13.8	0.146±0.102	12.1	0.119±0.102	12.0
ALA, g/d	1.35±0.33	25.5	1.52±0.50	72.2	1.32±0.38	23.4	1.43±0.33	68.0	1.21±0.23	16.5	1.34±0.27	64.1
n-6 PUFA, % energy	7.1±1.2	NA	7.5±1.6	NA	7.3±1.6	NA	7.6±1.5	NA	6.7±0.9	NA	6.9±1.4	NA
Saturated fat, % energy	11.4±2.2	33.3	11.4±2.1	36.0	10.8±1.7	39.8	10.6±2.0	43.3	10.1±2.0	50.4	10.4±1.7	48.5
Dietary cholesterol, mg/d	277±90	66.9	274±83	69.5	303±123	61.8	317±106	57.7	323±142	58.8	310±120	61.2
Total fat, % energy	34.1±5.1	54.2	33.9±4.7	53.6	33.8±4.7	51.1	33.5±4.6	54.2	31.6±5.1	65.7	31.8±4.9	65.4
Carbohydrate, % energy	47.3±7.2	NA	49.7±6.6	NA	48.6±6.2	NA	50.7±6.3	NA	50.3±6.7	NA	52.3±6.5	NA
Dietary fiber, g/d	15.0±5.0	4.2	17.2±5.8	7.0	13.1±4.6	2.4	14.2±5.0	3.8	17.7±6.1	9.3	18.9±4.7	11.2
Sodium, g/d	3.3±0.6	10.5	3.5±0.6	8.3	3.2±0.5	11.3	3.4±0.5	8.9	3.0±0.7	19.4	3.2±0.6	12.7

Based on data from National Health and Nutrition Examination Survey 2005–2006 and 2007–2008, derived from two 24-hour dietary recalls per person, with population SDs adjusted for within-person vs between-person variation. All values are energy-adjusted using individual regressions or percent energy, and for comparability, means and proportions are reported for a 2000-kcal/d diet. To obtain actual mean consumption levels, the group means for each food or nutrient can be multiplied by the group-specific total calories (kcal/d) divided by 2000 kcal/d.

SD indicates standard deviation; NH, non-Hispanic; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; ALA, α-linoleic acid; n-6-PUFA, ω-6-polyunsaturatedfatty acid; and NA, not available.

*Guidelines adjusted to a 2000-kcal/d diet. Whole grains (characterized as minimum 1.1 g of fiber per 10 g of carbohydrate), 3 or more 1-oz equivalent (1 oz of bread; 1 cup of dry cereal; 1/2 cup of cooked rice, pasta, or cereal) servings per day (Dietary Guidelines for Americans)[58a]; fish or shellfish, 2 or more 100-g (3.5-oz) servings per week[58a]; fruits, 2 cups per day[116]; vegetables, 2 1/2 cups per day, including up to 3 cups per week of starchy vegetables[116]; nuts, legumes, and seeds, 4 or more 50-g servings per week[58a]; processed meats (bacon, hot dogs, sausage, processed deli meats), 2 or fewer 100-g (3.5-oz) servings per week (1/4 of discretionary calories)[116]; sugar-sweetened beverages (defined as ≥50 cal/8 oz, excluding whole juices), ≤36 oz per week (≈1/4 of discretionary calories)[58a,116]; sweets and bakery desserts, 2.5 or fewer 50-g servings per week (≈1/4 of discretionary calories)[58a,116]; EPA/DHA, ≥0.250 g/d[120]; ALA, ≥1.6/1.1 g/d (men/women)[117]; saturated fat, <10% energy; dietary cholesterol, <300 mg/d[116]; total fat, 20% to 35% energy[116]; dietary fiber, ≥28/d[116]; and sodium, <2.3 g/d.[116]

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Table 5-2. Dietary Consumption in 2005–2008 Among US Children and Teenagers of Selected Foods and Nutrients Related to Cardiometabolic Health

	Boys (5–9 y)		Girls (5–9 y)		Boys (10 –14 y)		Girls (10 –14 y)		Boys (15–19 y)		Girls (15–19 y)	
	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*
Foods												
Whole grains, servings/d	0.5±0.5	2.7	0.5±0.3	1.1	0.6±0.6	4.0	0.5±0.4	1.8	0.4±0.4	1.6	0.5±0.5	2.6
Fruits, servings/d	1.5±1.1	8.3	1.5±0.8	8.5	1.2±1.0	6.9	1.4±1.1	8.4	0.9±0.8	3.9	0.9±0.8	4.6
Fruits including 100% juices, servings/d	3.3±1.7	35.7	3.1±1.4	28.5	2.4±1.7	21.7	2.8±1.9	26.0	2.2±1.7	21.1	2.4±1.7	21.7
Vegetables including starch, servings/d	0.9±0.4	0.5	1.0±0.5	0.9	1.0±0.6	1.1	1.1±0.6	1.6	1.0±0.1	1.1	1.1±0.2	1.1
Vegetables including starch and juices/ sauces, servings/d	1.1±0.4	0.8	1.1±0.6	1.3	1.1±0.6	1.2	1.3±0.6	1.9	1.3±0.9	1.5	1.3±0.4	2.4
Fish and shellfish, servings/wk	0.5±0.7	9.9	0.7±0.7	11.7	0.9±0.7	13.5	0.6±0.7	10.3	0.7±0.9	11.0	0.7±0.9	12.2
Nuts, legumes, and seeds, servings/wk	1.4±0.4	12.5	1.3±2.5	9.6	2.1±2.8	14.1	1.4±1.0	10.1	1.1±1.0	9.4	0.8±1.0	6.8
Processed meats, servings/wk	2.3±1.1	55.5	2.1±1.0	60.0	2.6±1.0	54.9	2.3±1.0	52.1	3.2±1.5	45.6	2.4±1.0	55.8
Sugar-sweetened beverages, servings/wk	8.5±5.9	38.6	8.3±5.1	37.9	13.3±7.0	23.5	10.9±7.3	31.6	18.2±11.1	16.7	13.9±10.1	32.4
Sweets and bakery desserts, servings/wk	10.1±2.1	19.5	9.3±2.1	18.3	9.0±2.1	23.5	8.1±2.0	30.2	6.0±5.2	41.9	8.2±5.2	31.5
Nutrients												
Total calories, kcal/d	1946±328	NA	1743±330	NA	2139±403	NA	1849±432	NA	2670±903	NA	1845±453	NA
EPA/DHA, g/d	0.045±0.025	3.1	0.056±0.025	5.9	0.074±0.030	7.3	0.052±0.030	4.7	0.071±0.022	5.2	0.065±0.021	5.7
ALA, g/d	1.12±0.15	9.5	1.15±0.20	46.3	1.11±0.20	9.7	1.19±0.28	49.1	1.14±0.18	13.2	1.34±0.18	59.2
n-6 PUFA, % energy	6.4±1.1	NA	6.5±1.0	NA	6.5±1.0	NA	6.7±0.9	NA	6.4±0.6	NA	7.1±1.3	NA
Saturated fat, % energy	11.7±1.4	24.9	11.8±0.8	21.3	11.6±0.7	27.8	11.5±1.8	28.6	11.8±1.4	24.8	11.3±1.7	34.1
Dietary cholesterol, mg/d	225±69	81.6	239±57	78.4	245±57	76.6	226±114	81.6	244±114	76.9	240±114	77.7
Total fat, % energy	33.0±3.3	67.6	33.3±3.0	66.6	33.1±2.6	65.6	33.1±4.2	61.6	33.6±3.1	58.9	33.3±4.9	57.0
Carbohydrate, % energy	54.5±4.1	NA	53.8±3.7	NA	53.1±3.6	NA	53.8±4.9	NA	51.4±4.1	NA	52.9±6.3	NA
Dietary fiber, g/d	13.6±2.3	0.2	13.9±2.2	0.7	13.3±3.4	1.1	13.9±3.3	1.8	11.9±2.4	0.6	13.3±2.9	1.9
Sodium, g/d	3.1±0.3	8.2	3.2±0.4	8.7	3.2±0.2	9.8	3.3±0.2	7.4	3.2±0.4	11.9	3.4±0.5	9.1

Based on data from National Health and Nutrition Examination Survey 2005–2006 and 2007–2008, derived from two 24-hour dietary recalls per person, with population SDs adjusted for within-person vs between-person variation. All values are energy-adjusted using individual regressions or percent energy, and for comparability, means and proportions are reported for a 2000-kcal/d diet. To obtain actual mean consumption levels, the group means for each food or nutrient can be multiplied by the group-specific total calories (kcal/d) divided by 2000 kcal/d.

SD indicates standard deviation; NA, not available; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; ALA, α-linoleic acid; and n-6-PUFA, ω-6-polyunsaturated fatty acid.

*See Table 5-1 for food group, serving size, and guideline definitions. For different age and sex subgroups here, the guideline cutpoints are standardized to a 2000-kcal/d diet to account for differences in caloric intake in these groups.

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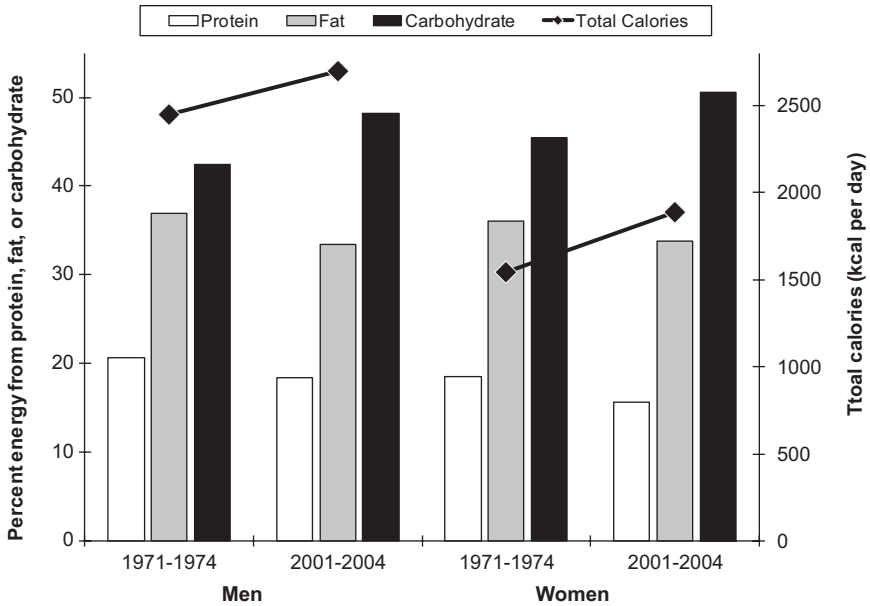

Chart 5-1. Age-adjusted trends in macronutrients and total calories consumed by US adults (20–74 years of age), 1971–2004. Data derived from National Center for Health Statistics. *Health, United States 2007, With Chartbook on Trends in the Health of Americans.*[13]

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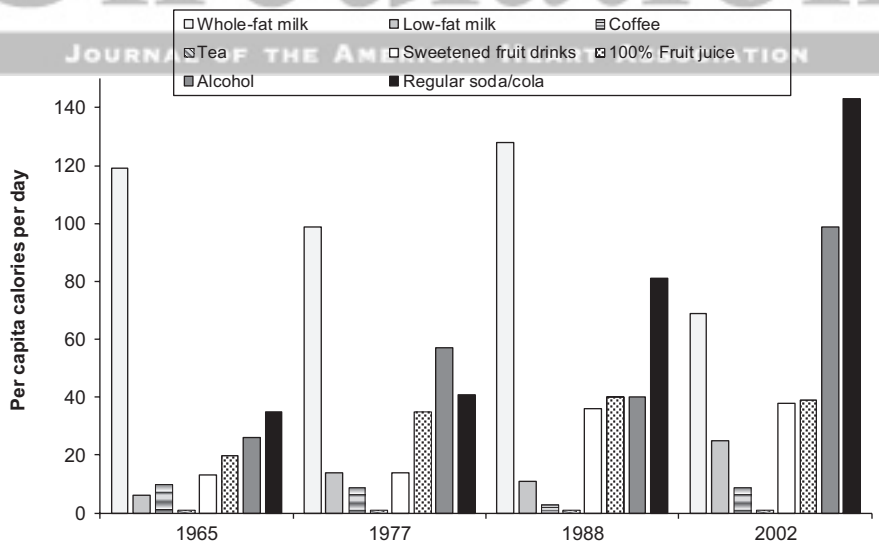

Chart 5-2. Per-capita calories consumed from different beverages by US adults (≥19 years of age), 1965–2002. Data derived from Nationwide Food Consumption Surveys (1965, 1977–1978), National Health and Nutrition Examination Survey (1988–1994, 1999–2002), and Duffey and Popkin.[57]

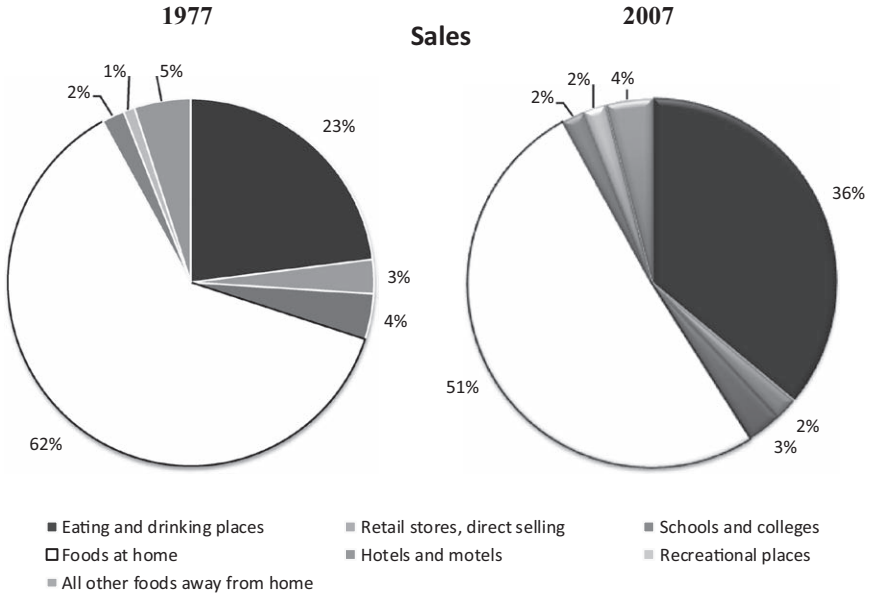

Chart 5-3. Total US food expenditures away from home and at home, 1977 and 2007. Data derived from Davis et al.[59]

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6. Overweight and Obesity

See Table 6-1 and Charts 6-1 through 6-3

Prevalence

Youth

(See Table 6-1 and Chart 6-1)

- The prevalence of overweight and obesity in children 2 to 5 years of age, based on a BMI-for-age value ≥85th percentile of the 2000 CDC growth charts, was 26% for non-Hispanic white boys and 21% for non-Hispanic white girls, 31% for non-Hispanic black boys and 27% for non-Hispanic black girls, and 32% for Mexican American boys and 33% for Mexican American girls according to 2009–2010 data from NHANES (NCHS). In children 6 to 11 years of age, the prevalence was 30% for non-Hispanic white boys and 25% for non-Hispanic white girls, 41% for non-Hispanic black boys and 44% for non-Hispanic black girls, and 39% for Mexican American boys and 40% for Mexican American girls. In children 12 to 19 years of age, the prevalence was 32% for non-Hispanic white boys and 28% for non-Hispanic white girls, 37% for non-Hispanic black boys and 45% for non-Hispanic black girls, and 46% for Mexican American boys and 41% for Mexican American girls.[2]
- The prevalence of obesity in children 2 to 5 years of age, based on BMI-for-age values ≥95th percentile of the 2000 CDC growth charts, was 12% for non-Hispanic white boys and 6% for non-Hispanic white girls, 21% for non-Hispanic black boys and 17% for non-Hispanic black girls, and 19% for Mexican American boys and 12% for Mexican American girls according to 2009–2010 data from NHANES (NCHS). In children 6 to 11 years of age, the prevalence was 17% for non-Hispanic white boys and 11% for non-Hispanic white girls, 30% for non-Hispanic black boys and 28% for non-Hispanic black girls, and 22% for Mexican American boys and 22% for Mexican American girls. In children 12 to 19 years of age, the prevalence was 18% for non-Hispanic white boys and 15% for non-Hispanic white girls, 23% for non-Hispanic black boys and 25% for non-Hispanic black girls, and 29% for Mexican American boys and 19% for Mexican American girls.[2]
- Overall, 18% of US children and adolescents 6 to 19 years of age have BMI-for-age values ≥95th percentile of the 2000 CDC growth charts for the United States (NHANES [2009–2010], NCHS).[2]
- NHANES 2009–2010 found that 16.9% (15.4%–18.4%) of youth aged 2 to 19 years were obese, which was unchanged from 2007–2008. Rates of overweight and obesity (≥85th BMI percentile) were 39.1% for Hispanics, 39.4% for Mexican Americans, 27.9% for non-Hispanic whites, and 39.1% for non-Hispanic blacks.[2]
- A study of >8500 4-year-olds in the Early Childhood Longitudinal Study, Birth Cohort (National Center for Education Statistics) found that 1 in 5 were obese. Almost 13% of Asian children, 16% of white children, nearly 21% of black children, 22% of Hispanic children, and 31% of American Indian children were obese. Children were considered obese if their BMI was ≥95th percentile on the basis of CDC BMI growth charts. For 4-year-olds, that would be a BMI of ≈18 kg/m^2.[3]
- Childhood sociodemographic factors may contribute to sex disparities in obesity prevalence. A study of data from the National Longitudinal Study of Adolescent Health found that parental education consistently modified sex disparity in blacks. The sex gap was largest in those with low parental education (16.7% of men compared with 45.4% of women were obese) and smallest in those with high parental education (28.5% of men compared with 31.4% of women were obese). In whites, there was little overall sex difference in obesity prevalence.[4]
- The obesity epidemic is disproportionally more rampant among children living in low-income, low-education, and higher-unemployment households, according to data from the National Survey of Children's Health.[5]
- Data from 2011 show that American Indian/Alaskan Native youth have an obesity rate of 17.7%, whereas rates are 14.7% for Hispanics, 10.6% for non-Hispanic blacks, 10.3% for non-Hispanic whites, and 9.3% for Asian/Pacific Islanders.[6]
- According to 1999–2008 NHANES survey data, lowest-income girls had an obesity prevalence of 17.9% compared

Abbreviations Used in Chapter 6

AF	atrial fibrillation
AFFIRM	Atrial Fibrillation Follow-up Investigation of Rhythm Management
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CAD	coronary artery disease
CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CHF	congestive heart failure
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
FHS	Framingham Heart Study
HbA_{1c}	hemoglobin A_{1c}
HDL	high-density lipoprotein
HR	hazard ratio
HUNT 2	Nord-Trøndelag Health Study
IMT	intima-media thickness
MEPS	Medical Expenditure Panel Survey
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
OR	odds ratio
PA	physical activity
RR	relative risk
SBP	systolic blood pressure
SD	standard deviation
STEMI	ST-segment–elevation myocardial infarction
WHO	World Health Organization

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with 13.1% among those with higher income; similar observations were observed for boys (20.6% versus 15.6%, respectively).[7]

- According to the US National Longitudinal Study of Adolescent Health, 1.0% of adolescents were severely obese in 1996 (defined as age <20 years and BMI ≥95th sex-specific BMI-for-age growth chart or BMI ≥30 kg/m^2); the majority (70.5%) maintained this weight status into adulthood. Obese adolescents had a 16-fold increased risk of becoming severely obese adults compared with those with normal weight or those who were overweight.[8]
- NHANES 2003–2004 and 2005–2006 data were used to determine overweight and obesity prevalence in rural versus urban youth; the results showed that 39% of rural versus 32% of urban children had BMI >85th percentile.[9]

Adults

(See Table 6-1 and Chart 6-2)

- According to NHANES 2007-2010 (unpublished NHLBI tabulations):
  - —Overall, 68% of US adults were overweight or obese (73% of men and 64% of women).
  - —Among men, Mexican-Americans (81%) and non-Hispanic whites (73%) were more likely to be overweight or obese than non-Hispanic blacks (69%).
  - —Among women, non-Hispanic blacks (80%) and Mexican-Americans (78%) were more likely to be overweight or obese than non-Hispanic whites (60%).
  - —Among US adults, 35% were obese (35% of men and 36% of women).
  - —Among men, non-Hispanic blacks (38%) and Mexican-Americans (36%) were more likely to be obese than non-Hispanic whites (34%).
  - —Among women, non-Hispanic blacks (54%) and Mexican-Americans (45%) were more likely to be obese than non-Hispanic whites (33%).
- When estimates were based on self-reported height and weight in the BRFSS/CDC survey in 2011, the prevalence of obesity ranged from 20.7% in Colorado to 34.9% in Mississippi. The median percentage by state was 27.8%.[10] Additionally, no state met the Healthy People 2010 goal of reducing obesity to 15% of adults.[11]
- On the basis of 2011 data on self-reported weights and heights from the 2011 NHIS[12]:
  - —Blacks ≥18 years of age (26.4%), American Indians or Alaska Natives (27.6%), and whites (36.6%) were less likely than Asians (56.7%) to be at a healthy weight.[12]
  - —Blacks ≥18 years of age (38.9%) and American Indians or Alaska Natives (40.8%) were more likely to be obese than were whites (27.2%) and Asians (9.3%).[12]
- Most adults in Asian subgroups were in the healthy weight range, with rates ranging from 51% for Filipino adults to 68% for Chinese adults. Although the prevalence of obesity is low within the Asian adult population, Filipino adults (14%) were more than twice as likely to be obese (BMI ≥30 kg/m^2) as Asian Indian (6%), Vietnamese (5%), or Chinese (4%) adults.[13]
- According to the 2008 National Healthcare Disparities Report (on the basis of NHANES 2003–2006)[14]:
  - —Approximately 64.8% of obese adults were told by a doctor or health professional that they were overweight.
  - —The proportion of obese adults told that they were overweight was significantly lower for non-Hispanic blacks (60.5%) and Mexican Americans (57.1%) than for non-Hispanic whites (66.4%), for middle-income people than for high-income people (62.4% versus 70.6%), and for adults with less than a high school education than for those with any college education (59.2% versus 70.3%).[14]
- As judged by an analysis of data from the Multi-Ethnic Study of Atherosclerosis (MESA), a large proportion of white, black, and Hispanic participants were overweight (60%–85%) or obese (30%–50%), whereas fewer Chinese American participants were overweight (33%) or obese (5%).[15]
- In a cross-sectional study of 7000 elderly subjects at high cardiovascular risk, adherence to a Mediterranean diet, moderate alcohol consumption, the expenditure of ≥200 kcal/d in PA, and nonsmoking were associated with a 1.3-kg/m^2 (0.9–1.7) lower BMI and a 4.3-cm (3.1–5.4) lower waist circumference than having ≤1 healthy lifestyle factor.[16]
- Among severely obese (class II or III) patients, a 1-year intensive lifestyle intervention of diet and PA achieved clinically significant weight loss and improvement in cardiometabolic factors in addition to reduction in visceral and hepatic fat content.[17]

Trends

Youth

(See Chart 6-3)

- Among infants and children between 6 and 23 months of age, the prevalence of high weight for recumbent length was 7% in 1976–1980 and 12% in 2003–2006 (NHANES, NCHS).[18]
- The obesity epidemic in children continues to grow on the basis of recent data from the Bogalusa Heart Study. Compared with 1973 to 1974, the proportion of children 5 to 17 years of age who were obese was 5 times higher in 2008 to 2009.[19]
- A comparison of NHANES 2009–2010 data with 1999–2000 data demonstrates an increase in obesity prevalence in male youth of 5% (OR, 1.05; 95% CI, 1.01–1.10) but not in female youth (OR, 1.02; 95% CI, 0.98–1.07).[2]

Adults

- On the basis of 2009 self-reported BRFSS data, overall obesity prevalence was 26.7% in the United States, with rates of 27.4% in men and 26.0% in women. By race/ethnicity, the prevalence of obesity among non-Hispanic whites was 25.2%, whereas it was 36.8% among non-Hispanic blacks and 30.7% among Hispanics. There was an inverse association by education level: College graduates had a 20.8% rate of obesity, whereas those who attained less than a high school education had an obesity prevalence of 32.9%.[20]
- The prevalence of obesity increased by 5.6% or ≈2.7 million people from 1997 to 2002 among Medicare beneficiaries.

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By 2002, 21.4% of beneficiaries and 39.3% of disabled beneficiaries were obese compared with 16.4% and 32.5%, respectively, in 1997.[21]

- The population attributable fraction for CHD associated with reducing current population mean BMI to 21 kg/m^2 in the Asia-Pacific region ranged from 2% in India to 58% in American Samoa; the population attributable fraction for ischemic stroke ranged from 3% in India to 64% in American Samoa. These data from 15 countries show the proportion of CVD that would be prevented if the population mean BMI were reduced below the current overweight cut point.[22a]
- The CARDIA study showed that young adults who were overweight or obese had lower health-related quality of life than normal-weight participants 20 years later. On the basis of data from the Medical Outcomes Study 12-item short-form health survey, overweight and obese participants had lower multivariable-adjusted scores on the physical component summary score but not on the mental component summary score.[22]
- Forecasts through 2030 using the BRFSS 1990–2008 data set suggest that by 2030, 51% of the population will be obese, with 11% with severe obesity, an increase of 33% for obesity and 130% for severe obesity.[23]

Morbidity

- Overweight children and adolescents are at increased risk for future adverse health effects, including the following[24]:
  - —Increased prevalence of traditional cardiovascular risk factors such as hypertension, hyperlipidemia, and DM.
  - —Poor school performance, tobacco use, alcohol use, premature sexual behavior, and poor diet.
  - —Other associated health conditions, such as asthma, hepatic steatosis, sleep apnea, stroke, some cancers (breast, colon, and kidney), musculoskeletal disorders, and gallbladder disease.
- According to data from the Bogalusa Heart Study and the Young Finns study, adolescents with high BMI in the overweight or obese range are at a 2.5-fold increased risk of developing metabolic syndrome, a 2.2-fold increased risk of high carotid IMT, and a 3.4-fold increased risk of DM in adulthood.[25]
- The increasing prevalence of obesity is driving an increased incidence of type 2 DM. Data from the FHS indicate a doubling in the incidence of DM over the past 30 years, most dramatically during the 1990s and primarily among individuals with a BMI >30 kg/m^2.[26]
- Obesity was the most powerful predictor of DM in the Nurses' Health Study. Women with a BMI of ≥35 kg/m^2 had an RR for DM of 38.8 compared with women with a BMI of <23 kg/m^2.[27]
- Obesity is also a strong predictor of sleep-disordered breathing, itself strongly associated with the development of CVD, as well as with myriad other health conditions, including numerous cancers, nonalcoholic fatty liver disease, gallbladder disease, musculoskeletal disorders, and reproductive abnormalities.[28]
- A recent meta-analysis of 15 prospective studies demonstrated the increased risk for Alzheimer disease or vascular dementia and any dementia was 1.35 and 1.26 for overweight, respectively, and 2.04 and 1.64 for obesity, respectively.[29]
- A randomized clinical trial of 130 severely obese adult individuals randomized to either 12 months of diet and PA or only 6 months of PA resulted in 12.1 and 9.9 kg, respectively, of weight loss at 1 year, with improvements in waist circumference, visceral fat, BP, and insulin resistance.[17]
- Data from 4 Finnish cohort studies examining childhood and adult BMI with a mean follow-up of 23 years found that overweight or obese children who remained obese in adulthood had increased risks of type 2 DM, hypertension, dyslipidemia, and carotid atherosclerosis. However, those who became normal weight by adulthood had risks comparable to individuals who were never obese.[30]
- In a meta-analysis from 58 cohorts, representing 221 934 people in 17 developed countries with 14 297 incident CVD outcomes, BMI, waist circumference, and waist-to-hip ratio were only minimally associated with cardiovascular outcomes after controlling for baseline SBP, DM, and total and HDL cholesterol in addition to age, sex, and smoking status. Measures of adiposity also did not improve risk discrimination or reclassification when risk factor data were included.[31]
- Ten-year follow-up data from the Swedish Obese Subjects intervention study indicated that to maintain a favorable effect on cardiovascular risk factors, more than the short-term goal of 5% weight loss is needed to overcome secular trends and aging effects.[32]
- An obesity paradox has been reported, with obese patients demonstrating favorable outcomes in CHF, hypertension, peripheral vascular disease, and coronary artery disease (CAD). In the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) study, a multicenter trial of atrial fibrillation (AF), obese patients had lower all-cause mortality (HR=0.77, P=0.01) than normal-weight patients after multivariable adjustment over a 3-year follow-up period.[33]
- A systematic review of prospective studies examining overweight and obesity as predictors of major stroke subtypes in >2 million participants over ≥4 years found an adjusted RR for ischemic stroke of 1.22 (1.05–1.41) in overweight individuals and an RR of 1.64 (1.36–1.99) for obese individuals relative to normal-weight individuals. RRs for hemorrhagic stroke were 1.01 (0.88–1.17) and 1.24 (0.99–1.54) for overweight and obese individuals, respectively. These risks were graded with increasing BMI and were independent of age, lifestyle, and other cardiovascular risk factors.[34]

Mortality

- Elevated childhood BMIs in the highest quartile were associated with premature death as an adult in a cohort of 4857 American Indian children during a median follow-up of 23.9 years.[35]
- Among adults, obesity was associated with nearly 112 000 excess deaths (95% CI, 53 754–170 064) relative to normal weight in 2000. Grade I obesity (BMI 30 to <35 kg/m^2) was associated with almost 30 000 of these excess deaths (95% CI, 8534–68 220) and grade II to III obesity (BMI ≥35 kg/m^2) with >82 000 (95% CI, 44 843–119 289). Underweight was associated with nearly 34 000 excess deaths (95% CI,

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15 726–51 766). As other studies have found,[36] overweight (BMI 25 to <30 kg/m^2) was not associated with excess deaths.[37]

- Overweight was associated with significantly increased mortality resulting from DM or kidney disease and was not associated with increased mortality resulting from cancer or CVD in an analysis of 2004 data from NHANES. Obesity was associated with significantly increased mortality caused by CVD, some cancers, and DM or kidney disease. Obesity was associated with 13% of CVD deaths in 2004.[38]
- In a collaborative analysis of data from almost 900 000 adults in 57 prospective studies, mostly in western Europe and North America, overall mortality was lowest at a BMI of ≈22.5 to 25 kg/m^2 in both sexes and at all ages, after exclusion of early follow-up and adjustment for smoking status. Above this range, each 5-kg/m^2-higher BMI was associated with ≈30% higher all-cause mortality, and no specific cause of death was inversely associated with BMI. Below 22.5 to 25 kg/m^2, the overall inverse association with BMI was predominantly related to strong inverse associations for smoking-related respiratory disease, and the only clearly positive association was for ischemic heart disease.[39]
- In a meta-analysis of 1.46 million white adults, over a mean follow-up period of 10 years, all-cause mortality was lowest at BMI levels of 20.0 to 24.9 kg/m^2. Among women, compared with a BMI of 22.5 to 24.9 kg/m^2, the HRs for death were as follows: BMI 15.0 to 18.4 kg/m^2, 1.47; 18.5 to 19.9 kg/m^2, 1.14; 20.0 to 22.4 kg/m^2, 1.0; 25.0 to 29.9 kg/m^2, 1.13; 30.0 to 34.9 kg/m^2, 1.44; 35.0 to 39.9 kg/m^2, 1.88; and 40.0 to 49.9 kg/m^2, 2.51. Similar estimates were observed in men.[40]
- Calculations based on NHANES data from 1978–2006 suggest that the gains in life expectancy from smoking cessation are beginning to be outweighed by the loss of life expectancy related to obesity.[41]
- Because of the increasing prevalence of obesity, the number of quality-adjusted life-years lost as a result of obesity is similar to or greater than that lost as a result of smoking, according to data from the BRFSS.[42]
- Recent estimates suggest that reductions in smoking, cholesterol, BP, and PA levels resulted in a gain of 2 770 500 life-years; however, these gains were reduced by a loss of 715 000 life-years caused by the increased prevalence of obesity and DM.[43]
- Using self-reported NHIS data with linked mortality files through 2006, the HR for all-cause mortality was 1.07 (95% CI, 0.91–1.26) for overweight individuals, 1.41 (95% CI, 1.16–1.73) for obese individuals, and 2.46 for extremely obese individuals (95% CI, 1.91–3.16).[44]
- In a comparison of 5 different anthropometric variables (BMI, waist circumference, hip circumference, waist-to-hip ratio, and waist-to-height ratio) in 62 223 individuals from Norway with 12 years of follow-up from the HUNT 2 study (Nord-Trøndelag Health Study), the risk of death per SD increase in each measure was 1.02 (95% CI, 0.99–1.06) for BMI, 1.10 (95% CI, 1.06–1.14) for waist circumference, 1.01 (95% CI, 0.97–1.05) for hip circumference, 1.15 (95% CI, 1.11–1.19) for waist-to-hip ratio, and 1.12 (95% CI, 1.08–1.16) for waist-to-height ratio. For CVD mortality, the risk of death per SD increase was 1.12 (95% 1.06–1.20) for BMI, 1.19 (1.12–1.26) for waist circumference, 1.06 (1.00–1.13) for hip circumference, 1.23 (95% CI, 1.16–1.30) for waist-to-hip ratio, and 1.24 (95% CI, 1.16–1.31) for waist-to-height ratio.[45]
- According to data from the National Cardiovascular Data Registry, among patients presenting with ST-segment–elevation myocardial infarction (STEMI) and a BMI of at least 40 kg/m^2, in-hospital mortality rates were higher for patients with class III obesity (OR, 1.64; 95% CI, 1.32–2.03) when class I obesity was used as the referent.[46]
- The Canadian Heart Health Surveys from 1986 to 1995 demonstrated an increased risk of cancer across BMI categories, with an HR of 1.34 (95% CI, 1.01–1.78) for those with BMI 30 to 34.9 kg/m^2 and an HR of 1.82 (95% CI, 1.22–2.71) for those with BMI ≥35 kg/m^2.[47]
- In a study of 22 203 women and men from England and Scotland, metabolically unhealthy obese individuals were at an increased risk of all-cause mortality compared with metabolically healthy obese individuals (HR, 1.72; 95% CI, 1.23–2.41).[48]

Cost

- Among children and adolescents, annual hospital costs related to obesity were $127 million between 1997 and 1999.[49]
- According to 1 study, overall estimates show that the annual medical burden of obesity has increased to almost 10% of all medical spending and could amount to $147 billion per year in 2008 (in 2008 dollars).[50]
- If current trends in the growth of obesity continue, total healthcare costs attributable to obesity could reach $861 to $957 billion by 2030, which would account for 16% to 18% of US health expenditures.[51]
- According to NHANES I data linked to Medicare and mortality records, obese 45-year-olds had lifetime Medicare costs of $163 000 compared with $117 000 among those with normal weight by the time they reached 65 years of age.[52]
- The total excess cost related to the current prevalence of adolescent overweight and obesity is estimated to be $254 billion ($208 billion in lost productivity secondary to premature morbidity and mortality and $46 billion in direct medical costs).[53]
- According to 2006 MEPS and 2006 BRFSS data, annual medical expenditures would be 6.7% to 10.7% lower in the absence of obesity.[54]
- According to data from the Medicare Current Beneficiary Survey from 1997–2006, in 1997, expenditures for a Part A and Part B services beneficiary were $6832 for a normal-weight individual, which was more than for overweight ($5473) or obese ($5790) individuals. However, over time, expenses increased more rapidly for overweight and obese individuals.[55]
- The costs of obesity are high: Obese people pay on average $1429 (42%) more for healthcare costs than normal-weight individuals. For obese beneficiaries, Medicare pays $1723 more, Medicaid pays $1021 more, and private insurances pay $1140 more than for beneficiaries who are at normal weight. Similarly, obese people have 46% higher inpatient costs and 27% more outpatient visits and spend 80% more on prescription drugs.[50]

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Bariatric Surgery

- Patients with BMI >40 kg/m^2 or >35 kg/m^2 with an obesity-related comorbidity are eligible for gastric bypass surgery, which is typically performed as either a Roux-en-Y gastric bypass or a biliopancreatic diversion.
- According to the 2006 NHDS, the incidence of bariatric surgery was estimated at 113 000 cases per year, with costs of ≈1.5 billion dollars annually.[56]
- Among obese Swedish patients undergoing bariatric surgery and followed up for up to 15 years, maximum weight loss was 32%. The risk of death was 0.76 among those who underwent bariatric surgery compared with matched control subjects.[57] More recent data examining MI and stroke showed that bariatric surgery was associated with fewer CVD deaths (HR, 0.47; 95% CI, 0.29–0.76) and fewer strokes (HR, 0.67; 95% CI, 0.54–0.83) than in the control group.[58]
- Among 641 patients followed up for 10 years compared with 627 matched control subjects, after 2 years of follow-up, 72% of the surgically treated patients versus 21% of the control patients had remission of their DM; at 10 years of follow-up, results were 36% and 13%, respectively. Similar results have been observed for hypertension, elevated triglycerides, and low HDL cholesterol.[59]
- According to retrospective data from the United States, among 9949 patients who underwent gastric bypass surgery, after a mean of 7 years, long-term mortality was 40% lower among the surgically treated patients than among obese control subjects. Specifically, cancer mortality was reduced by 60%, DM mortality by 92%, and CAD mortality by 56%. Non-disease death rates (eg, accidents, suicide) were 58% higher in the surgery group.[60]
- A recent retrospective cohort from the Veterans Affairs medical system showed that in a propensity-matched analysis, bariatric surgery was not associated with reduced mortality compared with obese control subjects (time-adjusted HR, 0.94; 95% CI, 0.64–1.39).[61]
- Two recent randomized controlled trials were performed that randomized bariatric surgery compared with intensive medical treatment among patients with type 2 DM. The first study randomized 150 patients and conducted 12-month follow-up; this study showed that glycemic control improved (6.4%) and weight loss was greater (29.4 versus 5.4 kg) in the surgical arm.[62] The second trial randomized 60 patients to bariatric surgery versus medical therapy and conducted follow-up for 24 months. The results showed that DM remission occurred in 75% of the group that underwent gastric bypass surgery compared with 0% of those in the medical treatment arm, with HbA_{1c} values of 6.35% in the surgical arm compared with 7.69% in the medical treatment arm.[63]
- A recent cost-effectiveness study of laparoscopic adjustable gastric banding showed that after 5 years, $4970 was saved in medical expenses; if indirect costs were included (absenteeism and presenteeism), savings increased to $6180 and $10 960, respectively.[64]

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Table 6-1. Overweight and Obesity

	Prevalence of Overweight and Obesity, 2007–2010, Age >20 y	Prevalence of Obesity in Adults, 2007–2010, Age >20 y	Prevalence of Overweight and Obesity in Children, 2009–2010, Ages 2–19 y	Prevalence of Obesity in Children, 2009–2010, Ages 2–19 y	Cost, 2008*
Both sexes, n (%)	154 700 000 (68.2)	78 400 000 (34.6)	23 900 000 (31.8)	12 700 000 (16.9)	$147 Billion
Males	79 900 000 (72.9)	36 800 000 (33.6)	12,700 000 (33.0)	7 200 000 (18.6)	…
Females	74 800 000 (63.7)	41 600 000 (35.6)	11 200 000 (30.4)	5 500,000 (15.0)	…
NH white males, %	73.1	33.8	30.1	16.1	…
NH white females, %	60.2	32.5	25.6	11.7	…
NH black males, %	68.7	37.9	36.9	24.3	…
NH black females, %	79.9	53.9	41.3	24.3	…
Mexican American males, %	81.3	36.0	40.5	24.0	…
Mexican American females, %	78.2	44.8	38.2	18.2	…

Overweight and obesity in adults is defined as body mass index (BMI) ≥25 kg/m^2. Obesity in adults is defined as BMI ≥30 kg/m^2. In children, overweight and obesity are based on BMI-for-age values at or above the 85th percentile of the 2000 Centers for Disease Control and Prevention (CDC) growth charts. In children, obesity is based on BMI-for-age values at or above the 95th percentile of the CDC growth charts. In January 2007, the American Medical Association's Expert Task Force on Childhood Obesity recommended new definitions for overweight and obesity in children and adolescents[65]; however, statistics based on this new definition are not yet available.

NH indicates non-Hispanic; ellipses (…), data not available.

*Data from *Health Affairs*.[50]

Sources: National Health and Nutrition Examination Survey (NHANES) 2007–2010 (adults), unpublished National Heart, Lung, and Blood Institute (NHLBI) tabulation; NHANES 2009–2010 (ages 2–19 years) from Ogden et al.[2]

Extrapolation for ages 2 to 19 years from NHLBI tabulation of US Census resident population on April 1, 2010.

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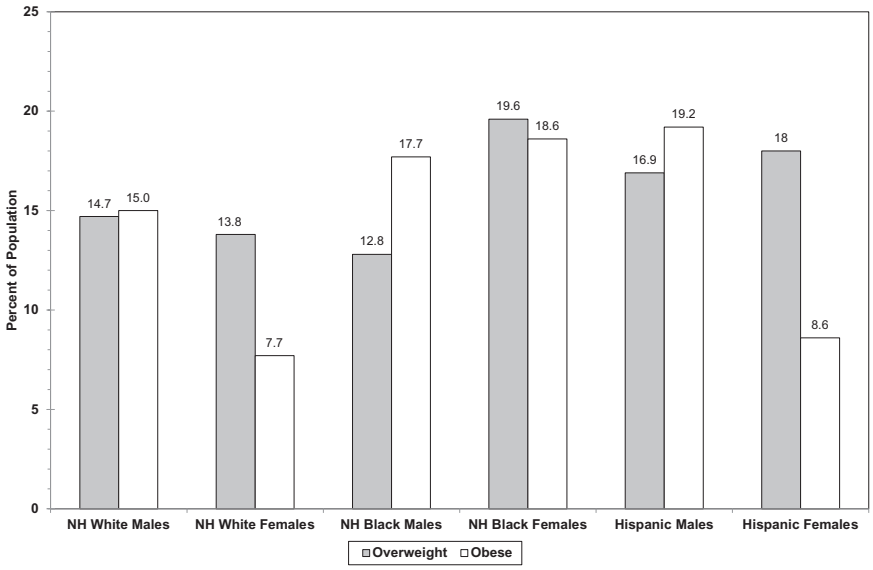

Chart 6-1. Prevalence of overweight and obesity among students in grades 9 through 12 by sex and race/ethnicity. NH indicates non-Hispanic. Data derived from Youth Risk Behavior Surveillance—United States, 2011, Table 101.[66]

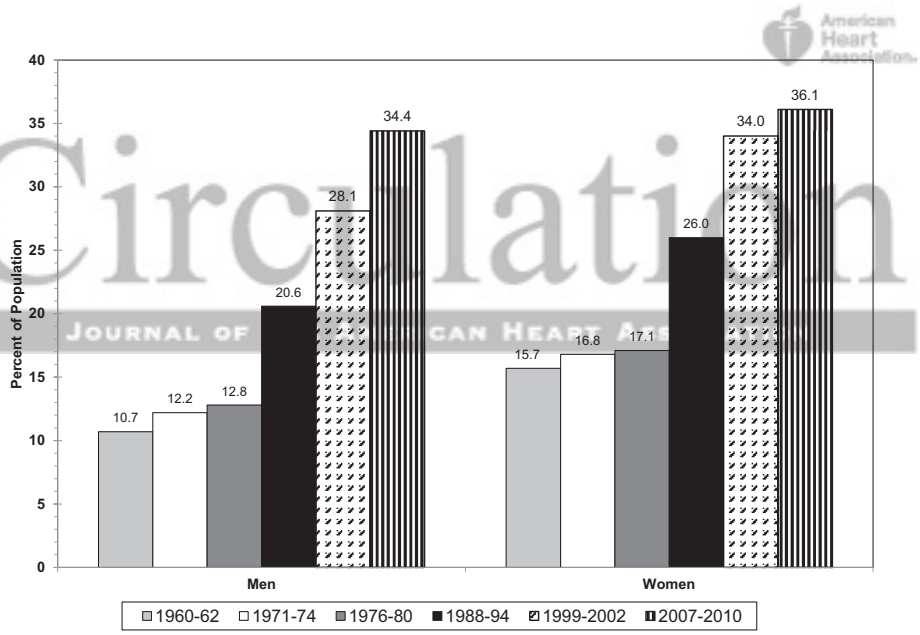

Chart 6-2. Age-adjusted prevalence of obesity in adults 20 to 74 years of age by sex and survey year (National Health Examination Survey: 1960–1962; National Health and Nutrition Examination Survey: 1971–1974, 1976–1980, 1988–1994, 1999–2002, and 2007–2010). Obesity is defined as body mass index of 30.0 kg/m^2. Data derived from Health, United States, 2011 (National Center for Health Statistics).[67]

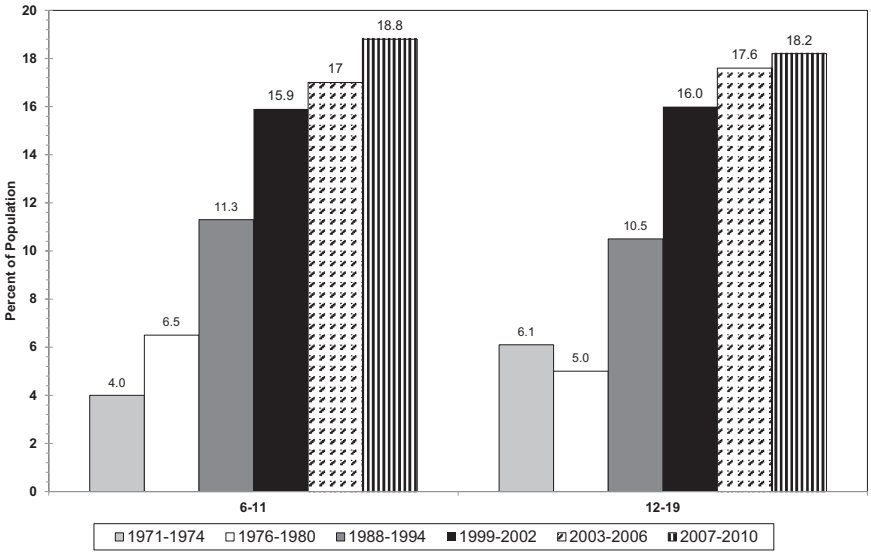

Chart 6–3. Trends in the prevalence of obesity among US children and adolescents by age and survey year (National Health and Nutrition Examination Survey: 1971–1974, 1976–1980, 1988–1994, 1999–2002 , 2003–2006 and 2007–2010). Data derived from Health, United States, 2011 (National Center for Health Statistics).[67]

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7. Family History and Genetics

See Tables 7-1 through 7-3.

Biologically related first-degree relatives (siblings, offspring and parents) share roughly 50% of their genetic variation with one another. This constitutes much greater sharing of genetic variation than with a randomly selected person from the population, and thus, when a trait aggregates within a family, this lends evidence for a genetic risk factor for the trait. Similarly, racial/ethnic minorities are more likely to share their genetic variation within their demographic than with other demographics. Familial aggregation of CVD may be related to aggregation of specific behaviors (eg, smoking, alcohol use) or risk factors (eg, hypertension, DM, obesity) that may themselves have environmental and genetic contributors. Unlike classic mendelian genetic risk factors, whereby usually 1 mutation directly causes 1 disease, a complex trait's genetic contributors may increase risk without necessarily always causing the condition. The effect size of any specific contributor to risk may be small but widespread throughout a population, or may be large but affect only a small population, or may have an enhanced risk when an environmental contributor is present. Although the breadth of all genetic research into CVD is beyond the scope of this chapter, we present a summary of evidence that a genetic risk for CVD is likely, as well as a summary of evidence on the most consistently replicated genetic markers for HD and stroke identified to date.

Abbreviations Used in Chapter 7

AAA	abdominal aortic aneurysm
ABI	ankle-brachial index
AF	atrial fibrillation
BMI	body mass index
CAC	coronary artery calcification
CARDIoGRAM	Coronary Artery Disease Genome-wide Replication and Meta-Analysis Consortium
CI	confidence interval
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
FHS	Framingham Heart Study
GFR	glomerular filtration rate
HbA_{1c}	glycosylated hemoglobin
HD	heart disease
HDL	high-density lipoprotein
HF	heart failure
LDL	low-density lipoprotein
MI	myocardial infarction
NHANES	National Health and Nutrition Examination Survey
OR	odds ratio
SBP	systolic blood pressure
SNP	single-nucleotide polymorphism

Family History

Prevalence

- Among adults ≥20 years of age, 12.6% reported having a parent or sibling with a heart attack or angina before the age of 50 years. The racial/ethnic breakdown is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - For non-Hispanic whites, 12.4% for men, 14.9% for women
  - For non-Hispanic blacks, 8.1% for men, 13.0% for women
  - For Mexican Americans, 8.1% for men, 10.0% for women
  - For other Hispanics, 8.8% for men, 12.0% for women
  - For other races, 8.7% for men, 10.7% for women
- HD occurs as people age, and those without a family history of HD may survive longer, so the prevalence of family history will vary depending on the age at which it is assessed. The breakdown of reported family history of heart attack by age in the US population as measured by NHANES is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - Age 20 to 39 years, 8.4% for men, 10.3% for women
  - Age 40 to 59 years, 12.8% for men, 15.3% for women
  - Age 60 to 79 years, 13.7% for men, 17.5% for women
  - Age ≥80 years, 9.8% for men, 13.7% for women
- In the multigenerational FHS, only 75% of participants with a documented parental history of a heart attack before age 55 years reported that history when asked.[1]

Impact of Family History

- Premature paternal history of a heart attack has been shown to approximately double the risk of a heart attack in men and increase the risk in women by ≈70%.[2,3]
- History of a heart attack in both parents increases the risk of heart attack, especially when 1 parent had a premature heart attack[4] (Table 7-1).
- Sibling history of HD has been shown to increase the odds of HD in men and women by ≈50%.[5]
- Premature family history of angina, MI, angioplasty, or bypass surgery increased the lifetime risk by ≈50% for both HD (from 8.9% to 13.7%) and CVD mortality (from 14.1% to 21%).[6]
- Similarly, parental history of AF is associated with ≈80% increased odds of AF in men and women,[7] and a history of stroke in a first-degree relative increased the odds of stroke in men and women by ≈50%.[8]

Genetics

Heart Disease

- Genome-wide association is a robust technique to identify associations between genotypes and phenotypes. Table 7-2 presents results from the CARDIoGRAM (Coronary ARteryDIsease Genome-wide Replication And Meta-analysis) consortium, which represents the largest genetic study of MI to date, with 22 233 MI case subjects and 64 762 control subjects and with independent validation in an additional 56 682 individuals.[9] Altogether, there are 23

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well-replicated loci for MI. The ORs are modest, ranging from 1.06 to 1.51 per copy of the risk allele (individuals may harbor up to 2 copies of a risk allele). However, these are common alleles, which suggests that the attributable risk may be substantial.

- Genetic markers discovered thus far have not been shown to add to cardiovascular risk prediction tools beyond current models that incorporate family history.[10] Genetic markers have also not been shown to improve prediction of subclinical atherosclerosis beyond traditional risk factors.[11]
- The most consistently replicated genetic marker for HD in European-derived populations is located at 9p21.3. At this single-nucleotide polymorphism, ≈27% of the white population is estimated to have 0 risk alleles, 50% is estimated to have 1 risk allele, and the remaining 23% is estimated to have 2 risk alleles.[12]
- The 10-year HD risk for a 65-year-old man with 2 risk alleles at 9p21.3 and no other traditional risk factors is ≈13.2%, whereas a similar man with 0 alleles would have a 10-year risk of ≈9.2%. The 10-year HD risk for a 40-year-old woman with 2 alleles and no other traditional risk factors is ≈2.4%, whereas a similar woman with 0 alleles would have a 10-year risk of ≈1.7%.[12]
- Variation at the 9p21.3 region also increases the risk of HF[13] and sudden death.[14] Associations have also been observed between the 9p21.3 region and coronary artery calcification (CAC).[15,16] Additionally, stronger associations have been found between variation at 9p21.3 and earlier[17,18] and more severe[19] heart attacks. The biological mechanism underpinning the association of genetic variation in the 9p21 region with disease outcomes is still under investigation.

Stroke

- The same 9p21.3 region has also been associated with intracranial aneurysm, abdominal aortic aneurysm (AAA),[20] and ischemic stroke.[21]
- For large-vessel ischemic stroke, a new association for large-vessel stroke with histone deacetylase 9 on chromosome 7p21.1 has been identified (>9000 subjects) and replicated (>12 000 subjects).[21]

CVD Risk Factors

- Heritability is the ratio of genetically caused variation to the total variation of a trait or measure. Table 7-3 presents heritability estimates for standard CVD risk factors using data generated from the FHS. These data suggest that most CVD risk factors have at least moderate heritability.

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Table 7-1. Odds Ratio for Combinations of Parental Heart Attack History

	OR (95% CI)
No family history	1.00
One parent with heart attack ≥50 y of age	1.67 (1.55–1.81)
One parent with heart attack <50 y of age	2.36 (1.89–2.95)
Both parents with heart attack ≥50 y of age	2.90 (2.30–3.66)
Both parents with heart attack, one <50 y of age	3.26 (1.72–6.18)
Both parents with heart attack, both <50 y of age	6.56 (1.39–30.95)

OR indicates odds ratio; CI, confidence interval.
Data derived from Chow et al.[4]

Table 7-2. Validated SNPs for MI, the Nearest Gene, and the OR From the CARDIoGRAM Consortium

SNP	Chromosomal Region	Gene	Effect Size (OR)	Minor Allele Frequency
rs599839	1p13.3	*SORT1*	1.11	0.22
rs17465637	1q41	*MIA3*	1.14	0.26
rs17114036	1p32.2	*PPAP2B*	1.17	0.09
rs11206510	1p32.3	*PCSK9*	1.08	0.18
rs6725887	2q33	*WDR12*	1.14	0.15
rs2306374	3q22.3	*MRAS*	1.12	0.18
rs17609940	6p21.31	*ANKS1A*	1.07	0.25
rs12526453	6p24.1	*PHACTR1*	1.10	0.33
rs12190287	6q23.2	*TCF21*	1.08	0.38
rs798220	6q25	*LPA*	1.51	0.02
rs11556924	7q32.2	*ZC3HC1*	1.09	0.38
rs4977574	9p21.3	*CDKN2A, CDKN2B*	1.29	0.46
rs579459	9q34.2	*ABO*	1.10	0.21
rs1746048	10q11	*CXCL12*	1.09	0.13
rs12413409	10q24.32	*CYP17A1-CNNM2-NT5C2*	1.12	0.11
rs964184	11q23.3	*ZNF259-APOA5-A4-C3-A1*	1.13	0.13
rs3184504	12q24	*Sh2b3*	1.07	0.44
rs4773144	13q34	*COL4A1-COL4A2*	1.07	0.44
rs2895811	14q32.2	*HHIPL1*	1.08	0.43
rs3825807	15q25.1	*ADAMTS7*	1.07	0.43
rs216172	17p13.3	*SMG6-SRR*	1.07	0.37
rs12936587	17p11.2	*RASD1-SMCR3-PEMT*	1.07	0.44
rs46522	17q21.32	*UBE2Z-GIP-ATP5G1-SNF8*	1.06	0.47
rs1122608	19q13.2	*LDLR*	1.14	0.23
rs9982601	21q22.11	*MRPS6*	1.18	0.15

SNPs indicates single-nucleotide polymorphisms; MI, myocardial infarction; OR, odds ratio; and CARDIoGRAM, Coronary Artery DIsease Genome-wide Replication And Meta-analysis Consortium.

Data derived from Schunkert et al.[9]

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Table 7-3. Heritability of CVD Risk Factors From the FHS

Trait	Heritability
ABI	0.21[22]
SBP	0.42[23]
DBP	0.39[23]
Left ventricular mass	0.24 to 0.32[24]
BMI	0.37 (mean age 40 y) to 0.52 (mean age 60 y)[25]
Waist circumference	0.41[26]
Visceral abdominal fat	0.36[27]
Subcutaneous abdominal fat	0.57[27]
Fasting glucose	0.34[28]
HbA_{1c}	0.27[28]
Triglycerides	0.48[29]
HDL cholesterol	0.52[29]
Total cholesterol	0.57[29]
LDL cholesterol	0.59[29]
Estimated GFR	0.33[30]

CVD indicates cardiovascular disease; FHS, Framingham Heart Study; ABI, ankle-brachial index; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; HbA1c, glycosylated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; and GFR, glomerular filtration rate.

8. High Blood Cholesterol and Other Lipids

See Table 8-1 and Charts 8-1 through 8-3.

Prevalence

For information on dietary cholesterol, total fat, saturated fat, and other factors that affect blood cholesterol levels, see Chapter 5 (Nutrition).

Youth

(See Chart 8-1.)

- Among children 4 to 11 years of age, the mean total blood cholesterol level is 161.9 mg/dL. For boys, it is 162.3 mg/dL; for girls, it is 161.5 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - For non-Hispanic whites, 160.9 mg/dL for boys and 161.6 mg/dL for girls
  - For non-Hispanic blacks, 165.2 mg/dL for boys and 157.9 mg/dL for girls
  - For Mexican Americans, 159.6 mg/dL for boys and 160.7 mg/dL for girls
- Among adolescents 12 to 19 years of age, the mean total blood cholesterol level is 158.2 mg/dL. For boys, it is 156.1 mg/dL; for girls, it is 160.3 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - For non-Hispanic whites, 156.8 mg/dL for boys and 161.1 mg/dL for girls
  - For non-Hispanic blacks, 154.1 mg/dL for boys and 160.6 mg/dL for girls
  - For Mexican Americans, 157.8 mg/dL for boys and 158.0 mg/dL for girls
- The prevalence of abnormal lipid levels among youths 12 to 19 years of age is 20.3%; 14.2% of normal-weight youths, 22.3% of overweight youths, and 42.9% of obese youths have at least 1 abnormal lipid level (NHANES 1999–2006, NCHS).[1]
- Approximately 7.8% of adolescents 12 to 19 years of age have total cholesterol levels ≥200 mg/dL (NHANES 2007–2010, unpublished NHLBI tabulation).

Abbreviations Used in Chapter 8

BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CVD	cardiovascular diseases
DM	diabetes mellitus
HD	heart disease
HDL	high-density lipoprotein
LDL	low-density lipoprotein
Mex. Am.	Mexican American
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute

- Fewer than 1% of adolescents are potentially eligible for pharmacological treatment on the basis of guidelines from the American Academy of Pediatrics.[1,2]

Adults

(See Table 8-1 and Charts 8-2 and 8-3.)

- An estimated 31.9 million adults ≥20 years of age have total serum cholesterol levels ≥240 mg/dL (extrapolated to 2010 by use of NCHS/NHANES 2007–2010 data), with a prevalence of 13.8% (Table 8-1; unpublished NHLBI tabulation).
- Approximately 5.6% of adults ≥20 years of age have undiagnosed hypercholesterolemia[3] (NHANES 2007–2010, unpublished NHLBI tabulation).
- Between the periods 1988–1994 and 1999–2002 (NHANES/NCHS), the age-adjusted mean total serum cholesterol level of adults ≥20 years of age decreased from 206 to 203 mg/dL, and LDL cholesterol levels decreased from 129 to 123 mg/dL.[4]
- Data from NHANES 2003–2008 (NCHS) showed the serum total crude mean cholesterol level in adults ≥20 years of age was 195 mg/dL for men and 201 mg/dL for women.[5]
- Data from the Minnesota Heart Survey (1980–1982 to 2000–2002) showed a decline in age-adjusted mean total cholesterol concentrations from 5.49 and 5.38 mmol/L for men and women, respectively, in 1980–1982 to 5.16 and 5.09 mmol/L, respectively, in 2000–2002; however, the decline was not uniform across all age groups. Middle-aged to older people have shown substantial decreases, but younger people have shown little overall change and recently had increased total cholesterol values. Lipid-lowering drug use rose significantly for both sexes among those 35 to 74 years of age. Awareness, treatment, and control of hypercholesterolemia have increased; however, more than half of those at borderline-high risk remain unaware of their condition.[6]
- According to data from NHANES 2005–2006, between the periods 1999 to 2000 and 2005 to 2006, mean serum total cholesterol levels in adults ≥20 years of age declined from 204 to 199 mg/dL. This decline was observed for men ≥40 years of age and for women ≥60 years of age. There was little change over this time period for other sex/age groups. In 2005 to 2006, ≈65% of men and 70% of women had been screened for high cholesterol in the past 5 years, and 16% of adults had serum total cholesterol levels ≥240 mg/dL.[7]
- According to data from NHANES, from 1999 to 2006, the prevalence of elevated LDL cholesterol levels (as defined by levels higher than the specified Adult Treatment Panel III risk category) in adults ≥20 years of age has decreased by ≈33%.[8]
- During the period from 1999 to 2006, 26.0% of adults had hypercholesterolemia, 9% of adults had both hypercholesterolemia and hypertension, 1.5% of adults had DM and hypercholesterolemia, and 3% of adults had all 3 conditions.[3]

Screening

- Data from the BRFSS study of the CDC in 2011 showed that the percentage of adults who had been screened for high blood cholesterol in the preceding 5 years ranged from 66.3% in Utah to 83.7% in Massachusetts. The median percentage among all 50 states was 75.5%.[9]

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- The percentage of adults who reported having had a cholesterol check increased from 68.6% during 1999 to 2000 to 74.8% during 2005 to 2006.[10]

Awareness

- Data from the BRFSS (CDC) survey in 2011 showed that among adults screened for high blood cholesterol, the percentage who had been told that they had high blood cholesterol ranged from 33.5% in Colorado to 42.3% in Mississippi. The median percentage among states was 38.4%.[9]
- Among adults with hypercholesterolemia, the percentage who had been told that they had high cholesterol increased from 42.0% during 1999 to 2000 to 50.4% during 2005 to 2006.[10]

Treatment

- NHANES data on the treatment of high LDL cholesterol showed an increase from 28.4% of individuals during 1999 to 2002 to 48.1% during 2005 to 2008.[11]
- Self-reported use of cholesterol-lowering medications increased from 8.2% during 1999 to 2000 to 14.0% during 2005 to 2006.[10]

Adherence

Youth

The American Academy of Pediatrics recommends screening for dyslipidemia in children and adolescents who have a family history of dyslipidemia or premature CVD, those whose family history is unknown, and those youths with risk factors for CVD, such as being overweight or obese, having hypertension or DM, or being a smoker.[1]

Analysis of data from NHANES 1999–2006 showed that the overall prevalence of abnormal lipid levels among youths 12 to 19 years of age was 20.3%.[1]

Adults

- On the basis of data from the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults[12]:
  - Fewer than half of all people who qualify for any kind of lipid-modifying treatment for CHD risk reduction are receiving it.
  - Fewer than half of even the highest-risk people (those with symptomatic CHD) are receiving lipid-lowering treatment.
  - Only approximately one third of treated patients are achieving their LDL goal; <20% of patients with CHD are at their LDL goal.
- Data from NHANES 2005–2006 indicate that among those with elevated LDL cholesterol levels, 35.5% had not been screened previously, 24.9% were screened but not told they had elevated cholesterol, and 39.6% were treated inadequately.[8]
- There were 33.2% of adults overall during 2005–2008 in NHANES who achieved LDL cholesterol goals. Among adults without health insurance, only 22.6% achieved LDL cholesterol goals; however, 82.8% of those adults with uncontrolled LDL cholesterol did have some form of health insurance.[11]

Lipid Levels

LDL (Bad) Cholesterol

Youth

- There are limited data available on LDL cholesterol for children 4 to 11 years of age.
- Among adolescents 12 to 19 years of age, the mean LDL cholesterol level is 89.5 mg/dL. For boys, it is 88.6 mg/dL, and for girls, it is 90.5 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - Among non-Hispanic whites, 90.4 mg/dL for boys and 90.9 mg/dL for girls
  - Among non-Hispanic blacks, 85.8 mg/dL for boys and 91.8 mg/dL for girls
  - Among Mexican Americans, 90.6 mg/dL for boys and 87.1 mg/dL for girls
- High levels of LDL cholesterol occurred in 7.3% of male adolescents and 7.6% of female adolescents during 2007 to 2010.[1]

Adults

- The mean level of LDL cholesterol for American adults ≥20 years of age was 115.8 mg/dL in 2007 to 2010.[8] Levels of 130 to 159 mg/dL are considered borderline high, levels of 160 to 189 mg/dL are classified as high, and levels of ≥190 mg/dL are considered very high according to Adult Treatment Panel III.
- According to NHANES 2007–2010 (unpublished NHLBI tabulation):
  - Among non-Hispanic whites, mean LDL cholesterol levels were 115.1 mg/dL for men and 115.7 mg/dL for women.
  - Among non-Hispanic blacks, mean LDL cholesterol levels were 115.9 mg/dL for men and 114.2 mg/dL for women.
  - Among Mexican Americans, mean LDL cholesterol levels were 119.7 mg/dL for men and 115.0 mg/dL for women.
- The age-adjusted prevalence of high LDL cholesterol in US adults was 26.6% in 1988 to 1994 and 25.3% in 1999 to 2004 (NHANES/NCHS). Between 1988 to 1994 and 1999 to 2004, awareness increased from 39.2% to 63.0%, and use of pharmacological lipid-lowering treatment increased from 11.7% to 40.8%. LDL cholesterol control increased from 4.0% to 25.1% among those with high LDL cholesterol. In 1999 to 2004, rates of LDL cholesterol control were lower among adults 20 to 49 years of age than among those ≥65 years of age (13.9% versus 30.3%, respectively), among non-Hispanic blacks and Mexican Americans than among non-Hispanic whites (17.2% and 16.5% versus 26.9%, respectively), and among men than among women (22.6% versus 26.9%, respectively).[13]

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- Mean levels of LDL cholesterol decreased from 126.1 mg/dL during 1999 to 2000 to 116.1 mg/dL during 2009 to 2010. The prevalence of high LDL cholesterol decreased from 31.5% during 1999 to 2000 to 28.2% during 2009 to 2010[8] (unpublished NHLBI tabulation).

HDL (Good) Cholesterol

Youth

- Among children 4 to 11 years of age, the mean HDL cholesterol level is 53.6 mg/dL. For boys, it is 55.1 mg/dL, and for girls, it is 51.9 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - Among non-Hispanic whites, 53.9 mg/dL for boys and 51.4 mg/dL for girls
  - Among non-Hispanic blacks, 59.9 mg/dL for boys and 55.3 mg/dL for girls
  - Among Mexican Americans, 53.5 mg/dL for boys and 50.5 mg/dL for girls
- Among adolescents 12 to 19 years of age, the mean HDL cholesterol level is 51.4 mg/dL. For boys, it is 49.2 mg/dL, and for girls, it is 53.6 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - Among non-Hispanic whites, 48.4 mg/dL for boys and 53.0 mg/dL for girls
  - Among non-Hispanic blacks, 53.9 mg/dL for boys and 55.4 mg/dL for girls
  - Among Mexican Americans, 47.5 mg/dL for boys and 53.3 mg/dL for girls
- Low levels of HDL cholesterol occurred in 21.7% of male adolescents and 10.7% of female adolescents during 2007 to 2010[1] (NHANES 2007–2010, unpublished NHLBI tabulation).

Adults

- An HDL cholesterol level <40 mg/dL in adult males and <50 mg/dL in adult females is considered low and is a risk factor for HD and stroke. The mean level of HDL cholesterol for American adults ≥20 years of age is 52.5 mg/dL (NHANES 2007–2010, unpublished NHLBI tabulation).
- According to NHANES 2007–2010 (unpublished NHLBI tabulation):
  - Among non-Hispanic whites, mean HDL cholesterol levels were 46.7 mg/dL for men and 58.1 mg/dL for women
  - Among non-Hispanic blacks, mean HDL cholesterol levels were 52.6 mg/dL for men and 58.7 mg/dL for women
  - Among Mexican Americans, mean HDL cholesterol levels were 45.4 mg/dL for men and 53.7 mg/dL for women

Triglycerides

Youth

- There are limited data available on triglycerides for children 4 to 11 years of age.
- Among adolescents 12 to 19 years of age, the mean triglyceride level is 82.9 mg/dL. For boys, it is 85.6 mg/dL, and for girls, it is 80.1 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2007–2010, unpublished NHLBI tabulation):
  - Among non-Hispanic whites, 89.6 mg/dL for boys and 83.5 mg/dL for girls
  - Among non-Hispanic blacks, 66.7 mg/dL for boys and 58.6 mg/dL for girls
  - Among Mexican Americans, 97.1 mg/dL for boys and 83.5 mg/dL for girls
- High levels of triglycerides occurred in 9.4% of male adolescents and 6.7% of female adolescents during 2007 to 2010.[1]

Adults

- A fasting triglyceride level ≥150 mg/dL in adults is considered elevated and is a risk factor for HD and stroke. The mean level of triglycerides for American adults ≥20 years of age is 130.3 mg/dL (NHANES 2007–2010, unpublished NHLBI tabulation).
  - Among men, the mean triglyceride level is 141.7 mg/dL (NHANES 2007–2010, unpublished NHLBI tabulation). The racial/ethnic breakdown is as follows:
    - 140.0 mg/dL for non-Hispanic white men
    - 111.3 mg/dL for non-Hispanic black men
    - 161.4 mg/dL for Mexican American men
  - Among women, the mean triglyceride level is 119.1 mg/dL, with the following racial/ethnic breakdown:
    - 121.5 mg/dL for non-Hispanic white women
    - 94.4 mg/dL for non-Hispanic black women
    - 134.1 mg/dL for Mexican American women
- Approximately 27% of adults ≥20 years of age had a triglyceride level ≥150 mg/dL during 2007 to 2010[14] (NHANES 2007–2010, unpublished NHLBI tabulation).
- Fewer than 3% of adults with a triglyceride level ≥150 mg/dL received pharmacological treatment during 1999 to 2004.[14]

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Table 8-1. High Total and LDL Cholesterol and Low HDL Cholesterol

Population Group	Prevalence of Total Cholesterol ≥200 mg/dL, 2010 Age ≥20 y	Prevalence of Total Cholesterol ≥240 mg/dL, 2010 Age ≥20 y	Prevalence of LDL Cholesterol ≥130 mg/dL, 2010 Age ≥20 y	Prevalence of HDL Cholesterol <40 mg/dL, 2010 Age ≥20 y
Both sexes*	98 900 000 (43.4%)	31 900 000 (13.8%)	71 000 000 (31.1%)	48 700 000 (21.8%)
Males*	45 300 000 (41.3%)	14 000 000 (12.7%)	35 200 000 (31.9%)	34 600 000 (31.8%)
Females*	53 600 000 (44.9%)	17 900 000 (14.7%)	35 800 000 (30.0%)	14 100 000 (12.3%)
NH white males, %	40.5	12.3	30.1	33.1
NH white females, %	45.8	15.6	29.3	12.4
NH black males, %	38.6	10.8	33.1	20.3
NH black females, %	40.7	11.7	31.2	10.2
Mexican-American males, %	48.1	15.2	39.9	34.2
Mexican-American females, %	44.7	13.5	30.4	15.1

Prevalence of total cholesterol ≥200 mg/dL includes people with total cholesterol ≥240 mg/dL. In adults, levels of 200 to 239 mg/dL are considered borderline high. Levels of ≥240 mg/dL are considered high.

LDL indicates low-density lipoprotein; HDL, high-density lipoprotein; and NH, non-Hispanic.

*Total data for total cholesterol are for Americans ≥20 y of age. Data for LDL cholesterol, HDL cholesterol, and all racial/ethnic groups are age adjusted for age ≥20 y.

Source for total cholesterol ≥200 mg/dL, ≥240 mg/dL, LDL, and HDL: National Health and Nutrition Examination Survey (2007–2010), National Center for Health Statistics, and National Heart, Lung, and Blood Institute. Estimates from National Health and Nutrition Examination Survey 2007–2010 (National Center for Health Statistics) applied to 2010 population estimates.

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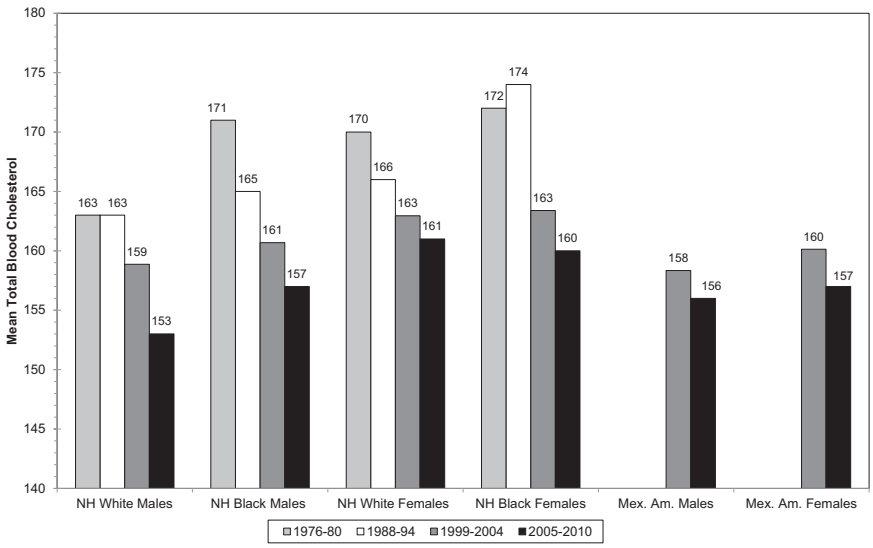

Chart 8-1. Trends in mean total serum cholesterol among adolescents 12 to 17 years of age by race, sex, and survey year (National Health and Nutrition Examination Survey: 1988–1994,* 1999–2004, and 2005-2010). Values are in mg/dL. NH indicates non-Hispanic; Mex. Am., Mexican American. *Data for Mexican Americans not available. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

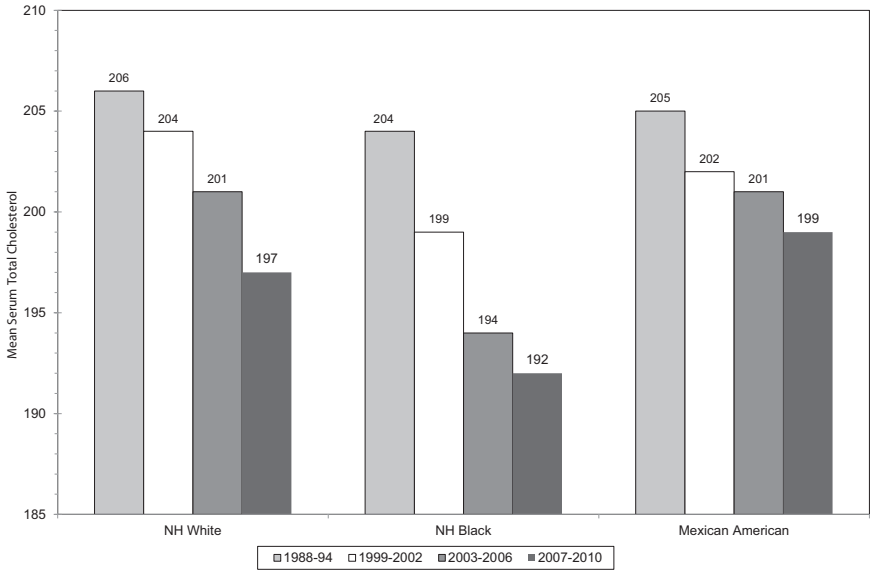

Chart 8-2. Trends in mean total serum cholesterol among adults ages ≥20 years old by race and survey year (National Health and Nutrition Examination Survey: 1988–1994, 1999–2002, 2003-2006, and 2007–2010). Values are in mg/dL. NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

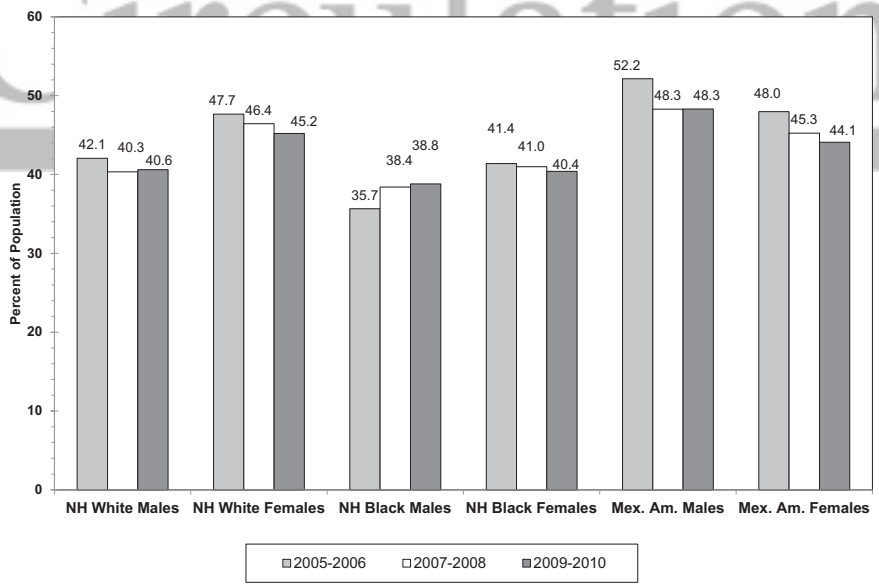

Chart 8-3. Age-adjusted trends in the prevalence of total serum cholesterol ≥200 mg/dL in adults ≥20 years of age by sex, race/ethnicity, and survey year (National Health and Nutrition Examination Survey 2005–2006, 2007–2008, and 2009–2010). NH indicates non-Hispanic; Mex. Am., Mexican American.

9. High Blood Pressure

ICD-9 401 to 404, ICD-10 I10 to I15. See Tables 9-1 and 9-2 and Charts 9-1 through 9-5.

Prevalence

(See Table 9-1 and Chart 9-1.)

- HBP is defined as:
  - — SBP ≥140 mm Hg or DBP ≥90 mm Hg or taking antihypertensive medicine, or
  - — Having been told at least twice by a physician or other health professional that one has HBP.

Abbreviations Used in Chapter 9

AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities Study
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHF	congestive heart failure
CHS	Cardiovascular Health Study
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
ED	emergency department
FHS	Framingham Heart Study
HBP	high blood pressure
HD	heart disease
ICD-9	International Classification of Diseases, 9th Revision
ICD-9-CM	International Classification of Diseases, Clinical Modification, 9th Revision
ICD-10	International Classification of Diseases, 10th Revision
LDL	low-density lipoprotein
MEPS	Medical Expenditure Panel Survey
MESA	Multi-Ethnic Study of Atherosclerosis
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHES	National Health Examination Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institute of Neurological Disorders and Stroke
NNHS	National Nursing Home Survey
PA	physical activity
REGARDS	REasons for Geographic And Racial Differences in Stroke study
SBP	systolic blood pressure
SEARCH	Search for Diabetes in Youth Study
WHI	Women's Health Initiative

- One in 3 US adults has HBP (unpublished NHLBI tabulation).
- Data from NHANES 2007–2010 found that ≈6% of US adults have undiagnosed hypertension. Data from the 2007–2008 BRFSS, NHIS, and NHANES surveys found 27.8%, 28.5%, and 30.7% US adults were told they had hypertension, respectively.[1]
- An estimated 77.9 million adults ≥20 years of age have HBP, extrapolated to 2010 with NHANES 2007–2010 data (Table 9-1).
- NHANES data show that a higher percentage of men than women have hypertension until 45 years of age. From 45 to 54 and from 55 to 64 years of age, the percentages of men and women with hypertension are similar. After that, a higher percentage of women have hypertension than men (Chart 9-1).
- HBP is 2 to 3 times more common in women taking oral contraceptives, especially among obese and older women, than in women not taking them.[2]
- Data from NHANES 2005–2006 found that 29% of US adults ≥18 years of age were hypertensive. The prevalence of hypertension was nearly equal between men and women; 7% of adults had HBP but had never been told that they had hypertension. Among hypertensive adults, 78% were aware of their condition, 68% were using antihypertensive medication, and 64% of those treated had their hypertensioncontrolled.[3]
- Data from the 2011 BRFSS/CDC indicate that the percentage of adults ≥18 years of age who had been told that they had HBP ranged from 22.9% in Utah to 40.1% in Alabama. The median percentage was 30.8%.[4]
  - — According to NHANES data 2003–2008, among US adults with hypertension, 8.9% met the criteria for resistant hypertension (BP was ≥140/90 mm Hg, and they reported using antihypertensive medications from 3 different drug classes or drugs from ≥4 antihypertensive drug classes regardless of BP). This represents 12.8% of the population taking antihypertensive medication.[5]
- According to data from NHANES 1988–1994 and 2007–2008, HBP control rates improved from 27.3% to 50.1%, treatment improved from 54.0% to 73.5%, and the control/treated rates improved from 50.6% to 72.3%.[6]
- Projections show that by 2030, prevalence of hypertension will increase 7.2% from 2013 estimates (unpublished AHA computation, based on methodology described by Heidenreich et al).[7]

Older Adults

- In 2009 to 2010, hypertension was among the diagnosed chronic conditions that were more prevalent among older (≥65 years of age) women than older men (57% for women, 54% for men). Ever-diagnosed conditions that were more prevalent among older men than older women included HD (37% for men, 26% for women) and DM (24% for men, 18% for women), on the basis of data from NHIS/NCHS.[8]
- The age-adjusted prevalence of hypertension (both diagnosed and undiagnosed) in 2003 to 2006 was 75% for older women and 65% for older men on the basis of data from NHANES/NCHS.[9]

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- Data from the 2004 NNHS revealed the most frequent chronic medical condition among this nationally representative sample of long-term stay residents aged ≥65 years was hypertension (53% of men and 56% of women). In men, prevalence of hypertension decreased with increasing age.[10]
- Among US adults ≥65 years of age (NHANES 1999–2004), prevalence of hypertension was 70.8%, awareness of hypertension was 75.9%, treatment for hypertension was 69.3%, and control of hypertension was 48.8%. Women had a slightly higher prevalence than men and a significantly lower rate of hypertension control.[11]

Children and Adolescents

- Analysis of the National Health Examination Survey (NHES), the Hispanic Health and Nutrition Examination Survey, and the NHANES/NCHS surveys of the NCHS (1963–2002) found that the BP, pre-HBP, and HBP trends in children and adolescents 8 to 17 years of age moved downward from 1963 to 1988 and upward thereafter. Pre-HBP and HBP increased 2.3% and 1%, respectively, between 1988 and 1999. Increased obesity (abdominal obesity more so than general obesity) partially explained the HBP and pre-HBP rise from 1988 to 1999. BP and HBP reversed their downward trends 10 years after the increase in the prevalence of obesity. In addition, an ethnic and sex gap appeared in 1988 for pre-HBP and in 1999 for HBP: Non-Hispanic blacks and Mexican Americans had a greater prevalence of HBP and pre-HBP than non-Hispanic whites, and the prevalence was greater in boys than in girls. In that study, HBP in children and adolescents was defined as SBP or DBP that was, on repeated measurement, ≥95th percentile.[12]
- A study in Ohio of >14 000 children and adolescents 3 to 18 years of age who were observed at least 3 times between 1999 and 2006 found that 3.6% had hypertension.[13] Of these, 26% had been diagnosed and 74% were undiagnosed. In addition, 3% of those with hypertension had stage 2 hypertension, and 41% of those with stage 2 hypertension were undiagnosed. Criteria for prehypertension were met by 485 children. Of these, 11% were diagnosed. In this study, HBP in children and adolescents was defined as SBP or DBP that was, on repeated measurement, ≥95th percentile.[13]
- A study from 1988–1994 through 1999–2000 of children and adolescents 8 to 17 years of age showed that among non-Hispanic blacks, mean SBP levels increased by 1.6 mm Hg among girls and by 2.9 mm Hg among boys compared with non-Hispanic whites. Among Mexican Americans, girls' SBP increased 1.0 mm Hg and boys' SBP increased 2.7 mm Hg compared with non-Hispanic whites.[14]
- Analysis of data from the Search for Diabetes in Youth Study (SEARCH), which included children 3 to 17 years of age with type 1 and type 2 DM, found the prevalence of elevated BP to be 5.9% among those with type 1 DM and 23.7% among those with type 2 DM.[15]
- A study of high school students in Houston, TX (mean age 15.4 years; 45.2% male, 49.3% Hispanic, 25.2% Caucasian, and 16.1% African American) found ≈30% of the students had at least 1 elevated BP measurement; elevated BP was significantly influenced by obesity.[16]
- Longitudinal BP outcomes from the National Childhood Blood Pressure database (ages 13–15 years) were examined after a single BP measurement. Among those determined to have prehypertension, 14% of boys and 12% of girls had hypertension 2 years later; the overall rate of progression from prehypertension to hypertension was ≈7%.[17]

Race/Ethnicity and HBP

(See Table 9-1 and Chart 9-2.)

- The prevalence of hypertension in blacks in the United States is among the highest in the world, and it is increasing. From 1988 to 1994 through 1999 to 2002, the prevalence of HBP in adults increased from 35.8% to 41.4% among blacks, and it was particularly high among black women at 44.0%. Prevalence among whites also increased, from 24.3% to 28.1%.[18]
- Compared with whites, blacks develop HBP earlier in life, and their average BPs are much higher. As a result, compared with whites, blacks have a 1.3-times greater rate of nonfatal stroke, a 1.8-times greater rate of fatal stroke, a 1.5-times greater rate of death attributable to HD, and a 4.2-times greater rate of end-stage kidney disease (fifth and sixth reports of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure).
- Within the black community, rates of hypertension vary substantially.[18,19]
  - Those with the highest rates are more likely to be middle-aged or older, less educated, overweight or obese, and physically inactive and are more likely to have DM.
  - Those with the lowest rates are more likely to be younger but also overweight or obese.
  - Those with uncontrolled HBP who are not taking antihypertensive medication tend to be male, to be younger, and to have infrequent contact with a physician.
- Analysis from the REasons for Geographic And Racial Differences in Stroke study (REGARDS) study of the NINDS suggests that efforts to raise awareness of prevalent hypertension among blacks apparently have been successful (31% greater odds in blacks relative to whites), and efforts to communicate the importance of receiving treatment for hypertension have been successful (69% greater odds among blacks relative to whites); however, substantial racial disparities remain with regard to the control of BP (SBP <140 mm Hg, DBP <90 mm Hg), with the odds of control being 27% lower in blacks than in whites. In contrast, geographic disparities in hypertension awareness, treatment, and control were minimal.[20]
- Data from the 2011 NHIS showed that black adults ≥18 years of age were more likely (33.4%) to have been told on ≥2 occasions that they had hypertension than white adults (23.3%), American Indian/Alaska Native adults (25.8%), or Asian adults (18.7%).[21]
- The CDC analyzed death certificate data from 1995 to 2002 (any-mention mortality; ICD-9 codes 401–404 and ICD-10 codes I10–I13). The results indicated that Puerto Rican Americans had a consistently higher hypertension-related death rate than all other Hispanic subpopulations and non-Hispanic whites. The age-standardized hypertension-related

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mortality rate was 127.2 per 100 000 population for all Hispanics, similar to that of non-Hispanic whites (135.9). The age-standardized rate for Hispanic females (118.3) was substantially lower than that observed for Hispanic males (135.9). Hypertension-related mortality rates for males were higher than rates for females for all Hispanic subpopulations. Puerto Rican Americans had the highest hypertension-related death rate among all Hispanic subpopulations (154.0); Cuban Americans had the lowest (82.5).[22]

- Some studies suggest that Hispanic Americans have rates of HBP similar to or lower than those of non-Hispanic white Americans. Findings from a new analysis of combined data from the NHIS of 2000–2002 point to a health disparity between black and white adults of Hispanic descent. Black Hispanics were at slightly greater risk than white Hispanics, although non-Hispanic black adults had by far the highest rate of HBP. The racial disparity among Hispanics also was evident in the fact that higher-income, better-educated black Hispanics still had a higher rate of HBP than lower-income, less-educated white Hispanics.[23] Data from the NHLBI's ARIC study found that hypertension was a particularly powerful risk factor for CHD in black people, especially black women.[24]
- Data from MESA found that being born outside the United States, speaking a language other than English at home, and living fewer years in the United States were each associated with a decreased prevalence of hypertension.[25]
- Filipino (27%) and Japanese (25%) adults were more likely than Chinese (17%) or Korean (17%) adults to have ever been told that they had hypertension.[26]

Mortality

(See Table 9-1.)

HBP mortality in 2009 was 61 762. Any-mention mortality in 2009 was 348 102. The 2009 death rate was 18.5.[27]

- From 1999 to 2009, the death rate caused by HBP increased 17.1%, and the actual number of deaths rose 43.6% (AHA tabulation).[27]
- The 2009 overall death rate resulting from HBP was 18.5. Death rates were 17.0 for white males, 51.6 for black males, 14.4 for white females, and 38.3 for black females. When any-mention mortality for 2009 was used, the overall death rate was 104.9. Death rates were 108.5 for white males, 219.7 for black males, 86.1 for white females, and 163.7 for black females.
- Analysis of NHANES I and II comparing hypertensive and nonhypertensive individuals found a reduction in age-adjusted mortality rate of 4.6/1000 person-years among people with hypertension compared with a reduction of 4.2/1000 person-years among those without hypertension.[28]
- Assessment of 30-year follow-up of the Hypertension Detection and Follow-up Program identified the long-term benefit of stepped care, as well as the increased survival for hypertensive African Americans.[29]
- Assessment of the Charleston Heart Study and Evans County Heart Study identified the excess burden of elevated BP for African Americans and its effect on long-term health outcomes.[30]
- Data from the Harvard Alumni Health Study found that higher BP in early adulthood was associated several decades later with higher risk for all-cause mortality, CVD mortality, and CHD mortality but not stroke mortality.[31]

Risk Factors

- Numerous risk factors and markers for development of hypertension, including age, ethnicity, family history of hypertension and genetic factors, lower education and socioeconomic status, greater weight, lower PA, tobacco use, psychosocial stressors, sleep apnea, and dietary factors (including dietary fats, higher sodium intake, lower potassium intake, and excessive alcohol intake), have been identified.
- A study of related individuals in the NHLBI's FHS suggested that different sets of genes regulate BP at different ages.[32]
- Recent data from the Nurses' Health Study suggest that a large proportion of incident hypertension in women can be prevented by controlling dietary and lifestyle risk factors.[33]
- A meta-analysis identified the benefit of a goal BP of 130/80 mm Hg for individuals with hypertension and type 2 DM but less evidence for treatment below this value.[34]

Aftermath

- Approximately 69% of people who have a first heart attack, 77% of those who have a first stroke, and 74% of those who have CHF have BP >140/90 mm Hg (NHLBI unpublished estimates from ARIC, CHS, and FHS Cohort and Offspring studies).
- Data from FHS/NHLBI indicate that recent (within the past 10 years) and remote antecedent BP levels may be an important determinant of risk over and above the current BP level.[35]
- Data from the FHS/NHLBI indicate that hypertension is associated with shorter overall life expectancy, shorter life expectancy free of CVD, and more years lived with CVD.[36]
  - —Total life expectancy was 5.1 years longer for normotensive men and 4.9 years longer for normotensive women than for hypertensive people of the same sex at 50 years of age.
  - —Compared with hypertensive men at 50 years of age, men with untreated BP <140/90 mm Hg survived on average 7.2 years longer without CVD and spent 2.1 fewer years of life with CVD. Similar results were observed for women.

Hospital Discharges/Ambulatory Care Visits

(See Table 9-1.)

- From 2000 to 2010, the number of inpatient discharges from short-stay hospitals with HBP as the first-listed diagnosis increased from 457 000 to 488 000 (no significant difference; NCHS, NHDS). The number of all-listed discharges increased from 8 034 000 to 11 282 000 (NHLBI, unpublished data from the NHDS, 2010; diagnoses in 2010 were truncated at 7 diagnoses for comparability with earlier year).
- Data from the Nationwide Inpatient Sample from the years 2000 to 2007 found the frequency of hospitalizations for

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adults aged ≥18 years of age with a hypertensive emergency increased from 101 to 111 per 100 000 in 2007 (average increase of 1.11%). In contrast to the increased number of hospitalizations, the all-cause in-hospital mortality rate decreased during the same period from 2.8% to 2.6%.[37]

- Data from ambulatory medical care utilization estimates for 2009 showed that the number of visits for essential hypertension was 55 148 000. Of these, 49 966 000 were physician office visits, 1 000 000 were ED visits, and 4 182 000 were outpatient department visits (NAMCS and NHAMCS, NHLBI tabulation).
- In 2010, there were 280 000 hospitalizations with a first-listed diagnosis of essential hypertension (ICD-9-CM code 401), but essential hypertension was listed as either a primary or a secondary diagnosis 11 048 000 times for hospitalized inpatients (unpublished data from the NHDS, NHLBI tabulation).

Awareness, Treatment, and Control

(See Table 9-2 and Charts 9-3 through 9-5.)

- Data from NHANES 2007–2010 showed that of those with hypertension who were ≥20 years of age, 81.5% were aware of their condition, 74.9% were under current treatment, 52.5% had their hypertension under control, and 47.5% did not have it controlled (NHLBI tabulation).
- Data from NHANES 1999–2006 showed that 11.2% of adults ≥20 years of age had treated and controlled BP levels.[38]
- Analysis of NHANES data from 1999–2004 through 2005–2006 found that there were substantial increases in awareness and treatment rates of hypertension. The control rates increased in both sexes, in non-Hispanic blacks, and in Mexican Americans. Among the group ≥60 years of age, awareness, treatment, and control rates of hypertension increased significantly.[3,39]
- In NHANES 2007–2010, rates of control were lower in Mexican Americans (39.3%) than in non-Hispanic whites (54.9%) and non-Hispanic blacks (47.6%; unpublished NHLBI tabulation).
- The awareness, treatment, and control of HBP among those ≥65 years of age in the CHS/NHLBI improved during the 1990s. The percentages of those aware of and treated for HBP were higher among blacks than among whites. Prevalence rates with HBP under control were similar. For both groups combined, the control of BP to <140/90 mm Hg increased from 37% in 1990 to 49% in 1999. Improved control was achieved by an increase in antihypertensive medications per person and by an increase in the proportion of the CHS population treated for hypertension from 34.5% to 51.1%.[40]
- Data from the FHS of the NHLBI show that among those ≥80 years of age, only 38% of men and 23% of women had BPs that met targets set forth in the National High Blood Pressure Education Program's clinical guidelines. Control rates in men <60, 60 to 79, and ≥80 years of age were 38%, 36%, and 38%, respectively; for women in the same age groups, they were 38%, 28%, and 23%, respectively.[41]
- Data from the WHI observational study of nearly 100 000 postmenopausal women across the country enrolled between 1994 and 1998 indicate that although prevalence rates ranged from 27% of women 50 to 59 years of age to 41% of women 60 to 69 years of age to 53% of women 70 to 79 years of age, treatment rates were similar across age groups: 64%, 65%, and 63%, respectively. Despite similar treatment rates, hypertension control is especially poor in older women, with only 29% of hypertensive women 70 to 79 years of age having clinic BPs <140/90 mm Hg compared with 41% and 37% of those 50 to 59 and 60 to 69 years of age, respectively.[42]
- Among a cohort of postmenopausal women taking hormone replacement, hypertension was the most common comorbidity, with a prevalence of 34%.[43]
- A study of >300 women in Wisconsin showed a need for significant improvement in BP and LDL levels. Of the screened participants, 35% were not at BP goal, 32.4% were not at LDL goal, and 53.5% were not at both goals.[44]
- In 2005, a survey of people in 20 states conducted by the BRFSS of the CDC found that 19.4% of respondents had been told on ≥2 visits to a health professional that they had HBP. Of these, 70.9% reported changing their eating habits; 79.5% reduced the use of or were not using salt; 79.2% reduced the use of or eliminated alcohol; 68.8% were exercising; and 73.4% were taking antihypertensive medication.[45]
- On the basis of NHANES 2003–2004 data, it was found that nearly three fourths of adults with CVD comorbidities have hypertension. Poor control rates of systolic hypertension remain a principal problem that further compromises their already high CVD risk.[46]
- According to data from NHANES 2001–2006, non-Hispanic blacks had 90% higher odds of poorly controlled BP than non-Hispanic whites. Among those who were hypertensive, non-Hispanic blacks and Mexican Americans had 40% higher odds of uncontrolled BP than non-Hispanic whites.[47]
- According to data from NHANES 1998–2008 for adults with DM, prevalence of hypertension increased, whereas awareness, treatment, and control improved during these time periods; however, for adults 20 to 44 years of age, there was no evidence of improvement.[48]
- "Resistant hypertension" is a treatment and control issue for nearly 1 in 10 hypertensive adults. This category of HBP represents individuals with uncontrolled HBP despite the use of ≥3 antihypertensive medications or with BP controlled with the use of ≥4 medications.[49,50]

Cost

(See Table 9-1.)

- The estimated direct and indirect cost of HBP for 2009 is $51.0 billion (MEPS, NHLBI tabulation).
- Projections show that by 2030, total cost of HBP will increase to an estimated $343 billion (unpublished AHA computation, based on methodology described in Heidenreich et al[7]).

Prehypertension

- Prehypertension is untreated SBP of 120 to 139 mm Hg or untreated DBP of 80 to 89 mm Hg and not having been told on 2 occasions by a physician or other health professional that one has hypertension.

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- Data from NHANES 1999–2006 estimate that 29.7% of adults ≥20 years of age have prehypertension.[38]
- Follow-up of 9845 men and women in the FHS/NHLBI who attended examinations from 1978 to 1994 revealed that at 35 to 64 years of age, the 4-year incidence of hypertension was 5.3% for those with baseline BP <120/80 mm Hg, 17.6% for those with SBP of 120 to 129 mm Hg or DBP of 80 to 84 mm Hg, and 37.3% for those with SBP of 130 to 139 mm Hg or DBP of 85 to 89 mm Hg. At 65 to 94 years of age, the 4-year incidences of hypertension were 16.0%, 25.5%, and 49.5% for these BP categories, respectively.[51]
- Data from FHS/NHLBI also reveal that prehypertension is associated with elevated relative and absolute risks for CVD outcomes across the age spectrum. Compared with normal BP (<120/80 mm Hg), prehypertension was associated with a 1.5- to 2-fold increased risk for major CVD events in those <60, 60 to 79, and ≥80 years of age. Absolute risks for major CVD associated with prehypertension increased markedly with age: 6-year event rates for major CVD were 1.5% in prehypertensive people <60 years of age, 4.9% in those 60 to 79 years of age, and 19.8% in those ≥80 years of age.[41]
- In a study of NHANES 1999–2000 (NCHS), people with prehypertension were more likely than those with normal BP levels to have above-normal cholesterol levels (≥200 mg/dL) and to be overweight or obese, whereas the probability of current smoking was lower. People with prehypertension were 1.65 times more likely to have 1 or more of these adverse risk factors than were those with normal BP.[52]
- Assessment of the REGARDS data identified high risk of prehypertension to be associated with increased age and black race.[53]
- A meta-analysis of 12 prospective cohort studies (including 518 520 participants) found prehypertension was associated with incident stroke. The risk was particularly noted in nonelderly persons and for those with BP values in the higher prehypertension range.[54]
- Prehypertension was found to be significantly associated with stroke.[54]
- Prehypertension was highest in blacks with other risk factors, including DM and elevated C-reactive protein.[53]

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Table 9-1. High Blood Pressure

Population Group	Prevalence, 2010, Age ≥20 y	Mortality,* 2009, All Ages	Hospital Discharges, 2010, All Ages	Estimated Cost, 2009
Both sexes	77 895 000 (33.0%)	61 762	488 000	$51.0 Billion
Males	37 195 000 (33.6%)	27 668 (44.8%)†	216 000	…
Females	40 700 000 (32.2%)	34 094 (55.2%)†	272 000	…
NH white males	33.4%	20 286	…	…
NH white females	30.7%	26 201	…	…
NH black males	42.6%	6574	…	…
NH black females	47.0%	6951	…	…
Mexican American males	30.1%	…	…	…
Mexican American females	28.8%	…	…	…
Hispanic or Latino	22.2%‡	3733	…	…
Asian	18.7%‡	1471	…	…
American Indian/Alaska Native	25.8%‡	279	…	…

Hypertension is defined in terms of National Health and Nutrition Examination Survey blood pressure measurements and health interviews. A subject was considered hypertensive if systolic blood pressure was ≥140 mm Hg or diastolic blood pressure was ≥90 mm Hg, if the subject said "yes" to taking antihypertensive medication, or if the subject was told on 2 occasions that he or she had hypertension.

Ellipses (. . .) indicate data not available; NH, non-Hispanic.

*Mortality data for the white, black, Asian or Pacific Islander, and American Indian/Alaska Native populations include deaths among persons of Hispanic and non-Hispanic origin. Numbers of deaths for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated.

†These percentages represent the portion of total high blood pressure mortality that is for males vs females.

‡National Health Interview Survey (2010), National Center for Health Statistics; data are weighted percentages for Americans ≥18 years of age. Source: Schiller et al.[21]

Sources: Prevalence: National Health and Nutrition Examination Survey (2007–2010), National Center for Health Statistics) and National Heart, Lung, and Blood Institute. Percentages for racial/ethnic groups are age adjusted for Americans ≥20 y of age. Age-specific percentages are extrapolated to the 2010 US population estimates. Mortality: CDC/National Center for Health Statistics, 2009 Mortality Multiple Cause–US, version July 19, 2012. These data represent underlying cause of death only. Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics; data include those discharged alive, dead, or status unknown. Cost: Medical Expenditure Panel Survey data include estimated direct costs for 2009; indirect costs calculated by National Heart, Lung, and Blood Institute for 2009.

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Table 9-2. Hypertension Awareness, Treatment, and Control: NHANES 1999–2004 and 2005–2010, by Race/Ethnicity and Sex

	Awareness, %		Treatment, %		Control, %	
	1999–2004	2005–2010	1999–2004	2005–2010	1999–2004	2005–2010
NH white males	71.2	77.5	61.2	69.4	41.0	50.1
NH white females	74.4	84.0	65.3	78.2	37.2	53.9
NH black male	69.1	77.5	58.1	66.9	32.3	39.7
NH black females	83.5	88.5	73.9	81.5	40.4	52.8
Mexican American males	57.0	64.8	41.8	54.0	23.3	35.1
Mexican American females	67.9	75.5	56.3	68.1	29.6	41.6

NHANES indicates National Health and Nutrition Examination Survey; NH, non-Hispanic.

Sources: NHANES (1999–2004, 2005–2010) and National Heart, Lung, and Blood Institute.

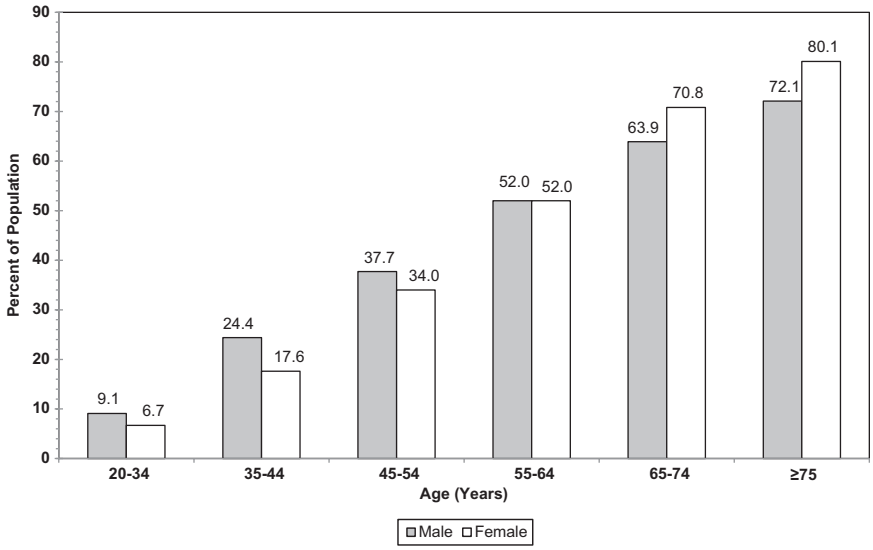

Chart 9-1. Prevalence of high blood pressure in adults ≥20 years of age by age and sex (National Health and Nutrition Examination Survey: 2007–2010). Hypertension is defined as systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg, if the subject said "yes" to taking antihypertensive medication, or if the subject was told on 2 occasions that he or she had hypertension. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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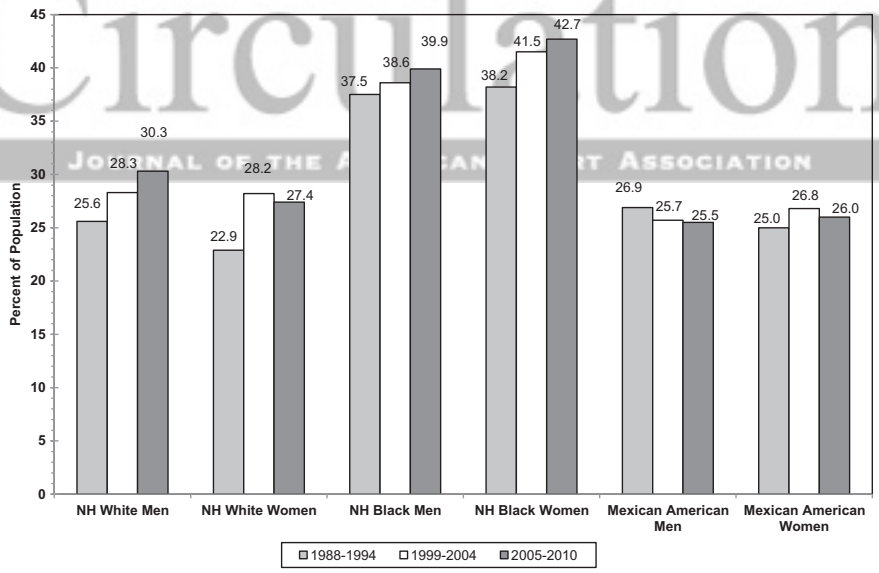

Chart 9-2. Age-adjusted prevalence trends for high blood pressure in adults ≥20 years of age by race/ethnicity, sex, and survey (National Health and Nutrition Examination Survey: 1988–1994, 1999–2004, and 2005–2010). NH indicates non-Hispanic. Source: National Center for Health Statics and National Heart, Lung and Blood Institute.

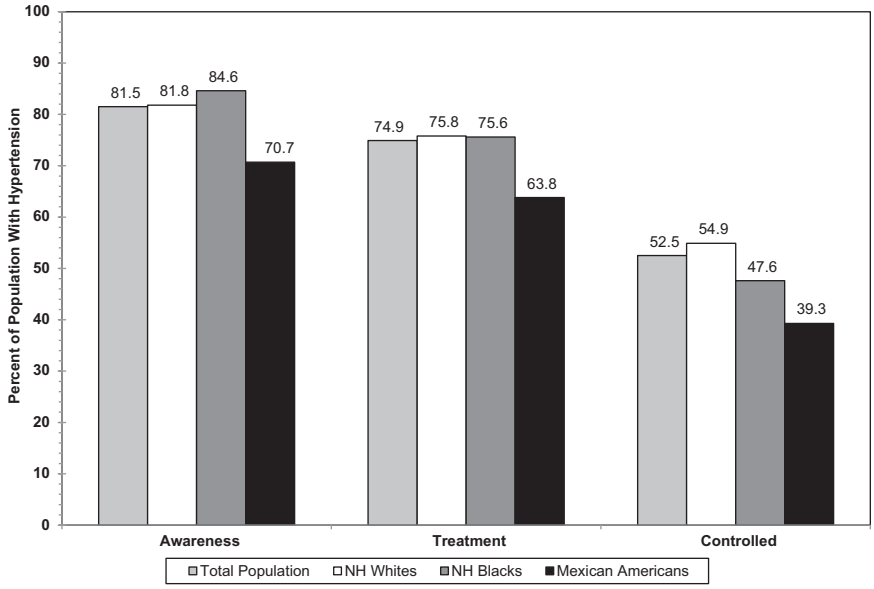

Chart 9-3. Extent of awareness, treatment, and control of high blood pressure by race/ethnicity (National Health and Nutrition Examination Survey: 2007–2010). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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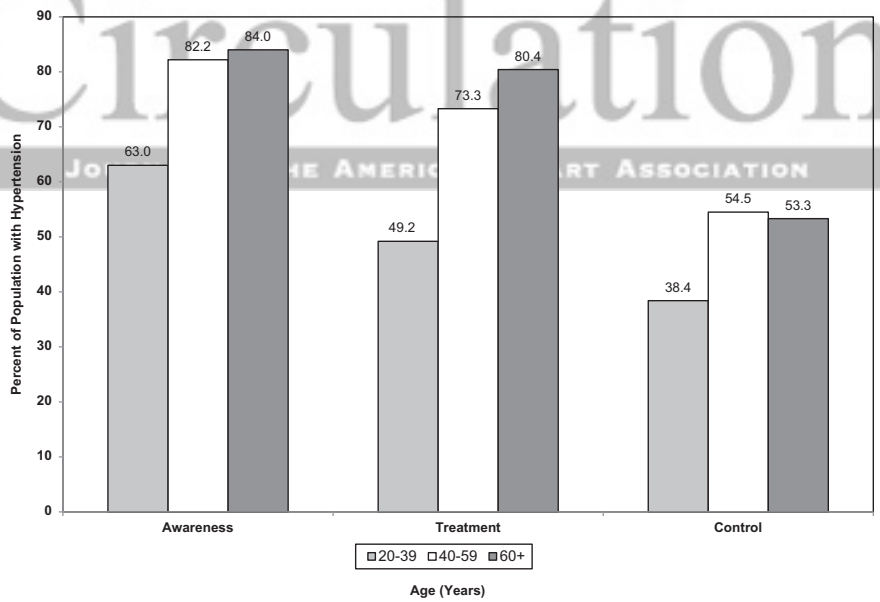

Chart 9-4. Extent of awareness, treatment, and control of high blood pressure by age (National Health and Nutrition Examination Survey: 2005–2008). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

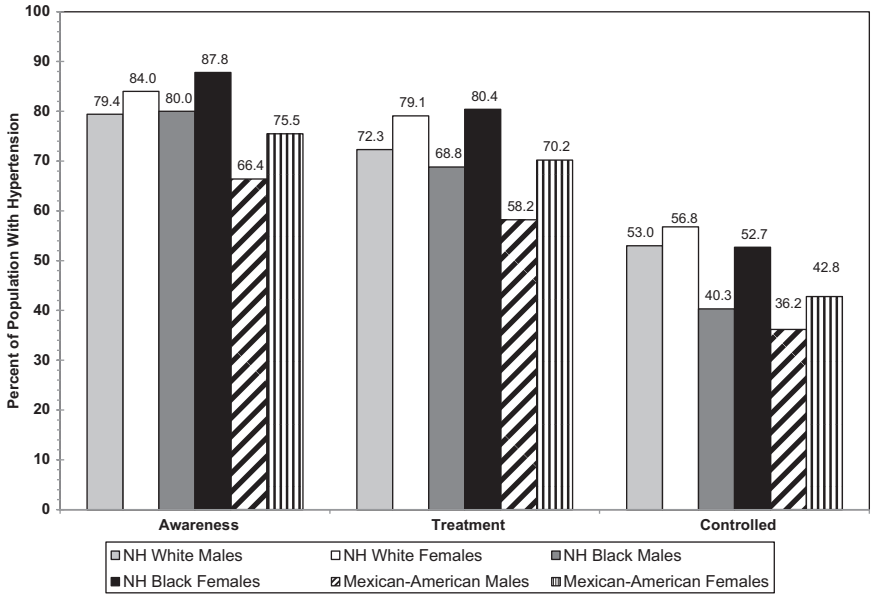

Chart 9-5. Extent of awareness, treatment, and control of high blood pressure by race/ethnicity and sex (National Health and Nutrition Examination Survey: 2007–2010). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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10. Diabetes Mellitus

ICD-9 250; ICD-10 E10 to E14. See Table 10-1 and Charts 10-1 through 10-4.

Prevalence

Youths

- In SEARCH, the prevalence of DM in youths <20 years of age in 2001 in the United States was 1.82 cases per 1000 youths (0.79 per 1000 among youths 0–9 years of age and 2.80 per 1000 among youths 10–19 years of age). Non-Hispanic white youths had the highest prevalence (1.06 per 1000) in the younger group. Among youths 10 to 19 years of age, black youths (3.22 per 1000) and non-Hispanic white youths (3.18 per 1000) had the highest rates, followed by American Indian youths (2.28 per 1000), Hispanic youths (2.18 per 1000), and Asian/Pacific Islander youths (1.34 per 1000). Among younger children, type 1 DM accounted for ≥80% of DM; among older youths, the proportion of type 2 DM ranged from 6% (0.19 per 1000 for non-Hispanic white youths) to 76% (1.74 per 1000 for American Indian youths). This translates to 154 369 youths with physician-diagnosed DM in 2001 in the United States, for an overall prevalence estimate for DM in children and adolescents of ≈0.18%.[1]
- Approximately 186 000 people <20 years of age have DM. Each year, ≈15 000 people <20 years of age are diagnosed with type 1 DM. Healthcare providers are finding more and more children with type 2 DM, a disease usually diagnosed in adults ≥40 years of age. Children who develop type 2 DM are typically overweight or obese and have a family history of the disease. Most are American Indian, black, Asian, or Hispanic/Latino.[2]
- During the period from 2002 to 2005, 3600 youth (age <20 years) were diagnosed with type 2 DM annually.[3]
- Among adolescents 10 to 19 years of age diagnosed with DM, 57.8% of blacks were diagnosed with type 2 versus type 1 DM compared with 46.1% of Hispanic and 14.9% of white youths.[4]
- According to the Bogalusa Heart Study, a long-term follow-up study of youths aging into adulthood, youths who were prediabetic or who had DM were more likely to have a constellation of metabolic disorders in young adulthood (19–44 years of age), including obesity, hypertension, dyslipidemia, and metabolic syndrome, all of which predispose to CHD.[5]
- Among youths with type 2 DM, 10.4% are overweight and 79.4% are obese.[6]
- According to NHANES data from 1999–2007, among US adolescents aged 12 to 19 years, the prevalence of prediabetes and DM increased from 9% to 23%.[7]
- The TODAY (Treatment Options for Type 2 Diabetes in Adolescents and Youth) cohort comprised youths aged 10 to 17 years (41.1% Hispanic and 31.5% non-Hispanic black) participating in a randomized controlled study of new-onset type 2 DM; 41.5% of participants had household income <$25 000.[8] The results of the clinical trial demonstrated that only half of the children maintained durable glycemic control with monotherapy,[9] a higher rate of treatment failure than observed in adult cohorts.

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Abbreviations Used in Chapter 10

ACCORD	Action to Control Cardiovascular Risk in Diabetes	IDDM	insulin-dependent diabetes mellitus
ACS	acute coronary syndrome	LDL	low-density lipoprotein
ADVANCE	Action in Diabetes and Vascular Disease: Preterax and Diamicron Modified Release Controlled Evaluation	MEPS	Medical Expenditure Panel Survey
AMI	acute myocardial infarction	MESA	Multi-Ethnic Study of Atherosclerosis
ARIC	Atherosclerosis Risk in Communities study	MI	myocardial infarction
BMI	body mass index	NAMCS	National Ambulatory Medical Care Survey
BP	blood pressure	NCHS	National Center for Health Statistics
BRFSS	Behavioral Risk Factor Surveillance System	NH	non-Hispanic
CDC	Centers for Disease Control and Prevention	NHAMCS	National Hospital Ambulatory Medical Care Survey
CHD	coronary heart disease	NHANES	National Health and Nutrition Examination Survey
CHS	Cardiovascular Health Study	NHDS	National Hospital Discharge Survey
CI	confidence interval	NHIS	National Health Interview Survey
CVD	cardiovascular disease	NHLBI	National Heart, Lung, and Blood Institute
DM	diabetes mellitus	NSTEMI	non–ST-segment–elevation myocardial infarction
ED	emergency department	OR	odds ratio
ESRD	end-stage renal disease	PA	physical activity
FHS	Framingham Heart Study	PAR	population-attributable risk
HbA_{1c}	hemoglobin A_{1c}	RR	relative risk
HD	heart disease	SBP	systolic blood pressure
HDL	high-density lipoprotein	SEARCH	Search for Diabetes in Youth Study
HR	hazard ratio	STEMI	ST-segment–elevation myocardial infarction
ICD-9	*International Classification of Diseases, 9th Revision*	TODAY	Treatment Options for Type 2 Diabetes in Adolescents and Youth
ICD-10	*International Classification of Diseases, 10th Revision*	UA	unstable angina

- Of 1514 SEARCH participants, 95% reported having undergone BP checks and 88% reported lipid-level checks, whereas slightly more than two thirds (68%) reported having had HbA_{1c} testing or eye examinations (66%).[10]

Adults

(See Table 10-1 and Charts 10-1 through 10-3.)

- On the basis of data from NHANES 2007–2010 (unpublished NHLBI tabulation), an estimated 19.7 million Americans ≥20 years of age have physician-diagnosed DM. An additional 8.2 million adults have undiagnosed DM, and ≈87.3 million adults have prediabetes (eg, fasting blood glucose of 100 to <126 mg/dL). The prevalence of prediabetes in the US adult population is 38%.
- The prevalence of diagnosed DM in adults ≥65 years of age was 26.9% in 2010, and an additional 50% (>20 million) had prediabetes based on fasting glucose, oral glucose tolerance testing, or HbA_{1c}. In addition, data from NHANES 2005–2006 show that 46% of DM cases remain undiagnosed in this group aged ≥65 years.[11]
- Men ≥20 years of age have a slightly higher prevalence of DM (11.8%) than women (10.8%).[6]
- After adjustment for population age differences, 2007 to 2009 national survey data for people ≥20 years of age indicate that 7.1% of non-Hispanic whites, 8.4% of Asian Americans, 11.8% of Hispanics, and 12.6% of non-Hispanic blacks had diagnosed DM.[3]
- Compared with non-Hispanic white adults, the risk of diagnosed DM was 18% higher among Asian Americans, 66% higher among Hispanics/Latinos, and 77% higher among non-Hispanic blacks.[3]
- In 2004 to 2006, the prevalence of diagnosed DM was more than twice as high for Asian Indian adults (14%) as for Chinese (6%) or Japanese (5%) adults.[12]
- Type 2 DM accounts for 90% to 95% of all diagnosed cases of DM in adults.[3]
- On the basis of 2010 BRFSS (CDC) data, the prevalence of adults in the United States who reported ever having been told by a physician that they had DM ranged from 5.3% in Alaska to 13.2% in Alabama. The median percentage among all states was 8.7%.[13]
- The CDC analyzed data from 1994 to 2004 collected by the Indian Health Service that indicated that the age-adjusted prevalence per 1000 population of DM increased 101.2% among American Indian/Alaska Native adults <35 years of age (from 8.5% to 17.1%). During this time period, the prevalence of diagnosed DM was greater among females than males in all age groups.[14]
- On the basis of projections from NHANES/NCHS studies between 1984 and 2004, the total prevalence of DM in the United States is expected to more than double from 2005 to 2050 (from 5.6% to 12.0%) in all age, sex, and race/ethnicity groups. Increases are projected to be largest for the oldest age groups (for instance, projected to increase by 220% among those 65–74 years of age and by 449% among those ≥75 years of age). DM prevalence is projected to increase by 99% among non-Hispanic whites, by 107% among non-Hispanic blacks, and by 127% among Hispanics. The age/race/ethnicity group with the largest increase is expected to be blacks ≥75 years of age (projected increase of 606%).[15]
- According to NHIS data from 1997 to 2008, the prevalence of DM was higher at both time points among Asian Americans (4.3%–8.2%, respectively) than among whites (3.8%–6.0%, respectively), with the Asian American group also having a greater proportional increase (1.9- versus 1.5-fold increase). This was observed despite lower BMI levels (23.6 versus 26.1 kg/m^2 in the earliest time period) among Asians.[16]
- The prevalence of DM for all age groups worldwide was estimated to be 2.8% in 2000 and is projected to be 4.4% in 2030. The total number of people with DM is projected to rise from 171 million in 2000 to 366 million in 2030.[17]
- According to international survey and epidemiologic data from 2.7 million participants, the prevalence of DM in adults increased from 8.3% in men and 7.5% in women in 1980 to 9.8% in men and 9.2% in women in 2008. The number of individuals affected with DM increased from 153 million in 1980 to 347 million in 2008.[18]

Incidence

Youths

- In the SEARCH study, the incidence of DM in youths overall was 24.3 per 100 000 person-years. Among children <10 years of age, most had type 1 DM, regardless of race/ethnicity. The highest rates of incident type 1 DM were observed in non-Hispanic white youths (18.6, 28.1, and 32.9 per 100 000 person-years for age groups of 0–4, 5–9, and 10–14 years, respectively). Overall, type 2 DM was relatively infrequent, with the highest rates (17.0–49.4 per 100 000 person-years) seen among 15- to 19-year-old minority groups.[4]
- Of 2291 individuals <20 years of age with newly diagnosed DM, slightly more than half (54.5%) had autoimmune, insulin-sensitive DM, and 15.9% had nonautoimmune, insulin-resistant DM.[19]

Adults

(See Table 10-1.)

- A total of 1.9 million new cases of DM (type 1 or type 2) were diagnosed in US adults ≥20 years of age in 2010.[3]
- Data from the FHS indicate a doubling in the incidence of DM over the past 30 years, most dramatically during the 1990s. Among adults 40 to 55 years of age in each decade of the 1970s, 1980s, and 1990s, the age-adjusted 8-year incidence rates of DM were 2.0%, 3.0%, and 3.7% among women and 2.7%, 3.6%, and 5.8% among men, respectively. Compared with the 1970s, the age-and sex-adjusted OR for DM was 1.40 in the 1980s and 2.05 in the 1990s (P for trend=0.0006). Most of the increase in absolute incidence of DM occurred in individuals with a BMI ≥30 kg/m^2 (P for trend=0.03).[20]
- DM incidence in adults also varies markedly by race. Over 5 years of follow-up in 45- to 84-year-olds in MESA, 8.2% of the cohort developed DM. The cumulative incidence was highest in Hispanics (11.3%), followed by black (9.5%), Chinese (7.7%), and white (6.3%) participants.[21]
- A recent meta-analysis of television viewing, including 4 studies consisting of 175 938 adult individuals and 6428 incident type 2 DM cases, showed that among those who

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watched 2 hours of television per day, the RR of DM was 20% higher (HR, 1.20; 95% CI, 1.14–1.27).[22]

- Gestational diabetes complicates 2% to 10% of pregnancies and increases the risk of developing type 2 DM by 35% to 60%.[3]

Mortality

(See Table 10-1.)

DM mortality in 2009 was 68 705. Any-mention mortality in 2009 was 230 642.[23]

- The 2009 overall underlying-cause death rate attributable to DM was 20.9. Death rates per 100 000 people were 23.3 for white males, 44.2 for black males, 15.7 for white females, and 35.9 for black females.[23]
- According to data from the National Diabetes Information Clearinghouse, the National Institute of Diabetes and Digestive and Kidney Diseases, and the National Institutes of Health

  —At least 68% of people >65 years of age with DM die of some form of HD; 16% die of stroke.
  —HD death rates among adults with DM are 2 to 4 times higher than the rates for adults without DM.[3]

- In a collaborative meta-analysis of 820 900 individuals from 97 prospective studies, DM was associated with the following risks: all-cause mortality, HR 1.80 (95% CI, 1.71–1.90); cancer death, HR 1.25 (95% CI, 1.19–1.31); and vascular death, HR 2.32 (95% CI, 2.11–2.56). In particular, DM was associated with death attributable to the following cancers: liver, pancreas, ovary, colorectal, lung, bladder, and breast. A 50-year-old with DM died on average 6 years earlier than an individual without DM.[24]
- FHS/NHLBI data show that having DM significantly increased the risk of developing CVD (HR 2.5 for women and 2.4 for men) and of dying when CVD was present (HR 2.2 for women and 1.7 for men). Diabetic men and women ≥50 years of age lived an average of 7.5 and 8.2 years less than their nondiabetic equivalents. The differences in life expectancy free of CVD were 7.8 and 8.4 years, respectively.[25]
- Analysis of data from NHANES 1971–2000 found that men with DM experienced a 43% relative reduction in the age-adjusted mortality rate, which was similar to that of nondiabetic men. Among women with DM, however, mortality rates did not decrease, and the difference in mortality rates between diabetic and nondiabetic women doubled.[26]
- During 1979 to 2004, DM death rates for black youths 1 to 19 years of age were approximately twice those for white youths. During 2003 to 2004, the annual average DM death rate per 1 million youths was 2.46 for black youths and 0.91 for white youths.[27]
- Analysis of data from the FHS from 1950 to 2005 found reductions in all-cause and CVD mortality among men and women with and without DM. However, all-cause and CVD mortality rates among individuals with DM remain ≈2-fold higher than for individuals without DM.[28]
- According to NHIS data from 1997 to 2006, the rate of CVD death among adults with DM decreased by 40% (95% CI, 23%–54%). Similarly, all-cause mortality decreased by 23% (95% CI, 10%–35%). Compared with adults without DM, the CVD mortality rate decreased by 60% and the all-cause mortality rate decreased by 44%.[29]
- Among 11 927 participants with DM who were part of the Translating Research into Action for Diabetes study, DM was recorded on 41% of the death certificates as the underlying cause of death for slightly more than one tenth (13%). It was noted that DM was more likely to be reported as a cause of death among individuals who died of CVD. These findings underscore that underreporting of DM exists on death certificates.[30]

Awareness

(See Chart 10-4.)

- Analysis of NHANES/NCHS data from 1988–1994 to 2005–2006 in adults ≥20 years of age showed that 40% of those with DM did not know they had it.[11] Although the prevalence of diagnosed DM has increased significantly over the past decade, the prevalence of undiagnosed DM and impaired fasting glucose has remained relatively stable. Minority groups remain disproportionately affected.[31]
- Analysis of NHANES/NCHS data collected during 2005–2008 indicated that the prevalence of DM was 8.2% among people ≥20 years of age. Prevalence of DM was defined as people who were told by a physician or other health professional that they have DM. Of the estimated 18.3 million adults with DM, 73.3% were told they had DM or were undergoing treatment, and 26.7% (5.7 million) were unaware of the diagnosis. Of 7.9 million people being treated (37.3% of the diabetic population), one third (2.6 million) had their DM under control (ie, they were undergoing treatment and had fasting plasma glucose <126 mg/dL), and 25.0% (5.3 million) were being treated but did not have their DM under control (fasting plasma glucose ≥126 mg/dL). An estimated 13.3 million individuals with DM are not treated. The untreated and unaware population (5.6 million) was 26.7% of the diabetic population (NHANES 2003–2006, NHLBI tabulation).

Aftermath

- Although the exact date of DM onset can be difficult to determine, increasing duration of DM diagnosis is associated with increasing CVD risk. Longitudinal data from FHS suggest that the risk factor–adjusted RR of CHD is 1.38 (95% CI, 0.99–1.92) times higher and the risk for CHD death is 1.86 (95% CI, 1.17–2.93) times higher for each 10-year increase in duration of DM.[32]
- On the basis of data from the NCHS/NHIS, 1997–2005[33]

  —The estimated number of people ≥35 years of age with DM with a self-reported cardiovascular condition increased 36%, from 4.2 million in 1997 to 5.7 million in 2005; however, the age-adjusted prevalence of self-reported CVD conditions among people with diagnosed DM who were ≥35 years of age decreased 11.2%, from 36.6% in 1997 to 32.5% in 2005.
  —Age-adjusted CVD prevalence was higher among men than women, among whites than blacks, and among non-Hispanics than Hispanics. Among women, the age-adjusted prevalence decreased by 11.2%; among men, it

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did not decrease significantly. Among blacks, the age-adjusted prevalence of self-reported CVD decreased by 25.3%; among whites, no significant decrease occurred; among non-Hispanics, the rate decreased by 12%. No clear trends were detected among Hispanics. If the total number of people with DM and self-reported CVD increased over this period but proportions with self-reported CVD declined, the data suggest that the mean age at which people have been diagnosed is decreasing, or the higher CVD mortality rate among older diabetic individuals is removing them from ability to self-report CVD. These and other data show a consistent increase over time in the United States of the number of people with DM and CVD.

- Data from FHS show that despite improvements in CVD morbidity and mortality, DM continues to be associated with incremental CVD risk. Participants 45 to 64 years of age from the FHS original and offspring cohorts who attended examinations in 1950 to 1966 ("earlier" time period) and 1977 to 1995 ("later" time period) were followed up for incident MI, CHD death, and stroke. Among participants with DM, the age- and sex-adjusted CVD incidence rate was 286.4 per 10 000 person-years in the earlier period and 146.9 per 10 000 person-years in the later period, a 35.4% decline. HRs for DM as a predictor of incident CVD were not significantly different in the earlier (risk factor–adjusted HR, 2.68; 95% CI, 1.88–3.82) versus later (HR, 1.96; 95% CI, 1.44–2.66) period.[34] Thus, although there was a 50% reduction in the rate of incident CVD events among adults with DM, the absolute risk of CVD remained 2-fold greater than among people without DM.[34]

  —Data from these earlier and later time periods in FHS also suggest that the increasing prevalence of DM is leading to an increasing rate of CVD, resulting in part from CVD risk factors that commonly accompany DM. The age- and sex-adjusted HR for DM as a CVD risk factor was 3.0 in the earlier time period and 2.5 in the later time period. Because the prevalence of DM has increased over time, the population attributable risk (PAR) for DM as a CVD risk factor increased from 5.4% in the earlier time period to 8.7% in the later time period (attributable risk ratio, 1.62; P=0.04). Adjustment for CVD risk factors (age, sex, hypertension, current smoking, high cholesterol, and obesity) weakened this attributable risk ratio to 1.5 (P=0.12).[35]

  —Other data from FHS show that over a 30-year period, CVD among women with DM was 54.8% among normal-weight women but 78.8% among obese women. Among normal-weight men with DM, the lifetime risk of CVD was 78.6%, whereas it was 86.9% among obese men.[36]

- DM increases the risk of stroke, with the RR ranging from 1.8- to 6-fold increased risk.[32,37]

  —DM is associated with increased ischemic stroke incidence at all ages, with the incremental risk associated with DM being most prominent before 55 years of age in blacks and before 65 years of age in whites.[37]

  —Ischemic stroke patients with DM are younger, more likely to be black, and more likely to have hypertension, prior MI, and high cholesterol than nondiabetic patients.[37]

- Data from the ARIC study of the NHLBI found that the magnitude of incremental CHD risk associated with DM was smaller in blacks than in whites.[38]
- A subgroup analysis was conducted of patients with DM enrolled in randomized clinical trials that evaluated acute coronary syndrome (ACS) therapies. The data included 62 036 patients from Thrombolysis in Myocardial Infarction studies (46 577 with STEMI and 15 459 with unstable angina [UA]/non–ST-segment–elevation myocardial infarction [NSTEMI]). Of these, 17.1% had DM. Modeling showed that mortality at 30 days was significantly higher among patients with DM than among those without DM who presented with UA/NSTEMI (2.1% versus 1.1%; $P \leq 0.001$) and STEMI (8.5% versus 5.4%; P=0.001), with adjusted risks for 30-day mortality in DM versus no DM of 1.78 for UA/NSTEMI (95% CI, 1.24–2.56) and 1.40 (95% CI, 1.24–1.57) for STEMI. DM was also associated with significantly higher mortality 1 year after UA/NSTEMI or STEMI. By 1 year after ACS, patients with DM presenting with UA/NSTEMI had a risk of death that approached that of patients without DM presenting with STEMI (7.2% versus 8.1%).[39]
- Other studies show that the increased prevalence of DM is being followed by an increasing prevalence of CVD morbidity and mortality. New York City death certificate data for 1989 to 1991 and 1999 to 2001 and hospital discharge data for 1988 to 2002 show increases in all-cause and cause-specific mortality between 1990 and 2000, as well as in annual hospitalization rates for DM and its complications among patients hospitalized with AMI and/or DM. During this decade, all-cause and cause-specific mortality rates declined, although not for patients with DM; rates increased 61% and 52% for diabetic men and women, respectively, as did hospitalization rates for DM and its complications. The percentage of all AMIs occurring in patients with DM increased from 21% to 36%, and the absolute number more than doubled, from 2951 to 6048. Although hospital days for AMI fell overall, for those with DM, they increased 51% (from 34 188 to 51 566). These data suggest that increases in DM rates threaten the long-established nationwide trend toward reduced coronary artery events.[40]
- In an analysis of provincial health claims data for adults living in Ontario, Canada, between 1992 and 2000, the rate of patients admitted for AMI and stroke decreased to a greater extent in the diabetic than the nondiabetic population (AMI, −15.1% versus −9.1%, P=0.0001; stroke, −24.2% versus −19.4%, P=0.0001). Diabetic patients experienced reductions in case fatality rates related to AMI and stroke similar to those without DM (−44.1% versus −33.2%, P=0.1; −17.1% versus −16.6%, P=0.9, respectively) and similarly comparable decreases in all-cause mortality. Over the same period, the number of DM cases increased by 165%, which translates to a marked increase in the proportion of CVD events occurring among patients with

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DM: AMI, 44.6%; stroke, 26.1%; AMI deaths, 17.2%; and stroke deaths, 13.2%.[41]

- In the same data set, the transition to a high-risk category (an event rate equivalent to a 10-year risk of 20% or an event rate equivalent to that associated with previous MI) occurred at a younger age for men and women with DM than for those without DM (mean difference 14.6 years). For the outcome of AMI, stroke, or death resulting from any cause, diabetic men and women entered the high-risk category at 47.9 and 54.3 years of age, respectively. The data suggest that DM confers a risk equivalent to aging 15 years. In North America, diverse data show lower rates of CVD among diabetic people, but as the prevalence of DM has increased, so has the absolute burden of CVD, especially among middle-aged and older individuals.[42]
- DM accounted for 44% of the new cases of end-stage renal disease (ESRD) in 2007. According to data from the US Renal Data System and BRFSS from 1996 to 2007, the incidence rate of ESRD attributed to DM decreased from 304.5 per 100 000 to 199.1 per 100 000.[43]
- According to NHANES data, the prevalence of diabetic kidney disease has increased from 2.2% in NHANES III to 3.3% in NHANES 2005–2008. These increases were observed in direct proportion to increases in DM.[44]
- HbA_{1c} levels ≥6.5% can be used to diagnose DM.[45] In the population-based ARIC study, HbA_{1c} levels ≥6.5% had a 14-year follow-up, multivariable-adjusted HR of 16.5 (95% CI, 14.2–19.1) for diagnosed DM and 1.95 (95% CI, 1.53–2.48) for CHD relative to those with HbA_{1c} <5.0%.[46]
- According to data from the ARIC study and NHANES III, the sensitivity and specificity for diagnosing DM (compared with a single fasting glucose measurement of ≥126 mg/dL) were 47% and 98%, respectively.

Risk Factors

- DM, especially type 2 DM, is associated with clustered risk factors for CHD, with a prevalence of 75% to 85% for hypertension among adults with DM, 70% to 80% for elevated LDL, and 60% to 70% for obesity.[47]
- Aggressive treatment of hypertension is recommended for adults with DM to prevent cardiovascular complications. Between NHANES III (1984–1992) and NHANES 1999–2004, the proportion of patients with DM whose BP was treated increased from 76.5% to 87.8%, and the proportion whose BP was controlled nearly doubled (15.9% to 29.6%).[48]
- Aggressive treatment of hypercholesterolemia is recommended for adults with DM, with the cornerstone of treatment being statin therapy, which is recommended for all patients with DM >40 years of age independent of baseline cholesterol, with targeted LDL cholesterol <100 mg/dL and optimally <70 mg/dL.[49]
- CHD risk factors among patients with DM remain suboptimally treated, although improvements have been observed over the past decade. Between 1999 and 2008, in up to 2623 adult participants with DM, data from NHANES showed that improvements were observed for the achieved targets for control of HbA_{1c} (from 37.0% to 55.2%), BP (from 35.2% to 51.0%), and LDL cholesterol (from 32.5% to 52.9%).[50]
- Data from the 2004 National Healthcare Disparities Report (Agency for Healthcare Research and Quality, US Department of Health and Human Services) found that only approximately one third of adults with DM received all 5 interventions to reduce risk factors recommended for comprehensive DM care in 2001. The proportion receiving all 5 interventions was lower among blacks than whites and among Hispanics than non-Hispanic whites.[51]

  —In multivariable models that controlled for age, sex, income, education, insurance, and residence location, blacks were 38% less likely and Hispanics were 33% less likely than their respective comparison groups to receive all recommended risk factor interventions in 2001.[51]

- In 1 large academic medical center, outpatients with type 2 DM were observed during an 18-month period for proportions of patients who had HbA_{1c} levels, BP, or total cholesterol levels measured; who had been prescribed any drug therapy if HbA_{1c} levels, SBP, or LDL cholesterol levels exceeded recommended treatment goals; and who had been prescribed greater-than-starting-dose therapy if these values were above treatment goals. Patients were less likely to have cholesterol levels measured (76%) than HbA_{1c} levels (92%) or BP (99%; $P<0.0001$ for either comparison). The proportion of patients who received any drug therapy was greater for above-goal HbA_{1c} (92%) than for above-goal SBP (78%) or LDL cholesterol (38%; $P<0.0001$ for each comparison). Similarly, patients whose HbA_{1c} levels were above the treatment goal (80%) were more likely to receive greater-than-starting-dose therapy than were those who had above-goal SBP (62%) and LDL cholesterol levels (13%; $P<0.0001$).[52]

  —Data from the same academic medical center also showed that CVD risk factors among women with DM were managed less aggressively than among men with DM. Women were less likely than men to have HbA_{1c} <7% (without CHD: adjusted OR for women versus men 0.84, $P=0.005$; with CHD: 0.63, $P<0.0001$). Women without CHD were less likely than men to be treated with lipid-lowering medication (0.82; $P=0.01$) or, when treated, to have LDL cholesterol levels <100 mg/dL (0.75; $P=0.004$) and were less likely than men to be prescribed aspirin (0.63; $P<0.0001$). Women with DM and CHD were less likely than men to be prescribed aspirin (0.70, $P<0.0001$) and, when treated for hypertension or hyperlipidemia, were less likely to have BP levels <130/80 mm Hg (0.75; $P<0.0001$) or LDL cholesterol levels <100 mg/dL (0.80; $P=0.006$).[53]

- Analysis of data from the CHS of the NHLBI found that lifestyle risk factors, including PA level, dietary habits, smoking habits, alcohol use, and adiposity measures, assessed late in life, were each independently associated with risk of new-onset DM. Participants whose PA level and dietary, smoking, and alcohol habits were all in the low-risk group had an 82% lower incidence of DM than all other participants. When absence of adiposity was added to the other 4 low-risk lifestyle factors, incidence of DM was 89% lower.[54]
- According to 2007 data from the BRFSS, only 25% of adults with DM achieved recommended levels of total PA based on the 2007 American Diabetes Association guidelines.[55]

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Hospitalizations

(See Table 10-1.)

Youths

- Nationwide Inpatient Sample data from 1993 to 2004 were analyzed for individuals 0 to 29 years of age with a diagnosis of DM. Rates of hospitalizations increased by 38%. Hospitalization rates were higher for females (42%) than for males (29%). Inflation-adjusted total charges for DM hospitalizations increased 130%, from $1.05 billion in 1993 to $2.42 billion in 2004.[56]

Adults

- According to NHDS data reported by the CDC in an analysis of data from 2009, DM was a listed diagnosis in ≈15% of US adult hospital discharges, among which circulatory diseases was the most common first-listed diagnosis (26.2%; 1.4 million discharges) and DM the second most common (12.0%; 655 000 discharges).[57]

Hypoglycemia

- Hypoglycemia is a common side effect of DM treatment, typically defined as a blood glucose level <50 mg/dL; severe hypoglycemia is additionally defined as patients needing assistance to treat themselves.
- In the ADVANCE (Action in Diabetes and Vascular Disease: Preterax and Diamicron Modified Release Controlled Evaluation) trial, 2.1% of patients had an episode of severe hypoglycemia. Severe hypoglycemia was associated with an increased risk of major macrovascular events (HR, 2.88; 95% CI, 2.01–4.12), cardiovascular death (HR, 2.68; 95% CI, 1.72–4.19), and all-cause death (HR, 2.69; 95% CI, 1.97–3.67), including nonvascular outcomes. The lack of specificity of hypoglycemia with vascular outcomes suggests that it might be a marker for susceptibility. Risk factors for hypoglycemia included older age, DM duration, worse renal function, lower BMI, lower cognitive function, multiple glucose-lowering medications, and randomization to the intensive glucose control arm.[58]
- According to data from the 2004 to 2008 MarketScan database of type 2 DM, which consisted of 536 581 individuals, the incidence rate of hypoglycemia was 153.8 per 10 000 person-years and was highest in adults aged 18 to 34 years (218.8 per 10 000 person-years).[59]
- According to data from 2956 adults ≥55 years of age from the ACCORD (Action to Control Cardiovascular Risk in Diabetes) trial, poor cognitive function, defined as a 5-point poorer baseline score on the Digit Symbol Substitution Test, was associated with a 13% increased risk of severe hypoglycemia that required medical assistance.[60]

Cost

(See Table 10-1.)

- In 2007, the direct ($116 billion) and indirect ($58 billion) cost attributable to DM was $174 billion.[3] These estimates include not just DM as a primary diagnosis but also long-term complications that are attributed to DM.[61]
- After adjustment for age and sex, medical costs for patients with DM were 2.3 times higher than for people without DM.[3]
- A study of data from NHANES 2003–2006, Ingenix Research Data Mart, 2003–2005 NAMCS, the 2003–2005 NHAMCS, the 2004–2005 Nationwide Inpatient Sample, and the 2003–2005 MEPS found that the estimated economic cost of undiagnosed DM in 2007 was $18 billion, including medical costs of $11 billion and indirect costs of $7 billion.[62]
- According to 2003–2005 MEPS data (household component data), reductions in DM and hypertension of 5% could save ≈9 billion dollars annually in the short-term. Longer term, savings could total nearly $25 billion.[63]
- When the 2007 MarketScan database was used to assess costs associated with youths who have DM, mean annual costs per person with DM were $9061 compared with $1468 for those without DM; 43% of this cost was for prescription drugs. For type 1 compared with type 2 DM, costs were $9333 versus $5683, respectively.[64]
- Using the insurance claims and MarketScan data from 7556 youths <19 years of age with insulin-treated DM, costs for youths with hypoglycemia were $12 850 compared with $8970 for youths without hypoglycemia. For diabetic ketoacidosis, costs were $14 236 for youths with versus $8398 for youths without diabetic ketoacidosis.[65]
- The cost of hypoglycemia, according to data from 536 581 individuals with type 2 DM from the 2004–2008 MarketScan database, was $52 223 675, which accounted for 1.0% of inpatient costs, 2.7% of ED costs, and 0.3% of outpatient costs. This resulted in a mean cost of $17 564 for an inpatient admission, $1387 for an ED visit, and $394 for an outpatient visit.[59]

Type 1 DM

- Type 1 DM constitutes 5% to 10% of DM in the United States.[66]
- The Colorado IDDM (insulin-dependent DM) Study Registry (1978-1988) and SEARCH for Diabetes in Youth (2002-2004) demonstrated an increasing incidence of type 1 DM among Colorado youths ≤17 years of age, with a 2.3% (95% CI, 1.6%–3.1%) increase per year over the past 26 years.[67]
- Among youths with type 1 DM, the prevalence of overweight is 22.1% and the prevalence of obesity is 12.6%.[6]
- A long-term study of patients with type 1 DM that began in 1966 showed that over 30 years of follow-up, overall risk of mortality associated with type 1 DM was 7 times greater than that of the general population. Females had a 13.2-fold incremental mortality risk compared with a 5.0-fold increased risk in males. During the course of study, the incremental mortality risk associated with type 1 DM declined from 9.3 to 5.6 times that of nondiabetic control subjects.[68]
- According to 30-year mortality data from Allegheny County, PA, those with type 1 DM have a mortality rate 5.6 times higher than the general population.[69]
- The leading cause of death among patients with type 1 DM is CVD, which accounted for 22% of deaths among those in the Allegheny County, PA, type 1 DM registry, followed by renal (20%) and infectious (18%) causes.[70]

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- Long-term follow-up data from the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Study Research Group showed that intensive versus conventional treatment in the Diabetes Control and Complications Trial was associated with a 42% reduced risk of CVD (P=0.02) and a 57% reduced risk of the composite end point (P=0.02; included nonfatal MI, stroke, and CVD death).[71]
- Observational data from the Swedish National Diabetes Register showed that most CVD risk factors were more adverse among patients with HbA_{1c} between 8.0% and 11.9% than among those with HbA_{1c} between 5.0% and 7.9%. Per 1% unit increase in HbA_{1c}, the HR of fatal and nonfatal CHD was 1.30 in multivariable adjusted models and 1.27 for fatal and nonfatal CVD. Among patients with HbA_{1c} 8.0% to 11.9% compared with those with HbA_{1c} 5.0% to 7.9%, the HR of fatal/nonfatal CHD was 1.71 and the risk of fatal/nonfatal CVD was 1.59.[72]
- Among 2787 patients from the EURODIAB Prospective Complications Study, age, waist-hip ratio, pulse pressure, non-HDL cholesterol, microalbuminuria, and peripheral and autonomic neuropathy were risk factors for all-cause, CVD, and non-CVD mortality.[61]
- Among 3610 older patients (>60 years of age) with type 1 DM, the risk of severe hypoglycemia was twice as high as for those <60 years of age (40.1 versus 24.3 per 100 patient-years).[73]

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Table 10-1. Diabetes Mellitus

Population Group	Prevalence of Physician-Diagnosed DM, 2010: Age ≥20 y	Prevalence of Undiagnosed DM, 2010: Age ≥20 y	Prevalence of Prediabetes, 2010: Age ≥20 y	Incidence of Diagnosed DM: Age ≥20 y†	Mortality (DM), 2009: All Ages‡	Hospital Discharges, 2010: All Ages	Cost, 2007†
Both sexes*	19 700 000 (8.3%)	8 200 000 (3.5%)	87 300 000 (38.2%)	1 900 000	68 705	630 000	$174 Billion
Males*	9 600 000 (8.7%)	5 300 000 (4.7%)	50 700 000 (46.0%)	…	35 054	311 000	…
Females*	10 100 000 (7.9%)	2 900 000 (2.3%)	33 600 000 (30.5%)	…	33 651	319 000	…
NH white males, %	7.7	4.5	47.7	…	28 205	…	…
NH white females, %	6.2	1.8	30.0	…	25 908	…	…
NH black males, %	13.5	4.8	35.7	…	5488	…	…
NH black females, %	15.4	2.9	29.0	…	6472	…	…
Mexican-American males, %	11.4	6.6	47.0	…	…	…	…
Mexican-American females, %	12.0	4.7	31.9	…	…	…	…
Asian	…	…	…	…	1846	…	…
American Indian/Alaska Native	…	…	…	…	786	…	…

DM indicates diabetes mellitus; NH, non-Hispanic. Ellipses (…) indicate data not available.

Undiagnosed DM is defined as those whose fasting glucose is ≥126 mg/dL but who did not report being told by a healthcare provider that they had DM. Prediabetes is a fasting blood glucose of 100 to <126 mg/dL (impaired fasting glucose); prediabetes includes impaired glucose tolerance.

*These percentages represent the portion of total DM mortality that is for males vs females.

†Centers for Disease Control and Prevention, National Diabetes Fact Sheet, 2011.[3]

‡Mortality data include Hispanics.

Sources: Prevalence: Prevalence of diagnosed and undiagnosed diabetes: National Health and Nutrition Examination Survey 2007–2010, National Center for Health Statistics (NCHS), and National Heart, Lung, and Blood Institute. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolations to the 2010 US population estimates. Mortality: Centers for Disease Control and Prevention/NCHS, 2009 Mortality Multiple Cause–US, Version July 19, 2012. These data represent underlying cause of death only. Hospital discharges: National Hospital Discharge Survey, NCHS; data include those inpatients discharged alive, dead, or status unknown.

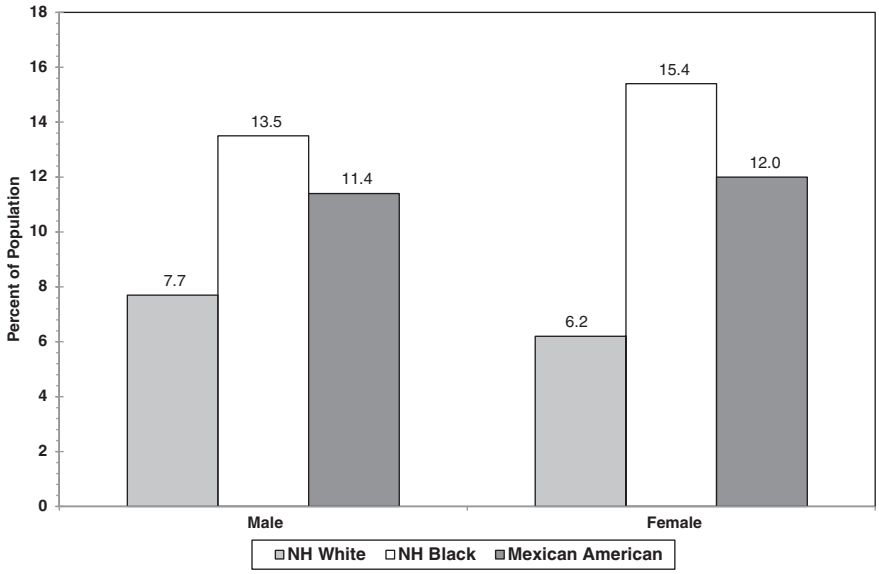

Chart 10-1. Age-adjusted prevalence of physician-diagnosed diabetes mellitus in adults ≥20 years of age by race/ethnicity and sex (National Health and Nutrition Examination Survey: 2007–2010). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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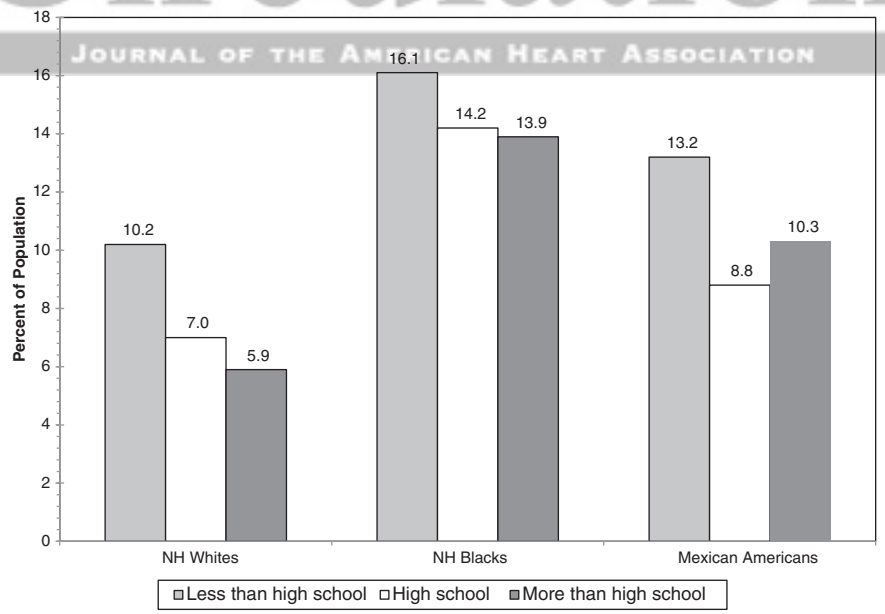

Chart 10-2. Age-adjusted prevalence of physician-diagnosed type 2 diabetes mellitus in adults ≥20 years of age by race/ethnicity and years of education (National Health and Nutrition Examination Survey: 2007–2010). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

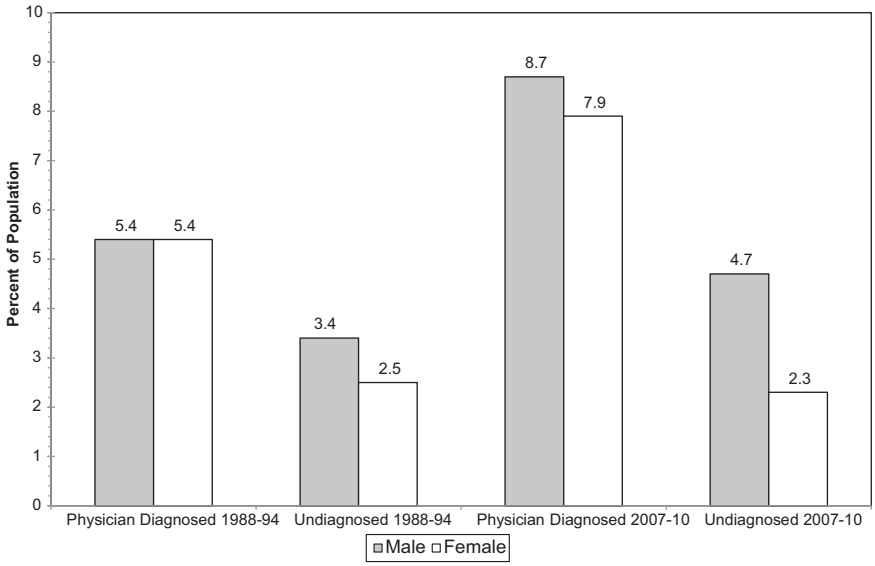

Chart 10-3. Trends in diabetes mellitus prevalence in adults ≥20 years of age by sex (National Health and Nutrition Examination Survey: 1988–1994 and 2007–2010). Source: National Center for Health Statistics, National Heart, Lung, and Blood Institute.

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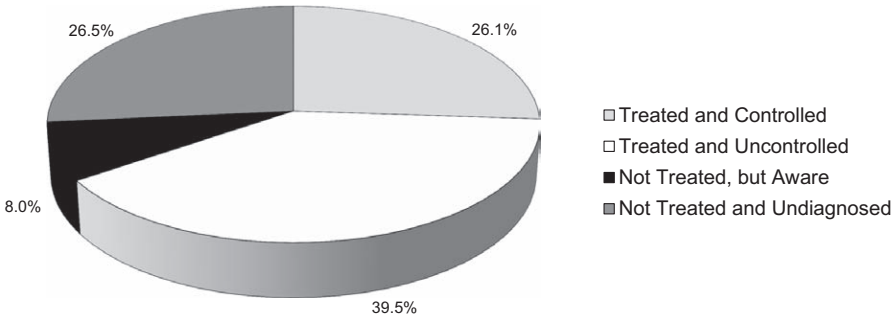

Chart 10-4. Diabetes mellitus awareness, treatment, and control (National Health and Nutrition Examination Survey: 2007–2010). Source: National Heart, Lung, and Blood Institute.

11. Metabolic Syndrome

- Metabolic syndrome refers to a cluster of risk factors for CVD and type 2 DM. Although several different definitions for metabolic syndrome have been proposed, the International Diabetes Federation, NHLBI, AHA, and others recently proposed a harmonized definition for metabolic syndrome.[1] By this definition, metabolic syndrome is diagnosed when ≥3 of the following 5 risk factors are present (most but not all people with DM will be classified as having metabolic syndrome by this definition because they will have at least 2 other factors besides the glucose criterion; many will prefer to separate those with DM into a separate group for risk stratification or treatment purposes):

  —Fasting plasma glucose ≥100 mg/dL or undergoing drug treatment for elevated glucose
  —HDL cholesterol <40 mg/dL in men or <50 mg/dL in women or undergoing drug treatment for reduced HDL cholesterol.
  —Triglycerides ≥150 mg/dL or undergoing drug treatment for elevated triglycerides
  —Waist circumference ≥102 cm in men or ≥88 cm in women in the United States.
  —BP ≥130 mm Hg systolic or ≥85 mm Hg diastolic or undergoing drug treatment for hypertension or antihypertensive drug treatment in a patient with a history of hypertension.

- Identification of metabolic syndrome represents a call to action for the healthcare provider and patient to address the underlying lifestyle-related risk factors, including abdominal obesity, physical inactivity, and atherogenic diet, as well as clinical management to address the characteristic atherogenic dyslipidemia, elevated BP, elevated glucose, and prothrombotic state that are common to people with metabolic syndrome. A multidisciplinary team of healthcare professionals is desirable to adequately address these multiple issues in patients with the metabolic syndrome.[2]

Abbreviations Used in Chapter 11

AF	atrial fibrillation
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities
BMI	body mass index
BP	blood pressure
CAC	coronary artery calcification
CAD	coronary artery disease
CHD	coronary heart disease
CI	confidence interval
COURAGE	Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation
CVD	cardiovascular disease
DM	diabetes mellitus
FRS	Framingham Risk Score
HDL	high-density lipoprotein
HF	heart failure
HR	hazard ratio
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute
OR	odds ratio
PA	physical activity
PAR	population attributable risk
PCI	percutaneous coronary intervention
RR	relative risk
WHO	World Health Organization

Adults

The following estimates include many of those who have DM, in addition to those with metabolic syndrome without DM.

- Prevalence of metabolic syndrome varies by the definition used, with definitions such as that from the International Diabetes Federation that suggest lower thresholds for defining central obesity in European whites, Asians, and Hispanics resulting in higher prevalence estimates.[3]
- On the basis of NHANES 2003–2006 data and National Cholesterol Education Program/Adult Treatment Panel III guidelines, ≈34% of adults ≥20 years of age met the criteria for metabolic syndrome.[4]
- Also based on NHANES 2003–2006 data[4]

  —The age-adjusted prevalence was 35.1% for men and 32.6% for women.
  —Among men, the age-specific prevalence ranged from 20.3% among people 20 to 39 years of age to 40.8% for people 40 to 59 years of age and 51.5% for people ≥60 years of age. Among women, the age-specific prevalence ranged from 15.6% among people 20 to 39 years of age to 37.2% for people 40 to 59 years of age and 54.4% for those ≥60 years of age.
  —The age-adjusted prevalences of people with metabolic syndrome were 37.2%, 25.3%, and 33.2% for non-Hispanic white, non-Hispanic black, and Mexican American men, respectively. Among women, the percentages were 31.5%, 38.8%, and 40.6%, respectively.
  —The age-adjusted prevalence was ≈53% higher among non-Hispanic black women than among non-Hispanic black men and ≈22% higher among Mexican American women than among Mexican American men.

- The prevalence of metabolic syndrome is also high among immigrant Asian Indians, ranging between 26.8% and 38.2% depending on the definition used.[5]
- The prevalence of metabolic syndrome among pregnant women increased to 26.5% during 1999 to 2004 from 17.8% during 1988 to 1994.[6]
- Despite its prevalence, the public's recognition of metabolic syndrome is limited.[7] The above estimates include many of those who have DM in addition to those with metabolic syndrome without DM.

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Children/Adolescents

- According to the 2009 AHA scientific statement about metabolic syndrome in children and adolescents, metabolic syndrome should be diagnosed with caution in this age group because metabolic syndrome categorization in adolescents is not stable.[8] Approximately half of the 1098 adolescent participants in the Princeton School District Study diagnosed with pediatric Adult Treatment Panel III metabolic syndrome lost the diagnosis over 3 years of follow-up.[9]
- Additional evidence of the instability of the diagnosis of metabolic syndrome in children exists. In children 6 to 17 years of age participating in research studies in a single clinical research hospital, the diagnosis of metabolic syndrome was unstable in 46% of cases after a mean of 5.6 years of follow-up.[10]
- On the basis of NHANES 1999–2002 data, the prevalence of metabolic syndrome in adolescents 12 to 19 years of age was 9.4%, which represents ≈2.9 million people. It was 13.2% in boys, 5.3% in girls, 10.7% in whites, 5.2% in blacks, and 11.1% in Mexican Americans.[11]
- In 1999 to 2004, ≈4.5% of US adolescents 12 to 17 years of age had metabolic syndrome according to the definition developed by the International Diabetes Federation.[12] In 2006, this prevalence would have represented ≈1.1 million adolescents 12 to 17 years of age with metabolic syndrome. It increased from 1.2% among those 12 to 13 years of age to 7.1% among those 14 to 15 years of age and was higher among boys (6.7%) than girls (2.1%). Furthermore, 4.5% of white adolescents, 3.0% of black adolescents, and 7.1% of Mexican American adolescents had metabolic syndrome. The prevalence of metabolic syndrome remained relatively stable during successive 2-year periods: 4.5% for 1999 to 2000, 4.4% to 4.5% for 2001 to 2002, and 3.7% to 3.9% for 2003 to 2004.
- In 1999 to 2002, among overweight or obese adolescents, 44% had metabolic syndrome.[11] In 1988 to 1994, two thirds of all adolescents had at least 1 metabolic abnormality.[13]
- Of 31 participants in the NHLBI Lipid Research Clinics Princeton Prevalence Study and the Princeton Follow-Up Study who had metabolic syndrome at baseline, 21 (68%) had metabolic syndrome 25 years later.[14] After adjustment for age, sex, and race, the baseline status of metabolic syndrome was significantly associated with an increased risk of having metabolic syndrome during adulthood (OR, 6.2; 95% CI, 2.8–13.8).
- In the Bogalusa Heart Study, 4 variables (BMI, homeostasis model assessment of insulin resistance, ratio of triglycerides to HDL cholesterol, and mean arterial pressure) considered to be part of the metabolic syndrome clustered together in blacks and whites and in children and adults.[15] The degree of clustering was stronger among adults than among children. The clustering of rates of change in the components of the metabolic syndrome in blacks exceeded that in whites. Cardiovascular abnormalities are associated with metabolic syndrome in children and adolescents.[16,17]

Risk

Adults

- Consistent with 2 earlier meta-analyses, a recent meta-analysis of prospective studies concluded that metabolic syndrome increased the risk of developing CVD (summary RR, 1.78; 95% CI, 1.58–2.00).[18] The risk of CVD tended to be higher in women (summary RR, 2.63) than in men (summary RR, 1.98; P=0.09). On the basis of results from 3 studies, metabolic syndrome remained a predictor of cardiovascular events after adjustment for the individual components of the syndrome (summary RR, 1.54; 95% CI, 1.32–1.79). A more recent meta-analysis among 87 studies comprising 951 083 subjects showed an even higher risk of CVD associated with metabolic syndrome (summary RR, 2.35; 95% CI, 2.02–2.73), with significant increased risks (RRs ranging from 1.6 to 2.9) for all-cause mortality, CVD mortality, MI, and stroke, as well as for those with metabolic syndrome without DM.[19]
- In one of the earlier studies among US adults, mortality follow-up of the second NHANES showed a stepwise increase in risk of CHD, CVD, and total mortality across the spectrum of no disease, metabolic syndrome (without DM), DM, prior CVD, and those with CVD and DM, with an HR for CHD mortality of 2.02 (95% CI, 1.42–2.89) associated with metabolic syndrome. Increased risk was seen with increased numbers of metabolic syndrome risk factors.[20]
- Several studies suggest that the Framingham Risk Score (FRS) is a better predictor of incident CVD than metabolic syndrome.[21–23] In the San Antonio Heart Study, the area under the receiver-operating characteristic curve was 0.816 for the FRS and 0.811 for the FRS plus the metabolic syndrome.[21] Furthermore, the sensitivity for CVD at a fixed specificity was significantly higher for the FRS than for the metabolic syndrome. In ARIC, inclusion of the metabolic syndrome did not improve the risk prediction achieved by the FRS.[22] In the British Regional Heart Study, the area under the receiver-operating characteristic curve for the FRS was 0.73 for incident CHD during 10 years of follow-up, and the area under the receiver-operating characteristic curve for the number of metabolic syndrome components was 0.63.[23] For CHD events during 20 years of follow-up, the areas under the receiver-operating characteristic curves were 0.68 for the FRS and 0.59 for the number of metabolic syndrome components.
- Estimates of RR for CVD generally increase as the number of components of metabolic syndrome increases.[23] Compared with men without an abnormal component in the Framingham Offspring Study, the HRs for CVD were 1.48 (95% CI, 0.69–3.16) for men with 1 or 2 components and 3.99 (95% CI, 1.89–8.41) for men with ≥3 components.[24] Among women, the HRs were 3.39 (95% CI, 1.31–8.81) for 1 or 2 components and 5.95 (95% CI, 2.20–16.11) for ≥3 components. Compared with men without a metabolic abnormality in the British Regional Heart Study, the HRs were 1.74 (95% CI, 1.22–2.39) for 1 component, 2.34 (95% CI, 1.65–3.32) for 2 components, 2.88 (95% CI, 2.02–4.11) for 3 components, and 3.44 (95% CI, 2.35–5.03) for 4 or 5 components.[23]
- The cardiovascular risk associated with the metabolic syndrome varies on the basis of the combination of metabolic syndrome components present. Of all possible ways to have 3 metabolic syndrome components, the combination of central obesity, elevated BP, and hyperglycemia conferred the greatest risk for CVD (HR, 2.36; 95% CI, 1.54–3.61) and mortality (HR, 3.09; 95% CI, 1.93–4.94) in the Framingham Offspring Study.[25]

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- Data from the Aerobics Center Longitudinal Study indicate that risk for CVD mortality is increased in men without DM who have metabolic syndrome (HR, 1.8; 95% CI, 1.5–2.0); however, among those with metabolic syndrome, the presence of DM is associated with even greater risk for CVD mortality (HR, 2.1; 95% CI, 1.7–2.6).[26] Analysis of data from NCHS was used to determine the number of disease-specific deaths attributable to all nonoptimal levels of each risk factor exposure by age and sex. The results of the analysis of dietary, lifestyle, and metabolic risk factors show that targeting a handful of risk factors has large potential to reduce mortality in the United States.[27]
- Among stable CAD patients in the COURAGE trial (Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation), the presence of metabolic syndrome was associated with an increased risk of death or MI (unadjusted HR, 1.41; 95% CI, 1.15–1.73; *P*=0.001); however, after adjustment for its individual components, metabolic syndrome was no longer significantly associated with outcome (HR, 1.15; 95% CI, 0.79–1.68; *P*=0.46). Early percutaneous coronary intervention (PCI) in addition to medical therapy did not significantly reduce the risk of death or MI regardless of metabolic syndrome or DM status.[28]
- In the INTERHEART case-control study of 26 903 subjects from 52 countries, metabolic syndrome was associated with an increased risk of MI, both according to the WHO (OR, 2.69; 95% CI, 2.45–2.95) and International Diabetes Federation (OR, 2.20; 95% CI, 2.03–2.38) definitions, with a PAR of 14.5% (95% CI, 12.7%–16.3%) and 16.8% (95% CI, 14.8%–18.8%), respectively, and associations that were similar across all regions and ethnic groups. In addition, the presence of ≥3 risk factors with subthreshold values was associated with increased risk of MI (OR, 1.50; 95% CI, 1.24–1.81) compared with having "normal" values. Similar results were observed when the International Diabetes Federation definition was used.[29]
- In MESA, among 6603 people aged 45 to 84 years (1686 [25%] with metabolic syndrome without DM and 881 [13%] with DM), subclinical atherosclerosis assessed by CAC was more severe in people with metabolic syndrome and DM than in those without these conditions, and the extent of CAC was a strong predictor of CHD and CVD events in these groups.[30] Furthermore, the progression of CAC was greater in people with metabolic syndrome and DM than in those without.[31]
- In addition to CVD, the metabolic syndrome has been associated with incident AF[32] and HF.[33]
- The metabolic syndrome is associated with increased healthcare use and healthcare-related costs among individuals with and without DM. Overall, healthcare costs increase by ≈24% for each additional metabolic syndrome component present.[34]

Children

- Few prospective pediatric studies have examined the future risk for CVD or DM according to baseline metabolic syndrome status. Data from 771 participants 6 to 19 years of age from the NHLBI's Lipid Research Clinics Princeton Prevalence Study and the Princeton Follow-Up Study showed that the risk of developing CVD was substantially higher among those with metabolic syndrome than among those without this syndrome (OR, 14.6; 95% CI, 4.8–45.3) who were followed up for 25 years.[14]
- Another analysis of 814 participants of this cohort showed that those 5 to 19 years of age who had metabolic syndrome at baseline had an increased risk of having DM 25 to 30 years later compared with those who did not have the syndrome at baseline (OR, 11.5; 95% CI, 2.1–63.7).[35]
- Additional data from the Princeton Follow-Up Study, the Fels Longitudinal Study, and the Muscatine Study suggest that the absence of components of the metabolic syndrome in childhood had a high negative predictive value for the development of metabolic syndrome or DM in adulthood.[36]

Risk Factors

- In prospective or retrospective cohort studies, the following factors have been reported as being directly associated with incident metabolic syndrome, defined by one of the major definitions: age,[35,37–39] low educational attainment,[37,40] low socioeconomic status,[41] smoking,[40–43] low levels of PA,[40–46] low levels of physical fitness,[44,47–49] intake of soft drinks,[50] intake of diet soda,[51] magnesium intake,[52] energy intake,[46] carbohydrate intake,[37,42,53] total fat intake,[37,53] Western dietary pattern,[51] meat intake,[51] intake of fried foods,[51] heavy alcohol consumption,[54] abstention from alcohol use,[37] parental history of DM,[35] long-term stress at work,[55] pediatric metabolicsyndrome,[35] obesity or BMI,[37,38,42,46,56] childhood obesity,[57] waist circumference,[39,53,58–61] intra-abdominal fat,[62] gain in weight or BMI,[37,63] change in weight or BMI,[39,42,64] weight fluctuation,[65] BP,[39,53,60,66] heart rate,[67] homeostasis model assessment,[58,68] fasting insulin,[58] 2-hour insulin,[58] proinsulin,[58] fasting glucose or hyperglycemia,[39,58,60] 2-hour glucose,[58] impaired glucose tolerance,[58] triglycerides,[39,53,56,58–60] low HDL cholesterol,[39,53,57,58,60] oxidized LDL,[68] uric acid,[64,69] γ-glutamyltransferase,[64,70,71] alanine transaminase,[64,70,72,73] plasminogen activator inhibitor-1,[74] aldosterone,[74] leptin,[75] C-reactive protein,[76,77] adipocyte–fatty acid binding protein,[78] and free testosterone index.[79]
- The following factors have been reported as being inversely associated with incident metabolic syndrome, defined by one of the major definitions, in prospective or retrospective cohort studies: muscular strength,[80] change in PA or physical fitness,[42,47] alcohol intake,[40,46] Mediterranean diet,[81] dairy consumption,[51] insulin sensitivity,[58] ratio of aspartate aminotransferase to alanine transaminase,[72] total testosterone,[79,82,83] sex hormone–binding globulin,[79,82,83] and Δ5-desaturase activity.[84]
- Furthermore, men were more likely than women to develop metabolic syndrome,[37,39] and blacks were shown to be less likely to develop metabolic syndrome than whites.[37]
- In >6 years of follow-up in the ARIC Study, 1970 individuals (25%) developed metabolic syndrome, and compared with the normal-weight group (BMI <25 kg/m^2), the ORs of developing metabolic syndrome were 2.81 (95% CI, 2.50–3.17) and 5.24 (95% CI, 4.50–6.12) for the overweight (BMI 25–30 kg/m^2) and obese (BMI ≥30 kg/m^2) groups, respectively. Compared with the lowest quartile of leisure-time PA, the ORs of developing metabolic syndrome were 0.80 (95% CI, 0.71–0.91) and 0.92 (95% CI, 0.81–1.04) for people in the highest and middle quartiles, respectively.[85]

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12. Chronic Kidney Disease

ICD-10 N18.0. See Tables 12-1 through 12-3.

End-Stage Renal Disease

Prevalence, Incidence, and Risk
(See Tables 12-1 and 12-2.)

ESRD is a condition that is most commonly associated with DM or HBP, occurs when the kidneys are functioning at a very low level, and is currently defined as the receipt of chronic renal replacement treatment such as hemodialysis, peritoneal dialysis, or kidney transplantation. The ESRD population is increasing in size and cost as those with chronic kidney disease (CKD) transition to ESRD and as a result of changing practice patterns in the United States.

- Data from the 2012 annual report of the US Renal Data System showed that in 2010:
  - —The prevalence of ESRD was 593 086, with 65% of these prevalent cases being treated with hemodialysis.[1]
  - —114 083 new cases of ESRD were reported.[1]
  - —16 843 kidney transplants were performed.[1]
- Data from a large cohort of insured patients found that in addition to established risk factors for ESRD, lower hemoglobin levels, higher serum uric acid levels, self-reported history of nocturia, and family history of kidney disease are independent risk factors for ESRD.[2]

Abbreviations Used in Chapter 12

ACTION	Acute Coronary Treatment and Intervention Outcomes Network
AF	atrial fibrillation
AMI	acute myocardial infarction
BMI	body mass index
BP	blood pressure
CHD	coronary heart disease
CHF	congestive heart failure
CKD	chronic kidney disease
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
eGFR	estimated glomerular filtration rate
ESRD	end-stage renal disease
GFR	glomerular filtration rate
HBP	high blood pressure
HF	heart failure
ICD-10	*International Classification of Diseases, 10th Revision*
JNC V	fifth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
MI	myocardial infarction
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
PAD	peripheral arterial disease
RR	relative risk

- Data from a large insured population revealed that among adults with a GFR >60 mL·min^{-1}·1.73 m^{-2} and no evidence of proteinuria or hematuria at baseline, risks for ESRD increased dramatically with higher baseline BP level, and in this same patient population, BP-associated risks were greater in men than in women and in blacks than in whites.[3]
- Compared with white patients with similar levels of kidney function, black patients are much more likely to progress to ESRD and are on average 10 years younger when they reach ESRD.[4,5]
- Results from a large community-based population showed that higher BMI also independently increased the risk of ESRD. The higher risk of ESRD with overweight and obesity was consistent across age, sex, and race and in the presence or absence of DM, hypertension, or known baseline kidney disease.[6]

Age, Sex, Race, and Ethnicity

- The median age of the population with ESRD in 2010 varied across different racial/ethnic groups: 57.9 years for Native Americans, 58.1 years for blacks, 59.7 years for Asians, and 61.2 years for whites.[1]
- Treatment of ESRD is more common in men than in women.[1]
- Blacks, Hispanics, Asian Americans, and Native Americans have higher rates of ESRD than do whites. However, since 2000 new ESRD rates have increased 6.1% for whites and 2.5 percent among Asians, while rates have decreased 7.0% for blacks.[1]

Chronic Kidney Disease

Prevalence

- CKD, defined as reduced GFR, excess urinary protein excretion, or both, is a serious health condition and a worldwide public health problem. The incidence and prevalence of CKD are increasing in the United States and are associated with poor outcomes and a high cost to the US healthcare system. Controversy exists about whether CKD itself independently causes incident CVD, but it is clear that people with CKD, as well as those with ESRD, represent a population at very high risk for CVD events. In fact, individuals with CKD are more likely to die of CVD than to transition to ESRD. The US Renal Data System estimates that by 2020, >700 000 Americans will have ESRD, with >500 000 requiring dialysis and >250 000 receiving a transplant.
- The National Kidney Foundation Kidney Disease Outcome Quality Initiative developed guidelines in 2002 that provided a standardized definition for CKD. Prevalence estimates may differ depending on assumptions used in obtaining estimates, including which equation is used to estimate GFR and methods for measuring proteinuria.[7]
- The most recent US prevalence estimates of CKD come from NHANES 1988–1994 and 1999–2004 (NCHS) in adults ≥20 years of age[8]:
  - —The prevalence of CKD in 1999–2004 (stages I to V)[9] is 13.1%. This represents an increase from the 10.0% prevalence estimate from NHANES 1988–1994 (NCHS).

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—The prevalence of stage I CKD (estimated GFR [eGFR] ≥90 mL·min^{-1}·1.73 m^{-2} with kidney damage, ie, presence of albuminuria) is 1.8%.
—The prevalence of stage II CKD (eGFR 60–89 mL·min^{-1}·1.73 m^{-2} with kidney damage) is 3.2%.
—The prevalence of stage III CKD (eGFR 30–59 mL·min^{-1}·1.73 m^{-2}) is 7.7%.
—The prevalence of stages IV and V CKD (eGFR<29 mL·min^{-1}·1.73 m^{-2}) is 0.4%.

- More than 26 million people (13%) in the United States have CKD, and most are undiagnosed.[8] Another 20 million are at increased risk for CKD.[10]

Demographics

- According to current definitions, the prevalence of CKD is higher with older age[1]:

—5.7% for those 20 to 39 years of age
—9.1% for those 40 to 59 years of age
—35.0% for those ≥60 years of age

- CKD prevalence was greater among those with DM (40.1%) and hypertension (23.2%) than among those without these chronic conditions.[1]
- The prevalence of CKD was slightly higher among Mexican Americans (18.7%) and non-Hispanic blacks (19.9%) than among non-Hispanic whites (16.1%). This disparity was most evident for those with stage I CKD; non-Hispanic whites had a CKD prevalence of 4.2% compared with prevalences among Mexican Americans and non-Hispanic blacks of 10.2% and 9.4%, respectively.[11]

Risk Factors

- Many traditional CVD risk factors are also risk factors for CKD, including older age, male sex, hypertension, DM, smoking, and family history of CVD.
- Recent evidence suggests that BMI is associated with worsening CKD.

—In a cohort of 652 African American individuals with hypertensive nephrosclerosis, BMI was independently associated with urine total protein and albumin excretion.[12]

- In addition, both the degree of CKD (ie, eGFR) and urine albumin are strongly associated with the progression from CKD to ESRD. Furthermore, urine albumin level is associated with progression to CKD across all levels of reduced eGFR.[13]
- Other risk factors include systemic conditions such as autoimmune diseases, systemic infections, and drug exposure, as well as anatomically local conditions such as urinary tract infections, urinary stones, lower urinary tract obstruction, and neoplasia. Even after adjustment for these risk factors, excess CVD risk remains.[14]

ESRD/CKD and CVD

(See Table 12-3.)

- CVD is the leading cause of death among those with ESRD, although the specific cardiovascular cause of death may be more likely to be arrhythmic than an AMI, end-stage HF, or stroke. CVD mortality is 5 to 30 times higher in dialysis patients than in subjects from the general population of the same age, sex, and race.[15,16]

—Individuals with less severe forms of kidney disease are also at significantly increased CVD risk independent of typical CVD risk factors.[17]
—CKD is a risk factor for recurrent CVD events.[18]
—CKD is also a risk factor for AF.[19]

- Studies from a broad range of cohorts demonstrate an association between reduced eGFR and elevated risk of CVD, CVD outcomes, and all-cause death[17,20–25] that appears to be largely independent of other known major CVD risk factors.
- Although clinical practice guidelines recommend management of mineral and bone disorders secondary to CKD, a recent meta-analysis suggests that there is no consistent association between calcium and parathyroid hormone and the risk of death or cardiovascular events.[26]
- Any degree of albuminuria, starting below the microalbuminuria cutpoint, has been shown to be an independent risk factor for cardiovascular events, CHF hospitalization, PAD, and all-cause death in a wide variety of cohorts.[27–32]
- A recent meta-analysis of 21 published studies of albuminuria involving 105 872 participants (730 577 person-years) from 14 studies with urine albumin/creatinine ratio measurements and 1.1 million participants (4.7 million person-years) from 7 studies with urine dipstick measurements showed that excess albuminuria or proteinuria is independently associated with a higher risk of CVD and all-cause mortality.[33]

—People with both albuminuria/proteinuria and reduced eGFR are at particularly high risk for CVD, CVD outcomes, and death.[34]
—The exact reasons why CKD and ESRD increase the risk of CVD have not been completely delineated but are clearly multifactorial and likely involve pathological alterations in multiple organ systems and pathways.

- One potential explanation for the higher CVD event rate in patients with CKD is the low uptake of standard therapies for patients presenting with MI. In a recent analysis from the Acute Coronary Treatment and Intervention Outcomes Network (ACTION) registry, patients presenting with CKD had a substantially higher mortality rate. In addition, patients with CKD were less likely to receive standard therapies for the treatment of MI.[35]

Cost: ESRD

- The total annual cost of treating ESRD in the United States was $26.8 billion in 2008, which represents nearly 6% of the total Medicare budget.[1]
- The total annual cost associated with CKD has not been determined accurately to date.

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Cystatin C: Kidney Function and CVD

Serum cystatin C, another marker of kidney function, has been proposed to be a more sensitive indicator of kidney function than serum creatinine and creatinine-based estimating formulas at higher levels of GFR. It is a low-molecular-weight protein produced at a constant rate by all nucleated cells and appears not to be affected significantly across age, sex, and levels of muscle mass. Cystatin C is excreted by the kidneys, filtered through the glomerulus, and nearly completely reabsorbed by proximal tubular cells.[36] Several equations have been proposed using cystatin C alone and in combination with serum creatinine to estimate kidney function.[37,38]

All-Cause Mortality

Elevated levels of cystatin C have been shown to be associated with increased risk for all-cause mortality in studies from a broad range of cohorts.[39–41]

- In addition to GFR and urine albumin-to-creatinine ratio, cystatin C provides incremental information for the prediction of ESRD and mortality.
  - — In a recent analysis of 26 643 US adults, the addition of cystatin C to the combination of creatinine and albumin-to-creatinine ratio resulted in a significant improvement in the prediction of both all-cause mortality and the development of ESRD.[42]

Cardiovascular Disease

- Data from a large national cohort found higher values of cystatin C to be associated with prevalent stroke, angina, and MI,[43] as well as higher BMI.[44]
- Elevated cystatin C was an independent risk factor for HF,[45,46] PAD events,[47] clinical atherosclerosis, and subclinical measures of CVD in older adults,[48] as well as for cardiovascular events among those with CHD.[39,49]
- In several diverse cohorts, elevated cystatin C has been found to be associated with CVD-related mortality,[41,50,51] including sudden cardiac death.[52]
- In a recent clinical trial of 9270 patients with CKD, the effect of lipid-lowering therapy with simvastatin/ezetimibe was associated with a lower risk for major atherosclerotic events compared with placebo.[53]

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Table 12-1. BP and the Adjusted Risk of ESRD Among 316 675 Adults Without Evidence of Baseline Kidney Disease

JNC V BP Category	Adjusted RR (95% CI)
Optimal	1.00 (Reference)
Normal, not optimal	1.62 (1.27–2.07)
High normal	1.98 (1.55–2.52)
Hypertension	
Stage 1	2.59 (2.07–3.25)
Stage 2	3.86 (3.00–4.96)
Stage 3	3.88 (2.82–5.34)
Stage 4	4.25 (2.63–6.86)

BP indicates blood pressure; ESRD, end-stage renal disease; JNC V, fifth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; RR, relative risk; and CI, confidence interval.

Table 12-2. Multivariable Association Between BMI and Risk of ESRD Among 320 252 Adults

BMI, kg/m^2	Adjusted RR (95% CI)
18.5–24.9 (Normal weight)	1.00 (Reference)
25.0–29.9 (Overweight)	1.87 (1.64–2.14)
30.0–34.9 (Class I obesity)	3.57 (3.05–4.18)
35.0–39.9 (Class II obesity)	6.12 (4.97–7.54)
≥40.0 (Extreme obesity)	7.07 (5.37–9.31)

BMI indicates body mass index; ESRD, end-stage renal disease; RR, relative risk; and CI, confidence interval.

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Table 12-3. Adjusted HR (95% CI) for Death of Any Cause, Cardiovascular Events, and Hospitalization Among 1 120 295 Ambulatory Adults, According to eGFR*

eGFR, mL·min^{-1}·1.73 m^{-2}	Death of Any Cause	Any Cardiovascular Event	Any Hospitalization
≥60†	1.00	1.00	1.00
45–59	1.2 (1.1–1.2)	1.4 (1.4–1.5)	1.1 (1.1–1.1)
30–44	1.8 (1.7–1.9)	2.0 (1.9–2.1)	1.5 (1.5–1.5)
15–29	3.2 (3.1–3.4)	2.8 (2.6–2.9)	2.1 (2.0–2.2)
<15	5.9 (5.4–6.5)	3.4 (3.1–3.8)	3.1 (3.0–3.3)

HR indicates hazard ratio; CI, confidence interval; and eGFR, estimated glomerular filtration rate.

*The analyses were adjusted for age, sex, income, education, use or nonuse of dialysis, and presence or absence of prior coronary heart disease, prior chronic heart failure, prior ischemic stroke or transient ischemic attack, prior peripheral arterial disease, diabetes mellitus, hypertension, dyslipidemia, a serum albumin level ≤3.5 g/dL, dementia, cirrhosis or chronic liver disease, chronic lung disease, documented proteinuria, and prior hospitalizations.

†This group served as the reference group.

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13. Total Cardiovascular Diseases

ICD-9 390 to 459, 745 to 747; ICD-10 I00 to I99, Q20 to Q28; see Glossary (Chapter 25) for details and definitions.

See Tables 13-1 through 13-4 and Charts 13-1 through 13-21.

Abbreviations Used in Chapter 13

AHA	American Heart Association
AMI	acute myocardial infarction
AP	angina pectoris
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CABG	cardiac revascularization (coronary artery bypass graft)
CAD	coronary artery disease
CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHF	congestive heart failure
CLRD	chronic lower respiratory disease
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
FHS	Framingham Heart Study
HBP	high blood pressure
HCM	hypertrophic cardiomyopathy
HD	heart disease
HDL	high-density lipoprotein
HF	heart failure
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
LDL	low-density lipoprotein
MEPS	Medical Expenditure Panel Survey
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHIS	National Health Interview Survey
NHHCS	National Home and Hospice Care Survey
NHLBI	National Heart, Lung, and Blood Institute
NNHS	National Nursing Home Survey
PA	physical activity
PCI	percutaneous coronary intervention
RR	relative risk
SBP	systolic blood pressure
UA	unstable angina

Prevalence

(See Table 13-1 and Chart 13-1.)

An estimated 83.6 million American adults (>1 in 3) have 1 or more types of CVD. Of these, 42.2 million are estimated to be ≥60 years of age. Total CVD includes diseases listed in the bullet points below, with the exception of congenital CVD. Because of overlap across conditions, it is not possible to add these conditions to arrive at a total of unique individuals.

- HBP—77.9 million (defined as systolic pressure ≥140 mm Hg and/or diastolic pressure ≥90 mm Hg, use of antihypertensive medication, or being told at least twice by a physician or other health professional that one has HBP).
- CHD—15.4 million
  - —MI (heart attack)—7.6 million
  - —AP (chest pain)—7.8 million
  - —HF—5.1 million
  - —Stroke (all types)—6.8 million
  - —Congenital cardiovascular defects— 650 000 to 1.3 million
- The following age-adjusted prevalence estimates from the NHIS, NCHS are for diagnosed conditions for people ≥18 years of age in 2011[1]:
  - —Among whites only, 11.1% have HD, 6.3% have CHD, 23.3% have hypertension, and 2.3% have had a stroke.
  - —Among blacks or African Americans, 10.7% have HD, 6.9% have CHD, 33.4% have hypertension, and 4.5% have had a stroke.
  - —Among Hispanics or Latinos, 8.6% have HD, 5.9% have CHD, 22.2% have hypertension, and 2.8% have had a stroke.
  - —Among Asians, 7.4% have HD, 4.3% have CHD, 18.7% have hypertension, and 2.7% have had a stroke.
  - —Among American Indians or Alaska Natives, 12.7% have HD, 7.2% have CHD, 25.8% have hypertension, and 4.6% have had a stroke.
  - —Among Native Hawaiians or other Pacific Islanders, 21.8% have hypertension. Statistics for the other conditions for this group are not shown because of unreliability.
- Asian Indian adults (9%) are ≈2-fold more likely than Korean adults (4%) to have ever been told they have HD, based on data for 2004 to 2006.[2]
- By 2030, 40.8% of the US population is projected to have some form of CVD (unpublished AHA computation, based on methodology described in Heidenreich et al[3]).

Incidence

(See Chart 13-2.)

- On the basis of the NHLBI's FHS original and offspring cohort data from 1980 to 2003[4]
  - —The average annual rates of first cardiovascular events rise from 3 per 1000 men at 35 to 44 years of age to 74 per 1000 men at 85 to 94 years of age. For women, comparable rates occur 10 years later in life. The gap narrows with advancing age.
  - —Before 75 years of age, a higher proportion of CVD events attributable to CHD occur in men than in women, and a higher proportion of events attributable to stroke occur in women than in men.

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- Among American Indian men 45 to 74 years of age, the incidence of CVD ranges from 15 to 28 per 1000 population. Among women, it ranges from 9 to 15 per 1000.[5]
- Data from the FHS indicate that the subsequent lifetime risk for all CVD in recipients starting free of known disease is almost 2 in 3 for men and >1 in 2 for women at 45 years of age (Table 13-4).[5a]
- Analysis of FHS data among participants free of CVD at 50 years of age showed the lifetime risk for developing CVD was 51.7% for men and 39.2% for women. Median overall survival was 30 years for men and 36 years for women.[6]

Mortality

(See Tables 13-1 through 13-3 and Charts 13-3 through 13-18.)

ICD-10 I00 to I99, Q20 to Q28 for CVD (CVD mortality includes congenital cardiovascular defects); C00 to C97 for cancer; C33 to C34 for lung cancer; C50 for breast cancer; J40 to J47 for chronic lower respiratory disease (CLRD); G30 for Alzheimer disease; E10 to E14 for DM; and V01 to X59, Y85 to Y86 for accidents.

- Mortality data show that CVD (I00–I99, Q20–Q28) as the listed underlying cause of death (including congenital cardiovascular defects) accounted for 32.3% (787 931) of all 2 437 163 deaths in 2009, or ≈1 of every 3 deaths in the United States. CVD any-mentions (1 330 137 deaths in 2009) constituted 54.6% of all deaths that year (NHLBI; NCHS public use data files).[7]
- In every year since 1900 except 1918, CVD accounted for more deaths than any other major cause of death in the United States.[8,9]
- On average, >2150 Americans die of CVD each day, an average of 1 death every 40 seconds. CVD currently claims more lives each year than cancer and CLRD combined.[7]
- The 2009 death rate attributable to CVD (I00–I99) was 236.1 (excluding congenital cardiovascular defects) (NCHS).[7] The rates were 281.4 for white males, 387.0 for black males, 190.4 for white females, and 267.9 for black females. From 1999 to 2009, death rates attributable to CVD (ICD-10 I00–I99) declined 32.7%. In the same 10-year period, the actual number of CVD deaths per year declined by 17.8% (AHA computation).[7]
- Among other causes of death in 2009, cancer caused 567 628 deaths; CLRD, 137 353; accidents, 118 021; and Alzheimer disease, 79 003.[7]
- The 2009 CVD (I00–I99) death rates were 287.2 for males and 196.1 for females. There were 40 678 deaths attributable to breast cancer in females in 2009; lung cancer claimed 70 389 females. Death rates for females were 22.3 for breast cancer and 38.5 for lung cancer. One in 30 deaths of females was attributable to breast cancer, whereas 1 in 6.9 was attributable to CHD. For comparison, 1 in 4.5 females died of cancer, whereas 1 in 3.0 died of CVD (I0–I99, Q20–Q28). On the basis of 2009 mortality data, CVD caused ≈1 death per minute among females, or 401 495 deaths. That represents approximately the same number of female lives that were claimed by cancer, CLRD, and Alzheimer disease combined (unpublished AHA tabulation).[7]
- Approximately 150 000 Americans died of CVD (I00–I99) in 2009 who were <65 years of age, and 34% of deaths attributed to CVD occurred before the age of 75 years,[7] which is well below the average life expectancy of 78.5 years.[8]
- According to the NCHS, if all forms of major CVD were eliminated, life expectancy could rise by almost 7 years. If all forms of cancer were eliminated, the estimated gain could be 3 years. According to the same study, the probability at birth of eventually dying of major CVD (I00–I78) is 47%, and the chance of dying of cancer is 22%. Additional probabilities are 3% for accidents, 2% for DM (unrelated to CVD), and 0.7% for HIV.[10]
- In 2009, the leading causes of death in women ≥65 years of age were diseases of the heart (No. 1), cancer (No. 2), stroke (No. 3), and CLRD (No. 4). In older men, they were diseases of the heart (No. 1), cancer (No. 2), CLRD (No. 3), and stroke (No. 4).[7]
- A study of the decrease in US deaths attributable to CHD from 1980 to 2000 suggests that ≈47% of the decrease was attributable to increased use of evidence-based medical therapies and 44% to changes in risk factors in the population attributable to lifestyle and environmental changes.[9]
- Analysis of data from NCHS was used to determine the number of disease-specific deaths attributable to all nonoptimal levels of each risk factor exposure, by age and sex. In 2005, tobacco smoking and HBP were estimated to be responsible for 467 000 deaths, accounting for ≈1 in 5 or 6 deaths among US adults. Overweight/obesity and physical inactivity were each estimated to be responsible for nearly 1 in 10 deaths. High dietary salt, low dietary omega-3 fatty acids, and high dietary *trans* fatty acids were the dietary risks with the largest estimated excess mortality effects.[10]

Aftermath

- Among an estimated 45 million people with functional disabilities in the United States, HD, stroke, and hypertension are among the 15 leading conditions that caused those disabilities. Disabilities were defined as difficulty with activities of daily living or instrumental activities of daily living, specific functional limitations (except vision, hearing, or speech), and limitation in ability to do housework or work at a job or business.[11]

Awareness of Warning Signs and Risk Factors for CVD

- Surveys conducted by the AHA in 1997, 2000, 2003, 2006, and 2009 to evaluate trends in women's awareness, knowledge, and perceptions related to CVD found that in 2009, awareness of HD as the leading cause of death among women was 54%, significantly higher than in 1997.[12] Awareness was 66% lower among black and Hispanic women than among white women,[13] and the racial/ethnic difference has not changed appreciably over time. Awareness of HD as the leading cause of death among women has approximately doubled among white and Hispanic women and tripled among black women between the first survey in 1997 and the current 2009 survey. However, after adjustment for age and education level, African American, Hispanic, and Asian women were significantly less likely to be aware that HD/heart attack is the leading cause of death than white women.[12] Awareness of heart attack signs was low for all racial/ethnic and age groups surveyed.[13]

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- A total of 875 students in 4 Michigan high schools were given a survey to obtain data on the perception of risk factors and other knowledge-based assessment questions about CVD. Accidents were rated as the greatest perceived lifetime health risk (39%). Nearly 17% selected CVD as the greatest lifetime risk, which made it the third most popular choice after accidents and cancer. When asked to identify the greatest cause of death for each sex, 42% correctly recognized CVD for men, and 14% correctly recognized CVD for women; 40% incorrectly chose abuse/use behavior with a substance other than cigarettes as the most important CVD risk behavior.[14]

Awareness of Cardiopulmonary Resuscitation

- Seventy-nine percent of the lay public are confident that they know what actions to take in a medical emergency; 98% recognize an automated external defibrillator as something that administers an electric shock to restore a normal heart beat among victims of sudden cardiac arrest; and 60% are familiar with cardiopulmonary resuscitation (Harris Interactive survey conducted on behalf of the AHA among 1132 US residents >18 years of age, January 8, 2008, through January 21, 2008).

Disparities in CVD Risk Factors

(See Chart 13-19.)

- Data from the 2003 CDC BRFSS survey of adults ≥18 years of age showed the prevalence of respondents who reported having ≥2 risk factors for HD and stroke was successively higher at higher age groups. The prevalence of having ≥2 risk factors was highest among blacks (48.7%) and American Indian/Alaska Natives (46.7%) and lowest among Asians (25.9%); prevalence was similar in women (36.4%) and men (37.8%). The prevalence of multiple risk factors ranged from 25.9% among college graduates to 52.5% among those with less than a high school diploma (or its equivalent). People reporting household income of ≥$50 000 had the lowest prevalence (28.8%), and those reporting household income of <$10 000 had the highest prevalence (52.5%). Adults who reported being unable to work had the highest prevalence (69.3%) of ≥2 risk factors, followed by retired people (45.1%), unemployed adults (43.4%), homemakers (34.3%), and employed people (34.0%). Prevalence of ≥2 risk factors varied by state/territory and ranged from 27.0% (Hawaii) to 46.2% (Kentucky). Twelve states and 2 territories had a multiple risk factor prevalence of ≥40%: Alabama, Arkansas, Georgia, Indiana, Kentucky, Louisiana, Mississippi, North Carolina, Ohio, Oklahoma, Tennessee, West Virginia, Guam, and Puerto Rico.[15]
- Analysis of several data sets by the CDC showed that in adults ≥18 years of age, disparities were common in all risk factors examined. In men, the highest prevalence of obesity (29.7%) was found in Mexican Americans who had completed a high school education. Black women with or without a high school education had a high prevalence of obesity (48.4%). Hypertension prevalence was high among blacks (41.2%) regardless of sex or educational status. Hypercholesterolemia was high among white and Mexican American men and white women regardless of educational status. CHD and stroke were inversely related to education, income, and poverty status. Hospitalization for total HD and AMI was greater among men, but hospitalization for CHF and stroke was greater among women. Among Medicare enrollees, CHF hospitalization was higher among blacks, Hispanics, and American Indian/Alaska Natives than among whites, and stroke hospitalization was highest among blacks. Hospitalizations for CHF and stroke were highest in the southeastern United States. Life expectancy remains higher in women than in men and in whites than in blacks by ≈5 years. CVD mortality at all ages tended to be highest in blacks.[16]
- Analysis of >14 000 middle-aged subjects between 1987 and 2002 in the ARIC study sponsored by the NHLBI showed that >90% of CVD events in black subjects, compared with ≈70% in white subjects, appeared to be explained by elevated or borderline risk factors. Furthermore, the prevalence of participants with elevated risk factors was higher in black subjects; after accounting for education and known CVD risk factors, the incidence of CVD was identical in black and white subjects. Thus, the observed higher CVD incidence rate in black subjects appears to be largely attributable to a greater prevalence of elevated risk factors. These results suggest that the primary prevention of elevated risk factors might substantially impact the future incidence of CVD, and these beneficial effects would likely be applicable not only for white but also for black subjects.[17]
- Data from the MEPS 2004 Full-Year Data File showed that nearly 26 million US adults ≥18 years of age were told by a doctor that they had HD, stroke, or any other heart-related disease[18]:
  - —38.6% maintained a healthy weight. Among those told that they had HD, 33.9% had a healthy weight compared with 39.3% who had never been told they had HD.
  - —78.8% did not currently smoke. Among those ever told that they had indicators of HD, 18.3% continued to smoke.
  - —More than 93% engaged in at least 1 recommended behavior for prevention of HD: 75.5% engaged in 1 or 2; 18% engaged in all 3; and 6.5% did not engage in any of the recommended behaviors.
  - —Age-based variations:
    - Moderate to vigorous PA ≥3 times per week varied according to age. Younger people (18–44 years of age) were more likely (59.9%) than those who were older (45–64 and ≥65 years of age, 55.3% and 48.5%, respectively) to engage in regular PA.
    - A greater percentage of those 18 to 44 years of age had a healthy weight (43.7%) than did those 45 to 64 years of age and ≥65 years of age (31.4% and 37.3%, respectively).
    - People ≥65 years of age were more likely to be current nonsmokers (89.7%) than were people 18 to 44 years of age and 45 to 64 years of age (76.1% and 77.7%, respectively).
  - —Race/ethnicity-based variations:
    - Non-Hispanic whites were more likely than Hispanics or non-Hispanic blacks to engage in moderate to vigorous PA (58.5% versus 51.4% and 52.5%, respectively).

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- Non-Hispanic whites were more likely to have maintained a healthy weight than were Hispanics or non-Hispanic blacks (39.8% versus 32.1% and 29.7%, respectively).
- Hispanics were more likely to be nonsmokers (84.2%) than were non-Hispanic whites and non-Hispanic blacks (77.8% and 76.3%, respectively).

—Sex-based variations:

- Men were more likely to have engaged in moderate to vigorous PA ≥3 times per week than women (60.3% versus 53.1%, respectively).
- Women were more likely than men to have maintained a healthy weight (45.1% versus 31.7%, respectively).
- 81.7% of women did not currently smoke, compared with 75.7% of men.

—Variations based on education level:

- A greater percentage of adults with at least some college education engaged in moderate to vigorous PA ≥3 times per week (60.8%) than did those with a high school education or less than a high school education (55.3% and 48.3%, respectively).
- A greater percentage of adults with at least some college education had a healthy weight (41.2%) than did those with a high school or less than high school education (36.2% and 36.1%, respectively).
- There was a greater percentage of nonsmokers among those with a college education (85.5%) than among those with a high school or less than high school education (73.8% and 69.9%, respectively).

- A study of nearly 1500 participants in the MESA study found that Hispanics with hypertension, hypercholesterolemia, and/or DM who speak Spanish at home and/or have spent less than half a year in the United States have higher SBP, LDL cholesterol, and fasting blood glucose, respectively, than Hispanics who speak English and who have lived a longer period of time in the United States.[19]

Family History of CVD

- A family history of CVD increases risk of CVD, with the largest increase in risk if the family member's CVD was premature.[20]
- There is consistent evidence from multiple large-scale prospective epidemiology studies for a strong and significant association of a reported family history of premature parental CHD with incident MI or CHD in offspring. In the FHS, the occurrence of a validated premature atherosclerotic CVD event in either a parent[21] or a sibling[22] was associated with an ≈2-fold elevated risk for CVD, independent of other traditional risk factors.
- Addition of family history of premature CVD to a model that contained traditional risk factors provided modestly improved prognostic value in the FHS.[21] Family history of premature MI is also an independent risk factor in other multivariable risk models that contain traditional risk factors in large cohorts of women[23] and men.[24]
- Parental history of premature CHD is associated with increased burden of subclinical atherosclerosis in the coronary arteries and the abdominal aorta.[25,26]
- In the FHS, a parental history of validated HF was associated with a 1.7-fold higher risk of HF in offspring, after multivariable adjustment.[27]
- A family history of early-onset sudden cardiac death in a first-degree relative is associated with a >2-fold higher risk for sudden cardiac death in offspring on the basis of available case-control studies.[28]
- The 2004 HealthStyles survey of 4345 people in the United States indicated that most respondents believe that knowing their family history is important for their own health, but few are aware of the specific health information from relatives necessary to develop a family history.[29]
- An accurate and complete family history may identify rare mendelian conditions such as hypertrophic cardiomyopathy (HCM), long-QT syndrome, or familial hypercholesterolemia. However, in the majority of people with a family history of a CVD event, a known rare mendelian condition is not identified.
- Studies are under way to determine genetic variants that may help identify individuals at increased risk of CVD.

Outcomes Associated With Healthy Lifestyle and Low Risk Factor Levels

Much of the literature on CVD has focused on factors associated with increasing risk for CVD and on factors associated with poorer outcomes in the presence of CVD; however, in recent years, a number of studies have defined the potential beneficial effects of healthy lifestyle factors and lower CVD risk factor burden on CVD outcomes and longevity. These studies suggest that prevention of risk factor development at younger ages may be the key to "successful aging," and they highlight the need for evaluation of the potential benefits of intensive prevention efforts at younger and middle ages once risk factors develop to increase the likelihood of healthy longevity.

- Data from the Cardiovascular Lifetime Risk Pooling Project, which involved 18 cohort studies and combined data on 257 384 black men and women and white men and women, indicate that at 45 years of age, participants with an optimal risk factor profile had a substantially lower lifetime risk of CVD events than those with 1 major risk factor (1.4% versus 39.6% among men, 4.1% versus 20.2% among women). The presence of ≥2 major risk factors further increased lifetime risk to 49.5% in men and 30.7% in women.[30]
- A recent study examined the association between low lifetime predicted risk for CVD (ie, having all optimal or near-optimal risk factor levels) and burden of subclinical atherosclerosis in younger adults in the CARDIA and MESA studies of the NHLBI. Among participants <50 years of age, nearly half had low and half had high predicted lifetime risks for CVD. Those with low predicted lifetime risk had lower prevalence and less severe amounts of coronary calcification and less carotid intima-media thickening, even at these younger ages, than those with high predicted lifetime risk. During follow-up, those with low predicted lifetime risk also had less progression of coronary calcium.[31]
- Among >7900 men and women from the FHS followed up for 111 000 person-years, median survival was highly associated with risk factor presence and burden at 50 years of age. Men and women with optimal risk factors had a

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median life expectancy ≥10 years longer than those with ≥2 major risk factors at age 50 years.[6]

- In another study, FHS investigators followed up 2531 men and women who were examined between the ages of 40 and 50 years and observed their overall rates of survival and survival free of CVD to 85 years of age and beyond. Low levels of the major risk factors in middle age were associated with overall survival and morbidity-free survival to ≥85 years of age.[32]
  - —Overall, 35.7% survived to the age of 85 years, and 22% survived to that age free of major morbidities.
  - —Factors associated with survival to the age of 85 years included female sex, lower SBP, lower total cholesterol, better glucose tolerance, absence of current smoking, and higher level of education attained. Factors associated with survival to the age of 85 years free of MI, UA, HF, stroke, dementia, and cancer were nearly identical.
  - —When adverse levels of 4 of these factors were present in middle age, <5% of men and ≈15% of women survived to 85 years of age.
- Data from the Chicago Heart Association Detection Project (1967–1973, with an average follow-up of 31 years) showed the following:
  - —In younger women (18–39 years of age) with favorable levels for all 5 major risk factors (BP, serum cholesterol, BMI, DM, and smoking), future incidence of CHD and CVD is rare, and long-term and all-cause mortality are much lower than for those who have unfavorable or elevated risk factor levels at young ages. Similar findings applied to men in this study.[33]
  - —Participants (18–64 years of age at baseline) without a history of MI were investigated to determine whether traditional CVD risk factors were similarly associated with CVD mortality in black and white men and women. In general, the magnitude and direction of associations were similar by race. Most traditional risk factors demonstrated similar associations with mortality in black and white adults of the same sex. Small differences were primarily in the strength and not the direction of the association.[34]
  - —Remaining lifetime risks for CVD death were noted to increase substantially and in a graded fashion according to the number of risk factors present in middle age (40–59 years of age). However, remaining lifetime risks for non-CVD death also increased dramatically with increasing CVD risk factor burden. These data help to explain the markedly greater longevity experienced by those who reach middle age free of major CVD risk factors.[35]
  - —Presence of a greater number of risk factors in middle age is associated with lower scores at older ages on assessment of social functioning, mental health, walking, and health perception in women, with similar findings in men.[36]
  - —Risk factor burden in middle age is associated with better quality of life at follow-up in older age (≈25 years later) and lower average annual Medicare costs at older ages.[36,37] Similarly, the existence of a greater number of risk factors in middle age is associated with higher average annual CVD-related and total Medicare costs (once Medicare eligibility is attained).[37]
- A study of 84 129 women enrolled in the Nurses' Health Study identified 5 healthy lifestyle factors, including absence of current smoking, drinking half a glass or more of wine per day (or equivalent alcohol consumption), half an hour or more per day of moderate or vigorous PA, BMI <25 kg/m^2, and dietary score in the top 40% (which included diets with lower amounts of *trans* fats, lower glycemic load, higher cereal fiber, higher marine omega-3 fatty acids, higher folate, and higher polyunsaturated to saturated fat ratio). When 3 of the 5 healthy lifestyle factors were present, the RR for CHD over a 14-year period was 57% lower; when 4 were present, RR was 66% lower; and when all 5 factors were present, RR was 83% lower.[38] However, data from NHANES 1999–2002 showed that only approximately one third of adults complied with ≥6 of the recommended heart-healthy behaviors. Dietary recommendations, in general, and daily fruit intake recommendations, in particular, were least likely to be followed.[39]
- Among individuals 70 to 90 years of age, adherence to a Mediterranean-style diet and greater PA are associated with 65% to 73% relatively lower rates of all-cause mortality, as well as lower mortality rates attributable to CHD, CVD, and cancer.[40]
- Seventeen-year mortality data from the NHANES II Mortality Follow-Up Study indicated that the RR for fatal CHD was 51% lower for men and 71% lower for women with none of 3 major risk factors (hypertension, current smoking, and elevated total cholesterol [≥240 mg/dL]) than for those with ≥1 risk factor. Had all 3 major risk factors not occurred, it is hypothesized that 64% of all CHD deaths among women and 45% of CHD deaths in men could theoretically have been avoided.[41]

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Hospital Discharges, Ambulatory Care Visits, Home Healthcare Patients, Nursing Home Residents, and Hospice Care Discharges

(See Table 13-1 and Charts 13-20 through 13-21.)

- From 2000 to 2010, the number of inpatient discharges from short-stay hospitals with CVD as the first-listed diagnosis decreased from 6 294 000 to 5 802 000 (NHDS, NHLBI tabulation). In 2010, CVD ranked highest among all disease categories in hospital discharges (NHDS, NHLBI tabulation).
- In 2010, there were 94 871 000 physician office visits with a primary diagnosis of CVD (NCHS, NAMCS, NHLBI tabulation). In 2009, there were 4 761 000 ED visits and 7 261 000 hospital outpatient department visits with a primary diagnosis of CVD (NHAMCS, NHLBI tabulation).
- In 2009, ≈1 of every 6 hospital stays, or 6 million, resulted from CVD (Agency for Healthcare Research and Quality, Nationwide Inpatient Sample). The total inpatient hospital cost for CVD was $71.2 billion, approximately one fourth of the total cost of inpatient hospital care in the United States. The average cost per hospitalization was ≈41% higher than the average cost for all stays. Hospital admissions that originated in the ED accounted for 60.7% of all hospital stays for CVD. This was 41% higher than the rate of 43.1% for all types of hospital stays; 3.3% of patients admitted to the hospital for CVD died in the hospital, which

was significantly higher than the average in-hospital death rate of 2.1% for all hospitalized patients.[42]

- In 2004, CAD was estimated to be responsible for 1.2 million hospital stays and was the most expensive condition treated. This condition resulted in >$44 billion in expenses. More than half of the hospital stays for CAD were among patients who also received PCI or coronary artery bypass graft (CABG) during their stay. AMI resulted in $31 billion of inpatient hospital charges for 695 000 hospital stays. The 1.1 million hospitalizations for CHF amounted to nearly $29 billion in hospital charges.[43]
- In 2003, ≈48.3% of inpatient hospital stays for CVD were for women, who accounted for 42.8% of the national cost ($187 billion) associated with these conditions. Although only 40% of hospital stays for AMI and CAD were for women, more than half of all stays for nonspecific chest pain, CHF, and stroke were for women. There was no difference between men and women in hospitalizations for cardiac dysrhythmias.[44]
- Circulatory disorders were the most frequent reason for admission to the hospital through the ED, accounting for 26.3% of all admissions through the ED. After pneumonia, the most common heart-related conditions (in descending order) were CHF, chest pain, hardening of the arteries, and heart attack, which together accounted for >15% of all admissions through the ED. Stroke and irregular heartbeat ranked seventh and eighth, respectively.[45]
- Among the 1.5 million nursing home residents each day in 2004, CVD was the leading primary diagnosis; approximately one fourth of nursing home residents had a primary diagnosis of CVD at admission (23.7% or 353 100 residents) or at the time of interview (25% or 373 000 residents) (NCHS, NNHS).[46]
- Among the 1.5 million home healthcare patients each day in 2007, CVD was the leading primary diagnosis; almost one fifth of home healthcare patients had a primary diagnosis of CVD at admission (18.3% or 267 300 residents) or at the time of interview (18.9% or 275 700 residents) (NCHS, NHHCS). The majority (62.9% or 918 900 patients) of home healthcare patients each day in 2007 had diagnosis of some type of CVD at the time of interview.[47]
- Among the 1.0 million patients discharged from hospice in 2007, CVD was the primary diagnosis for 15.8% (or 165 100 discharges) at admission and 15.9% (or 165 700 discharges) at discharge. Half (50% or 523 000) of all hospice discharges had a diagnosis of some type of CVD at the time of discharge.[47]

Operations and Procedures

- In 2010, an estimated 7 588 000 inpatient cardiovascular operations and procedures were performed in the United States; 4.4 million were performed on males, and 3.2 million were performed on females (NHDS, NHLBI tabulation).

Cost

- The estimated direct and indirect cost of CVD for 2009 is $312.6 billion (MEPS, NHLBI tabulation).
- By 2030, real (2010$) total direct medical costs of CVD are projected to increase to ≈$1.48 trillion (AHA computation, based on methodology described by Heidenreich et al[3]).

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Table 13-1. Cardiovascular Diseases

Population Group	Prevalence, 2010: Age ≥20 y	Mortality, 2009: All Ages*	Hospital Discharges, 2010: All Ages	Cost, 2009
Both sexes	83 600 000 (35.3%)	787 931	5 802 000	$312.6 Billion
Males	40 700 000 (36.7%)	386 436 (49.0%)†	3 021 000	
Females	42 900 000 (34.0%)	401 495 (51.0%)†	2 781 000	…
NH white males	36.6%	329 565	…	…
NH white females	32.4%	343 955	…	…
NH black males	44.4%	46 334	…	…
NH black females	48.9%	48 070	…	…
Mexican American males	33.4%	…	…	…
Mexican American females	30.7%	…	…	…
Asian	…	16 419‡	…	…
American Indian/Alaska Native	…	3588	…	…

Ellipses (…) indicate data not available; NH, non-Hispanic.

*Mortality data are for whites and blacks and include Hispanics.

†These percentages represent the portion of total cardiovascular disease mortality that is attributable to males vs females.

‡Includes Chinese, Filipino, Hawaiian, Japanese, and other Asian or Pacific Islander.

Sources: Prevalence: National Health and Nutrition Examination Survey 2007–2010, National Center for Health Statistics (NCHS) and National Heart, Lung, and Blood Institute (NHLBI). Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 y of age. Age-specific percentages are extrapolated to the 2010 US population estimates. Mortality: Centers for Disease Control and Prevention/NCHS, 2009 Mortality Multiple Cause–US, version July 19, 2012. These data represent underlying cause of death only. Data include congenital cardiovascular disease mortality. Hospital discharges: National Hospital Discharge Survey, NCHS. Data include those inpatients discharged alive, dead, or of unknown status. Cost: NHLBI. Data include estimated direct and indirect costs for 2009.

Table 13-2. Age-Adjusted Death Rates per 100 000 Population for CVD, CHD, and Stroke by State, 2007–2009

	CVD*			CHD†			Stroke‡		
State	Rank§	Death Rate	% Change, 1999–2001 to 2007–2009	Rank§	Death Rate	% Change, 1999–2001 to 2007–2009	Rank§	Death Rate	% Change, 1999–2001 to 2007–2009
Alabama	51	308.3	−20.9	24	108.5	−33.7	52	54.0	−22.4
Alaska	7	203.7	−28.6	6	87.2	−31.8	33	42.5	−34.2
Arizona	4	193.8	−32.3	21	106.3	−34.4	2	30.6	−41.3
Arkansas	47	293.0	−23.5	46	148.6	−20.5	51	53.4	−30.6
California	23	229.7	−30.0	31	119.5	−37.1	23	40.0	−36.7
Colorado	6	195.7	−29.1	5	87.0	−32.0	13	37.0	−34.5
Connecticut	18	218.8	−27.0	12	99.9	−37.5	5	33.7	−33.0
Delaware	27	237.6	−28.9	35	124.1	−37.2	19	39.1	−24.8
District of Columbia	45	284.2	−24.9	51	159.3	−21.2	10	35.6	−25.8
Florida	9	205.6	−33.5	26	110.8	−41.2	3	32.3	−34.3
Georgia	39	263.3	−30.0	9	92.7	−42.5	43	47.3	−33.8
Hawaii	5	195.3	−27.9	3	77.5	−31.8	21	39.4	−36.1
Idaho	14	213.9	−28.1	11	96.8	−34.2	27	41.5	−35.8
Illinois	32	247.8	−28.8	32	120.5	−36.9	30	41.8	−31.9
Indiana	40	263.4	−27.4	34	123.1	−34.1	38	44.8	−34.2
Iowa	28	237.9	−24.9	40	134.0	−28.0	32	42.0	−29.6
Kansas	30	239.7	−25.1	14	101.5	−34.2	39	45.6	−25.0
Kentucky	44	281.2	−28.3	43	135.7	−32.2	40	46.3	−31.4
Louisiana	48	294.6	−22.4	37	127.1	−32.5	45	47.6	−27.3
Maine	21	219.9	−29.0	18	103.7	−36.6	22	39.7	−31.1
Maryland	36	255.8	−25.0	41	134.6	−28.9	28	41.5	−33.2
Massachusetts	13	212.2	−26.8	16	102.0	−30.3	8	34.7	−30.7
Michigan	43	278.6	−24.9	45	146.6	−30.5	34	42.6	−30.5

(*Continued*)

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Table 13-2. Continued

State	CVD*			CHD†			Stroke‡		
	Rank§	Death Rate	% Change, 1999–2001 to 2007–2009	Rank§	Death Rate	% Change, 1999–2001 to 2007–2009	Rank§	Death Rate	% Change, 1999–2001 to 2007–2009
Minnesota	1	179.5	–31.0	2	72.8	–39.3	12	36.7	–34.5
Mississippi	52	331.1	–23.8	39	133.5	–35.8	47	50.6	–29.0
Missouri	42	275.1	–26.2	44	143.0	–30.3	42	46.6	–27.3
Montana	16	217.3	–24.2	10	93.0	–24.9	20	39.2	–34.7
Nebraska	20	219.8	–27.3	7	88.0	–33.8	26	41.2	–28.6
Nevada	34	252.8	–26.1	15	101.8	–37.7	17	37.8	–33.6
New Hampshire	10	211.4	–32.4	22	106.9	–41.1	6	33.7	–39.1
New Jersey	26	237.0	–28.3	38	127.6	–35.5	7	33.9	–27.7
New Mexico	8	204.2	–26.4	19	103.8	–31.8	11	36.2	–28.9
New York	38	262.3	–25.8	52	166.3	–30.0	1	27.5	–32.1
North Carolina	33	251.9	–29.4	29	117.6	–35.6	46	49.0	–35.4
North Dakota	15	216.6	–28.6	28	116.8	–29.5	15	37.3	–37.5
Ohio	41	265.0	–27.1	42	135.5	–32.9	37	44.0	–27.0
Oklahoma	50	306.0	–24.8	50	157.1	–31.6	50	51.3	–24.8
Oregon	12	212.0	–28.3	8	89.1	–33.6	36	43.7	–40.3
Pennsylvania	35	254.8	–26.9	36	126.2	–33.8	29	41.6	–27.4
Puerto Rico	2	184.9	–28.6	4	85.0	–31.8	16	37.6	–27.4
Rhode Island	29	239.4	–23.4	48	150.6	–28.1	4	33.2	–30.4
South Carolina	37	255.8	–30.2	23	108.2	–38.5	48	50.7	–36.5
South Dakota	17	217.3	–28.2	33	120.8	–27.5	18	38.4	–33.8
Tennessee	46	286.9	–26.7	49	156.2	–28.7	49	50.7	–33.0
Texas	31	247.1	–29.5	30	118.4	–38.6	41	46.4	–29.1
Utah	3	193.6	–28.5	1	70.4	–37.6	14	37.2	–38.8
Vermont	11	211.4	–29.0	27	112.1	–31.4	9	35.1	–34.5
Virginia	25	236.5	–28.5	17	103.1	–34.5	35	43.4	–35.3
Washington	19	219.5	–26.8	25	109.6	–30.5	25	40.5	–41.0
West Virginia	49	295.8	–25.5	47	149.9	–31.8	44	47.4	–22.7
Wisconsin	22	226.1	–29.2	20	105.9	–35.0	24	40.1	–37.1
Wyoming	24	230.6	–22.3	13	100.6	–30.9	31	41.8	–28.3
Total United States		243.9	–28.2		121.6	–34.6		40.6	–32.3

CVD indicates cardiovascular disease; CHD, coronary heart disease.

*CVD is defined here as *International Classification of Diseases, 10th Revision* (*ICD–10*) codes I00 to I99.

†CHD is defined here as *ICD-10* codes I20 to I25.

‡Stroke is defined here as *ICD-10* codes I60 to I69.

§Rank is lowest to highest.

Source: Centers for Disease Control and Prevention (CDC) WONDER, 2007–2009. Data provided by personal communication with the National Heart, Lung, and Blood Institute. The Agency for Healthcare Research and Quality has released state-level data for heart disease for all 50 states and the District of Columbia; the data are taken from the congressionally mandated National Healthcare Quality Report.[49] In addition, the *Women's Health and Mortality Chartbook* of the National Center for Health Statistics has state-related data for women.[50] Metropolitan/micropolitan area risk data are available for 500 such areas nationwide.[51] Behavioral Risk Factor Surveillance System data are also collected within each state.[52] The CDC has the Geographic Information Systems, which provides mortality rates down to the county level, by sex and ethnicity.[53] The 2008 *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries* is a new resource that provides data down to the county level, by sex and race.[54]

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Table 13-3. International Death Rates (Revised February 2012): Death Rates (Per 100 000 Population) for Total CVD, CHD, Stroke, and Total Deaths in Selected Countries (Most Recent Year Available)

	CVD Deaths	CHD Deaths	Stroke Deaths	Total Deaths
Men ages 35–74 y				
Russian Federation (2009)	1185.4	658.5	307.5	2438.3
Bulgaria (2008)	803.7	219.4	218.2	1554.3
Lithuania (2009)	734.7	444.6	138.3	1842.3
Romania (2010)	657.9	268.2	195.9	1548.4
Hungary (2009)	605.6	319.1	121.1	1652.3
Slovakia (2009)	553.2	318.3	117.1	1390.5
Poland (2009)	492.7	166.1	95.4	1389.7
Croatia (2009)	419.3	202.2	113.6	1184.7
Czech Republic (2009)	386.6	198.6	64.4	1080.8
Kuwait (2009)	319.6	187.0	62.1	563.9
Finland (2009)	284.4	170.0	43.8	833.2
United States (2009)	249.8	143.1	29.5	847.5
Greece (2009)	251.6	136.7	50.8	721.6
Germany (2010)	211.5	105.1	29.3	748.8
Ireland (2009)	210.0	140.6	29.2	701.3
Belgium (2005)	209.6	99.5	35.9	821.7
Denmark (2006)	206.6	84.8	45.6	865.6
New Zealand (2007)	204.2	135.6	29.1	635.7
United Kingdom (2009)	202.0	125.8	29.9	687.6
Canada (2004)	198.3	130.8	24.2	705.3
Austria (2010)	189.5	114.2	23.3	724.9
Portugal (2009)	168.7	61.3	62.1	825.3
Spain (2008)	168.2	77.6	33.7	714.0
Sweden (2010)	165.2	93.1	26.3	556.1
Italy (2008)	157.7	74.6	30.4	613.0
Norway (2009)	154.4	84.6	29.0	607.0
Switzerland (2007)	150.4	78.2	16.6	587.5
Netherlands (2010)	147.6	57.1	25.1	619.5
Japan (2009)	145.2	46.5	52.2	605.0
Australia (2006)	141.3	88.9	22.0	553.4
France (2008)	140.1	54.0	25.7	765.8
Korea, South (2009)	138.4	41.0	65.9	783.6
Israel (2008)	133.1	71.7	24.2	621.2
Women ages 35–74 y				
Russian Federation (2009)	463.0	221.2	157.5	918.5
Bulgaria (2008)	368.6	70.9	120.6	699.3
Romania (2010)	312.2	105.7	111.4	692.4
Lithuania (2009)	253.9	127.5	73.8	648.6
Kuwait (2009)	246.1	94.8	56.1	568.1
Hungary (2009)	239.2	113.7	56.0	719.4
Slovakia (2009)	228.5	120.7	57.5	590.7
Croatia (2009)	190.8	71.9	68.7	520.1
Poland (2009)	180.6	48.0	48.1	564.5
Czech Republic (2009)	164.3	69.9	34.8	506.6
United States (2009)	123.7	55.5	22.3	535.6
Denmark (2006)	100.0	32.4	32.1	557.8
Greece (2009)	97.1	33.3	29.3	319.0
Belgium (2005)	94.4	30.8	24.8	436.3

(*Continued*)

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Table 13-3. Continued

	CVD Deaths	CHD Deaths	Stroke Deaths	Total Deaths
New Zealand (2007)	89.8	43.9	21.7	418.2
United Kingdom (2009)	88.1	38.5	22.5	438.5
Ireland (2009)	86.8	40.9	21.9	419.8
Germany (2010)	84.1	30.4	17.7	392.0
Finland (2009)	83.4	36.1	23.0	377.8
Canada (2004)	83.1	42.8	17.3	432.7
Portugal (2009)	76.5	20.0	33.5	377.6
Austria (2010)	74.9	32.7	17.9	362.3
Netherlands (2010)	71.0	20.5	18.9	408.6
Sweden (2010)	67.8	31.1	16.2	357.1
Italy (2008)	64.0	20.4	18.5	320.7
Korea, South (2009)	63.5	41.0	33.2	312.3
Spain (2008)	62.4	18.7	17.8	304.4
Israel (2008)	61.5	23.1	14.4	376.0
Norway (2009)	60.5	26.3	15.2	377.0
Australia (2006)	60.4	26.8	16.3	327.5
Japan (2009)	54.4	12.8	22.7	266.9
Switzerland (2007)	54.1	19.4	12.4	327.6
France (2008)	51.4	11.9	14.2	349.9

CVD indicates cardiovascular disease; CHD, coronary heart disease.

Rates are adjusted to the European Standard population. *International Classification of Diseases, 10th Revision* (*ICD-10*) codes are used for all countries except Greece, for which *International Classification of Diseases, 9th Revision* (*ICD-9*) codes are used. For countries using ICD-9, the ICD-9 codes are 390 to 459 for CVD, 410 to 414 for CHD, and 430 to 438 for stroke. ICD-10 codes are I00 to I99 for CVD, I20 to I25 for CHD, and I60 to I69 for stroke. The following countries have been dropped from the table because data on number of deaths or population are no longer furnished to the World Health Organization: Argentina, China, Colombia, and Mexico.

Sources: The World Health Organization, National Center for Health Statistics, and National Heart, Lung, and Blood Institute.

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Table 13-4. Remaining Lifetime Risks for CVD and Other Diseases Among Men and Women Free of Disease at 40 and 70 Years of Age

	Remaining Lifetime Risk at Age 40 y		Remaining Lifetime Risk at Age 70 y	
Diseases	Men	Women	Men	Women
Any CVD[5a]	2 in 3*	1 in 2*	2 in 3†	1 in 2
CHD[6]	1 in 2	1 in 3	1 in 3	1 in 4
AF[17]	1 in 4	1 in 4	1 in 4	1 in 4
CHF[18]	1 in 5	1 in 5	1 in 5	1 in 5
Stroke[34]	1 in 6‡	1 in 5‡	1 in 6	1 in 5
Dementia[34]	…	…	1 in 7	1 in 5
Hip fracture[32]	1 in 20	1 in 6	…	…
Breast cancer[38,40]	1 in 1000	1 in 8	…	1 in 14
Prostate cancer[38]	1 in 6	…	…	…
Lung cancer[38]	1 in 12	1 in 17	…	…
Colon cancer[38]	1 in 16	1 in 17	…	…
DM[41]	1 in 3	1 in 3	1 in 9	1 in 7
Hypertension[36]	9 in 10‡	9 in 10‡	9 in 10†	9 in 10†
Obesity[37]	1 in 3	1 in 3	…	…

CVD indicates cardiovascular disease; ellipses (…), not estimated; CHD, coronary heart disease; AF, atrial fibrillation; CHF, congestive heart failure; DM, diabetes mellitus.

*Age 45.

†Age 65 y.

‡Age 55 y.

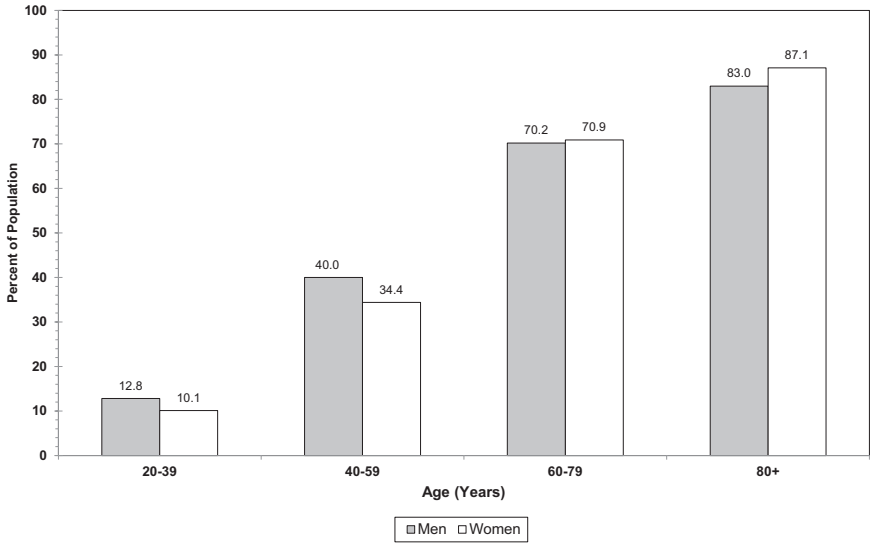

Chart 13-1. Prevalence of cardiovascular disease in adults ≥20 years of age by age and sex (National Health and Nutrition Examination Survey: 2007–2010). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute. These data include coronary heart disease, heart failure, stroke, and hypertension.

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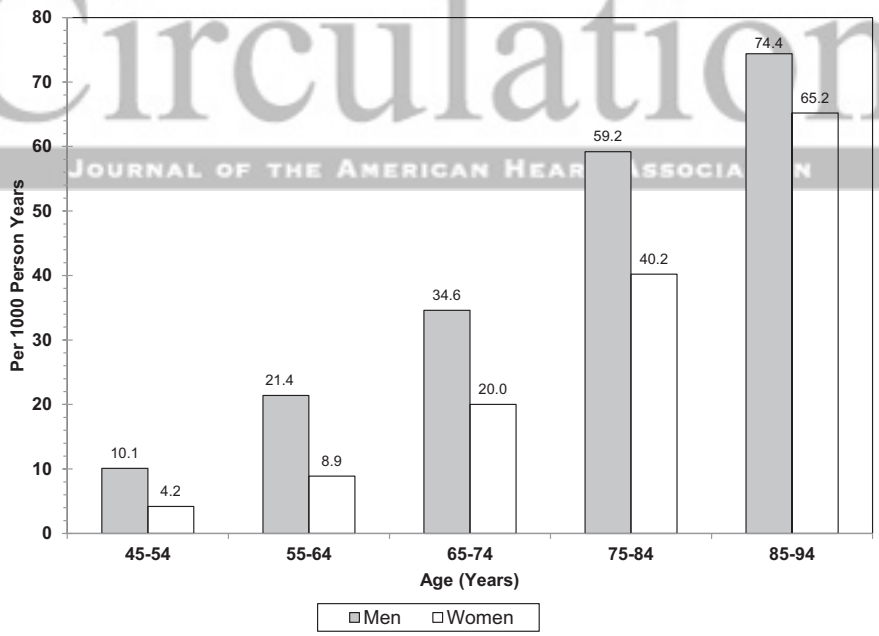

Chart 13-2. Incidence of cardiovascular disease* by age and sex (Framingham Heart Study, 1980–2003). *Coronary heart disease, heart failure, stroke, or intermittent claudication. Does not include hypertension alone. Source: National Heart, Lung, and Blood Institute.[4]

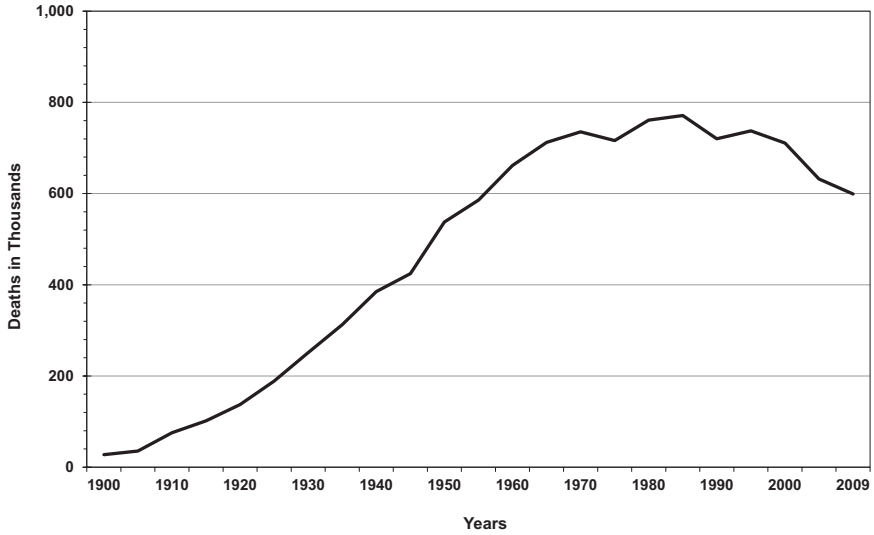

Chart 13-3. Deaths attributable to diseases of the heart (United States: 1900–2009). See Glossary (Chapter 25) for an explanation of "diseases of the heart." Note: In the years 1900–1920, the *International Classification of Diseases* codes were 77 to 80; for 1925, 87 to 90; for 1930–1945, 90 to 95; for 1950–1960, 402 to 404 and 410 to 443; for 1965, 402 to 404 and 410 to 443; for 1970–1975, 390 to 398 and 404 to 429; for 1980–1995, 390 to 398, 402, and 404 to 429; for 2000–2009, I00 to I09, I11, I13, and I20 to I51. Before 1933, data are for a death registration area and not the entire United States. In 1900, only 10 states were in the death registration area, and this increased over the years, so part of the increase in numbers of deaths is attributable to an increase in the number of states. Source: National Center for Health Statistics.

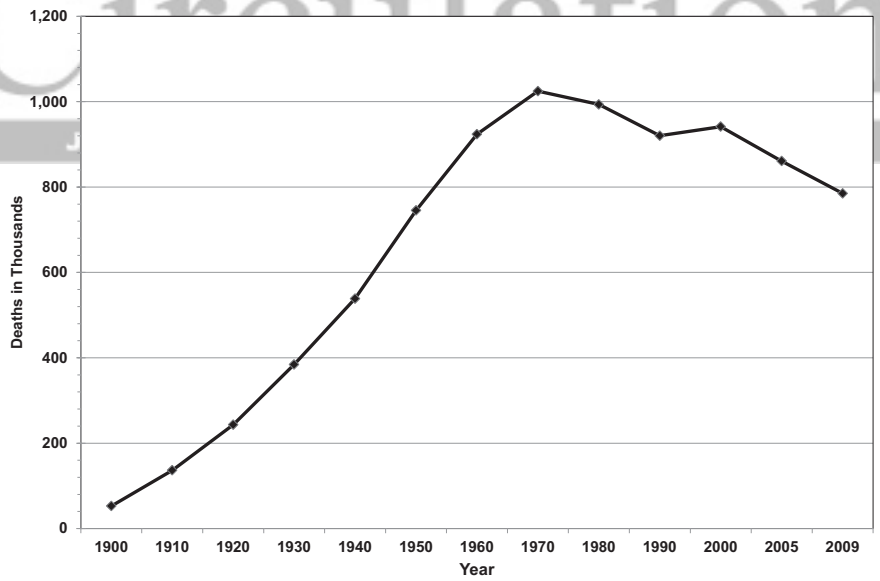

Chart 13-4. Deaths attributable to cardiovascular disease (United States: 1900–2009). Cardiovascular disease (*International Classification of Diseases, 10th Revision* codes I00–I99) does not include congenital. Before 1933, data are for a death registration area and not the entire United States. Source: National Center for Health Statistics.

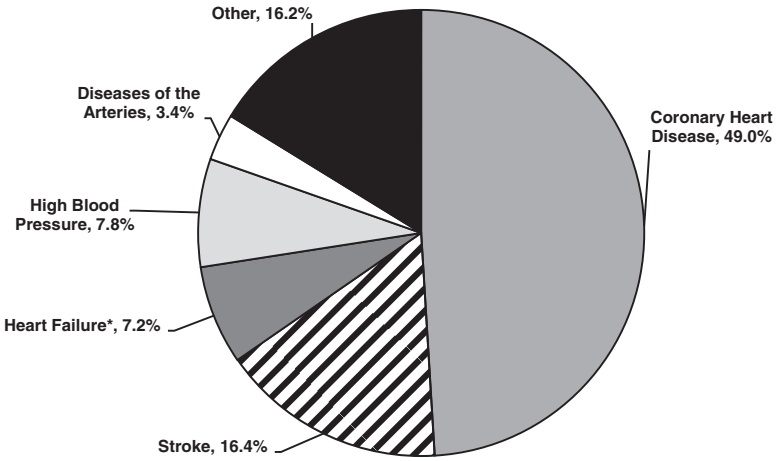

Chart 13-5. Percentage breakdown of deaths attributable to cardiovascular disease (United States: 2009). Source: National Heart, Lung, and Blood Institute from National Center for Health Statistics reports and data sets. *Not a true underlying cause. With any-mention deaths, heart failure accounts for 35% of cardiovascular disease deaths. Total may not add to 100 because of rounding. Coronary heart disease includes *International Classification of Diseases, 10th Revision* (*ICD-10*) codes I20 to I25; stroke, I60 to I69; heart failure, I50; high blood pressure, I10 to I15; diseases of the arteries, I70 to I78; and Other, all remaining *ICD-10* I categories.

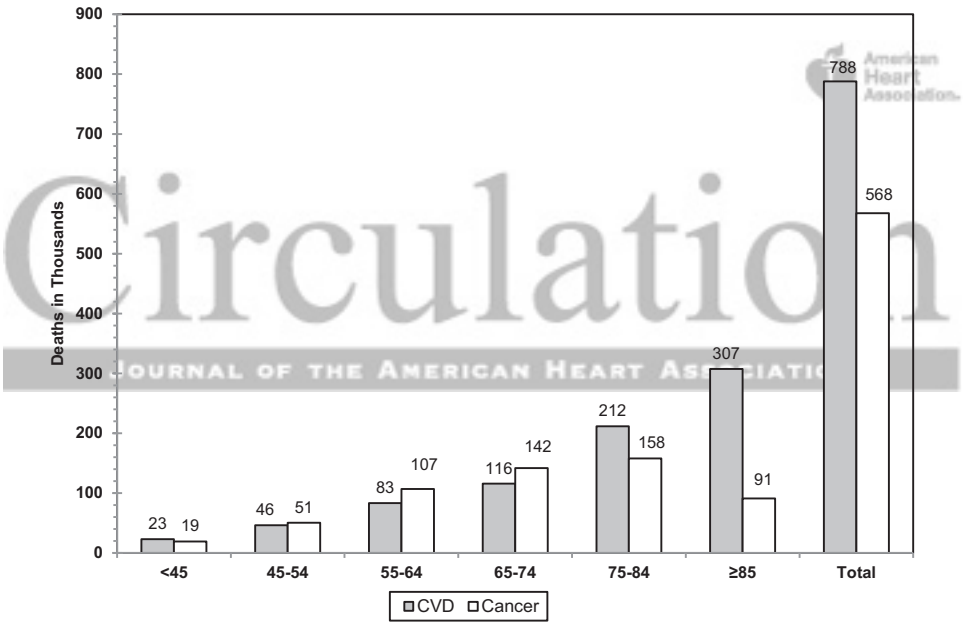

Chart 13-6. Cardiovascular disease (CVD) deaths vs cancer deaths by age (United States: 2009). Source: National Center for Health Statistics. CVD includes *International Classification of Diseases, 10th Revision* codes I00 to I99 and Q20 to Q28; cancer, C00 to C97.

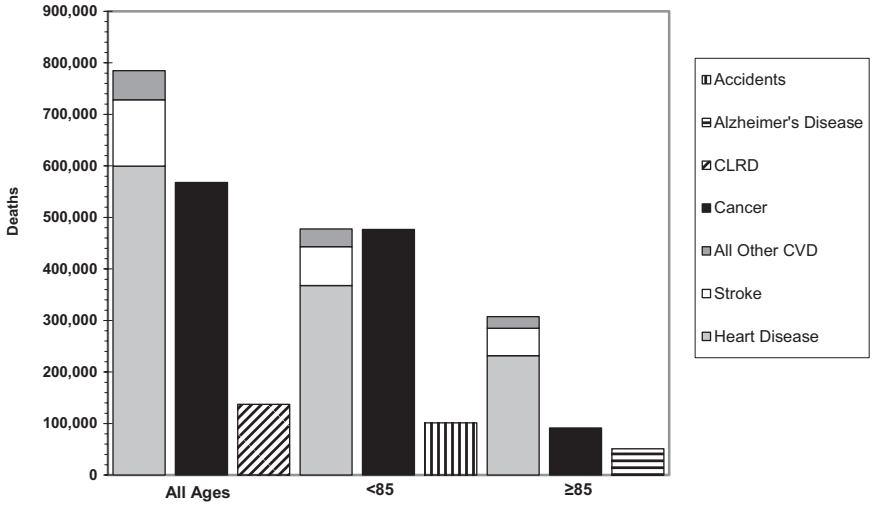

Chart 13-7. Cardiovascular disease (CVD) and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among both sexes, United States, 2009. CLRD indicates chronic lower respiratory disease. Heart disease includes *International Classification of Diseases, 10th Revision* codes I00 to I09, I11, I13, and I20 to I51; stroke, I60 to I69; all other CVD, I10, I12, I15, and I70 to I99; cancer, C00 to C97; CLRD, J40 to J47; Alzheimer disease, G30; and accidents, V01 to X59 and Y85 to Y86. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

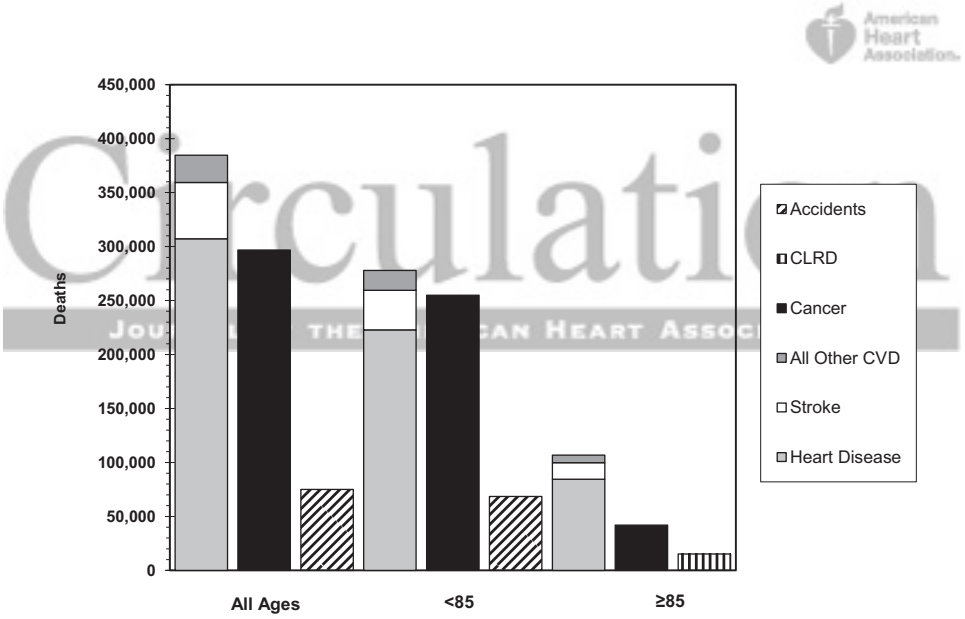

Chart 13-8. Cardiovascular disease (CVD) and other major causes of death in males: total, <85 years of age, and ≥85 years of age. Deaths among males, United States, 2009. CLRD indicates chronic lower respiratory disease. Heart disease includes *International Classification of Diseases, 10th Revision* codes I00 to I09, I11, I13, and I20 to I51; stroke, I60 to I69; all other CVD, I10, I12, I15, and I70 to I99; cancer, C00 to C97; CLRD, J40 to J47; and accidents, V01 to X59 and Y85 to Y86. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

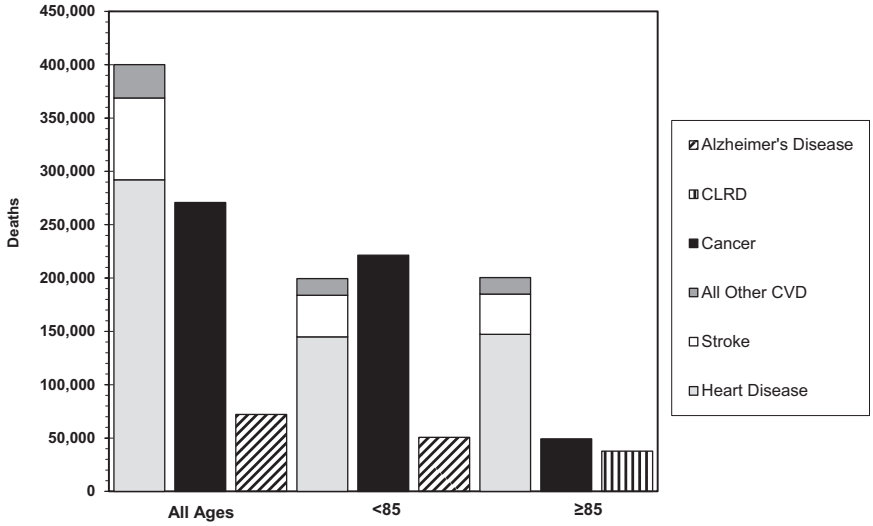

Chart 13-9. Cardiovascular disease (CVD) and other major causes of death in females: total, <85 years of age, and ≥85 years of age. Deaths among females, United States, 2009. CLRD indicates chronic lower respiratory disease. Heart disease includes *International Classification of Diseases, 10th Revision* codes I00 to I09, I11, I13, and I20 to I51; stroke, I60 to I69; all other CVD, I10, I12, I15, and I70 to I99; cancer, C00 to C97; CLRD, J40 to J47; and Alzheimer disease, G30. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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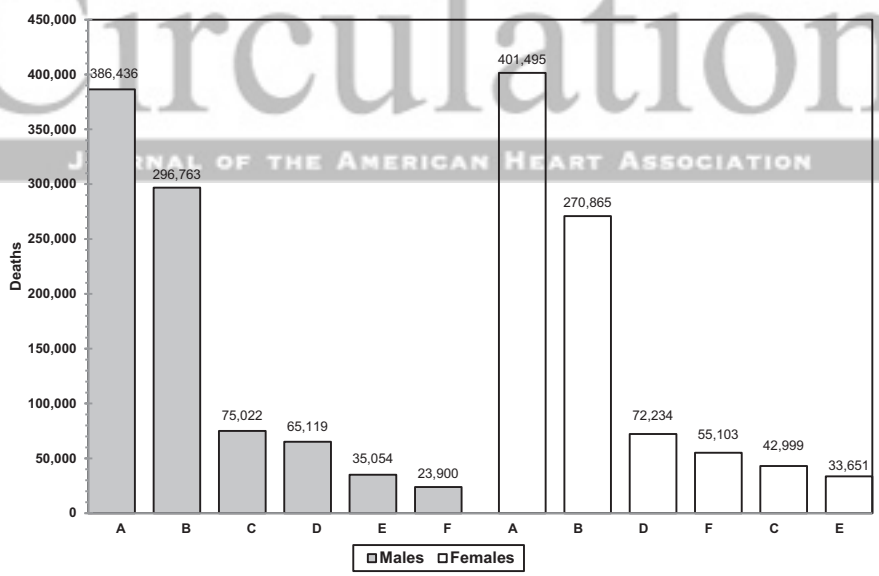

Chart 13-10. Cardiovascular disease and other major causes of death for all males and females (United States: 2009). A indicates cardiovascular disease plus congenital cardiovascular disease (*International Classification of Diseases, 10th Revision* codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, chronic lower respiratory disease (J40–J47); E, diabetes mellitus (E10–E14); and F, Alzheimer disease (G30). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

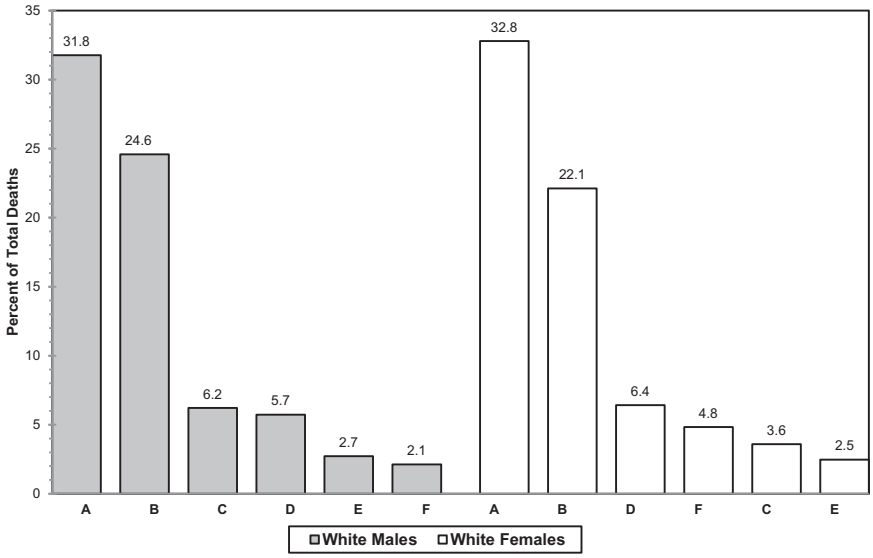

Chart 13-11. Cardiovascular disease and other major causes of death for white males and females (United States: 2009). A indicates cardiovascular disease plus congenital cardiovascular disease (*International Classification of Diseases, 10th Revision* codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, chronic lower respiratory disease (J40–J47); E, diabetes mellitus (E10–E14); and F, Alzheimer disease (G30). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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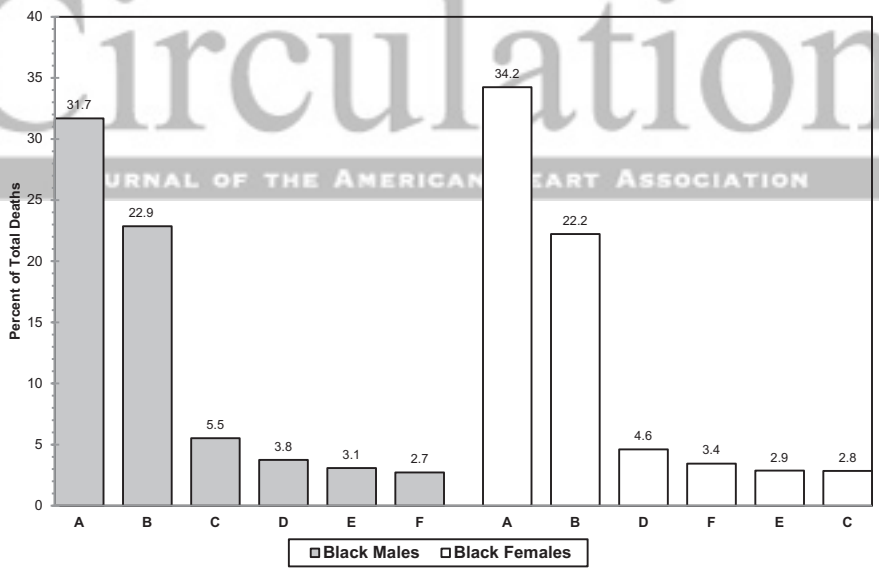

Chart 13-12. Cardiovascular disease and other major causes of death for black males and females (United States: 2009). A indicates cardiovascular disease plus congenital cardiovascular disease (*International Classification of Diseases, 10th Revision* codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, diabetes mellitus (E10–E14); E, chronic lower respiratory disease (J40–J47); and F, nephritis (N00–N07, N17–N19, N25–N27). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

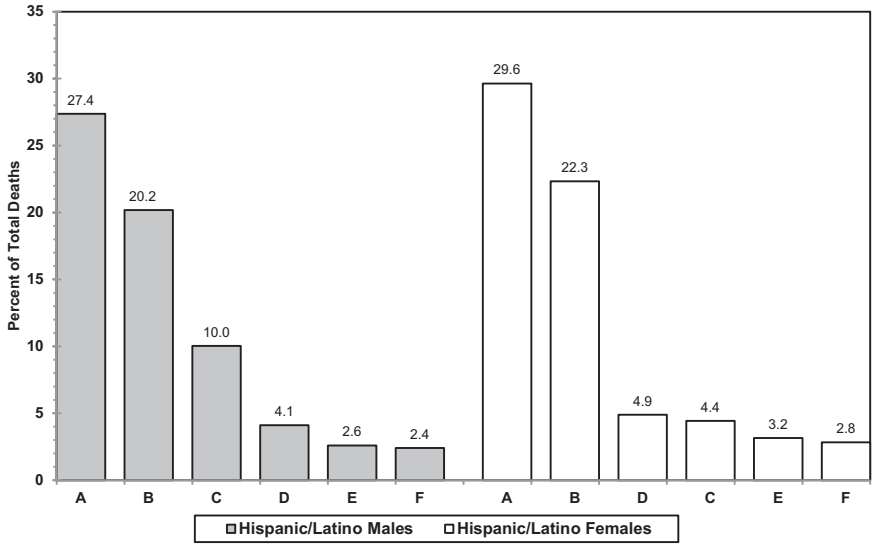

Chart 13-13. Cardiovascular disease and other major causes of death for Hispanic or Latino males and females (United States: 2009). A indicates cardiovascular disease plus congenital cardiovascular disease (*International Classification of Diseases, 10th Revision* codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, diabetes mellitus (E10–E14); E, chronic lower respiratory disease (J40–J47); and F, influenza and pneumonia (J09–J18). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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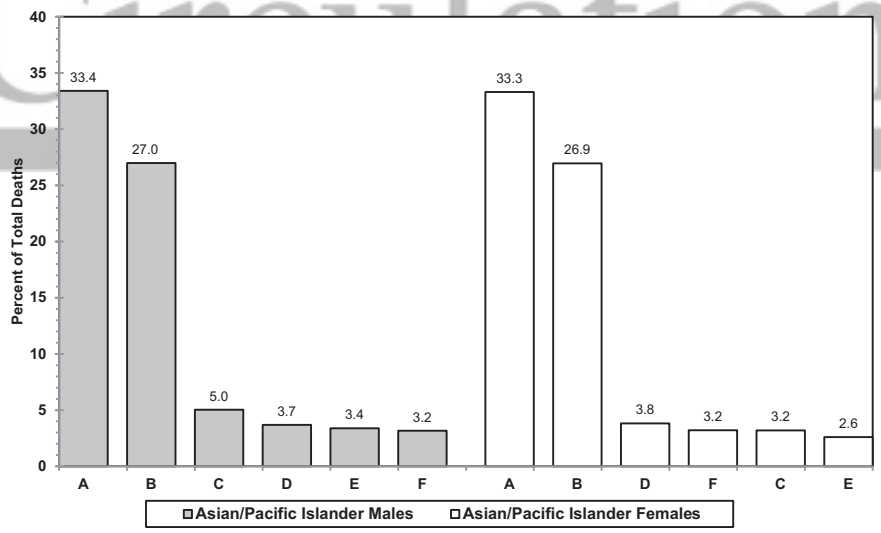

Chart 13-14. Cardiovascular disease and other major causes of death for Asian or Pacific Islander males and females (United States: 2009). "Asian or Pacific Islander" is a heterogeneous category that includes people at high cardiovascular disease risk (eg, South Asian) and people at low cardiovascular disease risk (eg, Japanese). More specific data on these groups are not available. A indicates cardiovascular disease plus congenital cardiovascular disease (*International Classification of Diseases, 10th Revision* codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, diabetes mellitus (E10–E14); E, chronic lower respiratory disease (J40–J47); and F, influenza and pneumonia (J09–J18). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

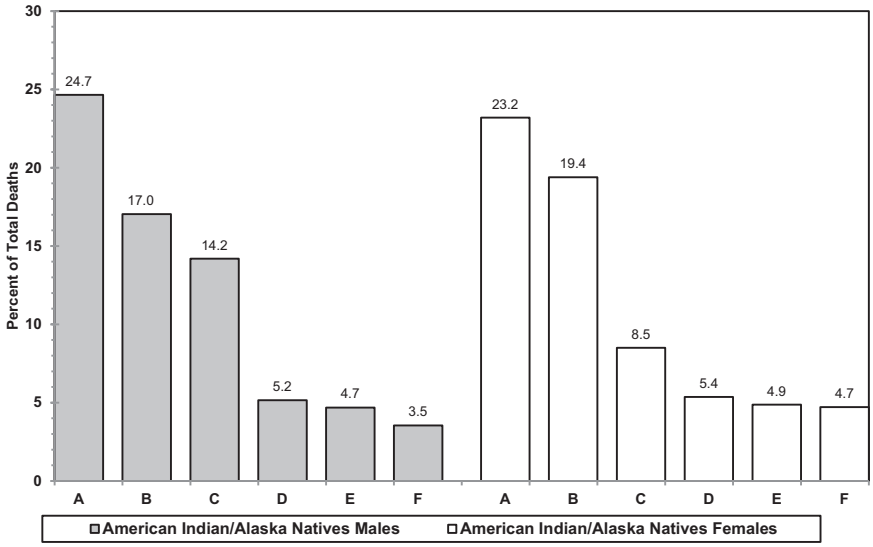

Chart 13-15. Cardiovascular disease and other major causes of death for American Indian or Alaska Native males and females (United States: 2009). A indicates cardiovascular disease plus congenital cardiovascular disease (*International Classification of Diseases, 10th Revision* codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, diabetes mellitus (E10–E14); E, chronic liver disease (K70, K73–K74); and F, chronic lower respiratory disease (J40–J47). Source: National Center for Health Statistics.

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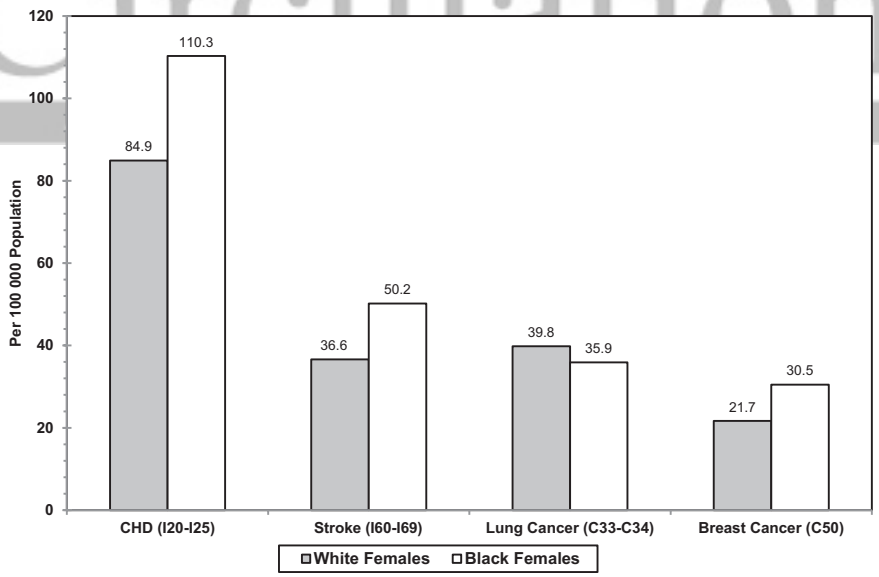

Chart 13-16. Age-adjusted death rates for coronary heart disease (CHD), stroke, and lung and breast cancer for white and black females (United States: 2009). CHD includes *International Classification of Diseases, 10th Revision* codes I20 to I25; stroke, I60 to I69; lung cancer, C33 to C34; and breast cancer, C50. Source: National Center for Health Statistics.

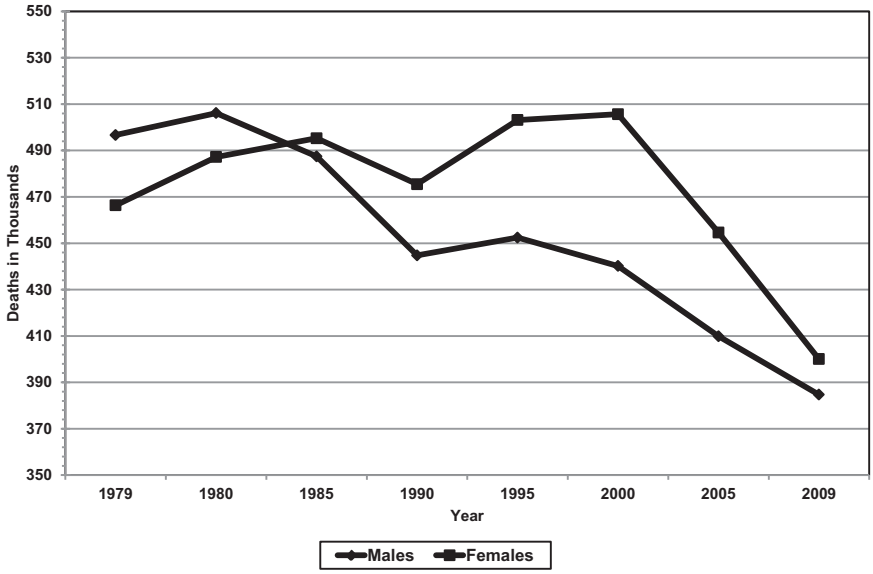

Chart 13-17. Cardiovascular disease mortality trends for males and females (United States: 1979–2009). CVD excludes congenital cardiovascular defects (*International Classification of Diseases, 10th Revision* codes I00–I99). The overall comparability for cardiovascular disease between the *International Classification of Diseases, 9th Revision* (1979–1998) and *International Classification of Diseases, 10th Revision* (1999–2009) is 0.9962. No comparability ratios were applied. Source: National Center for Health Statistics.

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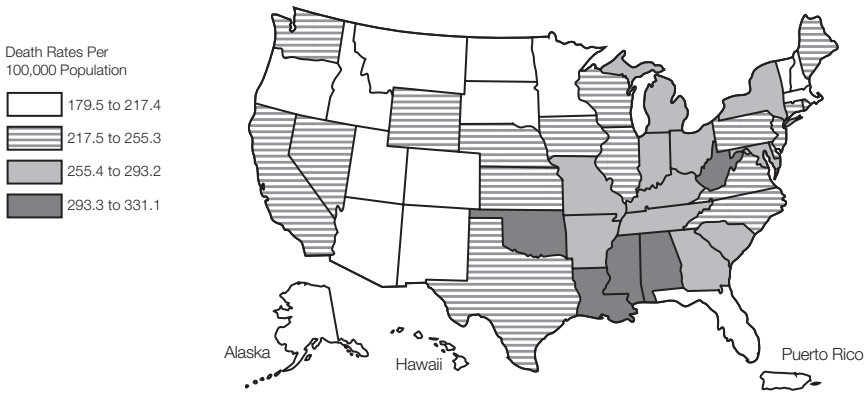
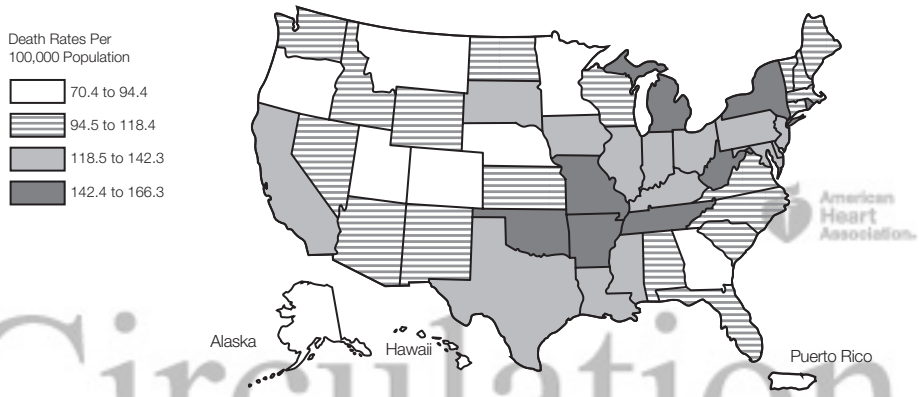
Major Cardiovascular Disease Age-Adjusted Death Rates by State



Coronary Heart Disease Age-Adjusted Death Rates by State



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Stroke Age-Adjusted Death Rates by State

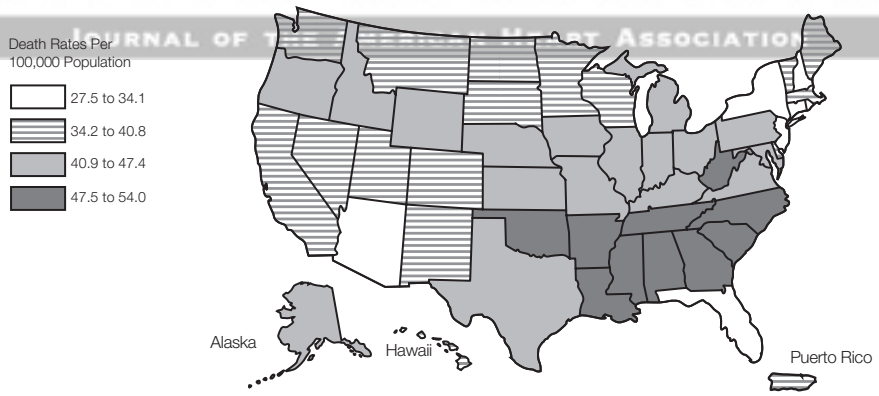
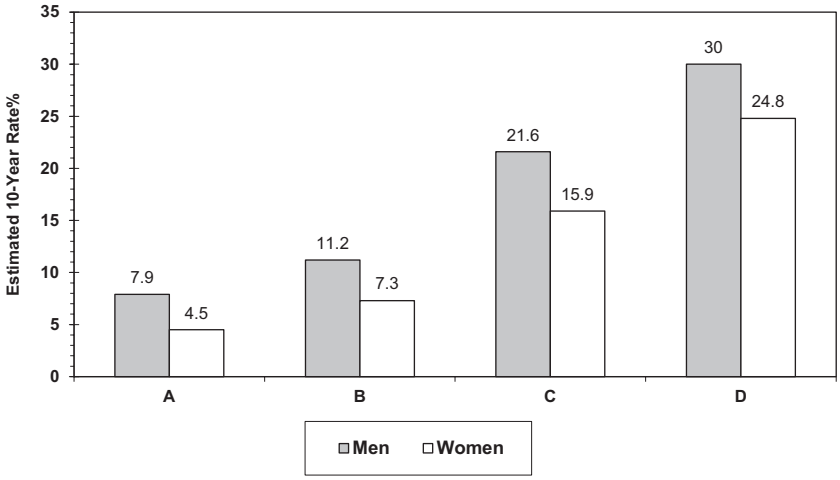


Chart 13-18. US maps corresponding to state death rates (including the District of Columbia), 2009.



	A	B	C	D
Age	50-54	50-54	50-54	50-54
HDL Cholesterol, mg/dL	45-49	45-49	35-44	35-44
Total Cholesterol, mg/dL	160-199	200-239	200-239	200-239
Systolic BP mm/Hg, no treatment	120-129	130-139	130-139	130-139
Smoker	No	No	No	Yes
Diabetes	No	No	Yes	Yes

Chart 13-19. Estimated average 10-year cardiovascular disease risk in adults 50 to 54 years of age according to levels of various risk factors (Framingham Heart Study). HDL indicates high-density lipoprotein; BP, blood pressure. Data derived from D'Agostino et al[48] with permission of the publisher. Copyright © 2008, American Heart Association.

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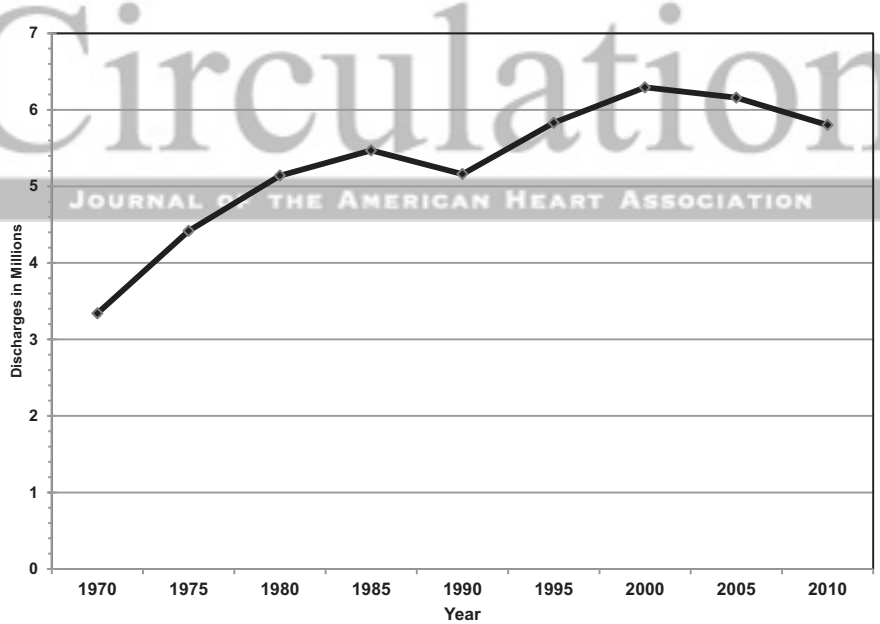

Chart 13-20. Hospital discharges for cardiovascular disease (United States: 1970–2010). Hospital discharges include people discharged alive, dead, and "status unknown." Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

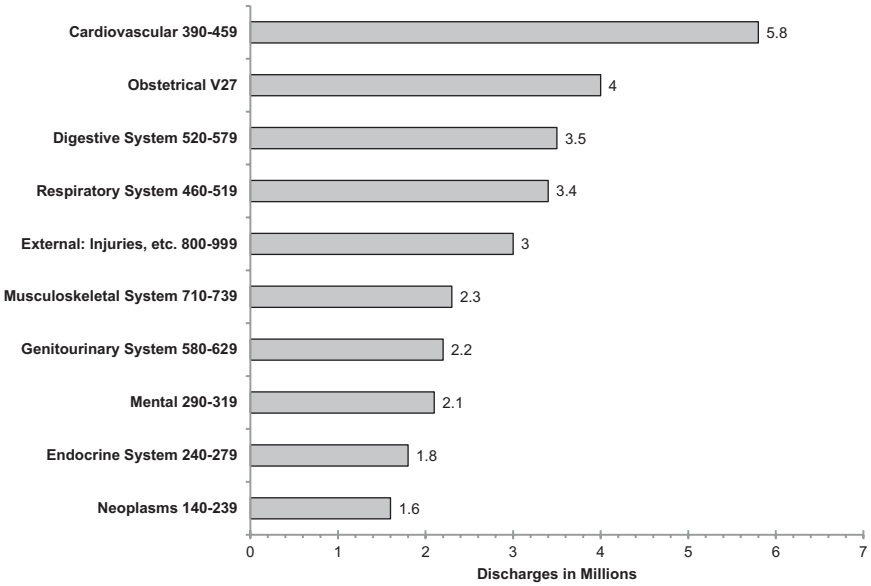

Chart 13-21. Hospital discharges for the 10 leading diagnostic groups (United States: 2010). Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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14. Stroke (Cerebrovascular Disease)

ICD-9 430 to 438; ICD-10 I60 to I69. See Tables 14-1 and 14-2 and Charts 14-1 through 14-13.

Stroke Prevalence

(See Table 14-1 and Chart 14-1.)

- An estimated 6.8 million Americans ≥20 years of age have had a stroke (extrapolated to 2010 using NHANES 2007–2010 data). Overall stroke prevalence during this period is an estimated 2.8%.
- According to data from the 2010 BRFSS (CDC), 2.7% of men and 2.6% of women ≥18 years of age had a history of stroke; 2.4% of non-Hispanic whites, 3.9% of non-Hispanic blacks, 1.5% of Asian/Pacific Islanders, 2.5% of Hispanics (of any race), 5.9% of American Indian/Alaska Natives, and 4.1% of other races or multiracial people had a history of stroke.[1]
- Over the time period 2006–2010, data from BRFSS show that the overall self-reported stroke prevalence did not change.[1]
- Older adults, blacks, people with lower levels of education, and people living in the southeastern United States had higher stroke prevalence.[1]
- The prevalence of silent cerebral infarction is estimated to range from 6% to 28%, with higher prevalence with increasing age.[2–4] The prevalence estimates also vary depending on the population studied (eg, ethnicity, sex, risk factor profile), definition of silent cerebral infarction, and imaging technique. It has been estimated that 13 million people had prevalent silent stroke in the 1998 US population.[5,6]
- The prevalence of stroke-related symptoms was found to be relatively high in a general population free of a prior diagnosis of stroke or transient ischemic attack (TIA). On the basis of data from 18 462 participants enrolled in a national cohort study, 17.8% of the population >45 years of age reported at least 1 symptom. Stroke symptoms were more likely among blacks than whites, among those with lower income and lower educational attainment, and among those with fair to poor perceived health status. Symptoms also were more likely in participants with higher Framingham stroke risk score (REGARDS, NINDS).[7]
- Projections show that by 2030, an additional 4 million people will have had a stroke, a 21.9% increase in prevalence from 2013 (AHA computation based on methodology described in Heidenreich et al[8]).

Stroke Incidence

(See Table 14-1 and Charts 14-2 through 14-5.)

- Each year, ≈795 000 people experience a new or recurrent stroke. Approximately 610 000 of these are first attacks, and 185 000 are recurrent attacks (GCNKSS, NINDS, and NHLBI; GCNKSS and NINDS data for 1999 provided July 9, 2008; estimates compiled by NHLBI).
- Of all strokes, 87% are ischemic and 10% are intracerebral hemorrhagic strokes, whereas 3% are subarachnoid hemorrhage strokes (GCNKSS, NINDS, 1999).
- On average, every 40 seconds, someone in the United States has a stroke (AHA computation based on the latest available data).

Abbreviations Used in Chapter 14

AF	atrial fibrillation
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities study
BASIC	Brain Attack Surveillance in Corpus Christi
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CAS	carotid artery stenting
CDC	Centers for Disease Control and Prevention
CEA	carotid endarterectomy
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CI	confidence interval
CLRD	chronic lower respiratory disease
CREST	Carotid Revascularization Endarterectomy versus Stenting Trial
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
eGFR	estimated glomerular filtration rate
EMS	emergency medical services
FHS	Framingham Heart Study
FRS	Framingham Risk Score
GCNKSS	Greater Cincinnati/Northern Kentucky Stroke Study
GFR	glomerular filtration rate
HD	heart disease
HDL	high-density lipoprotein
HR	hazard ratio
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
ICH	intracerebral hemorrhage
MCBE	Medicare beneficiaries
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institutes of Neurological Disorders and Stroke
NOMAS	Northern Manhattan Study
OR	odds ratio
PA	physical activity
PAR	population attributable risk
REGARDS	Reasons for Geographic and Racial Differences in Stroke study
RR	relative risk
SAH	subarachnoid hemorrhage
STOP	Stroke Prevention Trial in Sickle Cell Anemia
SWiTCH	Stroke With Transfusions Changing to Hydroxyurea
TIA	transient ischemic attack

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- Each year, ≈55 000 more women than men have a stroke (GCNKSS, NINDS).[9]
- Women have a higher lifetime risk of stroke than men. In the FHS, lifetime risk of stroke among those 55 to75 years of age was 1 in 5 for women (20% to 21%) and ≈1 in 6 for men (14% to 17%).[10]
- Women have lower age-adjusted stroke incidence than men; however, sex differences in stroke risk may be modified by age.[11] Data from FHS demonstrate that compared with white men, white women 45 to 84 years of age have lower stroke risk than men, but this association is reversed in older ages such that women >85 years of age have elevated risk compared with men.[12] Similarly, a population-based study in Sweden found stroke incidence to be lower for women than for men at ages 55 to 64 years, but at 75 to 85 years of age, this association reversed, and women had a higher incidence than men.[13] Other studies report an excess risk of stroke in men compared with women that persists throughout the life course or that diminishes but does not reverse with age.[14–18]
- In the national REGARDS cohort, in 27 744 participants followed up for 4.4 years (2003–2010), the overall age- and sex-adjusted black/white incidence rate ratio was 1.51, but for ages 45 to 54 years, it was 4.02, whereas for those ≥85 years of age, it was 0.86.[19] Similar trends for decreasing black/white incidence rate ratio with age were seen in the GCNKSS.[20]
- Analysis of data from the FHS suggests that stroke incidence is declining over time in this largely white cohort. Data from 1950 to 1977, 1978 to 1989, and 1990 to 2004 showed that the age-adjusted incidence of first stroke per 1000 person-years in each of the 3 periods was 7.6, 6.2, and 5.3 in men and 6.2, 5.8, and 5.1 in women, respectively. Lifetime risk for incident stroke at 65 years of age decreased significantly in the latest data period compared with the first, from 19.5% to 14.5% in men and from 18.0% to 16.1% in women.[21]
- In a similar fashion, data from the most recent GCNKSS show that compared with the 1990s, when incidence rates of stroke were stable, stroke incidence in 2005 was decreased for whites. A similar decline was not seen in blacks. These changes for whites were driven by a decline in ischemic strokes. There were no changes in incidence of ischemic stroke for blacks or of hemorrhagic strokes in blacks or whites.[9]
- The BASIC (Brain Attack Surveillance in Corpus Christi) project (NINDS) demonstrated an increased incidence of stroke among Mexican Americans compared with non-Hispanic whites in a community in southeast Texas. The crude 3-year cumulative incidence (2000–2002) was 16.8 per 1000 in Mexican Americans and 13.6 per 1000 in non-Hispanic whites. Specifically, Mexican Americans had a higher cumulative incidence for ischemic stroke at younger ages (45–59 years of age: RR, 2.04; 95% CI, 1.55–2.69; 60–74 years of age: RR, 1.58; 95% CI, 1.31–1.91) but not at older ages (≥75 years of age: RR, 1.12; 95% CI, 0.94–1.32). Mexican Americans also had a higher incidence of intracerebral hemorrhage and subarachnoid hemorrhage than non-Hispanic whites, adjusted for age.[22]
- The age-adjusted incidence of first ischemic stroke per 1000 was 0.88 in whites, 1.91 in blacks, and 1.49 in Hispanics according to data from the Northern Manhattan Study (NOMAS; NINDS) for 1993 to 1997. Among blacks, compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.85; of extracranial atherosclerotic stroke, 3.18; of lacunar stroke, 3.09; and of cardioembolic stroke, 1.58. Among Hispanics (primarily Cuban and Puerto Rican), compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.00; of extracranial atherosclerotic stroke, 1.71; of lacunar stroke, 2.32; and of cardioembolic stroke, 1.42.[23]
- Among 4507 American Indian participants without a prior stroke in the Strong Heart Study in 1989 to 1992, the age- and sex-adjusted incidence of stroke through 2004was 6.79 per 100 person-years, with 86% of incident strokes being ischemic.[24]
- In the GCNKSS, the annual incidence of anticoagulant-associated intracerebral hemorrhage per 100 000 people increased from 0.8 (95% CI, 0.3–1.3) in 1988 to 1.9 (95% CI, 1.1–2.7) in 1993/1994 and 4.4 (95% CI, 3.2–5.5) in 1999 (P<0.001 for trend). Among people ≥80 years of age, the rate of anticoagulant-associated intracerebral hemorrhage increased from 2.5 (95% CI, 0–7.4) in 1988 to 45.9 (95% CI, 25.6–66.2) in 1999 (P<0.001 for trend).[25]

TIA: Prevalence, Incidence, and Prognosis

- In a nationwide survey of US adults, the estimated prevalence of self-reported physician-diagnosed TIA was 2.3%, which translates into ≈5 million people. The true prevalence of TIA is greater, because many patients who experience neurological symptoms consistent with a TIA fail to report it to their healthcare provider.[26]
- In the GCNKS, using data from 1993 and 1994, the age-, sex-, and race-adjusted incidence rates for TIA were 0.83 per 10 000.[27] Age- and sex- adjusted incidence rates for TIA in Rochester, MN, were estimated at 0.68 per 1000 for the years 1985 through 1989.[28] In a more recent Italian community-based registry conducted in 2007 to 2009, the crude TIA incidence rate was 0.52 per 1000.[29]
- The prevalence of physician-diagnosed TIA increases with age.[26] Incidence of TIA increases with age and varies by sex and race/ethnicity. Men, blacks, and Mexican Americans have higher rates of TIA than their female and non-Hispanic white counterparts.[22,27,29]
- Approximately 15% of all strokes are heralded by a TIA.[30]
- TIAs confer a substantial short-term risk of stroke, hospitalization for CVD events, and death. Of 1707 TIA patients evaluated in the ED of Kaiser Permanente Northern California, a large, integrated healthcare delivery system, 180 (11%) experienced a stroke within 90 days. Ninety-one patients (5%) had a stroke within 2 days. Predictors of stroke included age >60 years, DM, focal symptoms of weakness or speech impairment, and TIA that lasted >10 minutes.[31]
- Meta-analyses of cohorts of patients with TIA have shown the short-term risk of stroke after TIA to be ≈3% to 10% at 2 days and 9% to 17% at 90 days.[32,33]
- Individuals who have a TIA and survive the initial high-risk period have a 10-year stroke risk of roughly 19% and a combined 10-year stroke, MI, or vascular death risk of 43% (4% per year).[34]

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- Within 1 year of TIA, ≈12% of patients will die.[27]
- It is estimated that one third of episodes characterized as TIA according to the classic definition (ie, focal neurological deficits that resolve within 24 hours) would be considered infarctions on the basis of diffusion-weighted magnetic resonance imaging findings.[35]

Stroke Mortality

(See Table 14-1 and Charts 14-6 through 14-7.)

- On average, every 4 minutes, someone dies of a stroke (NCHS, NHLBI).[36]
- Stroke accounted for ≈1 of every 19 deaths in the United States in 2009.[36]
- When considered separately from other CVDs, stroke ranks No. 4 among all causes of death, behind diseases of the heart, cancer, and CLRD (NCHS mortality data). Stroke mortality as an underlying cause of death in 2009 was 128 824; any-mention mortality in 2009 was 215 864, and the death rate was 38.9 per 100 000.[36]
- From 1999 to 2009, the annual stroke death rate decreased 36.9%, and the actual number of stroke deaths declined 22.9% (AHA computation).[36]
- Conclusions about changes in stroke death rates from 1980 to 2005 are as follows:
  - —There was a greater decline in stroke death rates in men than in women, with a male-to-female ratio decreasing from 1.11 to 1.03 (age adjusted).
  - —There were greater declines in stroke death rates in men than in women and among people ≥65 years of age than among younger ages.[37]
- In examining trends in stroke mortality by census divisions between 1999 and 2007 for people ≥45 years of age, the rate of decline varied by geographic region and race-ethnic group. Among black and white women and white men, rates declined by at least 2% annually in every census division, but among black men, rates declined little in the East and West South Central divisions.[38]
- Approximately 56% of stroke deaths in 2009 occurred out of the hospital (unpublished tabulation of NCHS 2009 Mortality Data Set).
- More women than men die of stroke each year because of the larger number of elderly women. Women accounted for almost 60% of US stroke deaths in 2009 (AHA tabulation).[36]
- From 1995 to 1998, age-standardized mortality rates for ischemic stroke, subarachnoid hemorrhage, and intracerebral hemorrhage were higher among blacks than whites. Death rates attributable to intracerebral hemorrhage also were higher among Asians/Pacific Islanders than among whites. All minority populations had higher death rates attributable to subarachnoid hemorrhage than did whites. Among adults 25 to 44 years of age, blacks and American Indian/Alaska Natives had higher risk ratios for stroke mortality than did whites for all 3 stroke subtypes. Age-standardized mortality rates for ischemic stroke and intracerebral hemorrhage were lower for Hispanics than for whites.[39]
- In 2002, death certificate data showed that the mean age at stroke death was 79.6 years; however, males had a younger mean age at stroke death than females. Blacks, American Indian/Alaska Natives, and Asian/Pacific Islanders had younger mean ages than whites, and the mean age at stroke death was also younger among Hispanics than non-Hispanics.[40]
- A report released by the CDC in collaboration with the Centers for Medicare & Medicaid Services, the *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries*, found that in Medicare beneficiaries over the time period 1995 to 2002, 30-day mortality rate varied by age: 9% in patients 65 to 74 years of age, 13.1% in those 74 to 84 years of age, and 23% in those ≥85 years of age.[41]
- There are substantial geographic disparities in stroke mortality, with higher rates in the southeastern United States, known as the "stroke belt." This area is usually defined to include the 8 southern states of North Carolina, South Carolina, Georgia, Tennessee, Mississippi, Alabama, Louisiana, and Arkansas. These geographic differences have existed since at least 1940,[42] and despite some minor shifts,[43] they persist.[41,44,45] Within the stroke belt, a "buckle" region along the coastal plain of North Carolina, South Carolina, and Georgia has been identified with an even higher stroke mortality rate than the remainder of the stroke belt.[46] The overall average stroke mortality is ≈20% higher in the stroke belt than in the rest of the nation and ≈40% higher in the stroke buckle.

Stroke Risk Factors

(See Table 14-2 and Chart 14-8.)

For prevalence and other information on any of these specific risk factors, refer to the specific risk factor chapters.

High Blood Pressure

(See Chapter 9 for more information.)

- BP is a powerful determinant of risk for both ischemic stroke and intracranial hemorrhage.
  - —Approximately 77% of those who have a first stroke have BP >140/90 mm Hg (NHLBI unpublished estimates from ARIC, CHS, and FHS Cohort and Offspring studies).
  - —Diabetic subjects with BP <120/80 mm Hg have approximately half the lifetime risk of stroke of subjects with hypertension. The treatment and lowering of BP among diabetic hypertensive individuals was associated with a significant reduction in stroke risk.[47]
- A meta-analysis of 12 prospective cohort studies (including 518 520 participants) found that prehypertension is associated with incident stroke. The risk is particularly noted in nonelderly people and for those with BP values in the higher prehypertension range.[48]
- Prehypertension was found to be highest in blacks with other risk factors, including DM and elevated C-reactive protein.[49]
- In REGARDS (NINDS), black participants were more aware than whites of their hypertension and more likely to be undergoing treatment if aware of their diagnosis, but among those treated for hypertension, they were less likely than whites to have their BP controlled.[50]
- REGARDS (NINDS) also showed no evidence of a difference between the stroke belt and other regions in awareness of hypertension, but there was a trend for better treatment

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and BP control in the stroke belt region. The lack of substantial geographic differences in hypertension awareness and the trend toward better treatment and control in the stroke belt suggest that differences in hypertension management may not be a major contributor to the geographic disparity in stroke mortality.[50]

Diabetes Mellitus

(See Chapter 10 for more information.)

- Age-specific incidence rates and rate ratios show that DM increases ischemic stroke incidence at all ages, but this risk is most prominent before 55 years of age in blacks and before 65 years of age in whites. Ischemic stroke patients with DM are younger, more likely to be black, and more likely to have hypertension, MI, and high cholesterol than nondiabetic patients.[51]
- In people with history of TIA or minor stroke, impaired glucose tolerance nearly doubled the stroke risk compared with those with normal glucose levels and tripled the risks for those with DM.[52]

Disorders of Heart Rhythm

(See Chapter 16 for more information.)

- AF is a powerful risk factor for stroke, independently increasing risk ≈5-fold throughout all ages. The percentage of strokes attributable to AF increases steeply from 1.5% at 50 to 59 years of age to 23.5% at 80 to 89 years of age.[53,54]
- Because AF is often asymptomatic[55,56] and likely frequently clinically undetected,[57] the stroke risk attributed to AF may be substantially underestimated.[58]
- Among 2580 participants ≥65 years of age with hypertension in whom a cardiac rhythm device that included an atrial lead was implanted, 35% developed subclinical tachyarrhythmias (defined as an atrial rate ≥190 beats per minute that lasted at least 6 minutes). These subclinical events were independently associated with a 2.5-fold increased risk of ischemic stroke or systemic embolism.[59]

High Blood Cholesterol and Other Lipids

(See Chapter 8 for more information.)

- Data from the Honolulu Heart Program//NHLBI found that in Japanese men 71 to 93 years of age, low concentrations of HDL cholesterol were more likely to be associated with a future risk of thromboembolic stroke than were high concentrations.[60] However, a meta-analysis of 23 studies performed in the Asia-Pacific Region showed no significant association between low HDL and stroke risk.[61]

Smoking

(See Chapter 3 for more information.)

- Current smokers have a 2 to 4 times increased risk of stroke compared with nonsmokers or those who have quit for >10 years.[62,63]
- Cigarette smoking is a risk factor for ischemic stroke and subarachnoid hemorrhage, but the data for intracerebral hemorrhage are less consistent.[62,63]
- Smoking is perhaps the most important modifiable risk factor in preventing subarachnoid hemorrhage, with the highest PAR of any subarachnoid hemorrhage risk factor.[64]
- Data also support a dose-response relationship across old and young age groups.[62,65]
- Discontinuation of smoking has been shown to reduce stroke risk across sex, race, and age groups.[65]
- Exposure to secondhand smoke (also termed *passive smoking* or *environmental tobacco smoke*) is a risk factor for stroke. Meta-analyses have estimated a pooled RR of 1.25 for exposure to spousal smoking (or nearest equivalent) and risk of stroke. A dose-response relationship between exposure to secondhand smoke and stroke risk has also been reported.[66,67]

Physical Inactivity

(See Chapter 4 for more information.)

- In NOMAS, a prospective cohort that included white, black, and Hispanic adults in an urban setting followed up for a median of 9 years, moderate to vigorous PA was associated with an overall 35% reduction in risk of ischemic stroke compared with no PA.[68] In this cohort, it was also found that only moderate- to vigorous-intensity exercise was associated with reduced stroke incidence, whereas light exercise (such as walking) showed no benefit.[69]
- In NOMAS, higher levels of PA were associated with lower risk of silent brain infarcts on magnetic resonance imaging, even after adjustment for other risk factors potentially in the pathway of action for PA.[70]
- Timing of PA in relation to stroke onset has also been examined in several studies. In a hospital-based case-control study from Heidelberg, Germany, recent activity (within the prior months) was associated with reduced odds of having a stroke or TIA, whereas sports activity during young adulthood that was not continued showed no benefit.[71] In a Danish case-control study, ischemic stroke patients were less physically active in the week preceding the stroke than age- and sex-matched control subjects, with the highest activity scores associated with the greatest reduction in odds of stroke.[72]

Family History and Genetics

(See Chapter 7 for more information.)

- In the FHS, a documented parental ischemic stroke by the age of 65 years was associated with a 3-fold increase in ischemic stroke risk in offspring, even after adjustment for other known stroke risk factors. The absolute magnitude of the increased risk was greatest in those in the highest quintile of the FRS. By age 65 years, people in the highest FRS quintile with an early parental ischemic stroke had a 25% risk of stroke compared with a 7.5% risk of ischemic stroke for those without such a history.[73]

Chronic Kidney Disease

(See Chapter 12 for more information.)

- The CHS (NHLBI) showed that people with creatinine ≥1.5 mg/dL were at increased risk for stroke, with an adjusted HR of 1.77 (95% CI, 1.08–2.91).[74] Participants in REGARDS with a reduced eGFR were also shown to have increased risk of stroke symptoms,[75] and a meta-analysis of >280 000 patients showed a 43% increased incident stroke risk among patients with a GFR <60 $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$.[76]

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Risk Factor Issues Specific to Women

- On average, women are older at stroke onset than men (≈75 years compared with 71 years).[12]
- Analysis of data from the FHS found that women with natural menopause before 42 years of age had twice the ischemic stroke risk of women with natural menopause after 42 years of age.[77] Investigators from the Nurse's Health Study, however, did not find an association between age at natural menopause and risk of ischemic or hemorrhagic stroke.[78]
- Overall, randomized clinical trial data indicate that the use of estrogen plus progestin, as well as estrogen alone, increases stroke risk in postmenopausal, generally healthy women and provides no protection for postmenopausal women with established CHD[79–82] and recent stroke or TIA.[83]
- In a nested case-control study of the United Kingdom's General Practice Research Database, the rate of stroke was not elevated when current users of transdermal estrogens, with or without progestins, were compared with nonusers (RR, 0.95; 95% CI, 0.75–1.20). This association was modified by dose such that stroke risk was not increased for users of low-dose (≤50 μg) estrogen patches (RR, 0.81; 95% CI, 0.62–1.05) but was increased for users of high-dose (>50 μg) patches (RR, 1.89; 95% CI, 1.15–3.11) compared with nonusers.[84]
- The risk of ischemic stroke or intracerebral hemorrhage during pregnancy and the first 6 weeks after giving birth was 2.4 times greater than for nonpregnant women of similar age and race, according to the Baltimore-Washington Cooperative Young Stroke Study. The risk of ischemic stroke during pregnancy was not increased during pregnancy per se but was increased 8.7-fold during the first 6 postpartum weeks. Intracerebral hemorrhage showed a small RR of 2.5 during pregnancy that increased dramatically to an RR of 28.3 in the first 6 postpartum weeks. The excess risk of stroke (all types except subarachnoid hemorrhage) attributable to the combined pregnancy/postpregnancy period was 8.1 per 100 000 pregnancies.[85]
- In the US Nationwide Inpatient Sample from 2000 to 2001, the rate of events per 100 000 pregnancies was 9.2 for ischemic stroke, 8.5 for intracerebral hemorrhage, 0.6 for cerebral venous thrombosis, and 15.9 for the ill-defined category of pregnancy-related cerebrovascular events, for a total rate of 34.2 per 100 000, not including subarachnoid hemorrhage. The risk was increased in blacks and among older women. Death occurred during hospitalization in 4.1% of women with these events and in 22% of survivors after discharge to a facility other than home.[86]
- Analyses of the US Nationwide Inpatient Sample from 1994 to 1995 and from 2006 to 2007 show a temporal increase in the proportion of pregnancy hospitalizations that were associated with a stroke, with a 47% increase for antenatal hospitalizations and an 83% increase for postpartum hospitalizations, but no increase for delivery hospitalizations. Increases in the prevalence of HD and hypertensive disorders accounted for almost all the increase in postpartum stroke hospitalizations but not the antenatal stroke hospitalizations.[88]
- Preeclampsia is a risk factor for ischemic stroke remote from pregnancy.[89] The subsequent stroke risk of preeclampsia maybe mediated by a 3.6- to 6.1-fold higher later risk of hypertension and a 3.1- to 3.7-fold higher later risk of DM, depending on whether the preeclampsia was mild or severe.[90]

Sleep Apnea

- Sleep apnea is an independent risk factor for stroke, increasing the risk of stroke or death 2-fold.[91–94]
- Increasing sleep apnea severity is associated with greater stroke risk; patients with severe sleep apnea have 3-to 4-fold increased odds of stroke.[91,93,94]
- Sleep apnea is common after stroke, occurring in two thirds to three quarters of poststroke patients. [95–106]
- Continuous positive airway pressure improves a variety of outcomes after stroke.[96,107–109] For example, continuous positive airway pressure reduces the risk of recurrent vascular events among patients with stroke (relative risk reduction, 81.4%; number needed to treat, 3.4).[108]

Awareness of Stroke Warning Signs and Risk Factors

- Correct knowledge of at least 1 stroke warning sign increased from 48% in 1995 to 68% in 2000, with no significant improvement to 2005 (68%) on the basis of a telephone survey conducted in a biracial population in the greater Cincinnati/Northern Kentucky region. Knowledge of 3 correct warning signs was low but increased over time: 5.4% in 1995, 12.0% in 2000, and 15.7% in 2005. Knowledge of at least 1 stroke risk factor increased from 59% in 1995 to 71% in 2000, but there was no improvement to 2005 (71%). Only 3.6% of those surveyed were able to independently identify tissue-type plasminogen activator as an available drug therapy, and only 9% of these were able to identify a window of <3 hours for treatment.[110]
- In the 2009 NHIS, 51.2% of subjects were aware of 5 stroke warning symptoms and would first call 911 if they thought that someone was having a stroke. Awareness of all 5 stroke warning symptoms and calling 911 was higher among whites than blacks and Hispanics (55.9%, 47.1%, and 36.5%, respectively), women than men (53.6% versus 48.6%), and people with higher versus lower educational attainment (59.0% for people with a bachelor's degree or more compared with 51.4% for people with a high school diploma or some college and 36.7% for those who had not received a high school diploma; unpublished NHLBI tabulation).
- A study was conducted of patients admitted to an ED with possible stroke to determine their knowledge of the signs, symptoms, and risk factors of stroke. Of the 163 patients able to respond, 39% did not know a single sign or symptom. Patients ≥65 years of age were less likely than those <65 years old to know a sign or symptom of stroke (28% versus 47%), and 43% did not know a single risk factor. Overall, almost 40% of patients did not know the signs, symptoms, and risk factors for stroke.[111]
- In 2004, 800 adults ≥45 years of age were surveyed to assess their perceived risk for stroke and their history of stroke risk factors. Overall, 39% perceived themselves to be at risk. Younger age, current smoking, a history of DM, high BP, high cholesterol, HD, and stroke/TIA were independently associated with perceived risk for stroke.

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Respondents with AF were no more likely to report being at risk than were respondents without AF. Perceived risk for stroke increased as the number of risk factors increased; however, 46% of those with ≥3 risk factors did not perceive themselves to be at risk.[112]

- A study of patients who had experienced a stroke found that only 60.5% were able to accurately identify 1 stroke risk factor and that 55.3% were able to identify 1 stroke symptom. Patients' median delay time from onset of symptoms to admission in the ED was 16 hours, and only 31.6% accessed the ED in <2 hours. Analysis showed that the appearance of nonmotor symptoms as the primary symptom and nonuse of the 911 system were significant predictors of delay >2 hours. Someone other than the patient made the decision to seek treatment in 66% of the cases.[113]
- Spanish-speaking Hispanics are less likely to know all stroke symptoms than English-speaking Hispanics, non-Hispanic blacks, and non-Hispanic whites. Lack of English proficiency is strongly associated with lack of stroke knowledge among Hispanics.[114]

Aftermath

(See Charts 14-9 through 14-12.)

- Stroke is a leading cause of serious long-term disability in the United States (Survey of Income and Program Participation, a survey of the US Bureau of the Census).[115]
- Among Medicare patients discharged from the hospital after stroke, ≈45% return directly home, 24% are discharged to inpatient rehabilitation facilities, and 31% are discharged to skilled nursing facilities. Of stroke patients returning directly home, 32% use home healthcare services.[116] For Medicare patients (including, but not limited to, stroke survivors), the likelihood of receiving inpatient rehabilitation facility care versus skilled nursing facility care is substantially influenced by the distance to and availability of inpatient rehabilitation facility beds.[117]
- Approximately one third of stroke survivors experience poststroke depression.[118]
- In the NHLBI's FHS, among ischemic stroke survivors who were ≥65 years of age, the following disabilities were observed at 6 months after stroke[119]:

  — 50% had some hemiparesis
  — 30% were unable to walk without some assistance
  — 46% had cognitive deficits
  — 35% had depressive symptoms
  — 19% had aphasia
  — 26% were dependent in activities of daily living
  — 26% were institutionalized in a nursing home

- Data from the BRFSS (CDC) 2005 survey on stroke survivors in 21 states and the District of Columbia found that 30.7% of stroke survivors received outpatient rehabilitation. The findings indicated that the prevalence of stroke survivors receiving outpatient stroke rehabilitation was lower than would be expected if clinical practice guideline recommendations for all stroke patients had been followed.[120]
- After stroke, women have greater disability than men. A cross-sectional analysis of 5888 community-living elderly people (>65 years of age) in the CHS who were ambulatory at baseline found that women were half as likely to be independent in activities of daily living after stroke, even after controlling for age, race, education, and marital status.[121] A prospective study from a Michigan-based stroke registry found that women had a 63% lower probability of achieving independence in activities of daily living 3 months after discharge, even after controlling for age, race, subtype, prestroke ambulatory status, and other patient characteristics.[122]
- Black stroke survivors had greater limitations in ambulation than did white stroke survivors, after adjustment for age, sex, and educational attainment but not stroke subtype, according to data from the NHIS (2000–2001, NCHS) as analyzed by the CDC.[123] A national study of inpatient rehabilitation after first stroke found that blacks were younger, had a higher proportion of hemorrhagic stroke, and were more disabled on admission. Compared with non-Hispanic whites, blacks and Hispanics also had a poorer functional status at discharge but were more likely to be discharged to home rather than to another institution, even after adjustment for age and stroke subtype. After adjustment for the same covariates, compared with non-Hispanic whites, blacks also had less improvement in functional status per inpatient day.[124]

Hospital Discharges/Ambulatory Care Visits

(See Table 14-1.)

- From 2000 to 2010, the number of inpatient discharges from short-stay hospitals with stroke as the first-listed diagnosis remained about the same, with discharges of 981 000 and 1 015 000, respectively (NHDS, NHLBI tabulation).
- Data from 2010 from the NHDS of the NCHS showed that the average length of stay for discharges with stroke as the first-listed diagnosis was 6.1 days compared with 9.5 days in 1990.
- In 2010, men and women accounted for roughly the same number of hospital stays for stroke in the 18- to 44-year-old age group. Among people 45 to 64 years of age, 57.1% of stroke patients were men. After 65 years of age, women were the majority. Among people 65 to 84 years of age, 53.4% of stroke patients were women, whereas among those ≥85 years of age, women constituted 66.2% of all stroke patients.[125]
- A first-ever county-level *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries* was released in 2008 by the CDC in collaboration with the Centers for Medicare & Medicaid Services. It found that the stroke hospitalization rate for blacks was 27% higher than for the US population in general, 30% higher than for whites, and 36% higher than for Hispanics. In contrast to whites and Hispanics, the highest percentage of strokes in blacks (42.3%) occurred in the youngest Medicare age group (65–74 years of age).[41]
- In 2009, there were 768 000 ED visits and 127 000 outpatient department visits with stroke as the first-listed diagnosis. In 2010, physician office visits for a first-listed diagnosis of stroke totaled 2 207 000 (NHAMCS, unpublished NHLBI tabulation).[126]

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Stroke in Children

- On the basis of pathogenic differences, pediatric strokes are typically classified as either perinatal (occurring at ≤28 days of life and including in utero strokes) or (later) childhood.
- Estimates of the overall annual incidence of stroke in US children are 6.4 per 100 000 children (0 to 15 years) in 1999 in the GCNKSS[127] and 4.6 per 100 000 children (0 to 19 years) in 1997 to 2003 according to data from Kaiser Permanente of Northern California, a large, integrated healthcare delivery system.[128] Approximately half of all incident childhood strokes are hemorrhagic.[127–129]
- The prevalence of perinatal strokes is 29 per 100 000 live births, or 1 per 3500 live births in the 1997–2003 Kaiser Permanente of Northern California population.[128]
- A history of infertility, preeclampsia, prolonged rupture of membranes, and chorioamnionitis are independent maternal risk factors for perinatal arterial ischemic stroke.[130] However, maternal health and pregnancies are normal in most cases.[131]
- The most common cause of arterial ischemic stroke in children is a cerebral arteriopathy, found in more than half of all cases.[132,133]
- Children with cardiac disease are at 16-fold increased risk of arterial ischemic stroke compared with children in the general population; those with single-ventricle physiology are at highest risk.[134]
- Thrombophilias (genetic and acquired) are risk factors for childhood stroke, with summary ORs ranging from 1.6 to 8.8 in a meta-analysis.[135]
- In a prospective Swiss registry, [136] atherosclerotic risk factors were less common in children with arterial ischemic stroke than in young adults; the most common of these factors in children was hyperlipidemia (15%). However, an analysis of the Nationwide Inpatient Sample suggests a low but rising prevalence of these factors among US adolescents and young adults hospitalized for ischemic stroke (1995 versus 2008).[137]
- From 1979 to 1998 in the United States, childhood mortality resulting from stroke declined by 58% overall, with reductions in all major subtypes.[138]
- The incidence of stroke in children has been stable over the past 10 years, whereas 30-day case fatality rates declined from 18% in 1988–1989 to 9% in 1993–1994 and 9% in 1999 in the GCNKSS population.[127]
- Compared with girls, boys have a 1.28-fold higher risk of stroke.[139] Compared with white children, black children have a 2-fold risk of both incident stroke and death attributable to stroke.[138,139] The increased risk among blacks is not fully explained by the presence of sickle cell disease, nor is the excess risk among boys fully explained by trauma.[139]
- At a mean follow-up time of 2.1 years, 37% of 123 childhood ischemic stroke survivors had full recovery, 20% had mild deficits, 26% had moderate deficits, and 16% had severe deficits.[140] Concomitant involvement of the basal ganglia, cerebral cortex, and posterior limb of the internal capsule predicts a persistent hemiparesis.[141]
- Despite current treatment, 1 of 10 children with ischemic or hemorrhagic stroke will have a recurrence within 5 years.[142,143] The 5-year recurrence risk is as high as 60% among children with cerebral arteriopathy. The recurrence risk after perinatal stroke, however, is negligible.[144]
- More than 25% of survivors of perinatal ischemic strokes develop delayed seizures within 3 years; children with larger strokes are at higher risk.[145]
- Sickle cell disease is the most important cause of ischemic stroke among black children. The Stroke Prevention Trial in Sickle Cell Anemia (STOP) demonstrated the efficacy of blood transfusions for primary stroke prevention in children with sickle cell disease identified as high-risk by transcranial Doppler; STOP II demonstrated that stopping transfusions after 30 months of treatment was associated with a high risk of stroke.[146]
- The Stroke With Transfusions Changing to Hydroxyurea (SWiTCH) trial recently reported that chronic blood transfusion therapy remains the superior option (over hydroxyurea) for secondary stroke prevention in children with sickle cell disease.[147] A similar trial of hydroxyurea as an alternative for primary stroke prevention is under way.

Stroke in the Very Elderly

- Stroke patients >85 years of age make up 17% of all stroke patients.[148]
- Very elderly patients have a higher risk-adjusted mortality,[148,149] have longer hospitalizations,[149] receive less evidenced-based care,[150,151] and are less likely to be discharged to their original place of residence.[149,152]

Barriers to Stroke Care

- On the basis of NHIS data, the inability to afford medications among stroke survivors increased significantly from 8.1% to 12.7% between 1997 and 2004, totaling 76 000 US stroke survivors in 2004. Compared with stroke survivors able to afford medications, those unable to afford them more frequently reported lack of transportation, no health insurance, no usual place of care, income <$20 000, and out-of-pocket medical expenses ≥$2000.[153]
- In 2002, ≈21% of US counties did not have a hospital, 31% lacked a hospital with an ED, and 77% did not have a hospital with neurological services.[154]
- Of patients with ischemic stroke in the California Acute Stroke Pilot Registry, 23.5% arrived at the ED within 3 hours of symptom onset, and 4.3% received thrombolysis. If all patients had called 911 immediately, the expected overall rate of thrombolytic treatment within 3 hours would have increased to 28.6%. If all patients with known onset had arrived within 1 hour and had been treated optimally, 57% could have received thrombolytic treatment.[155]
- Data from the Paul Coverdell National Acute Stroke Registry were analyzed from the 142 hospitals that participated in the 4 registry states. More patients were transported by ambulance than by other means (43.6%). Time of stroke symptom onset was recorded for 44.8% of the patients. Among these patients, 48% arrived at the ED within 2 hours of symptom onset. Significantly fewer blacks (42.4%) arrived within 2 hours of symptom onset than did whites (49.5%), and significantly fewer nonambulance patients (36.2%) arrived within 2 hours of symptom onset than did patients transported by ambulance (58.6%).[156]

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- NHIS data from 1998–2002 found that younger stroke survivors (45–64 years) self-reported worse access to physician care and medication affordability than older stroke survivors. Compared with older patients, younger stroke survivors were more likely to be male (52% versus 47%), to be black (19% versus 10%), and to lack health insurance (11% versus 0.4%). Lack of health insurance was associated with reduced access to care.[157]
- Results from the BASIC project found that women were less likely to arrive at the ED within 3 hours of stroke symptom onset than men (OR, 0.7; 95% CI, 0.5–0.9). Mexican Americans were 40% less likely to arrive by emergency medical services (EMS) than non-Hispanic whites, even after adjustment for age, National Institutes of Health Stroke Scale score, education, history of stroke, and insurance status. Language fluency was not associated with time to hospital arrival or use of EMS. The receipt of tissue-type plasminogen activator was low (1.5%) but did not differ by sex or race.[158]
- A national study of academic medical centers found no change in the proportion of patients with stroke arriving at hospitals within 2 hours of symptom onset between 2001 and 2004 (37% versus 38%); however, the rate of intravenous tissue-type plasminogen activator use increased over this time period (14% to 38%), which suggests system-level improvements in the organization of in-hospital care. In risk-adjusted analyses, black patients were 45% less likely to arrive within 2 hours than white patients.[159]

Operations and Procedures

(See Chart 14-13.)

- In 2010, an estimated 100 000 inpatient endarterectomy procedures were performed in the United States. Carotid endarterectomy is the most frequently performed surgical procedure to prevent stroke (NHDS, NHLBI tabulation).
- Although rates of carotid endarterectomy in the Medicare population decreased slightly between 1998 and 2004, the use of carotid artery stenting increased dramatically.[160]
- The practice of carotid stenting in the United States is expanding, from <3% of all carotid artery revascularization procedures in 1998 to 13% in 2008.[161]
- The randomized Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) compared carotid endarterectomy and stenting for symptomatic and asymptomatic carotid stenosis. There was no overall difference in the primary end point of stroke, MI, or death; however, carotid endarterectomy showed superiority with increasing age, with the crossover point at approximately age 70, and was associated with fewer strokes, which had a greater impact on quality of life than MI.[162,163]

Cost

(See Table 14-1.)

The direct and indirect cost of stroke in 2009 was $38.6 billion (MEPS, NHLBI tabulation).

- The estimated direct medical cost of stroke for 2009 is $22.8 billion. This includes hospital outpatient or office-based provider visits, hospital inpatient stays, ED visits, prescribed medicines, and home health care.[164]
- The mean expense per person for stroke care in the United States in 2009 was estimated at $6018.[164] The mean lifetime cost of ischemic stroke in the United States is estimated at $140 048. This includes inpatient care, rehabilitation, and follow-up care necessary for lasting deficits. (All numbers were converted to 1999 dollars by use of the medical component of the Consumer Price Index.)[165]
- The estimated cost of acute pediatric stroke in the United States was $42 million in 2003. The mean cost of short-term hospital care was $20 927 per discharge.[166]
- After adjustment for routine healthcare costs, the average 5-year cost of a neonatal stroke was $51 719 and that of a childhood stroke was $135 161. Costs among children with stroke continued to exceed those in age-matched control children even in the fifth year by an average of $2016.[167]
- In a study of stroke costs within 30 days of an acute event between 1987 and 1989 in the Rochester Stroke Study, the average cost was $13 019 for mild ischemic strokes and $20 346 for severe ischemic strokes (4 or 5 on the Rankin Disability Scale).[168]
- Inpatient hospital costs for an acute stroke event account for 70% of first-year poststroke costs.[128]
- The largest components of short-term care costs were room charges (50%), medical management (21%), and diagnostic costs (19%).[169]
- Death within 7 days, subarachnoid hemorrhage, and stroke while hospitalized for another condition are associated with higher costs in the first year. Lower costs are associated with mild cerebral infarctions or residence in a nursing home before the stroke.[168]
- Demographic variables (age, sex, and insurance status) are not associated with stroke cost. Severe strokes (National Institutes of Health Stroke Scale score >20) cost twice as much as mild strokes, despite similar diagnostic testing. Comorbidities such as ischemic HD and AF predict higher costs.[169,170]
- The total cost of stroke from 2005 to 2050, in 2005 dollars, is projected to be $1.52 trillion for non-Hispanic whites, $313 billion for Hispanics, and $379 billion for blacks. The per capita cost of stroke estimate is highest in blacks ($25 782), followed by Hispanics ($17 201) and non-Hispanic whites ($15 597). Loss of earnings is expected to be the highest cost contributor in each race/ethnic group.[171]
- In adjusted models that controlled for relevant covariates, the attributable 1-year cost of poststroke aphasia was estimated at $1703 in 2004 dollars.[172]
- Average cost for outpatient stroke rehabilitation services and medications the first year after inpatient rehabilitation discharge was $11 145. The corresponding average yearly cost of medication was $3376, whereas the average cost of yearly rehabilitation service utilization was $7318.[173]

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Table 14-1. Stroke

Population Group	Prevalence, 2010: Age ≥20 y	New and Recurrent Attacks, All Ages	Mortality, 2009: All Ages*	Hospital Discharges, 2010: All Ages	Cost, 2009
Both sexes	6 800 000 (2.8%)	795 000	128 842	1 015 000	$38.6 Billion
Males	3 000 000 (2.6%)	370 000 (46.5%)†	52 073 (40.4%)†	485 000	…
Females	3 800 000 (3.0%)	425 000 (53.5%)†	76 769 (59.6%)†	530 000	…
NH white males	2.4%	325 000‡	43 190	…	…
NH white females	2.9%	365 000‡	65 574	…	…
NH black males	4.3%	45 000‡	6962	…	…
NH black females	4.7%	60 000‡	8916	…	…
Mexican-American males	2.3%	…	…	…	…
Mexican-American females	1.4%	…	…	…	…
Hispanic or Latino	2.8%§	…	7065	…	…
Asian	2.7%§	…	3639‖	…	…
Native Hawaiian and other Pacific Islander	§¶	…	…	…	…
American Indian/ Alaska Native	4.6%§	…	561	…	…

NH indicates non-Hispanic. Ellipses (…) indicate data not available.

*Mortality data for the white, black, Asian or Pacific Islander, and American Indian/Alaska Native populations include deaths of persons of Hispanic and non-Hispanic origin. Numbers of deaths for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated.

†These percentages represent the portion of total stroke incidence or mortality that applies to males vs females.

‡Estimates include Hispanics and non-Hispanics. Estimates for whites include other nonblack races.

§National Health Interview Survey (2011), National Center for Health Statistics; data are weighted percentages for Americans >18 years of age.[174]

‖Includes Chinese, Filipino, Hawaiian, Japanese, and other Asian or Pacific Islander.

¶Estimate considered unreliable or does not meet standards of reliability or precision.

Sources: Prevalence: National Health and Nutrition Examination Survey 2007–2010, National Center for Health Statistics and National Heart, Lung, and Blood Institute. Percentages for racial/ethnic groups are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2010 US population. Incidence: Greater Cincinnati/Northern Kentucky Stroke Study/National Institutes of Neurological Disorders and Stroke data for 1999 provided on August 1, 2007. US estimates compiled by National Heart, Lung, and Blood Institute. See also Kissela et al.[142] Data include children. Mortality: Centers for Disease Control and Prevention/National Center for Health Statistics, 2009 Mortality Multiple Cause–United States, version July 19, 2012. These data represent underlying cause of death only. Mortality data for white and black males and females include Hispanics. Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics. Data include those inpatients discharged alive, dead, or status unknown. Cost: National Heart, Lung, and Blood Institute. Data include estimated direct and indirect costs for 2009.

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Table 14-2. Modifiable Stroke Risk Factors

Factor	Prevalence, %	PAR, %*	RR
Cigarette smoking			
Overall	19.8	12–14†	1.9
Men	22.3		
Women	17.4		
Hypertension		‡	8
Ages 20–34 y			
Men	13.4	99	
Women	6.2	98	
Ages 35–44 y			
Men	23.2	99	
Women	16.5	106	
Ages 45–54 y			
Men	36.2	100	
Women	35.9	103	
Ages 55–64 y			
Men	53.7	100	
Women	55.8	102	
Ages 65–74 y			

Continued

Table 14-2. Continued

Factor	Prevalence, %	PAR, %*	RR
Men	64.7	100	
Women	69.6	101	
Ages ≥75 y			
Men	64.1	100	
Women	76.4	101	
Diabetes	7.3	5–27	1.8–6.0
High total cholesterol	Data calculated for highest quintile (20%) vs lowest quintile	9.1 (5.7–13.8)	1.5 (95% CI, 1.3–1.8)
	Continuous risk for ischemic stroke	…	1.25 per 1-mmol/L (38.7 mg/dL) increase
Low HDL cholesterol			
<40 mg/dL			
Men	35		
Women	15		
	Data calculated for highest quintile (20%) vs lowest quintile	23.7	0.4
<35 mg/dL	26	20.6 (10.1–30.7)	2.00 (95% CI, 1.43–2.70)
	Continuous risk for ischemic stroke		≈0.5–0.6 for each 1-mmol/L increase
AF (nonvalvular)			
Overall age, y			
50–59	0.5	1.5	4.0
60–69	1.8	2.8	2.6
70–79	4.8	9.9	3.3
80–89	8.8	23.5	4.5
Asymptomatic carotid stenosis	2–8	2–7§	2.0
Sickle cell disease	0.25 (of blacks)	…	200–400‖
Postmenopausal hormone therapy	25 (Women 50–74 y of age)	9	1.4
Oral contraceptive use	13 (women 25–44 y)	9.4	2.3
Dietary factors			
Na intake >2300 mg	75–90	Unknown	Unknown
K intake <4700 mg	90–99	Unknown	Unknown
Physical inactivity	25	30	2.7
Obesity			1.39 Stroke death per increase of 5 kg/m^2
Men	33.3		
Women	35.3		
CHD			
Men	8.4	5.8	1.73 (1.68–1.78)
Women	5.6	3.9¶	1.55 (1.17–2.07)
Heart failure			
Men	2.6	1.4	
Women	2.1	1.1¶	
Peripheral arterial disease	4.9	3.0¶	

PAR indicates population attributable risk; RR, relative risk; CI, confidence interval; HDL, high-density lipoprotein; AF, atrial fibrillation; and CHD, coronary heart disease.

*PAR is the proportion of ischemic stroke in the population that can be attributed to a particular risk factor (see Goldstein et al[62] for formula).

†PAR is for stroke deaths, not ischemic stroke incidence.

‡PAR percent=100×{[prevalence (RR−1)/prevalence (RR−1) +1]}.

§Calculated on the basis of referenced data provided in the table or text.

‖Relative to stroke risk in children without sickle cell disease.

¶Calculated on the basis of point estimates of referenced data provided in the table. For peripheral arterial disease, calculation was based on average RR for men and women.

Adapted from Goldstein et al.[62]

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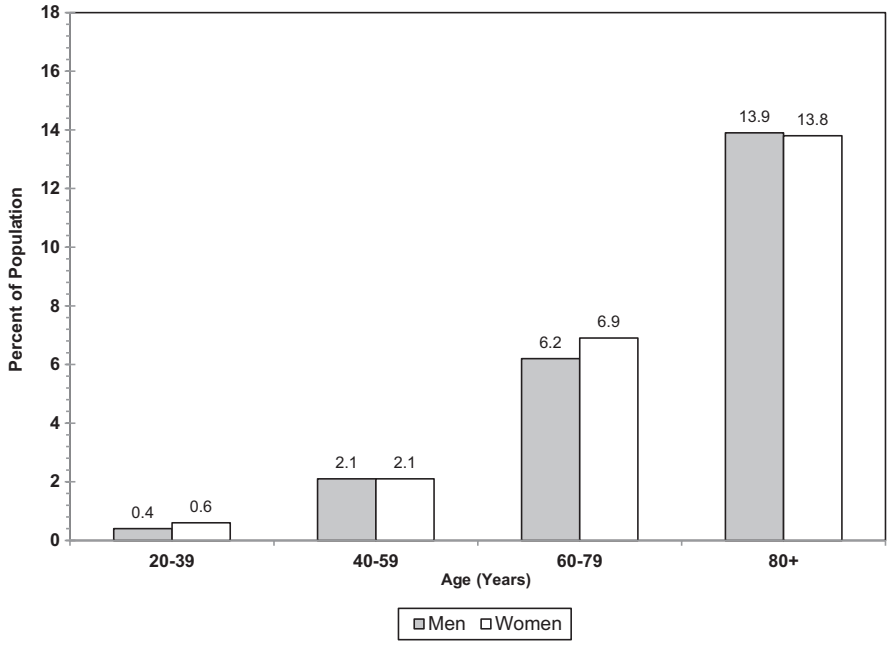


Chart 14-1. Prevalence of stroke by age and sex (National Health and Nutrition Examination Survey: 2007–2010). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

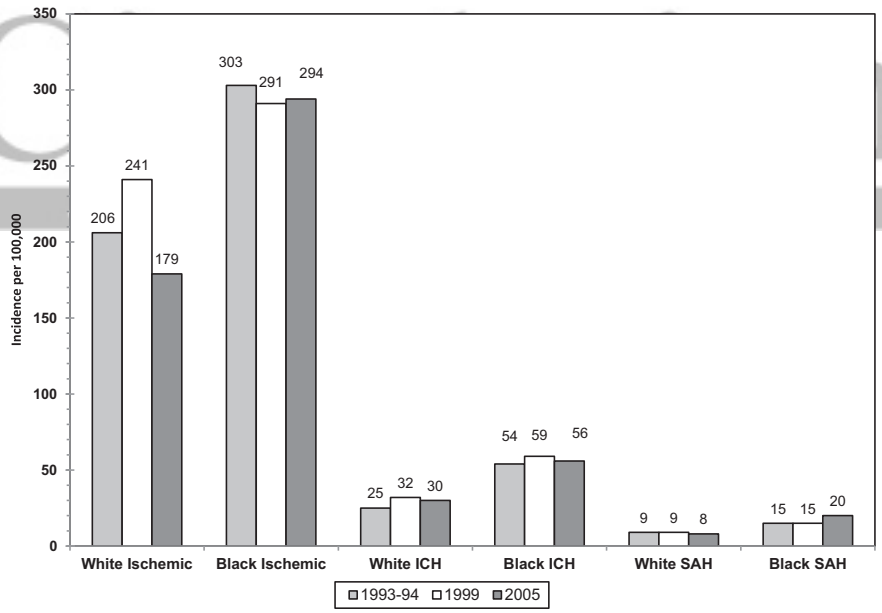


Chart 14-2. Annual age-adjusted incidence of first-ever stroke by race. Hospital plus out-of-hospital ascertainment, 1993 to 1994, 1999, and 2005. ICH indicates intracerebral hemorrhage; SAH, subarachnoid hemorrhage. Data derived from Kleindorfer et al.[9]

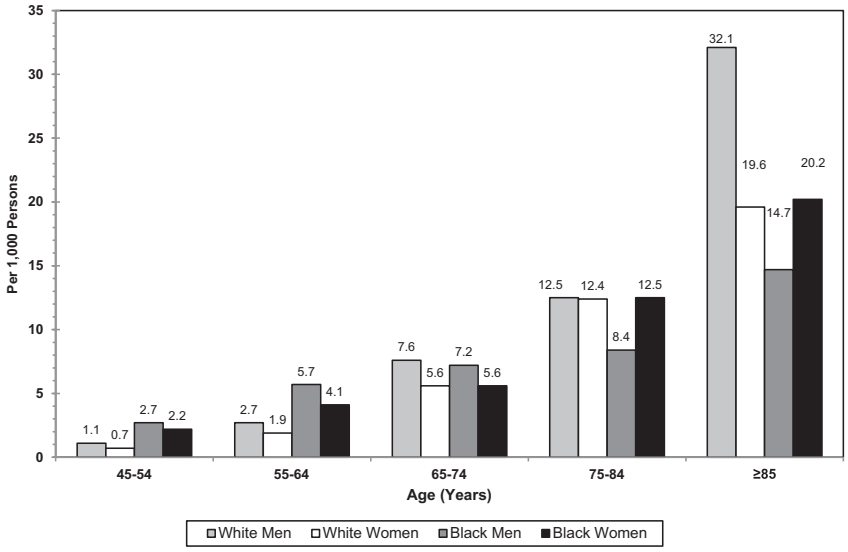


Chart 14-3. Annual rate of first cerebral infarction by age, sex, and race (Greater Cincinnati/Northern Kentucky Stroke Study: 1999). Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable. Source: Unpublished data from the Greater Cincinnati/Northern Kentucky Stroke Study.

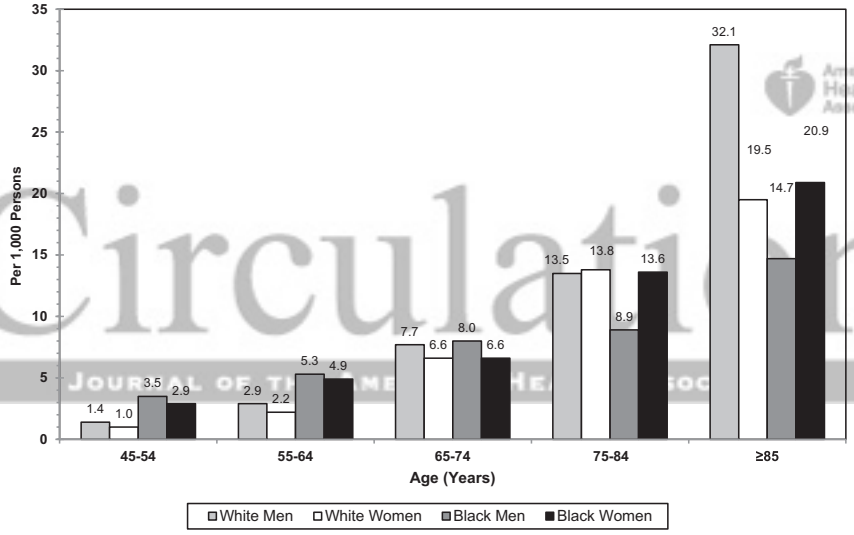


Chart 14-4. Annual rate of all first-ever strokes by age, sex, and race (Greater Cincinnati/Northern Kentucky Stroke Study: 1999). Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable.

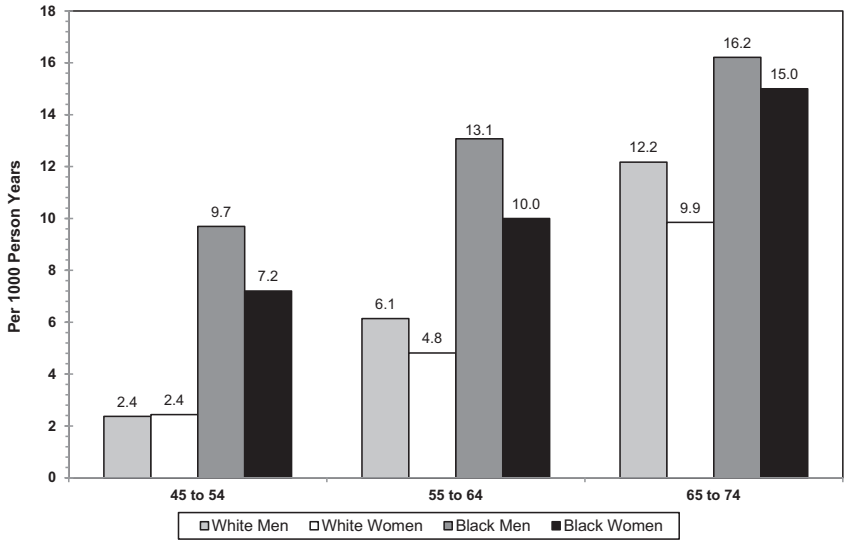


Chart 14-5. Age-adjusted incidence of stroke/transient ischemic attack* by race and sex, ages 45 to 74 years, Atherosclerosis Risk in Communities study cohort, 1987–2001. Data derived from the National Heart, Lung, and Blood Institute, *Incidence and Prevalence: 2006 Chart Book*.[175]

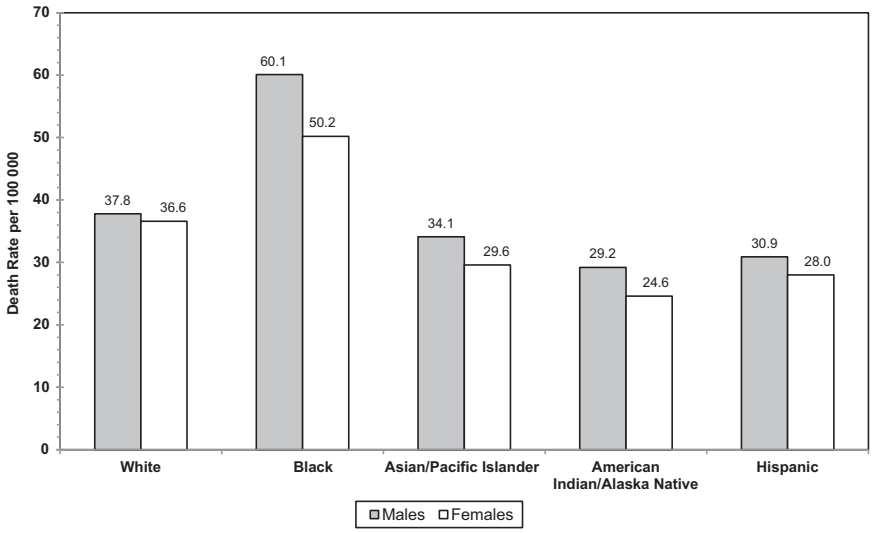


Chart 14-6. Age-adjusted death rates for stroke by sex and race/ethnicity, 2009. Death rates for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated. Stroke includes *International Classification of Diseases, 10th Revision* codes I60 to I69 (cerebrovascular disease). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

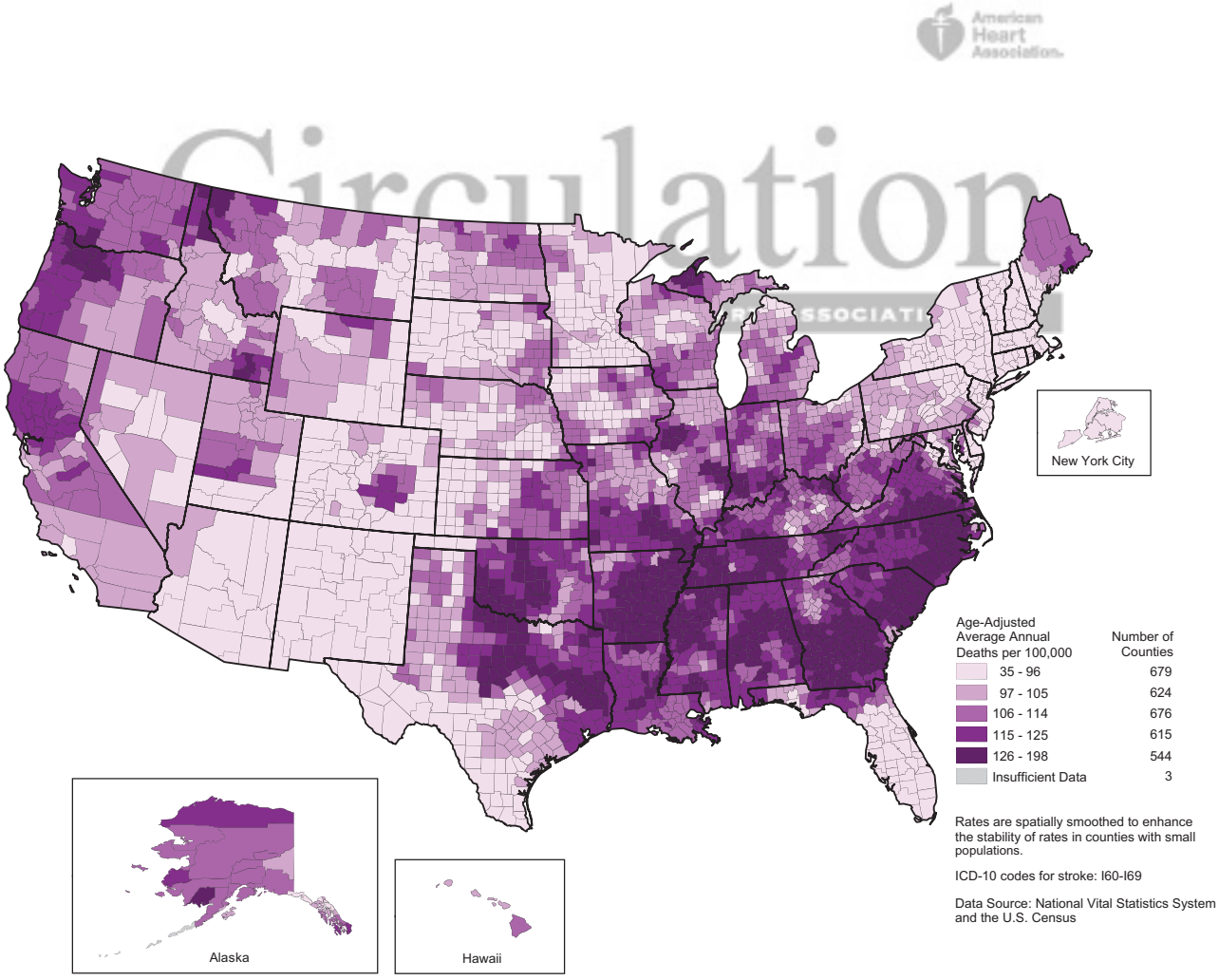
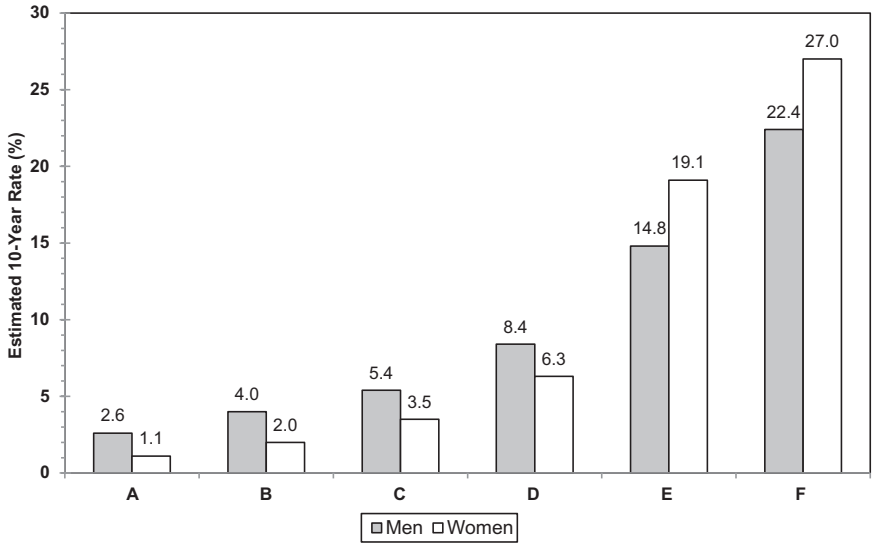


Chart 14-7. Stroke death rates, 2000 through 2006. Adults ≥35 years of age, by county. Rates are spatially smoothed to enhance the stability of rates in counties with small populations. *International Classification of Diseases, 10th Revision* codes for stroke: I60–I69. Data source: National Vital Statistics System and the US Census Bureau.



	A	B	C	D	E	F
Blood Pressure*	95-105	138-148	138-148	138-148	138-148	138-148
Diabetes	No	No	Yes	Yes	Yes	Yes
Cigarette Smoking	No	No	No	Yes	Yes	Yes
Prior AF	No	No	No	No	Yes	Yes
Prior CVD	No	No	No	No	No	Yes

* - Closest ranges for women are : 95-104 and 115-124.

Chart 14-8. Estimated 10-year stroke risk in adults 55 to 84 years of age according to levels of various risk factors (Framingham Heart Study). AF indicates atrial fibrillation; CVD, cardiovascular disease. Data derived from Wolf et al[176] with permission of the publisher. Copyright © 1991, American Heart Association.

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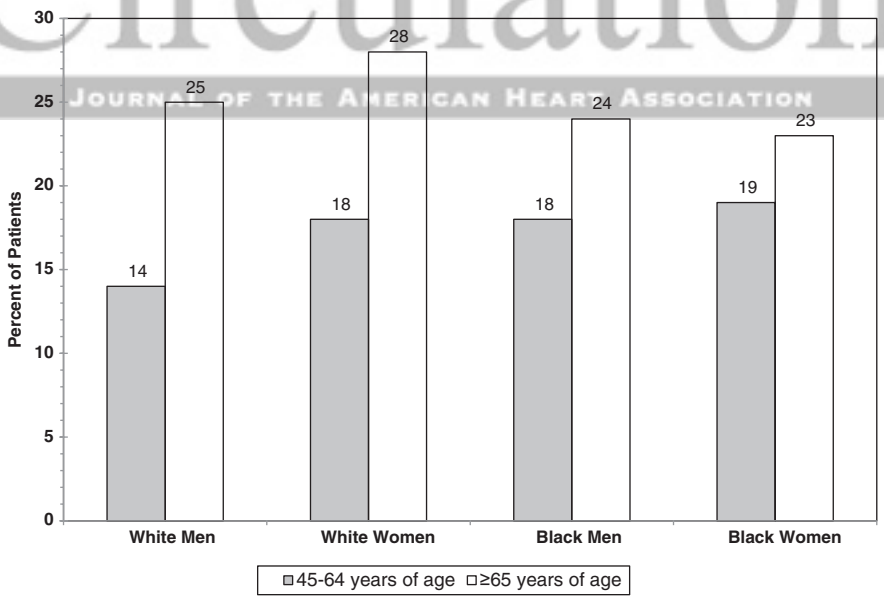

Chart 14-9. Proportion of patients dead 1 year after first stroke. Source: Pooled data from the Framingham Heart Study, Atherosclerosis Risk in Communities study, and Cardiovascular Health Study of the National Heart, Lung, and Blood Institute.

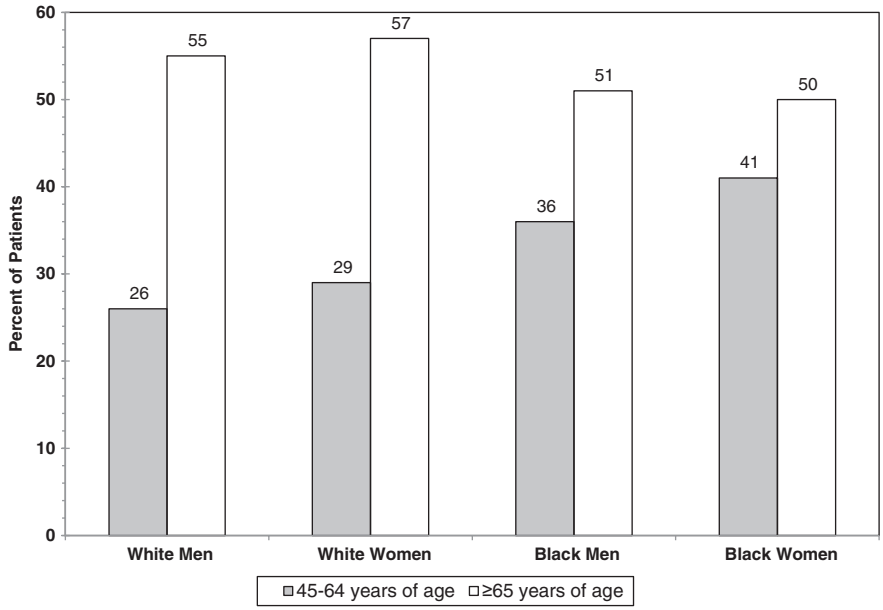

Chart 14-10. Proportion of patients dead within 5 years after first stroke. Source: Pooled data from the Framingham Heart Study, Atherosclerosis Risk in Communities study, and Cardiovascular Health Study of the National Heart, Lung, and Blood Institute.

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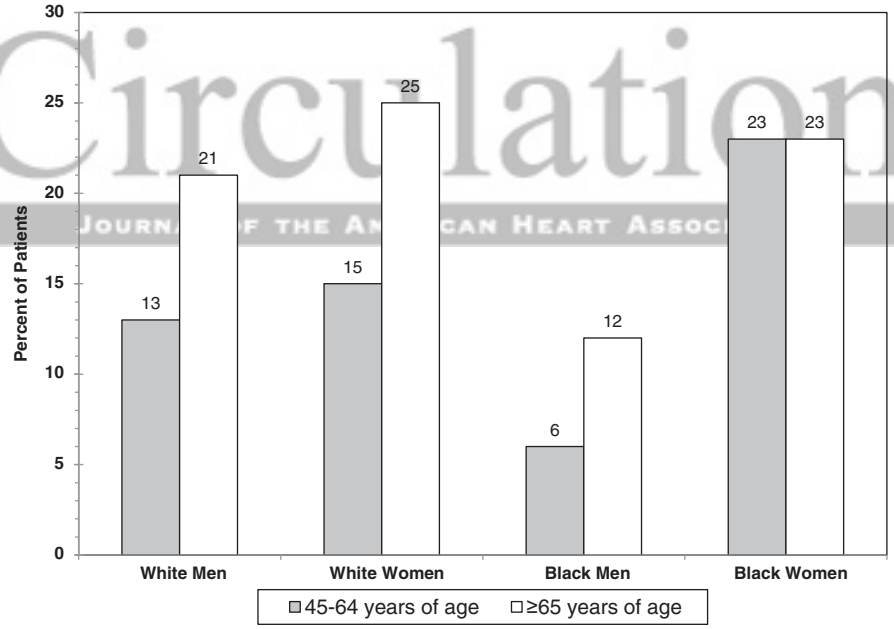

Chart 14-11. Proportion of patients with recurrent stroke in 5 years after first stroke. Source: Pooled data from the Framingham Heart Study, Atherosclerosis Risk in Communities study, and Cardiovascular Health Study of the National Heart, Lung, and Blood Institute.

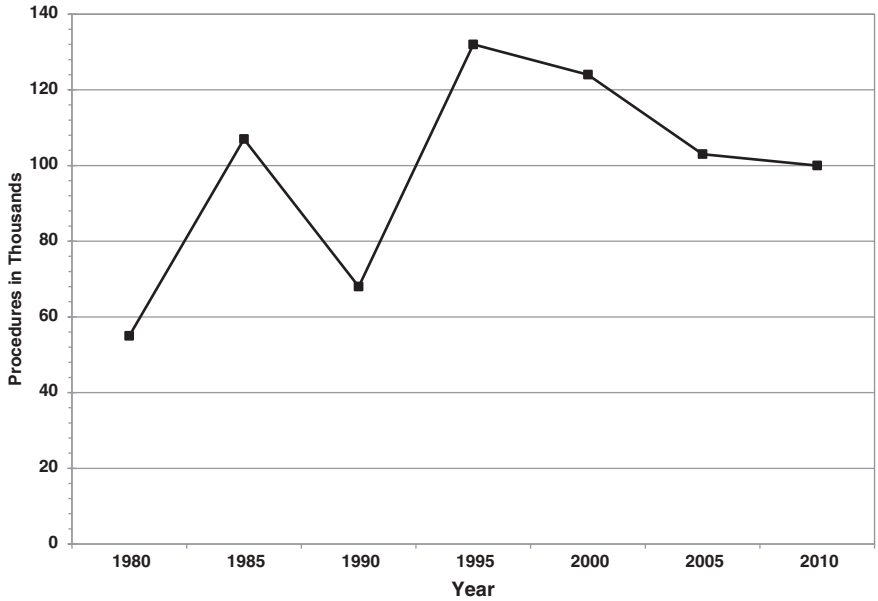

Chart 14-12. Trends in carotid endarterectomy procedures (United States: 1980–2010). Source: National Hospital Discharge Survey/ National Center for Health Statistics and National Heart, Lung, and Blood Institute.

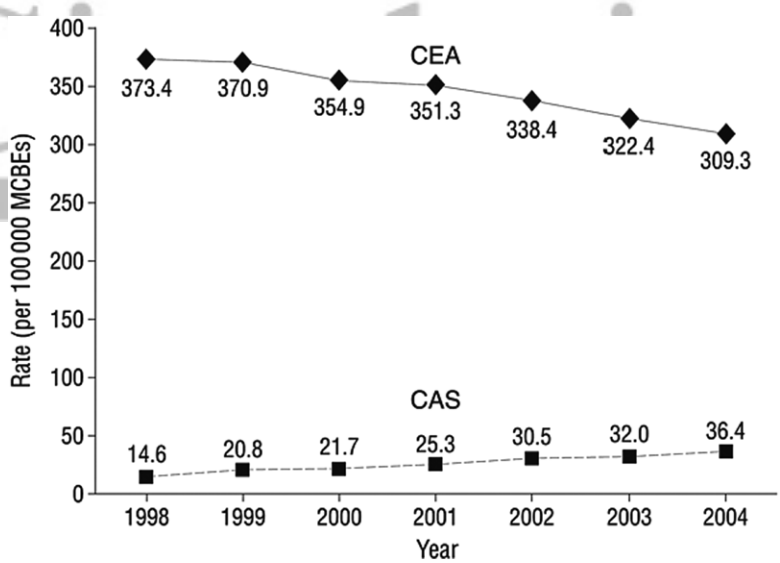

Chart 14-13. Trends in carotid revascularization procedures. MCBE indicates Medicare beneficiaries; CEA, carotid endarterectomy; and CAS, carotid artery stenting. Reproduced with permission from Goodney et al.[160] Copyright © 2008, American Medical Association. All rights reserved.

15. Congenital Cardiovascular Defects and Kawasaki Disease

ICD-9 745 to 747, ICD-10 Q20 to Q28. See Tables 15-1 through 15-4.

Congenital cardiovascular defects, also known as congenital heart defects, are structural problems that arise from abnormal formation of the heart or major blood vessels. ICD-9 lists 25 congenital heart defects codes, of which 21 designate specific anatomic or hemodynamic lesions.

Defects range in severity from tiny pinholes between chambers that may resolve spontaneously to major malformations that can require multiple surgical procedures before school age and may result in death in utero, in infancy, or in childhood. The common complex defects include the following:

- Tetralogy of Fallot (TOF)
- Transposition of the great arteries (TGA)
- Atrioventricular (AV) septal defects
- Coarctation of the aorta
- Hypoplastic left heart syndrome (HPLHS)

Congenital heart defects are serious and common conditions that have a significant impact on morbidity, mortality, and healthcare costs in children and in adults.[1–4] As health outcomes improve and survival increases for children living with congenital HD, the burden of care is shifting toward adult populations.[5]

Incidence

The most commonly reported incidence of congenital heart defects in the United States is between 4 and 10 per 1000, clustering around 8 per 1000 live births.[6,7] Continental variations in birth prevalence have been reported, from 6.9 per 1000 births in Europe to 9.3 per 1000 in Asia.[8] Variations in reported number of incident cases are largely accounted for by the age at detection and the method of diagnosis. Major defects may be apparent in the prenatal or neonatal period, but minor defects may not be detected until adulthood. Detection rates have increased since the advent of cardiac ultrasound.[4] Thus, true measures of the incidence of congenital HD would need to record new cases of defects that present from fetal life onward. Because most estimates are available for new cases detected between birth and the first year of life, birth prevalence is the best proxy for incident congenital heart defects. These are typically reported as cases per 1000 live births per year and do not distinguish between tiny defects that resolve without treatment and major malformations. To distinguish more serious defects, some studies also report new cases of sufficient severity to require an invasive procedure or that result in death within the first year of life. Despite the absence of true incidence figures, some data are available and are provided in Table 15-2.

Abbreviations Used in Chapter 15

ASD	atrial septal defect
AV	atrioventricular
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CI	confidence interval
DM	diabetes mellitus
HD	heart disease
HPLHS	hypoplastic left heart syndrome
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MACDP	Metropolitan Atlanta Congenital Defects Program
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHLBI	National Heart, Lung, and Blood Institute
RR	relative risk
TGA	transposition of the great arteries
TOF	tetralogy of Fallot
VSD	ventricular septal defect

- Using population-based data from the Metropolitan Atlanta Congenital Defects Program (MACDP) in metropolitan Atlanta, GA, congenital heart defects occurred in 1 of every 111 to 125 births (live, still, or >20 weeks' gestation) from 1995 to 1997 and from 1998 to 2005, with variations in sex and racial distribution of some lesions.[4,6]
- Data collected in Alberta, Canada, found the total prevalence of CHD to be 12.42 per 1000 total births (live, still, or >20 weeks' gestation).[9]
- The National Birth Defects Prevention Network for 13 states from 2004 to 2006 showed the average prevalence of 21 selected major birth defects. These data indicated that there are >6100 estimated annual cases of 5 cardiovascular defects: truncus arteriosus (0.7/10 000 births), TGA (3.0/10 000 births), TOF (4.0/10 000 births), AV septal defect (4.7/10 000 births), and HPLHS (2.3/10 000 births).[10]
- Analysis of contemporary birth cohorts with MACDP data revealed that the most common defects at birth were ventricular septal defect (VSD; 4.2/1000 births), atrial septal defect (ASD; 1.3/1000 births), valvar pulmonic stenosis (0.6/1000 births); TOF (0.5/1000 births), aortic coarctation (0.4/1000 births), AV septal defect (0.4/1000 births), and TGA (0.2/1000 births).[6,11]
- An estimated minimum of 32 000 infants are expected to be affected with congenital HD each year in the United States. Of these, an approximate 25%, or 2.4 per 1000 live births, require invasive treatment in the first year of life.[1]
- Estimates also are available for bicuspid aortic valves, which occur in 13.7 per 1000 people; these defects may not require treatment in infancy but can cause problems later in adulthood.[12]

Prevalence

(See Tables 15-1 through 15-3.)

The 32nd Bethesda Conference estimated that the total number of adults living with congenital HD in the United States in 2000 was 800 000.[2,3] In the United States, 1 in 150 adults are expected to have some form of congenital HD.[3] In population data from Canada, the measured prevalence of congenital cardiac defects in the general population was 11.89 per 1000 children and 4.09 per 1000 adults in the year 2000.[13] Extrapolated to the US population in the same

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year, this yields published estimates of 859 000 children and 850 000 adults for the year 2000.[11] The expected growth rates of the congenital heart defects population vary from 1% to 5% per year depending on the age and distribution of lesions.[2,13]

Estimates of the distribution of lesions in the congenital heart defects population using available data vary with assumptions made. If all those born with lesions between 1940 and 2002 were treated, there would be 750 000 survivors with simple lesions, 400 000 with moderate lesions, and 180 000 with complex lesions; in addition, there would be 3.0 million subjects alive with bicuspid aortic valves.[14] Without treatment, the number of survivors in each group would be 400 000, 220 000, and 30 000, respectively. The actual numbers surviving are projected to be between these 2 sets of estimates as of 1 decade ago.[14] Using measurements from population data in Canada, the prevalence of severe forms of congenital heart defects increased 85% in adults and 22% in children from 1985 to 2000.[13] The most common types of defects in children are (at a minimum) VSD, 620 000 people; ASD, 235 000 people; valvular pulmonary stenosis, 185 000 people; and patent ductus arteriosus, 173 000 people.[14] The most common lesions seen in adults are ASD and TOF.[2]

Risk Factors

- Numerous intrinsic and extrinsic nongenetic risk factors contribute to CHD.[15]
- Attributable risks or fractions have been shown to include paternal anesthesia in TOF (3.6%), sympathomimetic medication for coarctation of the aorta (5.8%), pesticides for VSD (5.5%), and solvents for HPLHS (4.6%).[16]
- A study of infants born with heart defects unrelated to genetic syndromes who were included in the National Birth Defects Prevention Study found that women who reported smoking in the month before becoming pregnant or in the first trimester were more likely to give birth to a child with a septal defect. Compared with the infants of mothers who did not smoke during pregnancy, infants of mothers who were heavy smokers (≥25 cigarettes daily) were twice as likely to have a septal defect.[17]
- Data from the Baltimore-Washington Infant Study reported that maternal smoking during the first trimester of pregnancy was associated with at least a 30% increased risk of the following lesions in the fetus: ASD, pulmonary valvar stenosis, truncus arteriosus, and TGA.[18]
- Associations between exposure to air pollutants during first-trimester pregnancy and risks of congenital heart defects were documented from 1986 to 2003 by the MACDP that related carbon monoxide, nitrogen dioxide, and sulfur dioxide measurements to the risk of ASD, VSD, TGA, and TOF.[19]
- The results of a population-based study examining pregnancy obesity found a weak to moderate positive association of maternal obesity with 7 of 16 categories of birth defects, including heart defects.[20]
- Although folic acid supplementation is recommended during pregnancy to potentially reduce the risk of congenital heart defects,[15] there has been only 1 US population-based case-control study, performed with the Baltimore-Washington Infant Study between 1981 and 1989, that showed an inverse relationship between folic acid use and the risk of TGA.[21] A study from Quebec, Canada, that analyzed 1.3 million births from 1990 to 2005 found a significant 6% per year reduction in severe congenital heart defects using a time-trend analysis before and after public health measures were instituted that mandated folic acid fortification of grain and flour products in Canada.[22]
- Pregestational DM was significantly associated with cardiac defects, both isolated and multiple. Gestational DM was associated with a limited group of birth defects.[23]
- Paternal risk of occupational exposure was addressed in a study published in 2012 documenting a higher incidence of congenital HD with paternal exposure to phthalates.[24]

Mortality

(See Table 15-1.)

Mortality related to congenital cardiovascular defects in 2009 was 3189 deaths. Any-mention mortality related to congenital cardiovascular defects in 2009 was 5051 deaths.[21]

- In 2009, congenital cardiovascular defects were the most common cause of infant death resulting from birth defects; 26.6% of infants who died of a birth defect had a heart defect.[25]
- The death rate attributable to congenital heart defects in the United States has continued to decline from 1979 to 1997 and from 1999 to 2006. Age-adjusted death rates attributable to all congenital heart defects declined 21% to 39%, and deaths tended to occur at progressively older ages. Nevertheless, mortality in infants <1 year of age continues to account for nearly half of the deaths.[18, 26]
- When CDC data on multiple causes of death were used to examine mortality in cyanotic and acyanotic lesions between 1979 and 2005, all-age death rates had declined by 60% for VSD and 40% for TOF.[27]
- In population-based data from Canada, 8123 deaths occurred in 71 686 congenital HD patients followed up for nearly 1 million patient-years. Overall mortality decreased by 31%, and the median age of death increased from 2 to 23 years between 1987 and 2005.[28]
- The 2009 age-adjusted death rate (deaths/100 000 people) attributable to congenital cardiovascular defects was 1.0. Death rates were 1.1 for white males, 1.4 for black males, 0.9 for white females, and 1.2 for black females. Crude infant mortality rates (<1 year of age) were 31.4 for white infants and 42.2 for black infants.[25]
- Mortality after congenital heart surgery also differs between races/ethnicities after adjustment for access to care. The risk of in-hospital mortality for minority patients compared with white patients is 1.22 (95% CI, 1.05–1.41) for Hispanics, 1.27 (95% CI, 1.09–1.47) for non-Hispanic blacks, and 1.56 (95% CI, 1.37–1.78) for other non-Hispanics.[29]
- According to CDC multiple-cause death data, from 1999 to 2006, sex differences in mortality over time varied with age. Between the ages of 18 and 34 years, mortality over time decreased significantly in females but not in males.[30]
- On the basis of data from the Healthcare Cost and Utilization Project's Kids' Inpatient Database from 2000, 2003, and 2006, male children had more congenital heart defect surgeries in infancy, more high-risk surgeries, and more procedures to correct multiple congenital heart

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defects. Female infants with high-risk congenital heart defects had a 39% higher adjusted mortality.[26]

- In 2007, 189 000 life-years were lost before 55 years of age because of deaths attributable to congenital cardiovascular defects. This is almost as many life-years as were lost from leukemia and asthma combined (NHLBI tabulation of NCHS mortality data).
- Data from the Pediatric Heart Network conducted in 15 North American centers revealed that even in lesions associated with the highest mortality among congenital lesions, such as HPLHS, aggressive palliation can lead to an increase in the 12-month survival rate, from 64% to 74%.[31]
- Data analysis for the Society of Thoracic Surgeons' Congenital Heart Surgery Database, a voluntary registry with self-reported data for a 4-year cycle (2007–2010) from 103 centers performing congenital heart surgery (98 from the United States, 3 from Canada, and 1 from Japan),[32] showed that of 95 357 total operations, the overall aggregate hospital discharge mortality rate was 3.5%.[33] Specifically, the mortality rate was 10.1% for neonates (0–30 days of age),[34] 2.9% for infants (31 days to 1 year of age),[35] 1.1% for children (>1 year to 18 years of age),[36] and 1.9% for adults (>18 years of age).[37]
- Using the Nationwide Inpatient Sample 1988–2003, mortality was examined for 12 congenital heart defect procedures. A total of 30 250 operations were identified, which yielded a national estimate of 152 277±7875 operations. Of these, 27% were performed in patients ≥18 years of age. The overall in-hospital mortality rate for adult patients with congenital heart defects was 4.71% (95% CI, 4.19%–5.23%), with a significant reduction in mortality observed when surgery was performed on such adult patients by pediatric versus nonpediatric heart surgeons (1.87% versus 4.84%; $P<0.0001$).[38]

Hospitalizations

(See Table 15-1.)

In 2004, birth defects accounted for >139 000 hospitalizations, representing 47.4 stays per 100 000 people. Cardiac and circulatory congenital anomalies accounted for 34% of all hospital stays for birth defects. Although the most common congenital lesions were shunts, including patent ductus arteriosus, VSDs, and ASDs, TOF accounted for a higher proportion of in-hospital death than any other birth defect. Between 1997 and 2004, hospitalization rates increased by 28.5% for cardiac and circulatory congenital anomalies.[39]

Cost

- From data from the Healthcare Cost and Utilization Project 2003 Kids' Inpatient Database and 2003 information on birth defects in the Congenital Malformations Surveillance Report, it was found that the most expensive average neonatal hospital charges were for 2 congenital heart defects: HPLHS ($199 597) and common truncus arteriosus ($192 781). Two other cardiac defects, coarctation of the aorta and TGA, were associated with average hospital charges in excess of $150 000. For the 11 selected cardiovascular congenital defects (of 35 birth defects considered), there were 11 578 hospitalizations in 2003 and 1550 in-hospital deaths (13.4%). Estimated total hospital charges for these 11 conditions were $1.4 billion.[40]
- In 2004, hospital costs for congenital cardiovascular defect conditions totaled $2.6 billion. The highest aggregate costs were for stays related to cardiac and circulatory congenital anomalies, which accounted for ≈$1.4 billion, more than half of all hospital costs for birth defects.[39]
- Data from 1941 neonates with HPLHS showed a median cost of $99 070 for stage 1 palliation (Norwood or Sano procedure), $35 674 for stage 2 palliation (Glenn procedure), $36 928 for stage 3 palliation (Fontan procedure), and $289 292 for transplantation.[41]
- In 2124 patients undergoing congenital heart operations between 2001 and 2007, total costs for the surgeries were $12 761 (ASD repair), $18 834 (VSD repair), $28 223 (TOF repair), and $55 430 (arterial switch operation).[42]

Kawasaki Disease

ICD-9 446.1; ICD-10 M30.3.

Mortality—5. Any-mention mortality—7.

- The incidence of Kawasaki disease is rising worldwide, including in the United States, where the hospitalization rate rose from 17.5/100 000 children aged <5 years to 20.8/100 000 children <5 years in 2006.[43] In 2010, Japan experienced its highest-ever incidence rate of 239.6 cases per 100 000 children aged <4 years.[44]
- US states with higher Asian American populations have higher rates of Kawasaki disease; for example, rates are 2.5-fold higher in Hawaii than in the continental United States.[45]
- Boys have a 1.5-fold higher incidence of Kawasaki disease than girls.[45]
- An estimated 5523 hospitalizations for Kawasaki disease occurred in the United States in 2006, with a mean patient age of 3 years. Race-specific incidence rates indicate that Kawasaki disease is most common among Americans of Asian and Pacific Island descent (30.3/100 000 children <5 years of age), occurs with intermediate frequency in non-Hispanic blacks (17.5/100 000 children <5 years of age) and Hispanics (15.7/100 000 children <5 years of age), and is least common in whites (12.0/100 000 children <5 years of age).[46]
- Kawasaki disease is more common during the winter and early spring months, except in Hawaii, where no clear seasonal trend is seen[47]; it occurs more often in boys than girls at a ratio of ≈1.5:1, and 76.8% of children with Kawasaki disease are <5 years of age.[43,45,46]
- Data from the Kids' Inpatient Database[45] show a hospitalization rate for Kawasaki disease for children <5 years of

  —19 per 100 000 in 2009
  —20.8 per 100 000 in 2006
  —17.3 per 100 000 in 2003
  —17.5 per 100 000 in 2000

- Addition of prednisolone to the standard regimen of intravenous immunoglobulin for patients with severe Kawasaki disease appears to result in a substantial reduction in the incidence of coronary artery anomalies (RR, 0.20; 95% CI, 0.12–0.28).[48]

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Table 15.1. Congenital Cardiovascular Defects

Population Group	Estimated Prevalence, 2002, All Ages	Mortality, 2009, All Ages	Hospital Discharges, 2009, All Ages
Both sexes	650 000 to 1.3 million[14]	3189	52 000
Males	…	1754 (55.0%)*	25 000
Females	…	1435 (45.0%)*	27 000
NH white males	…	1370	…
NH white females	…	1086	…
NH black males	…	304	…
NH black females	…	268	…
Asian	…	126	…
American Indian/Alaska Native	…	35	…

Ellipses (…) indicate data not available; NH, non-Hispanic.

*These percentages represent the portion of total congenital cardiovascular mortality that is for males vs females.

Sources: Mortality: Centers for Disease Control and Prevention/National Center for Health Statistics, 2009 Mortality Multiple Cause—United States, version July 19, 2012. These data represent underlying cause of death only; data include Hispanics. Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics; data include those inpatients discharged alive, dead, or status unknown.

Table 15.2. Annual Birth Prevalence of Congenital Cardiovascular Defects in the United States[1,4,7,12,49,50]

Type of Presentation	Rate per 1000 Live Births	Estimated Number (Variable With Yearly Birth Rate
Fetal loss	Unknown	Unknown
Invasive procedure during the first year	2.4	9200
Detected during first year*	8	36 000
Bicuspid aortic valve	13.7	54 800

*Includes stillbirths and pregnancy termination at <20 weeks' gestation; includes some defects that resolve spontaneously or do not require treatment.

Table 15.3. Estimated Prevalence of Congenital Cardiovascular Defects and Percent Distribution by Type, United States, 2002* (in Thousands)

	Prevalence, n			Percent of Total		
Type	Total	Children	Adults	Total	Children	Adults
Total	994	463	526	100	100	100
VSD†	199	93	106	20.1	20.1	20.1
ASD	187	78	109	18.8	16.8	20.6
Patent ductus arteriosus	144	58	86	14.2	12.4	16.3
Valvular pulmonic stenosis	134	58	76	13.5	12.6	14.4
Coarctation of aorta	76	31	44	7.6	6.8	8.4
Valvular aortic stenosis	54	25	28	5.4	5.5	5.2
TOF	61	32	28	6.1	7	5.4
AV septal defect	31	18	13	3.1	3.9	2.5
TGA	26	17	9	2.6	3.6	1.8
Hypoplastic right heart syndrome	22	12	10	2.2	2.5	1.9
Double-outlet right ventricle	9	9	0	0.9	1.9	0.1
Single ventricle	8	6	2	0.8	1.4	0.3
Anomalous pulmonary venous connection	9	5	3	0.9	1.2	0.6
Truncus arteriosus	9	6	2	0.7	1.3	0.5
HPLHS	3	3	0	0.3	0.7	0
Other	22	12	10	2.1	2.6	1.9

VSD indicates ventricular septal defect; ASD, atrial septal defect; TOF, tetralogy of Fallot; AV, atrioventricular; TGA, transposition of the great arteries; and HPLHS, hypoplastic left heart syndrome.

*Excludes an estimated 3 million bicuspid aortic valve prevalence (2 million in adults and 1 million in children).

†Small VSD, 117 000 (65 000 adults and 52 000 children); large VSD, 82 000 (41 000 adults and 41 000 children).

Source: Reprinted from Hoffman et al,[14] with permission from Elsevier. Average of the low and high estimates, two thirds from low estimate.[14]

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Table 15.4. Surgery for Congenital Heart Disease

	Sample	Population, Weighted
Surgery for congenital heart disease	14 888	25 831
Deaths	736	1253
Mortality rate, %	4.9	4.8
By sex (81 missing in sample)		
Male	8127	14 109
Deaths	420	714
Mortality rate, %	5.2	5.1
Female	6680	11 592
Deaths	315	539
Mortality rate, %	4.7	4.6
By type of surgery		
ASD secundum surgery	834	1448
Deaths	3	6
Mortality rate, %	0.4	0.4
Norwood procedure for HPLHS	161	286
Deaths	42	72
Mortality rate, %	26.1	25.2

ASD indicates atrial septal defect; HPLHS, hypoplastic left heart syndrome.

In 2003, 25 000 cardiovascular operations for congenital cardiovascular defects were performed on children <20 years of age. Inpatient mortality rate after all types of cardiac surgery was 4.8%. Nevertheless, mortality risk varies substantially for different defect types, from 0.4% for ASD repair to 25.2% for first-stage palliation for HPLHS. Fifty-five percent of operations were performed in males. In unadjusted analysis, mortality after cardiac surgery was somewhat higher for males than for females (5.1% vs 4.6%).

Source: Analysis of 2003 Kids' Inpatient Database[38] and personal communication with Kathy Jenkins, MD, Children's Hospital of Boston, MA, October 1, 2006.

16. Disorders of Heart Rhythm

See Table 16-1.

Bradyarrhythmias

ICD-9 426.0, 426.1, 427.81; ICD-10 I44.0 to I44.3, I49.5.
Mortality—791. Any-mention mortality—4678. Hospital discharges—110 000.

AV Block

Prevalence and Incidence

- The prevalence of first-degree AV block in NHANES III is 3.7% (313 of 8434 participants with electrocardiographic data readable for PR interval).[1]
- In a healthy sample of subjects from the ARIC study (mean age 53 years), the prevalence of first-degree AV block was 7.8% in black men, 3.0% in black women, 2.1% in white men, and 1.3% in white women.[2] Lower prevalence estimates were noted in the relatively younger population (mean age 45 years) of the CARDIA study at its year 20 follow-up examination: 2.6% in black men, 1.9% in black women, 1.2% in white men, and 0.1% in white women.[2]
- Mobitz II second-degree AV block is rare in healthy individuals (≈0.003%), whereas Mobitz I (Wenckebach) is observed in 1% to 2% of healthy young people, especially during sleep.[3]
- The prevalence of third-degree AV block in the general adult population is ≈0.02% to 0.04%.[4,5]
- Third-degree AV block is very rare in apparently healthy individuals. Johnson et al[6] found only 1 case among >67 000 symptom-free individuals; Rose et al,[7] in their study of >18 000 civil servants, did not find any cases. On the other hand, among 293 124 patients with DM and 552 624 with hypertension enrolled with Veterans Health Administration hospitals, third-degree AV block was present in 1.1% and 0.6% of those patients, respectively.[8]
- Congenital complete AV block is estimated to occur in 1 of 15 000 to 25 000 live births.[3]

Risk Factors

- Although first-degree AV block and Mobitz type I second-degree AV block can occur in apparently healthy individuals, presence of Mobitz II second-degree or third-degree AV block usually indicates underlying HD, including CHD and HF.[3]
- Reversible causes of AV block include electrolyte abnormalities, drug-induced AV block, perioperative AV block attributable to hypothermia, or inflammation near the AV conduction system after surgery in this region. Some conditions may warrant pacemaker implantation because of the possibility of disease progression even if the AV block reverses transiently (eg, sarcoidosis, amyloidosis, and neuromuscular diseases).[9]
- Long sinus pauses and AV block can occur during sleep apnea. In the absence of symptoms, these abnormalities are reversible and do not require pacing.[10]

Prevention

- Detection and correction of reversible causes of acquired AV block could be of potential importance in preventing symptomatic bradycardia and other complications of AV block.[9]

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Abbreviations Used in Chapter 16

AED	automated external defibrillator	HCM	hypertrophic cardiomyopathy
AHA	American Heart Association	HD	heart disease
AF	atrial fibrillation	HF	heart failure
AMI	acute myocardial infarction	HR	hazard ratio
ARIC	Atherosclerosis Risk in Communities study	ICD-9	*International Classification of Diseases, 9th Revision*
AV	atrioventricular	ICD-10	*International Classification of Diseases, 10th Revision*
BMI	body mass index	MI	myocardial infarction
BP	blood pressure	NCHS	National Center for Health Statistics
CABG	cardiac revascularization (coronary artery bypass graft)	NHANES	National Health and Nutrition Examination Survey
CAD	coronary artery disease	NHDS	National Hospital Discharge Survey
CARDIA	Coronary Artery Risk Development in Young Adults	NHLBI	National Heart, Lung, and Blood Institute
CHD	coronary heart disease	OR	odds ratio
CHS	Cardiovascular Health Study	PA	physical activity
CI	confidence interval	PAR	population attributable risk
CKD	chronic kidney disease	PVT	polymorphic ventricular tachycardia
CVD	cardiovascular disease	RR	relative risk
DM	diabetes mellitus	SBP	systolic blood pressure
ECG	electrocardiogram	SNP	single nucleotide polymorphism
ED	emergency department	SVT	supraventricular tachycardia
EMS	emergency medical services	TdP	torsade de pointes
FHS	Framingham Heart Study	VF	ventricular fibrillation
GWTG	Get With The Guidelines	VT	ventricular tachycardia

- In utero detection of congenital AV block is possible by echocardiography.[11]

Aftermath

- In the FHS, PR interval prolongation (>200 ms) was associated with an increased risk of AF (HR, 2.06; 95% CI, 1.36–3.12),[12,13] pacemaker implantation (HR, 2.89; 95% CI, 1.83–4.57),[13] and all-cause mortality (HR, 1.44; 95% CI, 1.09–1.91).[13] Compared with individuals with a PR ≤200 ms, individuals with PR interval >200 ms had an absolute increased risk per year of 1.04% for AF, 0.55% for pacemaker implantation, and 2.05% for death.
- Patients with abnormalities of AV conduction may be asymptomatic or may experience serious symptoms related to bradycardia, ventricular arrhythmias, or both.
- Decisions about the need for a pacemaker are influenced by the presence or absence of symptoms directly attributable to bradycardia. Permanent pacing improves survival in patients with third-degree AV block, especially if syncope has occurred.[9] Nevertheless, the overall prognosis depends to a large extent on the underlying HD.
- Although there is little evidence to suggest that pacemakers improve survival in patients with isolated first-degree AV block,[14] it is now recognized that marked first-degree AV block (PR >300 ms) can lead to symptoms even in the absence of higher degrees of AV block.[15]

Prognosis

- Investigators at Northwestern University compared older adult (age >60 years) outpatients with (n=470) and without (n=2090) asymptomatic bradycardia. Over a mean follow-up of 7.2 years, patients with asymptomatic bradycardia had a higher adjusted incidence of pacemaker insertion (HR, 2.14; 95% CI, 1.30–3.51; *P*=0.003), which appeared after a lag time of 4 years. However, the absolute rate of pacemaker implantation was low (<1% per year), and asymptomatic bradycardia was not associated with a higher risk of death.[16]

Sinus Node Dysfunction

Prevalence and Incidence

- The prevalence of sinus node dysfunction has been estimated to be between 403 and 666 per million, with an incidence rate of 63 per million per year requiring pacemaker therapy.[17]
- Sinus node dysfunction occurs in 1 of every 600 cardiac patients >65 years of age and accounts for ≈50% of implantations of pacemakers in the United States.[18,19]
- Sinus node dysfunction is commonly present with other causes of bradyarrhythmias (carotid sinus hypersensitivity in 33% of patients and advanced AV conduction abnormalities in 17%).[20,21]

Risk Factors

- The causes of sinus node dysfunction can be classified as intrinsic (secondary to pathological conditions involving the sinus node) or extrinsic (caused by depression of sinus node function by external factors such as drugs or autonomic influences).[22]
- Sinus node dysfunction may occur at any age but is primarily a disease of the elderly, with the average being ≈68 years of age.[18]
- Idiopathic degenerative disease is probably the most common cause of sinus node dysfunction.[23]
- Collected data from 28 different studies on atrial pacing for sinus node dysfunction showed a median annual incidence of complete AV block of 0.6% (range, 0%–4.5%) with a total prevalence of 2.1% (range, 0%–11.9%). This suggests that the degenerative process also affects the specialized conduction system, although the rate of progression is slow and does not dominate the clinical course of disease.[24]
- Ischemic HD can be responsible for one third of cases of sinus node dysfunction. Transient sinus node dysfunction can complicate MI, which is common during inferior MI, and is caused by autonomic influences. Cardiomyopathy, long-standing hypertension, infiltrative disorders (eg, amyloidosis and sarcoidosis), collagen vascular disease, and surgical trauma can also result in sinus node dysfunction.[25,26]

Aftermath

- The course of sinus node dysfunction is typically progressive, with 57% of patients experiencing symptoms over a 4-year period if untreated, and a 23% prevalence of syncope over the same time frame.[27]
- Approximately 50% of patients with sinus node dysfunction develop tachy-brady syndrome over a lifetime; such patients have a higher risk of stroke and death. The survival of patients with sinus node dysfunction appears to depend primarily on the severity of underlying cardiac disease and is not significantly changed by pacemaker therapy.[28–30]
- In a retrospective study,[31] patients with sinus node dysfunction who had pacemaker therapy were followed up for 12 years; at 8 years, mortality among those with ventricular pacing was 59% compared with 29% among those with atrial pacing. This discrepancy may be attributed to selection bias. For instance, the physiological or anatomic disorder (eg, fibrosis of conductive tissue) that led to the requirement for the particular pacemaker may have influenced prognosis, rather than the type of pacemaker used.
- The incidence of sudden death is extremely low, and sinus node dysfunction does not appear to affect survival whether untreated or treated with pacemaker therapy.[9]
- Supraventricular tachycardia (SVT) including AF occurs in 47% to 53% of patients with sinus node dysfunction.[30,32]
- On the basis of records from the NHDS, age-adjusted pacemaker implantation rates increased progressively from 370 per million in 1990 to 612 per million in 2002. This escalating implantation rate is attributable to increasing implantation for isolated sinus node dysfunction; implantation for sinus node dysfunction increased by 102%, whereas implantation for all other indications did not increase.[33]

SVT (Excluding AF and Atrial Flutter)

ICD-9 427.0; ICD-10 I47.1.

Mortality—130. Any-mention mortality—1220. Hospital discharges—23 000.

Prevalence and Incidence

- Data from the Marshfield Epidemiologic Study Area in Wisconsin suggested the incidence of documented paroxysmal

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SVT is 35 per 100 000 person-years. The mean age at SVT onset was 57 years, and both female sex and age >65 years were significant risk factors.[34]

- A review of ED visits from 1993 to 2003 revealed that 550 000 visits were for SVT (0.05% of all visits; 95% CI, 0.04%–0.06%), or ≈50 000 visits per year. Of these patients, 24% (95% CI, 15%–34%) were admitted to the hospital, and 44% (95% CI, 32%–56%) were discharged without specific follow-up.[35]
- The prevalence of SVT that is clinically undetected is likely much greater than the estimates from ED visits and electrophysiology procedures would suggest. For example, among a random sample of 604 participants in Finland, 7 (1.2%) fulfilled the diagnostic criteria for inappropriate sinus tachycardia.[36]
- Of 1383 participants in the Baltimore Longitudinal Study of Aging undergoing maximal exercise testing, 6% exhibited SVT during the test; increasing age was a significant risk factor. Only 16% exhibited >10 beats of SVT, only 4% were symptomatic, and the SVT participants were more likely to develop spontaneous SVT or AF.[37]
- From the surface ECG, the prevalence of atrial tachycardia is estimated to be 0.34% in asymptomatic patients and 0.46% in symptomatic patients.[38]

Aftermath

- The primary consequence of SVT for the majority of patients is a decline in quality of life.[39] However, rare cases of incessant SVT can lead to a tachycardia-induced cardiomyopathy,[40] and rare cases of sudden death attributed to SVT as a trigger have been described.[41]

Specific Types

- Among those presenting for invasive electrophysiological study and ablation, AV nodal reentrant tachycardia (a circuit that requires 2 AV nodal pathways) is the most common mechanism of SVT[42,43] and usually represents the majority of cases (56% of 1 series of 1754 cases from Loyola University Medical Center).[43]
- AV reentrant tachycardia (an arrhythmia that requires the presence of an extranodal connection between the atria and ventricles or specialized conduction tissue) is the second most common[42,43] (27% in the Loyola series[43]), and atrial tachycardia is the third most common (17% in the Loyola series[43]).
- In the pediatric population, AV reentrant tachycardia is the most common SVT mechanism, followed by AV nodal reentrant tachycardia and then atrial tachycardia.[44]
- AV reentrant tachycardia prevalence decreases with age, whereas AV nodal reentrant tachycardia and atrial tachycardia prevalences increase with advancing age.[43]
- The majority of AV reentrant tachycardia patients in the Loyola series were men (55%), whereas the majority of patients with AV nodal reentrant tachycardia (70%) or atrial tachycardia (62%) were women.[43]
- Multifocal atrial tachycardia is an arrhythmia that is commonly confused with AF and is characterized by 3 distinct P-wave morphologies, irregular R-R intervals, and a rate >100 beats per minute. It is uncommon in both children[45] and adults,[46] with a prevalence in hospitalized adults estimated at 0.05% to 0.32%.[46,47] The average age in adults is 70 to 72 years. Adults with multifocal atrial tachycardia have a mortality rate that is high, with estimates around 45%, but this is generally ascribed to the underlying condition(s).[46,47]

Wolff-Parkinson-White Syndrome

- Wolff-Parkinson-White syndrome, a diagnosis reserved for those with both ventricular preexcitation (evidence of an anterograde conducting AV accessory pathway on a 12-lead ECG) and tachyarrhythmias,[39] deserves special attention because of the associated risk of sudden death. Sudden death is generally attributed to rapid heart rates in AF conducting down an accessory pathway and leading to ventricular fibrillation (VF).[48,49] Of note, AF is common in Wolff-Parkinson-White patients, and surgical or catheter ablation of the accessory pathway often results in elimination of the AF.[50]
- Ventricular preexcitation with or without tachyarrhythmia was observed in 0.11% of 47 358 ECGs in adults participating in 4 large Belgian epidemiological studies[51] and in 0.17% of 32 837 Japanese high school students in ECGs obtained by law before the students entered school.[52]
- Asymptomatic adults with ventricular preexcitation appear to be at no increased risk of sudden death compared with the general population,[53–56] although certain characteristics found during invasive electrophysiological study (including inducibility of AV reentrant tachycardia or AF, accessory pathway refractory period, and the shortest R-R interval during AF) can help risk stratify these patients.[49,57]
- In a meta-analysis of 20 studies involving 1869 asymptomatic patients with a Wolff-Parkinson-White ECG pattern followed up for a total of 11 722 person-years, the risk of sudden death in a random effects model that was used because of heterogeneity across studies was estimated to be 1.25 (95% CI, 0.57–2.19) per 1000 person-years. Risk factors for sudden death included male sex, inclusion in a study of children (<18 years of age), and inclusion in an Italian study.[58]
- Symptomatic adult patients with the Wolff-Parkinson-White syndrome are at a higher risk of sudden death. In a study of 60 symptomatic patients in Olmsted County, Minnesota, including some who underwent curative surgery, 2 (3.3%) experienced sudden death over a 13-year period. Of 690 Wolff-Parkinson-White syndrome patients referred to a single hospital in the Netherlands, 15 (2.2%) had aborted sudden death, and VF was the first manifestation of the disease in 8 patients.[59]
- Of 379 Wolff-Parkinson-White patients with induced AV reentrant tachycardia during electrophysiology study who did not undergo ablation, 29 (8%) exhibited a "malignant presentation" over a mean 3.6 years of follow-up: syncope/presyncope in 25 patients, rapid preexcited AF causing hemodynamic collapse in 3 patients, and VF in 1 patient.[60] Those with such a presentation were more often male, had a shorter accessory pathway effective refractory period during electrophysiology study, more often had AV reentrant tachycardia that triggered AF during electrophysiology study, and more often had >1 accessory pathway.
- Although some studies in asymptomatic children with ventricular preexcitation suggest a benign prognosis,[55,61] others suggest that electrophysiological testing can identify a

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group of asymptomatic children with a risk of sudden death or VF as high as 11% over 19 months of follow-up.[62]

Subclinical Atrial Tachyarrhythmias

Pacemakers and defibrillators have increased clinician awareness of the frequency of subclinical AF and atrial high-rate episodes in individuals without a documented history of AF. Several studies have suggested that device-detected high-rate atrial tachyarrhythmias are surprisingly frequent and are associated with an increased risk of AF,[63] thromboembolism,[63,64] and total mortality.[63]

- Investigators in the ASSERT study (Asymptomatic Atrial Fibrillation and Stroke Evaluation in Pacemaker Patients and the Atrial Fibrillation Reduction Atrial Pacing Trial) prospectively enrolled 2580 patients with a recent pacemaker or defibrillator implantation who were ≥65 years of age, had a history of hypertension, and had no history of AF. They classified individuals by presence versus absence of subclinical atrial tachyarrhythmias (defined as atrial rate >190 beats per minute for >6 minutes in the first 3 months) and conducted follow-up for 2.5 years.[65] Subclinical atrial tachyarrhythmias in the first 3 months occurred in 10.1% of the patients and were associated with the following:
  - An almost 6-fold higher risk of clinical AF (HR, 5.56; 95% CI, 3.78–8.17; $P<0.001$)
  - A more than doubling in the adjusted risk of the primary end point, ischemic stroke or systemic embolism (HR, 2.50; 95% CI, 1.28–4.89; $P<0.008$)
  - An annual ischemic stroke or systemic embolism rate of 1.69% (versus 0.69% in those without)
  - A 13% PAR for ischemic stroke or systemic embolism
- Over the subsequent 2.5 years of follow-up, an additional 34.7% of the patients had subclinical atrial tachyarrhythmias, which were 8-fold more frequent than clinical AF episodes.
- The appropriate therapy of subclinical atrial tachyarrhythmias has not been rigorously studied.

AF and Atrial Flutter

ICD-9 427.3; ICD-10 I48.

Prevalence

- Estimates of the prevalence of AF in the United States ranged from ≈2.7 to 6.1 million in 2010, and AF prevalence is expected to rise to between ≈5.6 and 12 million in 2050.[66,67]
- Data from a California health plan suggest that compared with whites, blacks (OR, 0.49; 95% CI, 0.47–0.52), Asians (OR, 0.68; 95% CI, 0.64–0.72), and Hispanics (OR, 0.58; 95% CI, 0.55–0.61) have significantly lower adjusted prevalences of AF.[68]
- Data from the NHDS/NCHS (1996–2001) on cases that included AF as a primary discharge diagnosis found the following:
  - Approximately 44.8% of patients were men.
  - The mean age for men was 66.8 years versus 74.6 years for women.
  - The racial breakdown for admissions was 71.2% white, 5.6% black, and 2.0% other races (20.8% were not specified).
  - Black patients were much younger than patients of other races.
- Among Medicare patients aged ≥65 years, diagnosed from 1993 to 2007, the prevalence of AF increased ≈5% per year, from ≈41.1 per 1000 beneficiaries to 85.5 per 1000 beneficiaries.[69]

Incidence

- Data from the NHDS/NCHS (1996–2001) on cases that included AF as a primary discharge diagnosis found the following:
  - The incidence in men ranged from 20.6 per 100 000 people per year for patients between 15 and 44 years of age to 1077.4 per 100 000 people per year for patients ≥85 years of age.
  - In women, the incidence ranged from 6.6 per 100 000 people per year for patients between 15 and 44 years of age to 1203.7 per 100 000 people per year for those ≥85 years of age.
- In Olmsted County, Minnesota:
  - The age-adjusted incidence of clinically recognized AF in a white population increased by 12.6% between 1980 and 2000.[67,70]
  - The incidence of AF was greater in men (incidence ratio for men over women 1.86) and increased markedly with older age.[67]
- In a Medicare sample, the incidence of AF was ≈28 per 1000 person-years and did not change substantively between 1993 and 2007. Of individuals with incident AF in 2007, ≈55% were women, 91% were white, 84% had hypertension, 36% had HF, and 30% had cerebrovascular disease.[69]

Mortality

- In 2009, AF was mentioned on 100 196 US death certificates and was the underlying cause in 15 434 of those deaths (NCHS, NHLBI).
- In adjusted analyses from the FHS, AF was associated with an increased risk of death in both men (OR, 1.5; 95% CI, 1.2–1.8) and women (OR, 1.9; 95% CI, 1.5–2.2).[71] Furthermore, there was an interaction with sex, such that AF appeared to diminish the survival advantage typically observed in women.
- In Medicare beneficiaries ≥65 years of age with new-onset AF, mortality decreased modestly but significantly between 1993 and 2007. In 2007, the age- and sex-adjusted mortality at 30 days was 11%, and at 1 year, it was 25%.[69]
- A study of >4600 patients diagnosed with first AF showed that risk of death within the first 4 months after the AF diagnosis was high. The most common causes of CVD death were CAD, HF, and ischemic stroke, which accounted for 22%, 14%, and 10%, respectively, of the early deaths (within the first 4 months) and 15%, 16%, and 7%, respectively, of the late deaths.[70]

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- AF is also associated with mortality in individuals with other cardiovascular conditions and procedures, including HF,[72,73] MI,[74,75] CABG,[76,77] and stroke,[78] and with noncardiovascular conditions such as sepsis[79] and noncardiac surgery.[80]

Lifetime Risk and Cumulative Risk

- Participants in the NHLBI-sponsored FHS study were followed up from 1968 to 1999. At 40 years of age, remaining lifetime risks for AF were 26.0% for men and 23.0% for women. At 80 years of age, lifetime risks for AF were 22.7% for men and 21.6% for women. In further analysis, counting only those who had development of AF without prior or concurrent HF or MI, lifetime risk for AF was ≈16%.[81]
- By 80 years of age, investigators from the NHLBI-sponsored ARIC study observed that the cumulative risk of AF was 21% in white men, 17% in white women, and 11% in African Americans of both sexes.[82]

Risk Factors

- Standard risk factors
  - Both ARIC[83] and FHS (http://www.framinghamheartstudy.org/risk/atrial.html)[12,84] have developed risk prediction models to predict new-onset AF. Predictors of increased risk of new-onset AF include advancing age, European ancestry, body size (greater height and BMI), electrocardiography features (left ventricular hypertrophy, left atrial enlargement), DM, BP (SBP and hypertension treatment), and presence of CVD (CHD, HF, valvular HD).
  - Other consistently reported risk factors for AF include clinical and subclinical hyperthyroidism,[85,86] CKD,[87] and heavy alcohol consumption.[88]
- Family history
  - Although unusual, early-onset familial lone AF has long been recognized as a risk factor.[89,90]
  - In the past decade, the heritability of AF in the community has been appreciated. In studies from the FHS:
    - Adjusted for coexistent risk factors, having at least 1 parent with AF was associated with a 1.85-fold increased risk of AF in the adult offspring (multivariable-adjusted 95% CI, 1.12–3.06; P=0.02).[91]
    - A history of a first-degree relative with AF also was associated with an increased risk of AF (HR, 1.40; 95% CI, 1.13–1.74).[76] The risk was greater if the first-degree relative's age of onset was ≤65 years (HR, 2.01; 95% CI, 1.49–2.71) and with each additional affected first-degree relative (HR, 1.24; 95% CI, 1.05–1.46).[92]
- Genetics
  - Mutations in genes coding channels (sodium and potassium), gap junction proteins, and signaling have been described, often in lone AF or familial AF series, but they are responsible for few cases of AF in the community.[93]
  - Meta-analyses of genome-wide association studies have revealed SNPs on chromosomes 4q25 (upstream of *PITX2*),[94–96] 16q22 (*ZFHX3*),[95,97] and 1q21 (*KCNN3*),[96] as well as 6 other novel susceptibility loci (near *PRRX1*, *CAV1*, *C9orf3*, *SYNPO2L*, *SYNE2*, and *HCN4*).[98] Although an area of intensive inquiry, the causative SNPs and the functional basis of the associations have not been revealed.

Awareness

- In a US national biracial study of individuals with AF, compared with whites, blacks had approximately one third the likelihood (OR, 0.32; 95% CI, 0.20–0.52) of being aware that they had AF.[99]

Prevention

- Data from the ARIC study indicated that having at least 1 elevated risk factor explained 50% and having at least 1 borderline risk factor explained 6.5% of incident AF cases. The estimated overall incidence rate per 1000 person-years at a mean age of 54.2 years was 2.19 for those with optimal risk, 3.68 for those with borderline risk, and 6.59 for those with elevated risk factors.[100]
- Hypertension accounted for ≈14%[101] to 22%[100] of AF cases.
- Observational data from the CHS suggested that moderate-intensity exercise (such as regular walking) was associated with a lower risk of AF (HR, 0.72).[102] However, data from many studies suggested that vigorous-intensity exercise 5 to 7 days a week was associated with a slightly increased risk of AF (HR, 1.20; P=0.04).[103]
- Meta-analyses have suggested that renin-angiotensin system blockers may be useful in primary and secondary (recurrences) prevention of AF in trials of hypertension, after MI, in HF, and after cardioversion.[104,105] However, the studies were primarily secondary or post hoc analyses, and the results were fairly heterogeneous. Recently, in an analysis of the EMPHASIS-HF trial (Eplerenone in Mild Patients Hospitalization And SurvIval Study in Heart Failure), in one of many secondary outcomes, eplerenone was nominally observed to reduce the incidence of new-onset AF.[106]
- Although heterogeneous in their findings, modest-sized short-term studies suggested that the use of statins might prevent AF; however, larger longer-term studies do not provide support that statins are effective in AF prevention.[107]
- The NHLBI sponsored a workshop highlighting important research areas to advance the prevention of AF.[108]

Aftermath

- Hospitalization
  - Hospital discharges—479 000
    - From 1996 to 2001, hospitalizations with AF as the first-listed diagnosis increased by 34%.[109]
    - On the basis of Medicare and MarketScan databases, annually, individuals with AF (37.5%) are approximately twice as likely to be hospitalized as age- and sex-matched control subjects (17.5%).[110]
- Stroke
  - Stroke rates per 1000 patient-years declined in AF patients taking anticoagulants, from 46.7 in 1992 to 19.5

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in 2002, for ischemic stroke but remained fairly steady for hemorrhagic stroke (1.6–2.9).[111]
— When standard stroke risk factors were accounted for, AF was associated with a 4- to 5-fold increased risk of ischemic stroke.[112]
— Although the RR of stroke associated with AF did not vary (≈3–5-fold increased risk) substantively with advancing age, the proportion of strokes attributable to AF increased significantly. In FHS, AF accounted for ≈1.5% of strokes in individuals 50 to 59 years of age and ≈23.5% in those 80 to 89 years of age.[112]
— Paroxysmal, persistent, and permanent AF all appeared to increase the risk of ischemic stroke to a similar degree.[113]
— AF was also an independent risk factor for ischemic stroke severity, recurrence, and mortality.[78] In one study, people who had AF and were not treated with anticoagulants had a 2.1-fold increase in risk for recurrent stroke and a 2.4-fold increase in risk for recurrent severe stroke.[114]
— Studies have demonstrated an underutilization of warfarin therapy. In a recent meta-analysis, men and individuals with prior stroke were more likely to receive warfarin, whereas factors associated with lower use included alcohol and drug abuse, noncompliance, warfarin contraindications, dementia, falls, both gastrointestinal and intracranial hemorrhage, renal impairment, and advancing age.[115]

- Cognition
  — Individuals with AF have an adjusted 2-fold increased risk of dementia.[116]
  — A meta-analysis suggested that the risk was consistently high in the 7 studies of patients with recent stroke and a history of AF (OR, 2.4; 95% CI, 1.7–3.5; $P<0.001$; $I^2=87\%$). There was significant heterogeneity in the 7 studies of individuals without a history of stroke (OR, 1.6; 95% CI, 1.0–2.7; $P=0.05$; $I^2=87\%$).[117]
  — In individuals with AF in Olmsted County, Minnesota, the cumulative rate of dementia at 1 and 5 years was 2.7% and 10.5%, respectively.[97]

- Heart failure
  — AF and HF share many antecedent risk factors, and ≈40% of individuals with either AF or HF will develop the other condition.[72]
  — In the community, estimates of the incidence of HF in individuals with AF ranged from ≈3.3[72] to 4.4[118] per 100 person-years of follow-up.

Cost

Investigators examined Medicare and MarketScan databases (2004–2006) to estimate costs attributed to AF in 2008 US dollars:

- Annual total direct costs for AF patients were ≈$20 670 versus ≈$11 965 in the control group, for an incremental per-patient cost of $8705.[110]
- Extrapolating to the US population, it is estimated that the incremental cost of AF was ≈$26 billion, of which $6 billion was attributed to AF, $9.9 billion to other cardiovascular expenses, and $10.1 billion to noncardiovascular expenses.[110]

Tachycardia

ICD-9 427.0, 1, 2; ICD-10 I47.0, I47.1, I47.2, I47.9.
Mortality—607. Any-mention mortality—5902. Hospital discharges—78 000.

Monomorphic Ventricular Tachycardia

Prevalence and Incidence

- Of 150 consecutive patients with wide-complex tachycardia subsequently studied by invasive electrophysiological study, 122 (81%) had ventricular tachycardia (VT; the remainder had SVT).[119]
- Of patients with ventricular arrhythmias presenting for invasive electrophysiological studies, 11% to 21% had no structural HD, and the majority of those with structural HD had CAD.[120,121]
- In 634 patients with implantable cardioverter-defibrillators who had structural HD (including both primary and secondary prevention patients) followed up for a mean 11±3 months, ≈80% of potentially clinically relevant ventricular tachyarrhythmias were attributable to VT amenable to antitachycardia pacing (implying a stable circuit and therefore monomorphic VT).[122] Because therapy may have been delivered before spontaneous resolution occurred, the proportion of these VT episodes with definite clinical relevance is not known.
- Of those with VT in the absence of structural HD, right ventricular outflow tract VT is the most common form.[123]

Aftermath

- Although the prognosis of those with VT or frequent premature ventricular contractions in the absence of structural HD is good,[120,123] a potentially reversible cardiomyopathy may develop in patients with very frequent premature ventricular contractions,[124,125] and some cases of sudden death attributable to short-coupled premature ventricular contractions have been described.[126,127]

Polymorphic VT

Prevalence and Incidence

- The true prevalence and incidence of polymorphic VT (PVT) in the US general population is not known.
- During ambulatory cardiac monitoring, PVT prevalence ranged from 0.01% to 0.15%[128,129]; however, among patients who developed sudden cardiac death during ambulatory cardiac monitoring, PVT was detected in 30% to 43%.[129–131]
- A prevalence range of 15% to 19% was reported during electrophysiological study in patients resuscitated from cardiac arrest.[131–133]
- In the setting of AMI, the prevalence of PVT ranged from 1.2% to 2%.[134,135]
- Out-of-hospital PVT is estimated to be present in ≈25% of all cardiac arrest cases involving VT.[136,137]

Risk Factors

- PVT in the setting of a normal QT interval is most frequently seen in the context of acute ischemia or MI.[135,138]
- Less frequently, PVT with a normal QT interval can occur in patients without apparent structural HD. Catecholaminergic

PVT, which is discussed under inherited arrhythmic syndromes, is one such disorder.
- A prolonged QT, whether acquired (drug induced) or congenital, is a common cause of PVT. Drug-induced prolongation of QT causing PVT is discussed under torsade de pointes (TdP), whereas congenital prolonged QT is discussed under inherited arrhythmic syndromes.

Aftermath

- The presentation of PVT can range from a brief, asymptomatic, self-terminating episode to recurrent syncope or sudden cardiac death.[139]
- The overall hospital discharge rate (survival) of PVT has been estimated to be ≈28%.[140]

Prevention

- Prompt detection and correction of myocardial ischemia would potentially minimize the risk of PVT with normal QT in the setting of AMI.

Torsade de Pointes

Prevalence and Incidence

- The true incidence and prevalence of drug-induced TdP in the US general population is largely unknown.
- By extrapolating data from non-US registries,[141] it has been estimated that 12 000 cases of drug-induced TdP occur annually in the United States.[142]
- The prevalence of drug-induced prolongation of QT and TdP is 2 to 3 times higher in women than in men.[143]
- With the majority of QT-prolonging drugs, drug-induced TdP may occur in 3% to 15% of patients.[124]
- Antiarrhythmic drugs with QT-interval–prolonging potential carry a 1% to 3% risk of TdP over 1 to 2 years of exposure.[144]

Risk Factors

- TdP is usually related to administration of QT-prolonging drugs.[145] An up-to-date list of drugs with the potential to cause TdP may be found at http://www.azcert.org/medical-pros/drug-lists/drug-lists.cfm, a Web site maintained by the University of Arizona Center for Education and Research on Therapeutics.
- Specific risk factors for drug-induced TdP include prolonged QT, female sex, advanced age, bradycardia, hypokalemia, hypomagnesemia, left ventricular systolic dysfunction, and conditions that lead to elevated plasma concentrations of causative drugs, such as kidney disease, liver disease, drug interactions, or some combination of these.[142,146,147]
- Predisposition was also noted in patients who had a history of ventricular arrhythmia and who experienced a recent symptomatic increase in the frequency and complexity of ectopy.[148]
- Drug-induced TdP rarely occurs in patients without concomitant risk factors. An analysis of 144 published articles describing TdP associated with noncardiac drugs revealed that 100% of the patients had at least 1 risk factor, and 71% had at least 2 risk factors.[149]

Aftermath

- Drug-induced TdP may result in morbidity that requires hospitalization and in mortality attributable to sudden cardiac death in up to 31% of patients.[142,150]
- Patients with advanced HF with a history of drug-induced TdP had a significantly higher risk of sudden cardiac death during therapy with amiodarone than amiodarone-treated patients with no history of drug-induced TdP (55% versus 15%).[151] Current use of antipsychotic drugs was associated with a significant increase in the risk of sudden cardiac death attributable to TdP (OR, 3.3; 95% CI, 1.8–6.2).[152]
- Hospitalization was required in 47% and death occurred in 8% of patients with QT prolongation and TdP caused by administration of methadone.[153]

Prevention

- Keys to reducing the incidence of drug-induced cardiac arrhythmias include increased awareness among the medical, pharmaceutical, and nursing professions of the potential problems associated with the use of certain agents.
- Appropriate monitoring when a QT-prolonging drug is administered is essential. Also, prompt withdrawal of the offending agent should be initiated.[154]

VF and Ventricular Flutter

ICD-9 427.4; ICD-10 I49.0.

Mortality—1037. Any-mention mortality—9154.

Out-of-Hospital Cardiac Arrest: Adults

Out-of-hospital cardiac arrest is defined as a sudden and unexpected pulseless condition attributable to cessation of cardiac mechanical activity.[155] There are wide variations in the reported incidence of and outcomes for out-of-hospital cardiac arrest. These differences are caused in part by differences in definition and ascertainment of cardiac arrest data, as well as differences in treatment after the onset of cardiac arrest.

For additional details on out-of-hospital cardiac arrest treatment, please refer to Chapter 21, Quality of Care.

Incidence

(See Table 16-1.)

- The incidence of nontraumatic EMS-assessed, EMS-treated cardiac arrest and bystander-witnessed VF among individuals of any age during 2011 in the United States is best characterized by an ongoing registry from the Resuscitation Outcomes Consortium.
- The total resident population of the United States is 313 794 637 individuals (www.census.gov, accessed on June 22, 2012). Extrapolation of the incidence and case-fatality rate of EMS-assessed out-of-hospital cardiac arrest reported by the Resuscitation Outcomes Consortium (Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2012) to the total population of the United States suggests that each year, 359 400 (quasi CI, 350 200–368 200) people experience EMS-assessed out-of-hospital cardiac arrests in the United States.
- Approximately 60% of out-of-hospital cardiac arrests are treated by EMS personnel.[156]

- 25% of those with EMS-treated out-of-hospital cardiac arrest have no symptoms before the onset of arrest.[157]
- Among EMS-treated out-of-hospital cardiac arrests, 23% have an initial rhythm of VF or VT or are shockable by an automated external defibrillator.[158]
- The incidence of cardiac arrest with an initial rhythm of VF is decreasing over time; however, the incidence of cardiac arrest with any initial rhythm is not decreasing.[159]

Risk Factors

- A study conducted in New York City found the age-adjusted incidence of out-of-hospital cardiac arrest per 10 000 adults was 10.1 among blacks, 6.5 among Hispanics, and 5.8 among whites.[160]
- Prior HD is a major risk factor for cardiac arrest. A study of 1275 health maintenance organization enrollees 50 to 79 years of age who had cardiac arrest showed that the incidence of out-of-hospital cardiac arrest was 6.0 per 1000 person-years in subjects with any clinically recognized HD compared with 0.8 per 1000 person-years in subjects without HD. In subgroups with HD, incidence was 13.6 per 1000 person-years in subjects with prior MI and 21.9 per 1000 person-years in subjects with HF.[161]
- A family history of cardiac arrest in a first-degree relative is associated with an ≈2-fold increase in risk of cardiac arrest.[162,163]
- In a study of 81 722 women in the Nurses' Health Study, the PAR of sudden death associated with 4 lifestyle factors (smoking, PA, diet, and weight) was 81% (95% CI, 52%–93%).[164]

Aftermath

- Survival to hospital discharge in 2010, after EMS-treated nontraumatic cardiac arrest with any first recorded rhythm was 9.5% (95% CI, 8.8%–10.2%) for patients of any age, 9.8% (95% CI, 9.0%–10.6%) for adults, and 7.8% (4.2%–11.5%) for children (Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2012). Survival after bystander-witnessed VF was 28.4% (95% CI, 25.1%–31.8%) for patients of any age, 28.4% (95% CI, 25.1%–31.8%) for adults, and 57.1% (95% CI, 20.4%–93.8%) for children (Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2012).
- A study conducted in New York City found the age-adjusted survival to 30 days after discharge was more than twice as poor for blacks as for whites, and survival among Hispanics was also lower than among whites.[160]
- Seventy-nine percent of the lay public are confident that they know what actions to take in a medical emergency; 98% recognize an automated external defibrillator as something that administers an electric shock to restore a normal heartbeat among victims of sudden cardiac arrest; and 60% are familiar with cardiopulmonary resuscitation (Harris Interactive survey conducted on behalf of the AHA among 1132 US residents ≥18 years of age, January 8, 2008–January 21, 2008).

Out-of-Hospital Cardiac Arrest: Athletics

- Among 10.9 million registered participants in 40 marathons and 19 half marathons, the overall incidence of cardiac arrest was 0.54 per 100 000 participants (95% CI, 0.41–0.70).[165] Those with cardiac arrest were more often male and were running a marathon versus a half marathon. Seventy-one percent of those with cardiac arrest died; those who died were younger (mean 39±9 years of age) than those who did not die (mean 49±10 years of age), were more often male, and were more often running a full marathon.

Out-of-Hospital Cardiac Arrest: Children

(See Table 16-1.)

- The incidence of nontraumatic EMS-treated cardiac arrest and bystander-witnessed VF among individuals <18 years of age in the United States is best characterized by an ongoing registry. Survival to hospital discharge among children with EMS-treated, nontraumatic cardiac arrest is 7.8% (95% CI, 4.2%–11.5%; Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2011) and that of bystander-witnessed VF is 57.1% (95% CI, 20.4%–93.8%).
- Most sudden deaths in athletes were attributable to CVD (56%). Of the cardiovascular deaths that occurred, 29% occurred in blacks, 54% in high school students, and 82% with physical exertion during competition/training; only 11% occurred in females, although this proportion has increased over time.[166]
- A longitudinal study of students 17 to 24 years of age participating in National Collegiate Athletic Association sports showed that the incidence of nontraumatic out-of-hospital cardiac arrest was 1 per 22 903 athlete participant-years. The incidence of cardiac arrest tended to be higher among blacks than among whites and among men than among women.[167]

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In-Hospital Cardiac Arrest

- Extrapolation of the incidence of in-hospital cardiac arrest reported by Get With The Guidelines (GWTG)–Resuscitation to the total population of hospitalized patients in the United States suggests that each year, 209 000 (quasi CI, 192 000–211 000) people are treated for in-hospital cardiac arrest.[168]
- According to the GWTG-Resuscitation Investigators (unpublished data, June 22, 2012), 24.2% (95% CI, 23.5%–24.9%) of patients of any age or 23.9% (95% CI, 23.2%–24.6%) of adults and 40.2% (95% CI, 34.2%–46.2%) of children (excluding neonates who experienced in-hospital cardiac arrest with any first recorded rhythm) in 2011 survived to discharge.
- In 2011, 17.6% (95% CI, 17.0%–18.3%) of patients of any age or 17.7% (95% CI, 17.1%–18.4%) of adults and 14.1% (95% CI, 9.8%–18.3%) of children (excluding neonates) had in-hospital cardiac arrest with VF or pulseless VT as the first recorded rhythm. Of these, 43.0% (95% CI, 41.0%–45.0%) of adults and 52.8% (95% CI, 36.5%–69.1%) of children (excluding neonates) survived to discharge (GWTG-Resuscitation Investigators unpublished data, June 22, 2012). For additional details on in-hospital arrest treatment, please refer to Chapter 21, Quality of Care.

Monogenic Inherited Syndromes Associated With Sudden Cardiac Death

Long-QT Syndrome

- The hereditary long-QT syndrome is a genetic channelopathy characterized by prolongation of the QT interval (typically >460 ms) and susceptibility to ventricular tachyarrhythmias that lead to syncope and sudden cardiac death. Investigators have identified mutations in 13 genes leading to this phenotype (*LQT1* through *LQT13*). *LQT1* (*KCNQ1*), *LQT2* (*KCNH2*), and *LQT3* (*SCN5A*) mutations account for the majority (≈80%) of the typed mutations.[169,170]
- Prevalence of long-QT syndrome is estimated at 1 per 2000 live births from ECG-guided molecular screening of ≈44 000 mostly white infants born in Italy.[171] A similar prevalence was found among nearly 8000 Japanese school children screened by use of an ECG-guided molecular screening approach.[172]
- Long-QT syndrome has been reported among those of African descent, but its prevalence is not well assessed.[173]
- There is variable penetrance and a sex-time interaction for long-QT syndrome symptoms. Risk of cardiac events is higher among boys than girls (21% among boys and 14% among girls by age 12 years). Risk of events during adolescence is equivalent between sexes (≈25% for both sexes from ages 12–18 years). Conversely, risk of cardiac events in young adulthood is higher among women than men (39% among women from ages 18 to 40 years and 16% among men).[170]
- In addition to age and sex, the clinical course is influenced by prior syncope or aborted cardiac arrest, family history, QT-interval duration, genotype, number of mutations, and congenital deafness.[169,170,174]
- Risk of cardiac events is decreased during pregnancy but increased during the 9-month postpartum period.[175]
- The mainstay of therapy and prevention is β-blockade treatment.[170,174] Implantable defibrillators are considered for high-risk individuals.[176]

Short-QT Syndrome

- Short-QT syndrome is a recently described inherited mendelian condition characterized by shortening of the QT interval (typically QT <320 ms) and predisposition to AF and ventricular tachyarrhythmias and sudden death. Mutations in 5 ion channel genes have been described (*SQT1–SQT5*).[177]
- In a population of 41 767 young predominantly male Swiss transcripts, 0.02% of the population had a QT interval shorter than 320 ms.[178]
- Among 53 patients from the European Short QT Syndrome Registry (75% males, median age 26 years), a familial or personal history of cardiac arrest was present in 89%. Twenty-four patients received an implantable cardioverter-defibrillator, and 12 received long-term prophylaxis with hydroquinidine. During a median follow-up of 64 months, 2 patients received an appropriate implantable cardioverter-defibrillator shock, and 1 patient experienced syncope. Nonsustained PVT was recorded in 3 patients.[179]

The Brugada Syndrome

- The Brugada syndrome is an inherited channelopathy characterized by persistent ST-segment elevation in the precordial leads (V_1–V_3), right bundle-branch block, and susceptibility to ventricular arrhythmias and sudden cardiac death.[180]
- Mutations in several ion channel–related genes have been identified that lead to Brugada syndrome.[180]
- Prevalence is estimated at 1 to 5 per 10 000 individuals. Prevalence is higher in Southeast Asian countries, including Thailand and the Philippines. There is a strong male predominance (80% male).[180–185]
- Cardiac event rates for Brugada syndrome patients followed up prospectively in northern Europe (31.9 months) and Japan (48.7 months) were similar: 8% to 10% in patients with prior aborted sudden death, 1% to 2% in those with history of syncope, and 0.5% in asymptomatic patients.[186,187] Predictors of poor outcome included family history of sudden death and early repolarization pattern on ECG.[186,187]

Catecholaminergic PVT

- Catecholaminergic PVT is a familial condition characterized by adrenergically induced ventricular arrhythmias associated with syncope and sudden death. It is associated with frequent ectopy, bidirectional VT, and PVT with exercise or catecholaminergic stimulation (such as emotion, or medicines such as isoproterenol).
- Mutations in genes encoding the ryanodine type 2 receptor (*RYR2*)[188,189] are found in the majority, and mutations in genes encoding calsequestrin 2 (*CASQ2*)[190,191] are found in a small minority.[192] However, a substantial proportion of individuals with catecholaminergic PVT do not have an identified mutation.
- Statistics regarding catecholaminergic PVT are primarily from case series. Of 101 patients with catecholaminergic PVT, the majority had experienced symptoms before 21 years of age.[192]
- In small series (n=27 to n=101) of patients followed up over a mean of 6.8 to 7.9 years, 27% to 62% experienced cardiac symptoms, and fatal or near-fatal events occurred in 13% to 31%.[192–194]
- Risk factors for cardiac events included younger age of diagnosis and absence of β-blocker therapy. A history of aborted cardiac arrest and absence of β-blocker therapy were risk factors for fatal or near-fatal events.[192]

Arrhythmogenic Right Ventricular Cardiomyopathy

- Arrhythmogenic right ventricular cardiomyopathy is a form of genetically inherited structural HD that presents with fibrofatty replacement of the myocardium, with clinical presentation of palpitations, syncope, and sudden death.[195]
- Twelve arrhythmogenic right ventricular cardiomyopathy loci have been described (ARVC1–ARVC12). Disease-causing genes for 8 of these loci have been identified, the majority of which are in desmosomally related proteins.[195]
- Prevalence is estimated at 2 to 10 per 10 000 individuals.[195,196] Of 100 patients reported on from the Johns Hopkins Arrhythmogenic Right Ventricular Dysplasia Registry, 51 were men, and 95 were white, with the rest being of

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black, Hispanic, or Middle Eastern origin. Twenty-two percent of index cases had evidence of the familial form of arrhythmogenic right ventricular cardiomyopathy.[197]

- The most common presenting symptoms were palpitations (27%), syncope (26%), and sudden cardiac death (23%).[197]
- During a median follow-up of 6 years, 47 patients received an implantable cardioverter-defibrillator, 29 of whom received appropriate implantable cardioverter-defibrillator shocks. At the end of follow-up, 66 patients were alive. Twenty-three patients died at study entry, and 11 died during follow-up (91% of deaths were attributable to sudden cardiac arrest).[197] Similarly, the annual mortality rate was 2.3% for 130 patients with arrhythmogenic right ventricular cardiomyopathy from Paris, France, who were followed up for a mean of 8.1 years.[198]

Hypertrophic Cardiomyopathy

(Please refer to Chapter 19, Cardiomyopathy and Heart Failure, for statistics regarding the general epidemiology of HCM.)

- Over a mean follow-up of 8±7 years, 6% of HCM patients experienced sudden cardiac death.[199]
- Among 1866 sudden deaths in athletes between 1980 and 2006, HCM was the most common cause of cardiovascular sudden death (in 251 cases, or 36% of the 690 deaths that could be reliably attributed to a cardiovascular cause).[166]
- The risk of sudden death increases with increasing maximum left ventricular wall thickness,[200,201] and the risk for those with wall thickness ≥30 mm is 18.2 per 1000 patient-years (95% CI, 7.3–37.6),[200] or approximately twice that of those with maximal wall thickness <30 mm.[200,201] Of note, an association between maximum wall thickness and sudden death has not been found in every HCM population.[202]
- Nonsustained VT is a risk factor for sudden death,[203,204] particularly in younger patients. Nonsustained VT in those ≤30 years of age is associated with a 4.35-greater odds of sudden death (95% CI, 1.5–12.3).[203]
- A history of syncope is also a risk factor for sudden death in these patients,[205] particularly if the syncope was recent before the initial evaluation and not attributable to a neurally mediated event.[206]
- The presence of left ventricular outflow tract obstruction ≥30 mm Hg appears to increase the risk of sudden death by ≈2-fold.[207,208] The presence of left ventricular outflow tract obstruction has a low positive predictive value (7%–8%) but a high negative predictive value (92%–95%) for predicting sudden death.[207,209]
- The rate of malignant ventricular arrhythmias detected by implantable cardioverter-defibrillators appears to be similar between those with a family history of sudden death in ≥1 first-degree relatives and those with at least 1 of the risk factors described above.[210]
- The risk of sudden death increases with the number of risk factors.[211,212]

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Table 16-1. Incidence and Outcome of Out-of-Hospital Cardiac Arrest in the United States

	Overall (95% CI)	Adults (95% CI)	Children (95% CI)
Incidence (per 100 000)			
EMS assessed	126.4 (124.0–128.8)	147.7 (144.8–150.7)	11.0 (9.6–12.4)
EMS treated	63.8 (62.1–65.4)	80.1 (77.9–82.2)	8.8 (7.6–10.1)
Bystander-witnessed shockable rhythm (including VT, VF, shockable by AED)	10.0 (9.4–10.6)	9.8 (9.0–10.6)	0.3 (0.1–0.5)
Survival to discharge, %			
EMS assessed	4.8 (4.4–5.2)	5.3 (4.9–5.7)	6.3 (3.3–9.3)
EMS treated	9.5 (8.8–10.2)	9.8 (9.0–10.6)	7.8 (4.2–11.5)
Bystander-witnessed shockable rhythm	28.4 (25.1–31.8)	28.4 (25.1–31.8)	57.1 (20.4–93.8)

CI indicates confidence interval; EMS, emergency medical services; VT, ventricular tachycardia; VF, ventricular fibrillation; and AED, automated external defibrillator.
Source: Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2012.

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17. Subclinical Atherosclerosis

(See Table 17-1 and Chart 17-1 through 17-6.)

Atherosclerosis, a systemic disease process in which fatty deposits, inflammation, cells, and scar tissue build up within the walls of arteries, is the underlying cause of the majority of clinical cardiovascular events. Individuals who develop atherosclerosis tend to develop it in a number of different types of arteries (large and small arteries and those feeding the heart, brain, kidneys, and extremities), although they may have much more in some parts of the body than others. In recent decades, advances in imaging technology have allowed for improved ability to detect and quantify atherosclerosis at all stages and in multiple different vascular beds. Two modalities, computed tomography (CT) of the chest for evaluation of CAC and B-mode ultrasound of the neck for evaluation of carotid artery IMT, have been used in large studies with outcomes data and may help define the burden of atherosclerosis in individuals before they develop clinical events such as heart attack or stroke. Another commonly used method for detecting and quantifying atherosclerosis in the peripheral arteries is the ankle-brachial index (ABI), which is discussed in Chapter 20. Data on cardiovascular outcomes are starting to emerge for additional modalities that measure anatomic and functional measures of subclinical disease, including brachial artery reactivity testing, aortic and carotid magnetic resonance imaging, and tonometric methods of measuring vascular compliance or microvascular reactivity. Further research may help to define the role of these techniques in cardiovascular risk assessment. Some guidelines have recommended screening for subclinical atherosclerosis, especially by CAC, or IMT may be appropriate in people at intermediate risk for HD (eg, 10-year estimated risk of 10% to 20%) but not for lower-risk general population screening or for people with preexisting HD or most other high-risk conditions.[1,2] However, a recent guideline notes those with DM who are ≥40 years of age may be suitable for screening of risk by coronary calcium. There are still limited data demonstrating whether screening with these and other imaging modalities can improve patient outcomes or whether it only increases downstream medical care costs. A recently published report in a large cohort randomly assigned to coronary calcium screening or not showed such screening to result in an improved risk factor profile without increasing downstream medical costs.[3]

Abbreviations Used in Chapter 17

AAC	aortic artery calcification
ABI	ankle-brachial index
ACS	acute coronary syndrome
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
BP	blood pressure
CAC	coronary artery calcification
CAD	coronary artery disease
CARDIA	Coronary Artery Risk Development in Young Adults
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CONFIRM	Coronary CT Angiography Evaluation for Clinical Outcomes: An International Multicenter Registry
CT	computed tomography
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
EISNER	Early Identification of Subclinical Atherosclerosis by Noninvasive Imaging Research
FHS	Framingham Heart Study
FMD	flow-mediated dilation
FRS	Framingham Risk Score
HBP	high blood pressure
HDL	high-density lipoprotein
HD	heart disease
HR	hazard ratio
IMT	intima-media thickness
LDL	low-density lipoprotein
MESA	Multi-Ethnic Study of Atherosclerosis
NHLBI	National Heart, Lung, and Blood Institute
RR	relative risk
SBP	systolic blood pressure

Coronary Artery Calcification

Background

- CAC is a measure of the burden of atherosclerosis in the heart arteries and is measured by CT. Other components of the atherosclerotic plaque, including fatty (eg, cholesterol-rich components) and fibrotic components, often accompany CAC and may be present even in the absence of CAC.
- The presence of any CAC, which indicates that at least some atherosclerotic plaque is present, is defined by an Agatston score >0. Clinically significant plaque, frequently an indication for more aggressive risk factor management, is often defined by an Agatston score ≥100 or a score ≥75th percentile for one's age and sex. An Agatston score ≥400 has been noted to be an indication for further diagnostic evaluation (eg, exercise testing or myocardial perfusion imaging) for CAD.
- Current guidelines provide for a modest Class IIa, Level of Evidence B recommendation for screening for coronary calcium in people at intermediate risk (eg, 10%–20% 10-year CHD risk) or with DM; a Class IIb, Level of Evidence B recommendation for those at low intermediate risk (6%–10% 10-year CHD risk); but a contraindication (Class III, Level of Evidence B) for screening those at lower risk.[2]

Prevalence

(See Table 17-1 and Charts 17-1 and Chart 17-2.)

- The NHLBI's FHS reported CAC measured in 3238 white adults in age groups ranging from <45 years of age to ≥75 years of age.[4]
  - —Overall, 32.0% of women and 52.9% of men had prevalent CAC.
  - —Among participants at intermediate risk according to FRS, 58% of women and 67% of men had prevalent CAC.

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- The NHLBI's CARDIA study measured CAC in 3043 black and white adults 33 to 45 years of age (at the CARDIA year 15 examination).[5]
  - —Overall, 15.0% of men and 5.1% of women, 5.5% of those 33 to 39 years of age and 13.3% of those 40 to 45 years of age, had prevalent CAC. Overall, 1.6% of participants had an Agatston score that exceeded 100.
  - —The prevalence of CAC was lower in black men than in white men but was similar in black and white women at these ages.
- The NHLBI's MESA measured CAC in 6814 participants 45 to 84 years of age, including white (n=2619), black (n=1898), Hispanic (n=1494), and Chinese (n=803) men and women.[6]
  - —The prevalence and 75th percentile levels of CAC were highest in white men and lowest in black and Hispanic women. Significant ethnic differences persisted after adjustment for risk factors, with the RR of coronary calcium being 22% less in blacks, 15% less in Hispanics, and 8% less in Chinese than in whites.
  - —Table 17-1 shows the 75th percentile levels of CAC by sex and race at selected ages. These might be considered cut points above which more aggressive efforts to control risk factors (eg, elevated cholesterol or BP) could be implemented and/or at which treatment goals might be more aggressive (eg, LDL cholesterol <100 mg/dL instead of <130 mg/dL).
- The prevalence of CAC varies widely according to FRS. In a report from MESA,[7] the prevalence of CAC among individuals with very low FRS (10-year risk <5%) was low. These findings may have important implications for population screening for subclinical atherosclerosis.
- Investigators from the NHLBI's CARDIA study examined the association between neighborhood attributes and subclinical atherosclerosis in younger adult populations. Using 2000 US Census block-group–level data, among women, higher odds of CAC were associated with higher neighborhood deprivation and lower neighborhood cohesion. Among all men, neither neighborhood deprivation nor neighborhood cohesion was associated with CAC, whereas among men in deprived neighborhoods, low cohesion was associated with higher odds of CAC.[8]

CAC and Incidence of Coronary Events

(See Chart 17-3 and 17-4.)

- The NHLBI's MESA recently reported on the association of CAC scores with first CHD events over a median follow-up of 3.9 years among a population-based sample of 6722 men and women (39% white, 27% black, 22% Hispanic, and 12% Chinese).[9]
  - —People with CAC scores of 1 to 100 had ≈4 times greater risk and those with CAC scores >100 were 7 to 10 times more likely to experience a coronary event than those without CAC.
  - —CAC provided similar predictive value for coronary events in whites, Chinese, blacks, and Hispanics (HRs ranging from 1.15–1.39 for each doubling of coronary calcium).
- In another report of a community-based sample, not referred for clinical reasons, the South Bay Heart Watch examined CAC in 1461 adults (average age 66 years) with coronary risk factors, with a median of 7.0 years of follow-up.[10]
- In a study of healthy adults 60 to 72 years of age who were free of clinical CAD, predictors of the progression of CAC were assessed. Predictors tested included age, sex, race/ethnicity, smoking status, BMI, family history of CAD, C-reactive protein, several measures of DM, insulin levels, BP, and lipids. Insulin resistance, in addition to the traditional cardiac risk factors, independently predicts progression of CAC.[11] Clinically, however, it is not yet recommended to conduct serial scanning of CAC to measure effects of therapeutic interventions.
- A recent publication from MESA also used CAC, in particular, and carotid IMT to stratify CHD and CVD event risk in people with metabolic syndrome and DM; those with low levels of CAC or carotid IMT have CHD and CVD event rates as low as many people without metabolic syndrome and DM. Those with DM who have CAC scores <100 have annual CHD event rates of <1%.[12]
- It is noteworthy, as recently demonstrated in MESA in 5878 participants with a median of 5.8 years of follow-up, that the addition of CAC to standard risk factors resulted in significant improvement of classification of risk for incident CHD events, placing 77% of people in the highest or lowest risk categories compared with 69% based on risk factors alone. An additional 23% of those who experienced events were reclassified as high risk, and 13% with events were reclassified as low risk.[13]
- The contribution of CAC to risk prediction has also been observed in other cohorts, including both the Heinz Nixdorf Recall study[14] and the Rotterdam study.[15]
- In MESA, among 6603 people aged 45 to 84 years (1686 or 25% with metabolic syndrome without DM and 881 or 13% with DM), subclinical atherosclerosis assessed by CAC was more severe in people with metabolic syndrome and DM than in those without these conditions, and the extent of CAC was a strong predictor of CHD and CVD events in these groups, with CAC increasing the C statistic significantly ($P<0.001$) over other risk factors in each of these groups.[12]

CAC Progression and Risk

- A recent report in 4609 individuals who had baseline and repeat cardiac CT found that progression of CAC in predicting future all-cause mortality provided only incremental information over baseline score, demographics, and cardiovascular risk factors.[16]
- Furthermore, in MESA, among 5662 subjects aged 45 to 84 years who received baseline and follow-up CT scans, the progression of CAC was greater in people with metabolic syndrome and DM than in those without, and greater progression of CAC in these groups was also associated with greater risk for CHD events.[17]

Abdominal Aortic Calcification and Multisite Atherosclerosis

- Abdominal aortic calcification (AAC) can be measured by CT and at most age groups is at a higher prevalence than CAC. In MESA, AAC prevalence ranged from 34%

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in those aged 45 to 54 years to 94% in those aged 75 to 84 years. Prevalence was significantly higher in Caucasians (79%) and lowest in blacks (62%). The prevalence of CAC, increased carotid IMT, and abnormal ABI was also greater in those with versus without AAC, and by age 65 years, 97% of men and 91% of women had either AAC, CAC, increased carotid IMT, or low ABI. Three or more of these conditions ("multisite atherosclerosis") were present in 20% of women and 30% of men.[18]

- In addition, AAC was significantly associated with cigarette smoking and dyslipidemia, whereas CAC showed much weaker associations; age and hypertension were associated similarly and significantly with AAC and CAC. AAC was more strongly correlated with most CVD risk factors than was CAC.[19]
- At present, however, no recommendations exist for screening of AAC for assessment of cardiovascular risk because of the limited data available.

Carotid IMT

Background

- Carotid IMT measures the thickness of 2 layers (the intima and media) of the wall of the carotid arteries, the largest conduits of blood going to the brain. Carotid IMT is thought to be an even earlier manifestation of atherosclerosis than CAC, because thickening precedes the development of frank atherosclerotic plaque. Carotid IMT methods are still being refined, so it is important to know which part of the artery was measured (common carotid, internal carotid, or bulb) and whether near and far walls were both measured. This information can affect the average-thickness measurement that is usually reported.
- Unlike CAC, everyone has some thickness to the layers of their arteries, but people who develop atherosclerosis have greater thickness. Ultrasound of the carotid arteries can also detect plaques and determine the degree of narrowing of the artery they may cause. Epidemiological data, including the data discussed below, have indicated that high-risk levels of thickening might be considered as those in the highest quartile or quintile for one's age and sex, or ≥1 mm.
- Although ultrasound is commonly used to diagnose plaque in the carotid arteries in people who have had strokes or who have bruits (sounds of turbulence in the artery), guidelines are limited as to screening of asymptomatic people with carotid IMT to quantify atherosclerosis or predict risk. However, some organizations have recognized that carotid IMT measurement by B-mode ultrasonography may provide an independent assessment of coronary risk.[20] For those at intermediate CHD risk (eg, 10%–20% 10-year CHD risk), a Class IIa, Level of Evidence B recommendation has been noted for the use of carotid IMT in screening asymptomatic people.[2]

Prevalence and Association With Incident Cardiovascular Events

(See Chart 17-5 and 17-6.)

- The Bogalusa Heart Study measured carotid IMT in 518 black and white men and women at a mean age of 32±3 years. These men and women were healthy but overweight.[21]
  - —Men had significantly higher carotid IMT in all segments than women, and blacks had higher common carotid and carotid bulb IMTs than whites.
  - —Even at this young age, after adjustment for age, race, and sex, carotid IMT was associated significantly and positively with waist circumference, SBP, DBP, and LDL cholesterol. Carotid IMT was inversely correlated with HDL cholesterol levels. Participants with greater numbers of adverse risk factors (0, 1, 2, 3, or more) had stepwise increases in mean carotid IMT levels.
- In a subsequent analysis, the Bogalusa investigators examined the association of risk factors measured since childhood with carotid IMT measured in these young adults.[22] Higher BMI and LDL cholesterol levels measured at 4 to 7 years of age were associated with increased risk for being >75th percentile for carotid IMT in young adulthood. Higher SBP and LDL cholesterol and lower HDL cholesterol in young adulthood were also associated with having high carotid IMT. These data highlight the importance of adverse risk factor levels in early childhood and young adulthood in the early development of atherosclerosis.
- Among both women and men in MESA, blacks had the highest common carotid IMT, but they were similar to whites and Hispanics in internal carotid IMT. Chinese participants had the lowest carotid IMT, in particular in the internal carotid, of the 4 ethnic groups.[23]
- The NHLBI's CHS reported follow-up of 4476 men and women ≥65years of age (mean age 72 years) who were free of CVD at baseline.[24]
  - —Mean maximal common carotid IMT was 1.03±0.20 mm, and mean internal carotid IMT was 1.37±0.55 mm.
  - —After a mean follow-up of 6.2 years, those with maximal combined carotid IMT in the highest quintile had a 4-to 5-fold greater risk for incident heart attack or stroke than those in the bottom quintile. After adjustment for other risk factors, there was still a 2- to 3-fold greater risk for the top versus the bottom quintile.
- A study of 441 individuals ≤65 years of age without a history of CAD, DM, or hyperlipidemia who were examined for carotid IMT found 42% had high-risk carotid ultrasound findings (carotid IMT ≥75th percentile adjusted for age, sex, and race or presence of plaque). Among those with an FRS ≤5%, 38% had high-risk carotid ultrasound findings.[25]
- Conflicting data have been reported on the contribution of carotid IMT to risk prediction. In 13 145 participants in the NHLBI's ARIC study, the addition of carotid IMT combined with identification of plaque presence or absence to traditional risk factors reclassified risk in 23% of individuals overall, with a net reclassification improvement of 9.9%. There was a modest but statistically significant improvement in the area under the receiver operating characteristic curve, from 0.742 to 0.755.[26] In contrast, data reported recently from the Carotid Atherosclerosis Progression Study observed a net reclassification improvement of −1.4% that was not statistically significant.[27]

CAC and Carotid IMT

- In the NHLBI's MESA, a study of white, black, Chinese, and Hispanic adults 45 to 84 years of age, carotid IMT and

CAC were found to be commonly associated, but patterns of association differed somewhat by sex and race.[23]

—Common and internal carotid IMT were greater in women and men who had CAC than in those who did not, regardless of ethnicity.
—Overall, CAC prevalence and scores were associated with carotid IMT, but associations were somewhat weaker in blacks than in other ethnic groups.
—In general, blacks had the thickest carotid IMT of all 4 ethnic groups, regardless of the presence of CAC.
—Common carotid IMT differed little by race/ethnicity in women with any CAC, but among women with no CAC, IMT was higher among blacks (0.86 mm) than in the other 3 groups (0.76–0.80 mm).

- In a more recent analysis from the NHLBI's MESA, the investigators reported on follow-up of 6698 men and women in 4 ethnic groups over 5.3 years and compared the predictive utility of carotid IMT and CAC.[28]

—CAC was associated more strongly than carotid IMT with the risk of incident CVD.
—After adjustment for each other (CAC score and IMT) and for traditional CVD risk factors, the HR for CVD increased 2.1-fold for each 1-standard deviation increment of log-transformed CAC score versus 1.3-fold for each 1-standard deviation increment of the maximum carotid IMT.
—For CHD events, the HRs per 1-standard deviation increment increased 2.5-fold for CAC score and 1.2-fold for IMT.
—A receiver operating characteristic curve analysis also suggested that CAC score was a better predictor of incident CVD than was IMT, with areas under the curve of 0.81 versus 0.78, respectively.
—Investigators from the NHLBI's CARDIA and MESA studies examined the burden and progression of subclinical atherosclerosis among adults <50 years of age. Ten-year and lifetime risks for CVD were estimated for each participant, and the participants were stratified into 3 groups: (1) those with low 10-year (<10%) and low lifetime (<39%) predicted risk for CVD; (2) those with low 10-year (<10%) but high lifetime (≥39%) predicted risk; and (3) those with high 10-year risk (>10%). The latter group had the highest burden and greatest progression of subclinical atherosclerosis. Given the young age of those studied, ≈90% of participants were at low 10-year risk, but of these, half had high predicted lifetime risk. Compared with those with low short-term/low lifetime predicted risks, those with low short-term/high lifetime predicted risk had significantly greater burden and progression of CAC and significantly greater burden of carotid IMT, even at these younger ages. These data confirm the importance of early exposure to risk factors for the onset and progression of subclinical atherosclerosis.[29]

CT Angiography

CT angiography is widely used by cardiologists to aid in the diagnosis of CAD, particularly when other test results may be equivocal. It is also of interest because of its ability to detect and possibly quantitate overall plaque burden and certain characteristics of plaques that may make them prone to rupture, such as positive remodeling or low attenuation.

- In a study of 1059 subjects undergoing CT angiography, those who exhibited both positive remodeling and low-attenuation plaques <30 Hounsfield units on CT had the greatest risk of developing future ACS.[30]

However, because of the limited outcome data in asymptomatic people, as well as the associated expense and risk of CT angiography (including generally higher radiation levels than CT scanning to detect CAC), current guidelines do not recommend its use as a screening tool for assessment of cardiovascular risk in asymptomatic people (Class III, Level of Evidence C).[2]

- In symptomatic individuals, however, the CONFIRM registry (Coronary CT Angiography Evaluation for Clinical Outcomes: An International Multicenter Registry) showed that even if the CAC score was 0, obstructive CAD was possible and was associated with an increased risk of cardiovascular events (HR of 5.7 in those with ≥50% stenosis versus no obstructive CAD when the CAC score was 0).[31]
- In addition, a recent meta-analysis of 11 studies that included 7335 people with suspected CAD showed those with at least 1 significant coronary stenosis from CT angiography had an annualized event rate of 11.9% and an HR of 10.7 for incident cardiovascular events, which was not attenuated by adjustment for coronary calcium.[32]

Measures of Vascular Function and Incident CVD Events

Background

- Measures of arterial tonometry (stiffness) are based on the concept that pulse pressure has been shown to be an important risk factor for CVD. Arterial tonometry offers the ability to directly and noninvasively measure central pulse wave velocity in the thoracic and abdominal aorta.
- Brachial FMD is a marker for nitric oxide release from the endothelium that can be measured by ultrasound. Impaired FMD is an early marker of CVD.
- Recommendations have not been specific, however, as to which, if any, measures of vascular function may be useful for CVD risk stratification in selected patient subgroups. Because of the absence of significant prospective data relating these measures to outcomes, latest guidelines do not currently recommend measuring either FMD (Class III, Level of Evidence B) or arterial stiffness (Class III, Level of Evidence C) for cardiovascular risk assessment in asymptomatic adults.[2]

Arterial Tonometry and CVD

- The Rotterdam Study measured arterial stiffness in 2835 elderly participants (mean age 71 years).[33] They found that as aortic pulse wave velocity increased, the hazard risk of CHD was 1.72 (second versus first tertile) and 2.45 (third versus first tertile). Results remained robust even after accounting for carotid IMT, ABI, and pulse pressure.

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- A study from Denmark of 1678 individuals 40 to 70 years of age found that each 1–standard deviation increment in aortic pulse wave velocity increased CVD risk by 16% to 20%.[34]
- The FHS measured several indices of arterial stiffness, including pulse wave velocity, wave reflection, and central pulse pressure.[35] They found that not only was higher pulse wave velocity associated with a 48% increased risk of incident CVD events, but pulse wave velocity additionally improved CVD risk prediction (integrated discrimination improvement of 0.7%, $P<0.05$).

FMD and CVD

MESA measured FMD in 3026 participants (mean age 61 years) who were free of CVD. As FMD increased (ie, improved brachial function), the risk of CVD was 16% lower.[36] FMD also improved CVD risk prediction compared with the FRS by improving net reclassification by 29%.

Impact of Subclinical Disease Screening on Behavior and Risk

There are limited observational and clinical trial data to show whether screening for subclinical disease can result in initiation of beneficial lifestyle or preventive therapies.

- In a 1996 study of 703 men and women who underwent coronary calcium scanning, after adjustment for age, sex, preexisting high cholesterol, HBP, cigarette smoking, and a positive family history of CAD, the natural log of total calcium score remained associated with new aspirin usage, new cholesterol medication usage, consultation with a physician, weight loss, and decreases in dietary fat.[37]
- More recently, among 980 people referred for coronary calcium scanning, in multivariable analysis, greater baseline CAC score was strongly associated with initiation of aspirin therapy, dietary changes, and increased exercise.[38]
- A recent study examining the effects of an office-based ultrasound screening intervention in 355 subjects aged ≥40 years with at least 1 risk factor showed that the presence of an abnormal carotid ultrasound significantly altered physicians' prescription of aspirin and cholesterol medicine and subjects' awareness of CVD risk and intentions to make exercise and diet changes and quit smoking; an abnormal carotid ultrasound also predicted reduced dietary sodium and increased fiber intake.[39]
- In the only large-scale randomized clinical trial to date involving CAC scanning, the EISNER study (Early Identification of Subclinical Atherosclerosis by Noninvasive Imaging Research), among 2137 adults randomized 2:1 to scanning versus no scanning, those scanned showed a net favorable change in SBP, LDL cholesterol, and waist circumference among those with abnormal waist girth and no change in FRS compared with an increase in FRS in those who were not scanned. In addition, the higher the baseline CAC score, the greater the improvement in BP, total and LDL cholesterol, triglycerides, weight, and FRS. Importantly, downstream medical testing and costs were comparable between groups.[3]

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Table 17-1. CAC Scores for the 75th Percentile of Men and Women of Different Race/Ethnic Groups, at Specified Ages

	75th Percentile CAC Scores*			
Age, y	Black	Chinese	Hispanic	White
Women				
45	0	0	0	0
55	0	2	0	1
65	26	45	19	54
75	138	103	116	237
Men				
45	0	3	0	0
55	15	34	27	68
65	95	121	141	307
75	331	229	358	820

CAC indicates coronary artery calcification.

*The 75th percentile CAC score is the score at which 75% of people of the same age, sex, and race have a score at or below this level and 25% of people of the same age, sex, and race have a higher score. (Source: MESA CAC Tools Web site.[40])

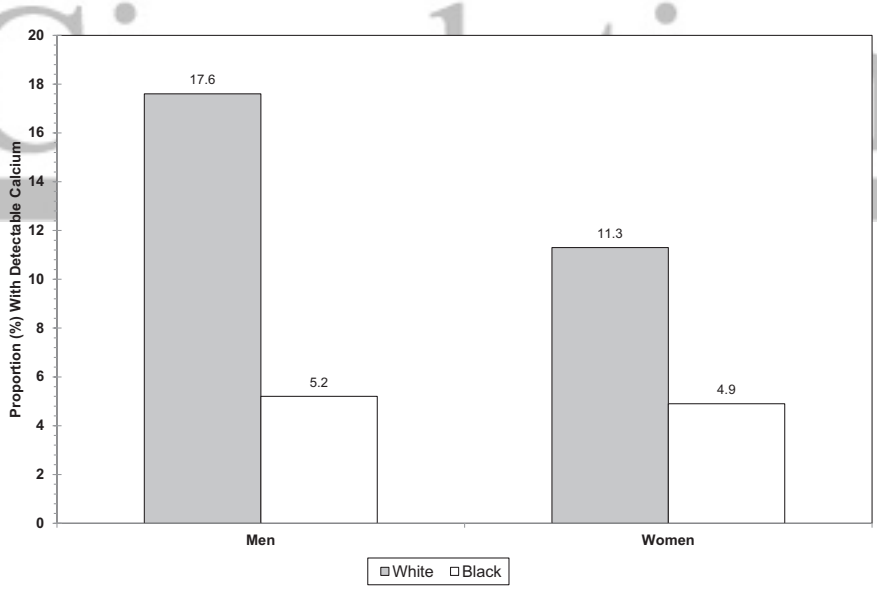

Chart 17-1. Prevalence (%) of coronary calcium: US adults 33 to 45 years of age. $P<0.0001$ across race-sex groups. Data derived from Loria et al.[5]

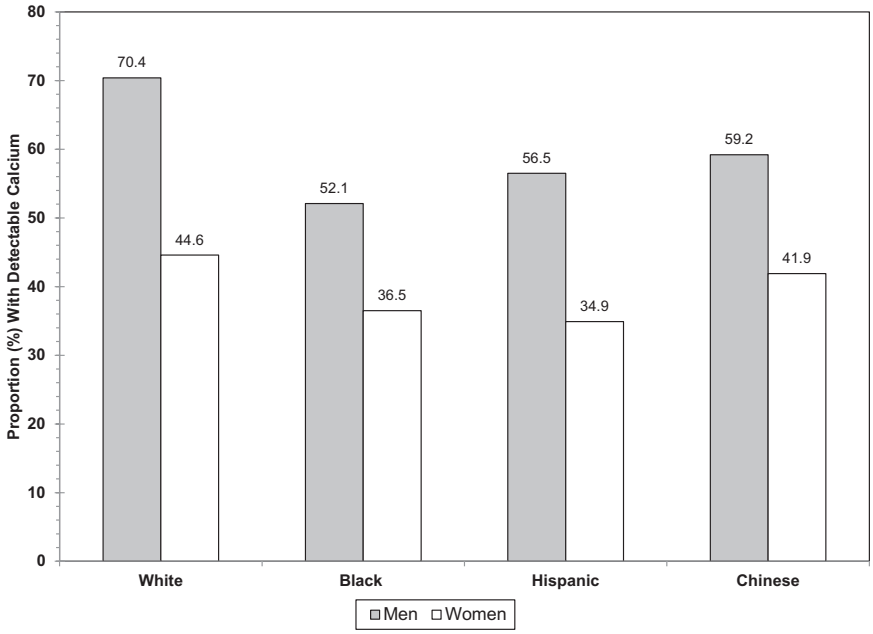

Chart 17-2. Prevalence (%) of coronary calcium: US adults 45 to 84 years of age. *P*<0.0001 across ethnic groups in both men and women. Data derived from Bild et al.[6]

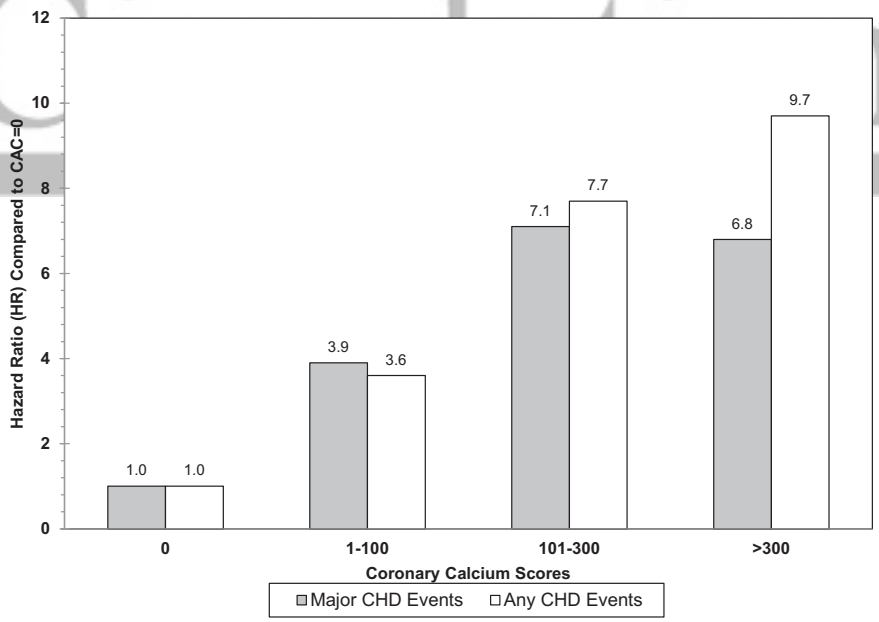

Chart 17-3. Hazard ratios for coronary heart disease (CHD) events associated with coronary calcium scores: US adults 45 to 84 years of age (reference group coronary artery calcification [CAC]=0). All hazard ratios *P*<0.0001. Major CHD events included myocardial infarction and death attributable to CHD; any CHD events included major CHD events plus definite angina or definite or probable angina followed by revascularization. Data derived from Detrano et al.[9]

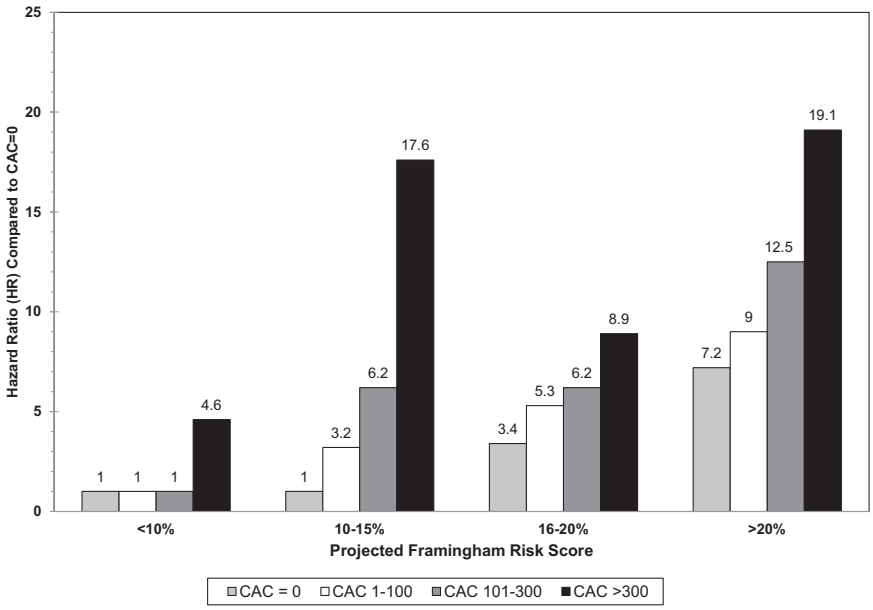

Chart 17-4. Hazard ratios for coronary heart disease events associated with coronary calcium scores: US adults (reference group coronary artery calcification [CAC]=0 and Framingham Risk Score <10%). Coronary heart disease events included nonfatal myocardial infarction and death attributable to coronary heart disease. Data derived from Greenland et al.[10]

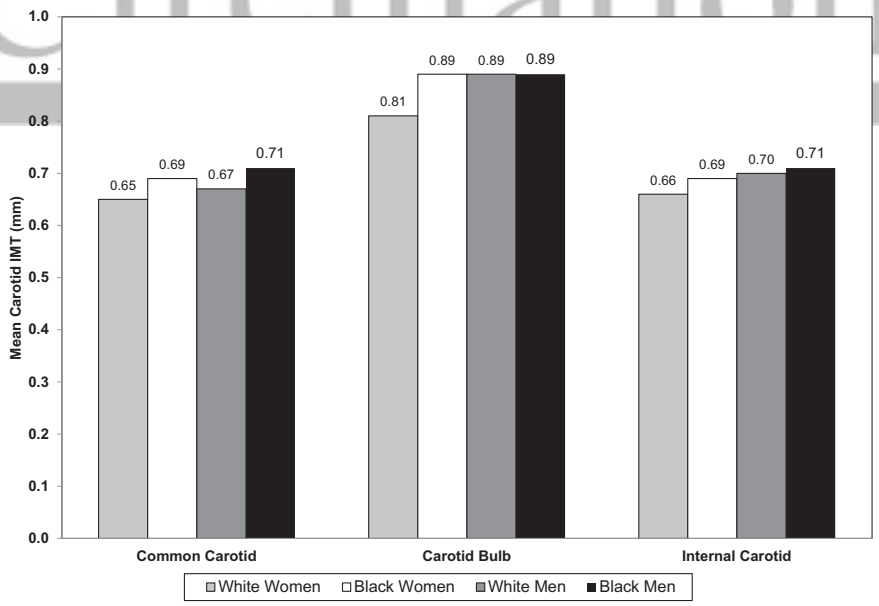

Chart 17-5. Mean values of carotid intima-media thickness (IMT) for different carotid artery segments in younger adults by race and sex (Bogalusa Heart Study). Data derived from Urbina et al.[21]

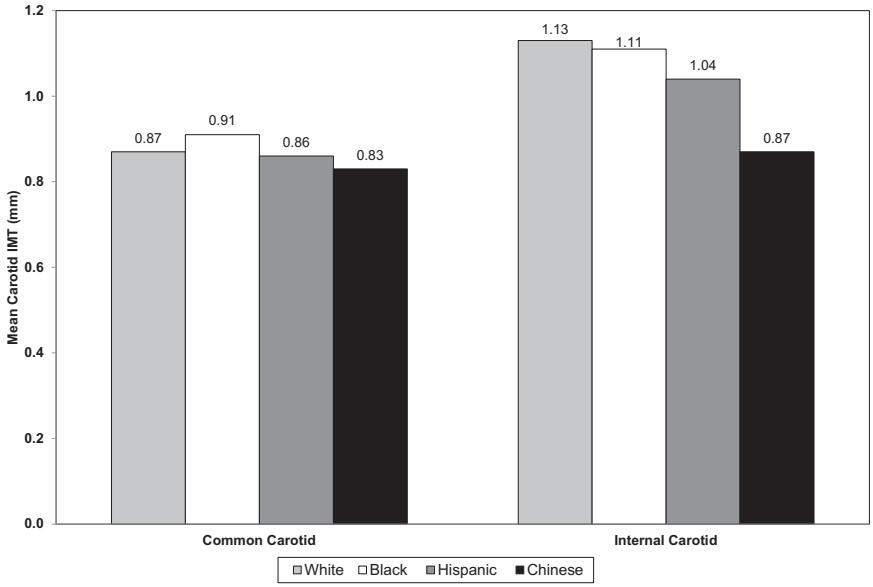

Chart 17-6. Mean values of carotid intima-media thickness (IMT) for different carotid artery segments in older adults, by race. Data derived from Manolio et al.[23]

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18. Coronary Heart Disease, Acute Coronary Syndrome, and Angina Pectoris

Coronary Heart Disease

ICD-9 410 to 414, 429.2; ICD-10 I20 to I25; see Glossary (Chapter 25) for details and definitions. See Tables 18-1 and 18-2. See Charts 18-1 through 18-10.

Prevalence

(See Table 18-1 and Charts 18-1 and 18-2.)

- On the basis of data from NHANES 2007–2010 (NHLBI tabulation), an estimated 15.4 million Americans ≥20 years of age have CHD.
  - —Total CHD prevalence is 6.4% in US adults ≥20 years of age. CHD prevalence is 7.9% for men and 5.1% for women.
  - —Among non-Hispanic whites, CHD prevalence is 8.2% for men and 4.6% for women.
  - —Among non-Hispanic blacks, CHD prevalence is 6.8% for men and 7.1% for women.
  - —Among Mexican Americans, CHD prevalence is 6.7% for men and 5.3% for women.
- On the basis of data from the 2011 NHIS:
  - —Among Hispanic or Latino individuals ≥18 years of age, CHD prevalence is 5.9%.[1]
  - —Among American Indian/Alaska Natives ≥18 years of age, it is estimated that 7.2% have CHD, and among Asians ≥18 years of age, the estimate is 4.3%.[1]
- According to data from NHANES 2007–2010 (NHLBI tabulation), the overall prevalence for MI is 2.9% in US adults ≥20 years of age. MI prevalence is 4.2% for men and 1.7% for women.
  - —Among non-Hispanic whites, MI prevalence is 4.4% for men and 1.5% for women.
  - —Among non-Hispanic blacks, MI prevalence is 3.9% for men and 2.3% for women.
  - —Among Mexican Americans, MI prevalence is 3.6% for men and 1.7% for women.
- Data from the BRFSS 2011 survey indicated that 4.3% of respondents had been told that they had had an MI. The highest prevalence was in Arkansas (6.4%) and West Virginia (6.2%). The lowest prevalence was in Colorado (2.7%) and Utah (3.0%). In the same survey, 4.2% of respondents were told that they had angina or CHD. The highest prevalence was in West Virginia (6.6%), and the lowest was in Colorado (2.4%).[2]
- Projections show that by 2030, prevalence of CHD will increase ≈18% from 2013 estimates (AHA computation, based on methodology described in Heidenreich et al[3]).

Incidence

(See Table 18-1 and Charts 18-3 through 18-5.)

- Approximately every 44 seconds, an American will have an MI (AHA computation) .
- On the basis of data from the ARIC study[4] of the NHLBI:

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Abbreviations Used in Chapter 18

AP	angina pectoris
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CABG	coronary artery bypass graft
CAD	coronary artery disease
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CI	confidence interval
CRUSADE	Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines
CVD	cardiovascular disease
DM	diabetes mellitus
ECG	electrocardiogram
ED	emergency department
EHS-ACS-II	Second Euro Heart Survey on Acute Coronary Syndromes
EMS	emergency medical services
FHS	Framingham Heart Study
GRACE	Global Registry of Acute Coronary Events
GWTG	Get With The Guidelines
HD	heart disease
HDL-C	high-density lipoprotein cholesterol
HF	heart failure
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHIS	National Health Interview Study
NHLBI	National Heart, Lung, and Blood Institute
NRMI	National Registry of Myocardial Infarction
NSTEMI	non–ST-segment–elevation myocardial infarction
OR	odds ratio
PCI	percutaneous coronary intervention
SBP	systolic blood pressure
STEMI	ST-segment–elevation myocardial infarction
UA	unstable angina
WISE	Women's Ischemia Syndrome Evaluation

—This year, ≈635 000 Americans will have a new coronary attack (defined as first hospitalized MI or CHD death), and ≈280 000 will have a recurrent attack. It is estimated that an additional 150 000 silent MIs occur each year. That assumes that ≈21% of the 715 000 first and recurrent MIs are silent.[5,6]
—The estimated annual incidence of MI is 525 000 new attacks and 190 000 recurrent attacks.
—Average age at first MI is 64.7 years for men and 72.2 years for women.

- On the basis of the NHLBI-sponsored FHS:

—CHD makes up more than half of all cardiovascular events in men and women <75 years of age.[5]
—The lifetime risk of developing CHD after 40 years of age is 49% for men and 32% for women.[7]
—The incidence of CHD in women lags behind men by 10 years for total CHD and by 20 years for more serious clinical events such as MI and sudden death.[5]

- In the NHLBI-sponsored ARIC study, in participants 35 to 74 years of age, the average age-adjusted first MI or fatal CHD rates per 1000 population were as follows: white men, 3.9; black men, 5.5; white women, 1.7; and black women, 3.4 (unpublished data from ARIC Surveillance 1987–2009, NHLBI).
- Incidence rates for MI in the NHLBI-sponsored ARIC study are displayed in Charts 18-3 and 18-4, stratified by age, race, and sex. The annual age-adjusted rates per 1000 population of first MI (1987–2009) in ARIC Surveillance (NHLBI) were 5.0 in black men, 3.0 in white men, 2.0 in black women, and 1.6 in white women (unpublished data from ARIC Surveillance 1987–2009, NHLBI).
- Analysis of more than 40 years of physician-validated AMI data in the FHS study of the NHLBI found that AMI rates diagnosed by electrocardiographic criteria declined ≈50%, with a concomitant 2-fold increase in rates of AMI diagnosed by blood markers. These findings may explain the paradoxical stability of AMI rates in the United States despite concomitant improvements in CHD risk factors.[8]
- Among American Indians 65 to 74 years of age, the annual rates per 1000 population of new and recurrent MIs were 7.6 for men and 4.9 for women.[9] Analysis of data from NHANES III (1988–1994) and NHANES 1999–2002 (NCHS) showed that in adults 20 to 74 years of age, the overall distribution of 10-year risk of developing CHD changed little during this time. Among the 3 racial/ethnic groups, blacks had the highest proportion of participants in the high-risk group.[10]
- On the basis of data from the NHDS, since the mid-1990s, the rate of hospitalization for MI and in-hospital case fatality rates have decreased.[11]
- From 2002 to 2007, the rates of hospitalization for MI decreased among Medicare beneficiaries; however, the degree of reduction was more significant in whites than African Americans.[12]

Trends in Incidence

- In the FHS, incidence rates of AMI diagnosed by electrocardiographic criteria decreased significantly in men 50 to 59 years of age and 70 to 79 years of age and in women 70 to 79 years of age from the period 1960–1969 to 1990–1999. In contrast, incidence rates of AMI diagnosed by serum biomarkers increased significantly in men 50 to 59 years of age and 70 to 79 years of age and in women 70 to 79 years of age.[13]
- Data from the Worcester Heart Attack Study showed that incidence rates for AMI were 277 per 100 000 person-years in 1975 and 209 per 100 000 person-years in 2005 (P=0.42 for overall trend).The incidence rate rose from 1975 to 1981, decreased from 1981 to 1988, increased from 1981 to 2001, and decreased from 2001 to 2005.[14]
- In Olmsted County, Minnesota, no significant change in the overall age- and sex-adjusted incidence rate for hospitalized MI was noted (186 per 100 000 person-years in 1987 and 180 per 100 000 person-years in 2006; P=0.171), but a significant decline in the age- and sex-adjusted incidence rate for hospitalized MI based on creatine kinase/creatine kinase-MB markers, to 141 per 100 000 person-years (P=0.020), was observed in 2006, which represents a 20% decrease during the study period.[15]
- Data from Kaiser Permanente Northern California showed that the age- and sex-adjusted incidence rate of hospitalizations for MI changed from 274 per 100 000 person-years in 1999 to 208 per 100 000 person-years in 2008. Furthermore, the age- and sex-adjusted incidence rate of hospitalizations for STEMI changed from 133 per 100 000 person-years in 1999 to 50 per 100 000 person-years in 2008 (P linear trend <0.001). The trajectory of the age- and sex-adjusted incidence rate of hospitalizations for NSTEMI did not change significantly.[16]
- From 1987 to 2008, the age- and biomarker-adjusted incidence rates of hospitalization for AMI or fatal CHD decreased by 4.9% per year (95% CI, 5.3%–4.5%) among white men, 3.9% per year (95% CI, 4.5%–3.4%) among white women, 1.8% per year (95% CI, 2.6%–1.0%) among black men, and 3.5% per year (95% CI, 4.4%–2.6%) among black women in the ARIC study.[17]

Mortality

- CHD caused ≈1 of every 6 deaths in the United States in 2009. CHD mortality was 386 324.[18]
- CHD any-mention mortality was 549 233. MI mortality (ICD-10 I21 to I22) was 125 464. MI any-mention mortality was 162 443 (NCHS, NHLBI tabulation).[18]
- In 2009, the overall CHD death rate was 116.1. From 1999 to 2009, the annual death rate attributable to CHD declined 40.3% and the actual number of deaths declined 27.1% (AHA computation).[19] The death rates were 155.9 for white males and 181.1 for black males; for white females, the rate was 84.9, and for black females, it was 110.3.[19]
- Approximately every 34 seconds, an American will experience a coronary event, and approximately every minute, someone will die of one (AHA computation).
- Approximately 34% of the people who experience a coronary attack in a given year will die of it, and ≈15% who experience a heart attack (MI) will die of it (AHA computation).
- The percentage of CHD deaths that occurred out of the hospital in 2009 was 73%. According to NCHS mortality data, 281 000 CHD deaths occur out of the hospital or in hospital

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EDs annually (2009, ICD-10 codes I20 to I25; NCHS, AHA tabulation).

- A study of 1275 health maintenance organization enrollees 50 to 79 years of age who had cardiac arrest showed that the incidence of out-of-hospital cardiac arrest was 6.0/1000 subject-years in subjects with any clinically recognized HD compared with 0.8/1000 subject-years in subjects without HD. In subgroups with HD, incidence was 13.6/1000 subject-years in subjects with prior MI and 21.9/1000 subject-years in subjects with HF.[20]
- Approximately 80% of people who die of CHD are ≥65 years of age (NCHS; AHA computation).
- The estimated average number of years of life lost because of an MI is 16.6 (NCHS, NHLBI tabulation).
- On the basis of data from the FHS of the NHLBI[5]:
  - —Fifty percent of men and 64% of women who die suddenly of CHD have no previous symptoms of this disease. Between 70% and 89% of sudden cardiac deaths occur in men, and the annual incidence is 3 to 4 times higher in men than in women; however, this disparity decreases with advancing age.
  - —People who have had an MI have a sudden death rate 4 to 6 times that of the general population.
- Researchers investigating variation in hospital-specific 30-day risk-stratified mortality rates for patients with AMI found teaching status, number of hospital beds, AMI volume, cardiac facilities available, urban/rural location, geographic region, hospital ownership type, and socioeconomic status profile of the patients were all significantly associated with mortality rates. However, a substantial proportion of variation in outcomes for patients with AMI between hospitals remains unexplained by measures of hospital characteristics.[21]

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Temporal Trends in CHD Mortality

- The decline in CHD mortality rates in part reflects the shift in the pattern of clinical presentations of AMI. In the past decade, there has been a marked decline in STEMI (from 133 to 50 cases per 100 000 person-years).[16]
- According to data from the National Registry of Myocardial Infarction[22]:
  - —From 1990 to 1999, in-hospital AMI mortality declined from 11.2% to 9.4%.
  - —Mortality rate increases for every 30 minutes that elapses before a patient with ST-segment elevation is recognized and treated.
- Other studies also reported declining case fatality rates after MI:
  - —In Olmsted County, Minnesota, the age- and sex-adjusted 30-day case-fatality rate decreased by 56% from 1987 to 2006.[15]
  - —In Worcester, MA, the hospital case fatality rates, 30-day postadmission case fatality rates, and 1-year postdischarge case fatality rates for STEMI were 11.1%, 13.2%, and 10.6%, respectively, in 1997 and 9.7%, 11.4%, and 8.4%, respectively, in 2005. The hospital case fatality rates, 30-day postadmission case fatality rates, and 1-year postdischarge case fatality rates for NSTEMI were 12.9%, 16.0%, and 23.1%, respectively, in 1997 and 9.5%, 14.0%, and 18.7%, respectively, in 2005.[23]
  - —Among enrollees of the Kaiser Permanente Northern California healthcare delivery system, the age- and sex-adjusted 30-day mortality rate for MI dropped from 10.5% in 1999 to 7.8% in 2008, and the 30-day mortality rate for NSTEMI dropped from 10.0% in 1999 to 7.6% in 2008.[16]
- CHD death rates have fallen from 1968 to the present. Analysis of NHANES (NCHS) data compared CHD death rates between 1980 and 2000 to determine how much of the decline in deaths attributable to CHD over that period could be explained by the use of medical and surgical treatments versus changes in CVD risk factors (resulting from lifestyle/behavior). After 1980 and 2000 data were compared, it was estimated that ≈47% of the decrease in CHD deaths was attributable to treatments, including the following[24]:
  - —Secondary preventive therapies after MI or revascularization (11%)
  - —Initial treatments for AMI or UA (10%)
  - —Treatments for HF (9%)
  - —Revascularization for chronic angina (5%)
  - —Other therapies (12%), including antihypertensive and lipid-lowering primary prevention therapies
- It was also estimated that a similar amount of the reduction in CHD deaths, ≈44%, was attributable to changes in risk factors, including the following[24]:
  - —Lower total cholesterol (24%)
  - —Lower SBP (20%)
  - —Lower smoking prevalence (12%)
  - —Decreased physical inactivity (5%)
  - —Nevertheless, these favorable improvements in risk factors were offset in part by increases in BMI and in DM prevalence, which accounted for an increased number of deaths (8% and 10%, respectively).
- Between 1980 and 2002, death rates attributable to CHD among men and women ≥65 years of age fell by 52% in men and 49% in women. Among men, the death rate declined on average by 2.9% per year in the 1980s, 2.6% per year during the 1990s, and 4.4% per year from 2000 to 2002. Among women, death rates fell by 2.6%, 2.4%, and 4.4%, respectively. However, when stratified by age, among men 35 to 54 years of age, the average annual rate of death fell by 6.2%, 2.3%, and 0.5%, respectively. Among women 35 to 54 years of age, the average annual rate of death fell by 5.4% and 1.2% and then increased by 1.5%, respectively. This increase was not statistically significant; however, in even younger women (35–44 years of age), the rate of death has been increasing by an average of 1.3% annually between 1997 and 2002, which is statistically significant.[25]
- An analysis of 28 studies published from 1977 to 2007 found that revascularization by CABG or PCI in conjunction with medical therapy in patients with nonacute CAD is associated with significantly improved survival compared with medical therapy alone.[26]
- A recent analysis of Centers for Medicare & Medicaid Services data suggests that between 1995 and 2006, the 30-day

mortality rate attributable to MI decreased, as did hospital variation in mortality attributable to MI.[27]

- Data from the Nationwide Inpatient Sample database suggest that mortality attributable to MI has decreased since 1988.[28]

Risk Factors

- Risk factors for CHD act synergistically to increase CHD risk, as shown in the example in Charts 18-6 and 18-7.

Awareness of Warning Signs and Risk Factors for HD

- Data from the Women Veterans Cohort showed that 42% of women ≥35 years of age were concerned about HD. Only 8% to 20% were aware that CAD is the major cause of death for women.[29]
- Among people in 14 states and Washington, DC, participating in the 2005 BRFSS, only 27% were aware of 5 heart attack warning signs and symptoms (1, pain in jaw, neck, or back; 2, weak, lightheaded, or faint; 3, chest pain or discomfort; 4, pain or discomfort in arms or shoulder; and 5, shortness of breath) and indicated that they would first call 911 if they thought someone was having a heart attack or stroke. Awareness of all 5 heart attack warning signs and symptoms and the need to call 911 was higher among non-Hispanic whites (30.2%), women (30.8%), and those with a college education or more (33.4%) than among non-Hispanic blacks and Hispanics (16.2% and 14.3%, respectively), men (22.5%), and those with less than a high school education (15.7%), respectively. By state, awareness was highest in West Virginia (35.5%) and lowest in Washington, DC (16.0%).[30]
- A 2004 national study of physician awareness and adherence to CVD prevention guidelines showed that fewer than 1 in 5 physicians knew that more women than men die each year of CVD.[31] Women's awareness that CVD is their leading cause of death increased from 30% in 1997 to 54% in 2009. The percentages of women identifying warning signs for a heart attack were as follows: pain in the chest, neck, shoulder, and arm—56%; shortness of breath—29%, chest tightness—17%, nausea—15%; and fatigue—7%. Compared with 1997, however, little change in knowledge of warning signs was noted. The 3 most commonly cited HD prevention strategies were getting adequate sleep (94%), consuming fish oil/omega 3 fatty acids (82%), and taking aspirin regularly (78%). The most common preventive actions taken by women during the preceding year were having BP checked (84%), trying to manage stress better (74%), seeing a doctor or other healthcare provider (73%), decreasing consumption of unhealthy foods (71%), and having cholesterol checked (66%).[32]

Delays Between Symptom Onset and Arrival at Hospital

- A recent community surveillance study in 4 US communities reported that in 2000, the overall proportion of people with delays of ≥4 hours from onset of AMI symptoms to hospital arrival was 49.5%. The study also reported that from 1987 to 2000, there was no statistically significant change in the proportion of patients whose delays were ≥4 hours, which indicates that there has been little improvement in the speed at which patients with MI symptoms arrive at the hospital after symptom onset. Although the proportion of patients with MI who arrived at the hospital by EMS increased over this period, from 37% in 1987 to 55% in 2000, the total time between onset and hospital arrival did not change appreciably.[33]
- Data from CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines) and the National Cardiovascular Data Registry ACTION Registry–GWTG showed a longer median time to hospital presentation in men (3 hours) than women (2.8 hours; $P<0.001$). From 2002 to 2007, presentation time did not change significantly in men or women.[13]
- Individuals with documented CHD have 5 to 7 times the risk of having a heart attack or dying as the general population. Survival rates improve after a heart attack if treatment begins within 1 hour; however, most patients are admitted to the hospital 2.5 to 3 hours after symptoms begin. More than 3500 patients surveyed with a history of CHD were asked to identify possible symptoms of heart attack. Despite their history of CHD, 44% had low knowledge levels. In this group, who were all at high risk of future AMI, 43% assessed their risk as less than or the same as others their age. More men than women perceived themselves as being at low risk, at 47% versus 36%, respectively.[34]
- Data from Worcester, MA, indicate that the average time from symptom onset to hospital arrival has not improved and that delays in hospital arrival are associated with less receipt of guidelines-based care. Mean and median prehospital delay times from symptom onset to arrival at the hospital were 4.1 and 2.0 hours in 1986 and 4.6 and 2.0 hours in 2005, respectively. Compared with those arriving within 2 hours of symptom onset, those with prolonged prehospital delay were less likely to receive thrombolytic therapy and PCI within 90 minutes of hospital arrival.[35]
- In an analysis from ARIC, low neighborhood household income (OR, 1.46; 95% CI, 1.09–1.96) and being a Medicaid recipient (OR, 1.87; 95% CI, 1.10–3.19) were associated with increased odds of having prolonged prehospital delays from symptom onset to hospital arrival for AMI compared with individuals with higher neighborhood household income and other insurance providers, respectively.[36]

Aftermath

- Depending on their sex and clinical outcome, people who survive the acute stage of an MI have a chance of illness and death 1.5 to 15 times higher than that of the general population. Among these people, the risk of another MI, sudden death, AP, HF, and stroke—for both men and women—is substantial (FHS, NHLBI).[5]
- On the basis of pooled data from the FHS, ARIC, and CHS studies of the NHLBI, within 1 year after a first MI:

  —At ≥45 years of age, 19% of men and 26% of women will die.

  —At 45 to 64 years of age, 5% of white men, 8% of white women, 14% of black men, and 9% of black women will die.

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—At ≥65 years of age, 25% of white men, 30% of white women, 25% of black men, and 30% of black women will die.
—In part because women have MIs at older ages than men, they are more likely to die of MIs within a few weeks.

- Within 5 years after a first MI:

—At ≥45 years of age, 36% of men and 47% of women will die.
—At 45 to 64 years of age, 11% of white men, 18% of white women, 22% of black men, and 28% of black women will die.
—At ≥65 years of age, 46% of white men, 53% of white women, 54% of black men, and 58% of black women will die.

- Of those who have a first MI, the percentage with a recurrent MI or fatal CHD within 5 years is:

—At 45 to 64 years of age, 15% of men and 22 of women
—At ≥65 years of age, 22% of men and women
—At 45 to 64 years of age, 14% of white men, 18% of white women, 22% of black men, and 28% of black women
—At ≥65 years of age, 21% of white men and women, 33% of black men, and 26% of black women

- The percentage of people with a first MI who will have HF in 5 years is:

—At 45 to 64 years of age, 8% of men and 18% of women
—At ≥65 years of age, 20% of men and 23% of women
—At 45 to 64 years of age, 7% of white men, 15% of white women, 13% of black men, and 25% of black women
—At ≥65 years of age, 19% of white men, 23% of white women, 31% of black men, and 24% of black women

- The percentage of people with a first MI who will have a stroke within 5 years is:

—At 45 to 64 years of age, 2% of men and 6% of women
—At ≥65 years of age, 5% of men and 8% of women
—At 45 to 64 years of age, 2% of white men, 4% of white women, 3% of black men, and 10% of black women
—At ≥65 years of age, 5% of white men, 8% of white women, 9% of black men, and 10% of black women

- The median survival time (in years) after a first MI is:

—At 55 to 64 years of age, 17.0 for men and 13.3 for women
—At 65 to 74 years of age, 9.3 for men and 8.8 for women
—At ≥75 years of age, 3.2 for men and 3.2 for women

- A Mayo Clinic study found that cardiac rehabilitation after an MI is underused, particularly in women and the elderly. Women were 55% less likely than men to participate in cardiac rehabilitation, and older study patients were less likely to participate than younger participants. Only 32% of men and women ≥70 years of age participated in cardiac rehabilitation compared with 66% of those 60 to 69 years of age and 81% of those <60 years of age.[37]
- Among survivors of an MI, in 2005, 34.7% of BRFSS respondents participated in outpatient cardiac rehabilitation. The prevalence of cardiac rehabilitation was higher among older age groups (≥50 years of age), among men versus women, among Hispanics, among those who were married, among those with higher education, and among those with higher levels of household income.[38]
- A recent analysis of Medicare claims data revealed that only 13.9% of Medicare beneficiaries enroll in cardiac rehabilitation after an AMI, and only 31% enroll after CABG. Older people, women, nonwhites, and individuals with comorbidities were less likely to enroll in cardiac rehabilitation programs.[39]

Hospital Discharges and Ambulatory Care Visits

(See Table 18-1 and Chart 18-8.)

- From 2000 to 2010, the number of inpatient discharges from short-stay hospitals with CHD as the first-listed diagnosis decreased from 2 165 000 to 1 346 000 (NHDS, NHLBI tabulation).
- In 2009, there were 14 044 000 ambulatory care visits with CHD as the first-listed diagnosis (NCHS, NAMCS, NHAMCS). There were 12 816 000 physician office visits, 639 000 ED visits, and 589 000 outpatient department visits with a primary diagnosis of CHD (NHAMCS, NHLBI tabulation). The majority of these visits (77.7%) were for coronary atherosclerosis.[40]
- The age-adjusted hospitalization rate for MI was 215 per 100 000 people in 1979 to 1981, increased to 342 in 1985 to 1987, stabilized for the next decade, and then declined after 1996 to 242 during the period from 2003 to 2005. Rates for men were almost twice those of women. Trends were similar for men and women. Hospitalization rates increased with age and were the highest among those ≥85 years of age.[11]
- Most hospitalized patients >65 years of age are women. For MI, 28.4% of hospital stays for people 45 to 64 years of age were for women, but 63.7% of stays for those ≥85 years of age were for women. Similarly, for coronary atherosclerosis, 32.7% of stays among people 45 to 64 years of age were for women; this figure increased to 60.7% of stays among those ≥85 years of age. For nonspecific chest pain, women were more numerous than men among patients <65 years of age. Approximately 54.4% of hospital stays among people 45 to 64 years of age were for women. Women constituted 73.9% of hospital stays for nonspecific chest pain among patients ≥85 years of age, higher than for any other condition examined. For AMI, one third more women than men died in the hospital: 9.3% of women died in the hospital compared with 6.2% of men.[41]

Operations and Procedures

- In 2010, an estimated 954 000 inpatient PCI procedures, 397 000 inpatient bypass procedures, 1 029 000 inpatient diagnostic cardiac catheterizations, 97 000 inpatient implantable defibrillator procedures, and 370 000 pacemaker procedures were performed for inpatients in the United States. (NHLBI tabulation).

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Cost
(See Table 18-1.)

- The estimated direct and indirect cost of CHD in 2009 is $195.2 billion (MEPS, NHLBI tabulation).
- In 2006, $11.7 billion was paid to Medicare beneficiaries for in-hospital costs when CHD was the principal diagnosis ($14 009 per discharge for AMI, $12 977 per discharge for coronary atherosclerosis, and $10 630 per discharge for other ischemic HD).[34,42]
- Between 2013 and 2030, medical costs of CHD (real 2010$) are projected to increase by ≈100%:

  —Indirect costs for all CVD (real 2010$) are projected to increase 52% (from $202.5 billion to $308.2 billion) between 2013 and 2030. Of these indirect costs, CHD is projected to account for ≈43% and has the largest indirect costs (AHA computation, based on methodology described in Heidenreich et al[3]).

Acute Coronary Syndrome

ICD-9 codes 410, 411; ICD-10 I20.0, I21, I22

The term ACS is increasingly used to describe patients who present with either AMI or UA. (UA is chest pain or discomfort that is accelerating in frequency or severity and may occur while at rest but does not result in myocardial necrosis.) The discomfort may be more severe and prolonged than typical AP or may be the first time a person has AP. UA, NSTEMI, and STEMI share common pathophysiological origins related to coronary plaque progression, instability, or rupture with or without luminal thrombosis and vasospasm.

- A conservative estimate for the number of discharges with ACS from hospitals in 2010 is 625 000. Of these, an estimated 363 000 are males and 262 000 are females. This estimate is derived by adding the first-listed inpatient hospital discharges for MI (595 000) to those for UA (30 000; NHDS, NHLBI).
- When secondary discharge diagnoses in 2010 were included, the corresponding number of inpatient hospital discharges was 1 141 000 unique hospitalizations for ACS; 653 000 were males, and 488 000 were females. Of the total, 813 000 were for MI alone, 322 000 were for UA alone, and 6000 hospitalizations received both diagnoses (NHDS, NHLBI).

Decisions about medical and interventional treatments are based on specific findings noted when a patient presents with ACS. Such patients are classified clinically into 1 of 3 categories according to the presence or absence of ST-segment elevation on the presenting ECG and abnormal ("positive") elevations of myocardial biomarkers, such as troponins, as follows:

- STEMI
- NSTEMI
- UA

The percentage of ACS or MI cases with ST-segment elevation varies in different registries/databases and depends heavily on the age of patients included and the type of surveillance used. According to the National Registry of Myocardial Infarction 4 (NRMI-4), ≈29% of patients with MI are patients with STEMI.[43] The AHA GWTG project found that 32% of the patients with MI in the CAD module are patients with STEMI (personal communication from AHA GWTG staff, October 1, 2007). The Global Registry of Acute Coronary Events (GRACE) study, which includes US patient populations, found that 38% of ACS patients have STEMI, whereas the second Euro Heart Survey on ACS (EHS-ACS-II) reported that ≈47% of patients with ACS have STEMI.[44]

In addition, the percentage of ACS or MI cases with ST-segment elevation appears to be declining. In an analysis of 46 086 hospitalizations for ACS in the Kaiser Permanente Northern California study, the percentage of MI cases with ST-segment elevation decreased from 48.5% to 24% between 1999 and 2008.[16]

- Analysis of data from the GRACE multinational observational cohort study of patients with ACS found evidence of a change in practice for both pharmacological and interventional treatments in patients with either STEMI or non–ST-segment–elevation ACS. These changes have been accompanied by significant decreases in the rates of in-hospital death, cardiogenic shock, and new MI among patients with non–ST-segment–elevation ACS. The use of evidence-based therapies and PCI interventions increased in the STEMI population. This increase was matched by a statistically significant decrease in the rates of death, cardiogenic shock, and HF or pulmonary edema.[45]
- A study of patients with non–ST-segment–elevation ACS treated at 350 US hospitals found that up to 25% of opportunities to provide American College of Cardiology (ACC)/AHA guideline–recommended care were missed in current practice. The composite guideline adherence rate was significantly associated with in-hospital mortality.[45]
- A study of hospital process performance in 350 centers of nearly 65 000 patients enrolled in the CRUSADE National Quality Improvement Initiative found that ACC/AHA guideline–recommended treatments were adhered to in 74% of eligible instances.[46]
- After adjustment for clinical differences and the severity of CAD by angiogram, 30-day mortality after ACS is similar in men and women.[47]

Angina Pectoris

ICD-9 413; ICD-10 I20.1 to I20.9.

Prevalence
(See Table 18-2 and Chart 18-9.)

- A study of 4 national cross-sectional health examination studies found that among Americans 40 to 74 years of age, the age-adjusted prevalence of AP was higher among women than men. Increases in the prevalence of AP occurred for Mexican American men and women and African American women but were not statistically significant for the latter.[48]

Incidence
(See Table 18-2 and Chart 18-10.)

- Only 18% of coronary attacks are preceded by long-standing AP (NHLBI computation of FHS follow-up since 1986).

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- The annual rates per 1000 population of new episodes of AP for nonblack men are 28.3 for those 65 to 74 years of age, 36.3 for those 75 to 84 years of age, and 33.0 for those ≥85 years of age. For nonblack women in the same age groups, the rates are 14.1, 20.0, and 22.9, respectively. For black men, the rates are 22.4, 33.8, and 39.5, and for black women, the rates are 15.3, 23.6, and 35.9, respectively (CHS, NHLBI).[49]
- On the basis of 1987 to 2001 data from the ARIC study of the NHLBI, the annual rates per 1000 population of new episodes of AP for nonblack men are 8.5 for those 45 to 54 years of age, 11.9 for those 55 to 64 years of age, and 13.7 for those 65 to 74 years of age. For nonblack women in the same age groups, the rates are 10.6, 11.2, and 13.1, respectively. For black men, the rates are 11.8, 10.6, and 8.8, and for black women, the rates are 20.8, 19.3, and 10.0, respectively.[49]

Mortality

- A small number of deaths resulting from CHD are coded as being attributable to AP. These are included as a portion of total deaths attributable to CHD.

Cost

- For women with nonobstructive CHD enrolled in the Women's Ischemia Syndrome Evaluation (WISE) study of the NHLBI, the average lifetime cost estimate was ≈$770 000 and ranged from $1.0 to $1.1 million for women with 1-vessel to 3-vessel CHD.[50]

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Table 18-1. Coronary Heart Disease

Population Group	Prevalence, CHD, 2010 Age ≥20 y	Prevalence, MI, 2010 Age ≥20 y	New and Recurrent MI and Fatal CHD, Age ≥35 y	New and Recurrent MI, Age ≥35 y	Mortality,* CHD, 2009 All Ages	Mortality,* MI, 2009 All Ages	Hospital Discharges CHD, 2010 All Ages
Both sexes	15 400 000 (6.4%)	7 600 000 (2.9%)	915 000	715 000	386 324	125 464	1 346 000
Males	8 800 000 (7.9%)	5 000 000 (4.2%)	535 000	410 000	210 069 (54.4%)†	68 814 (54.8%)†	828 000
Females	6 600 000 (5.1%)	2 600 000 (1.7%)	380 000	305 000	176 255 (45.6%)†	56 650 (45.2%)†	518 000
NH white males	8.2%	4.4%	465 000‡	…	183 453	60 316	…
NH white females	4.6%	1.5%	325 000‡	…	152 785	48 802	…
NH black males	6.8%	3.9%	65 000‡	…	21 051	6717	…
NH black females	7.1%	2.3%	60 000‡	…	19 470	6567	…
Mexican-American males	6.7%	3.6%	…	…	…	…	…
Mexican-American females	5.3%	1.7%	…	…	…	…	…
Hispanic or Latino	5.9%§	…	…	…	…	…	…
Asian	4.3%§	…	…	…	7752‖	2462‖	…
Native Hawaiian and other Pacific Islander	§¶	…	…	…	…	…	…
American Indian/ Alaska Native	7.2%§	…	…	…	1813	600	…

CHD includes people who responded "yes" to at least 1 of the questions in "Has a doctor or other health professional ever told you that you had coronary heart disease, angina or angina pectoris, heart attack, or myocardial infarction?" Those who answered "no" but were diagnosed with Rose angina are also included (the Rose questionnaire is only administered to survey participants >40 years of age).

CHD indicates coronary heart disease; MI, myocardial infarction; NH, non-Hispanic; and ellipses (...), data not available.

*Mortality data for the white, black, Asian or Pacific Islander, and American Indian/Alaska Native populations include deaths of persons of Hispanic and non-Hispanic origin. Numbers of deaths for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated.

†These percentages represent the portion of total CHD and MI mortality that is for males versus females.

‡Estimates include Hispanics and non-Hispanics. Estimates for whites include other nonblack races.

§National Health Interview Survey, National Center for Health Statistics 2010; data are weighted percentages for Americans ≥18 years of age.[1]

‖Includes Chinese, Filipino, Hawaiian, Japanese, and Other Asian or Pacific Islander.

¶Estimate considered unreliable or does not meet standards of reliability or precision.

Sources: Prevalence: National Health and Nutrition Examination Survey 2007–2010 (National Center for Health Statistics) and National Heart, Lung, and Blood Institute. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2010 US population estimates. These data are based on self-reports. Incidence: Atherosclerosis Risk in Communities study (2004–2009), National Heart, Lung, and Blood Institute. Mortality: Centers for Disease Control and Prevention/National Center for Health Statistics, 2009 Mortality Multiple Cause–US, version July 19, 2012. Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics (data include those inpatients discharged alive, dead, or status unknown).

Table 18-2. Angina Pectoris

Population Group	Prevalence, 2010, Age ≥20 y	Incidence of Stable AP, Age ≥45 y	Hospital Discharges, 2010, All Ages
Both sexes	7 800 000 (3.2%)	500 000	22 000
Males	3 700 000 (3.3%)	320 000	12 000
Females	4 100 000 (3.2%)	180 000	10 000
NH white males	3.3%	…	…
NH white females	2.8%	…	…
NH black males	2.4%	…	…
NH black females	5.4%	…	…
Mexican-American males	3.4%	…	…
Mexican-American females	3.3%	…	…

AP is chest pain or discomfort that results from insufficient blood flow to the heart muscle. Stable AP is predictable chest pain on exertion or under mental or emotional stress. The incidence estimate is for AP without myocardial infarction.

AP indicates angina pectoris; NH, non-Hispanic; and ellipses, data not available.

*There were 56 000 days of care for discharges of patients with AP from short-stay hospitals in 2010.

Sources: Prevalence: National Health and Nutrition Examination Survey 2007–2010 (National Center for Health Statistics) and National Heart, Lung, and Blood Institute; percentages for racial/ethnic groups are age-adjusted for US adults ≥20 years of age. AP includes persons who either answered "yes" to the question of ever having angina or AP or who were diagnosed with Rose angina (the Rose questionnaire is only administered to survey participants >40 years of age). Estimates from National Health and Nutrition Examination Survey 2007–2010 (National Center for Health Statistics) were applied to 2010 population estimates (≥20 years of age). Incidence: AP uncomplicated by a myocardial infarction or with no myocardial infarction (Framingham Heart Study 1980 to 2001–2003 of the original cohort and 1980 to 1998–2001 of the Offspring Cohort, National Heart, Lung, and Blood Institute). Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics; data include those inpatients discharged alive, dead, or status unknown.

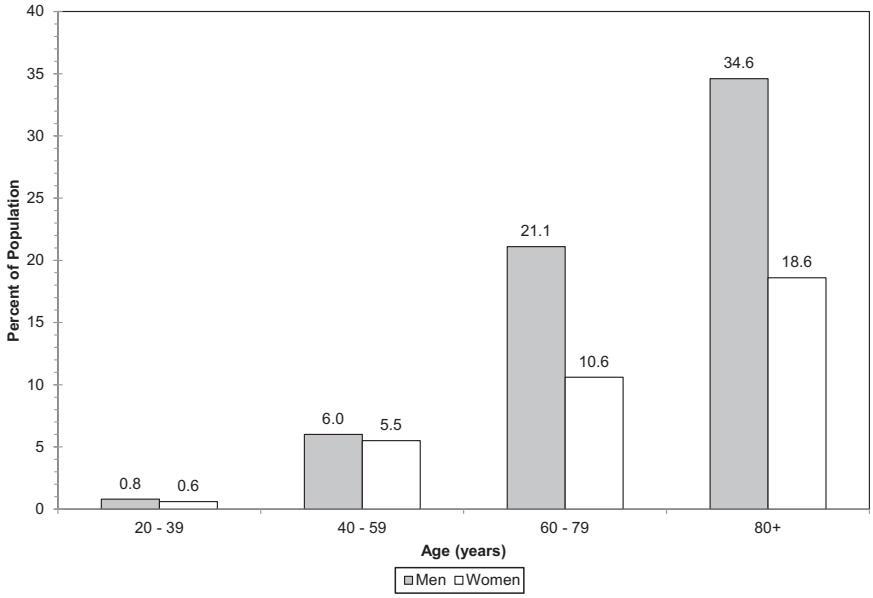


Chart 18-1. Prevalence of coronary heart disease by age and sex (National Health and Nutrition Examination Survey: 2007–2010). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

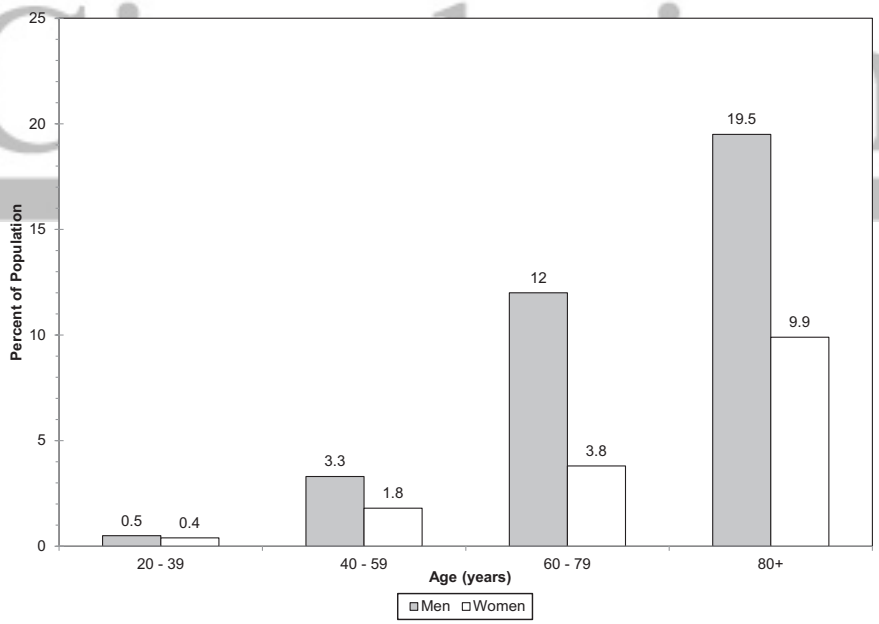


Chart 18-2. Prevalence of myocardial infarction by age and sex (National Health and Nutrition Examination Survey: 2007–2010). Myocardial infarction includes people who answered "yes" to the question of ever having had a heart attack or myocardial infarction. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

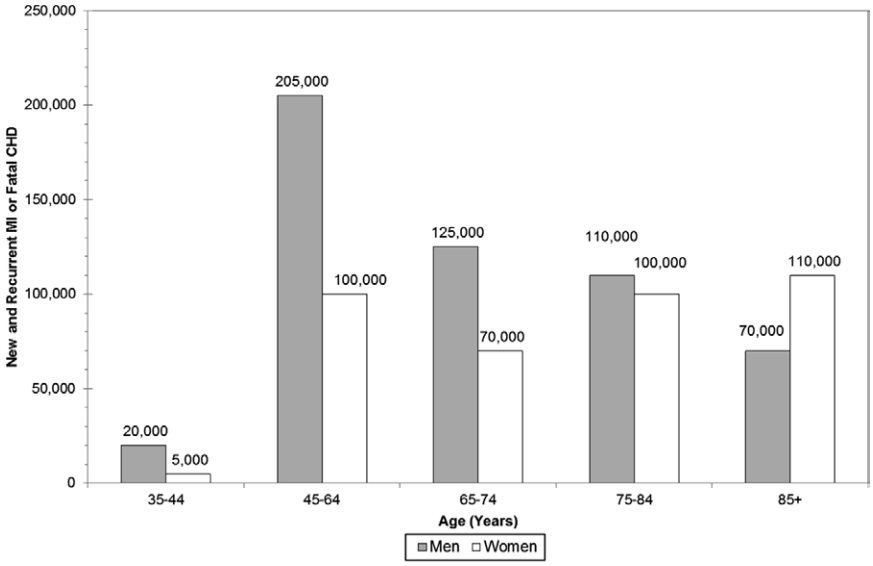
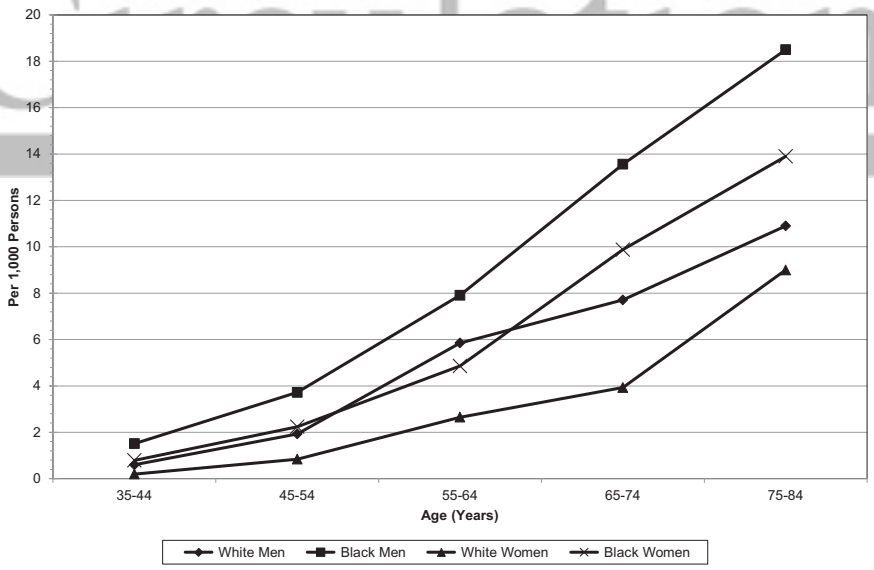
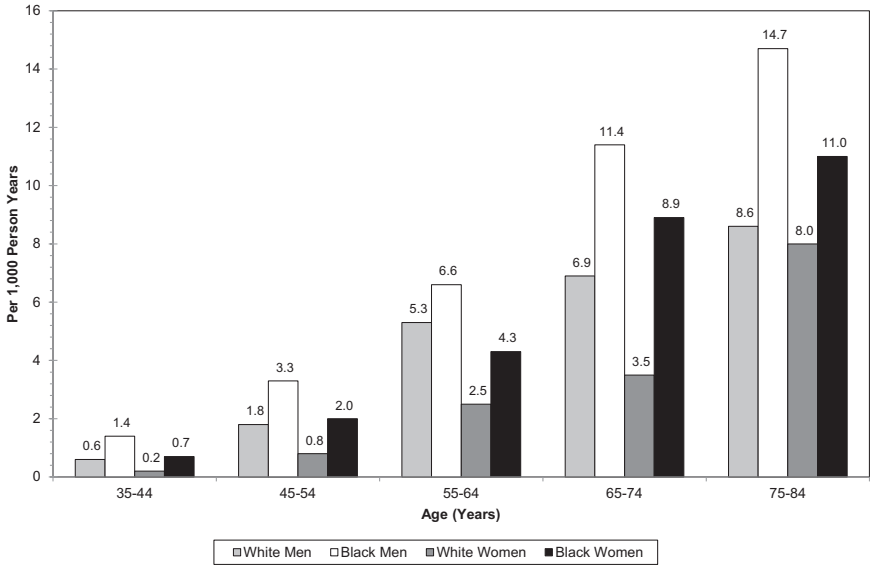


Chart 18-3. Annual number of adults having diagnosed heart attack or fatal coronary heart disease (CHD) by age and sex (Atherosclerosis Risk in Communities Surveillance: 2004–2009 and Cardiovascular Health Study). These data include myocardial infarction (MI) and fatal CHD but not silent MI. Source: National Heart, Lung, and Blood Institute.



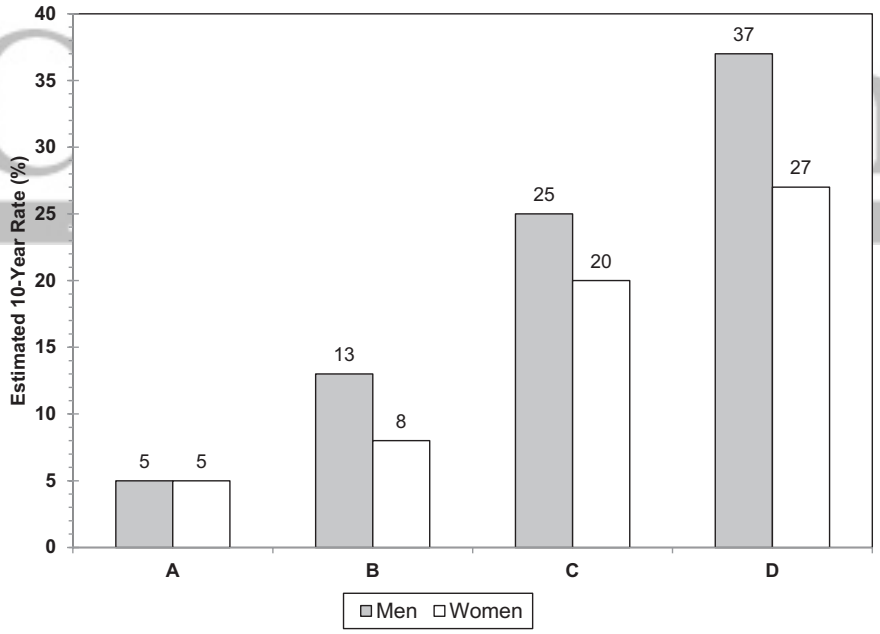
* 2005-2009 for ages 75-84 years

Chart 18-4. Incidence of heart attack or fatal coronary heart disease by age, sex, and race (Atherosclerosis Risk in Communities Surveillance: 2004–2009*). Source: National Heart, Lung, and Blood Institute. *2005–2009 for ages 75–84 years.



* 2005-2009 for ages 75-84 years

Chart 18-5. Incidence of myocardial infarction* by age, sex, and race (Atherosclerosis Risk in Communities Surveillance: 2004-2009*). Source: Unpublished data from Atherosclerosis Risk in Communities study, National Heart, Lung, and Blood Institute. *2005–2009 for ages 75–84 years.



	A	B	C	D
Blood Pressure	120/80	140/90	140/90	140/90
Cholesterol	200	240	240	240
HDL-C	50	50	40	40
Diabetes	No	No	Yes	Yes
Cigarettes	No	No	No	Yes

Chart 18-6. Estimated 10-year coronary heart disease risk in adults 55 years of age according to levels of various risk factors (Framingham Heart Study). HDL-C indicates high density lipoprotein-cholesterol. Data derived from Wilson et al.[51]

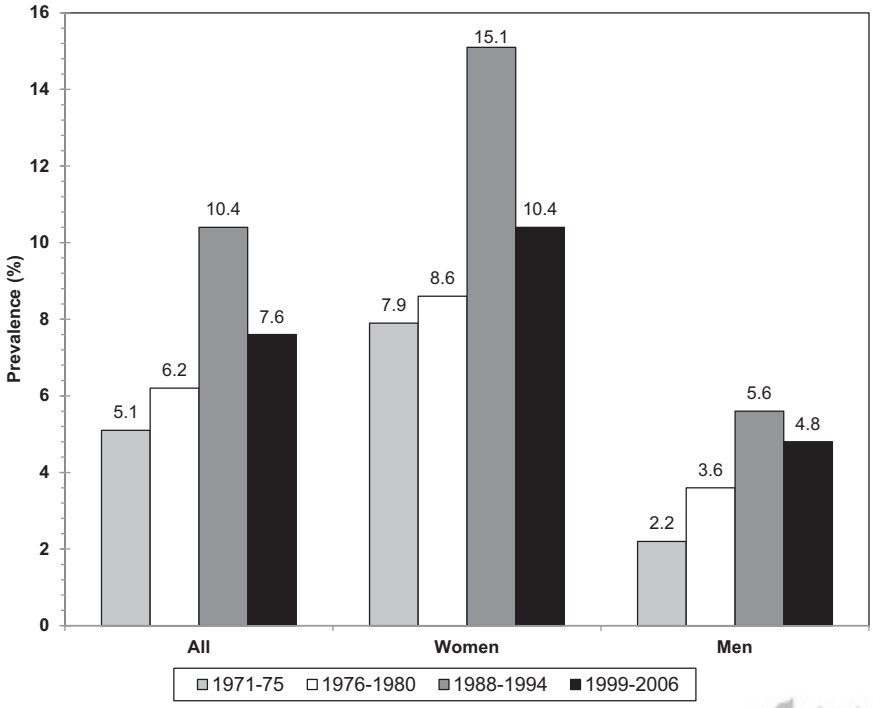


Chart 18-7. Prevalence of low coronary heart disease risk, overall and by sex (National Health and Nutrition Examination Survey: 1971–2006). Low risk is defined as systolic blood pressure <120 mm Hg and diastolic blood pressure <80 mm Hg; cholesterol <200 mg/dL; body mass index <25 kg/m^2; currently not smoking cigarettes; and no prior myocardial infarction or diabetes mellitus. Source: Personal communication with the National Heart, Lung, and Blood Institute, June 28, 2007.

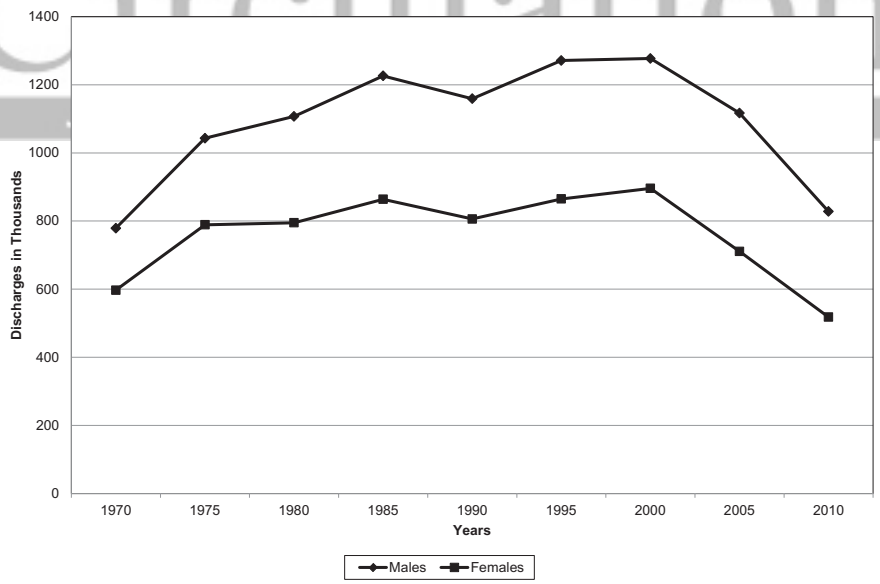


Chart 18-8. Hospital discharges for coronary heart disease by sex (United States: 1970–2010). Hospital discharges include people discharged alive, dead, and "status unknown." Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

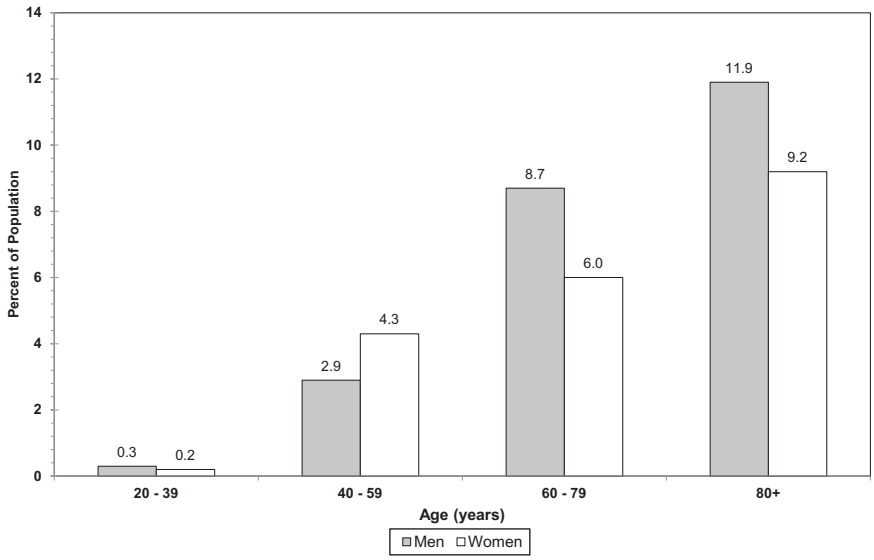


Chart 18-9. Prevalence of angina pectoris by age and sex (National Health and Nutrition Examination Survey: 2007–2010). Angina pectoris includes people who either answered "yes" to the question of ever having angina or angina pectoris or were diagnosed with Rose Angina. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

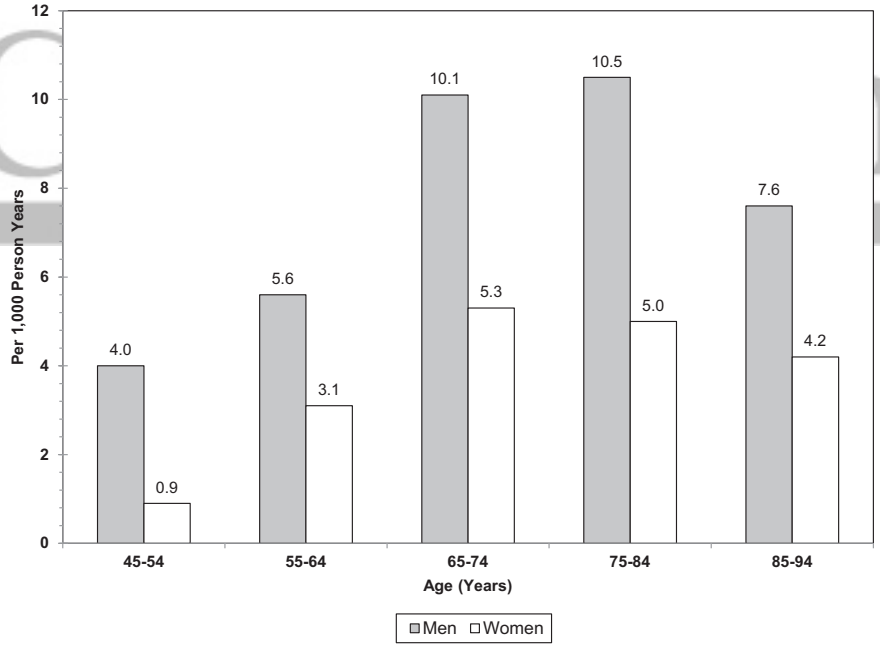


Chart 18-10. Incidence of angina pectoris* by age and sex (Framingham Heart Study 1980–2002/2003). *Angina pectoris deemed uncomplicated on the basis of physician interview of patient. (Rate for women 45–54 years of age considered unreliable.) Data derived from National Heart, Lung, and Blood Institute.[8]

19. Cardiomyopathy and Heart Failure

See Table 19-1 and Charts 19-1 through 19-3.

Cardiomyopathy

ICD-9 425; ICD-10 I42.

Mortality—23 831. Any-mention mortality—47 323. Hospital discharges—34 000.

- Since 1996, the NHLBI-sponsored Pediatric Cardiomyopathy Registry has collected data on all children with newly diagnosed cardiomyopathy in New England and the Central Southwest (Texas, Oklahoma, and Arkansas).[1]
  - —The overall incidence of cardiomyopathy is 1.13 cases per 100 000 among children <18 years of age.
  - —Among children <1 year of age, the incidence is 8.34, and among children 1 to 18 years of age, it is 0.70 per 100 000.
  - —The annual incidence is lower in white than in black children, higher in boys than in girls, and higher in New England (1.44 per 100 000) than in the Central Southwest (0.98 per 100 000).
- HCM is the most common inherited heart defect, occurring in 1 of 500 individuals. In the United States, ≈500 000 people have HCM, yet most are unaware of it.[2] See Chapter 16, Disorders of Heart Rhythm, for statistics regarding sudden death in HCM.
- In a recent report of the Pediatric Cardiomyopathy Registry, the overall annual incidence of HCM in children was 4.7 per 1 million children. There was a higher incidence in the New England than in the Central Southwest region, in boys than in girls, and in children diagnosed at <1 year of age than in older children.[3]
- Dilated cardiomyopathy is the most common form of cardiomyopathy. The Pediatric Cardiomyopathy Registry recently reported an annual incidence of dilated cardiomyopathy in children <18 years of age of 0.57 per 100 000 overall. The annual incidence was higher in boys than in girls (0.66 versus 0.47 cases per 100 000), in blacks than in whites (0.98 versus 0.46 cases per 100 000), and in infants (<1 year of age) than in children (4.40 versus 0.34 cases per 100 000). The majority of children (66%) had idiopathic disease. The most common known causes were myocarditis (46%) and neuromuscular disease (26%).[4]
- Risk factors for death and transplantation in children varied by cause of dilated cardiomyopathy. For idiopathic dilated cardiomyopathy, increased left ventricular end-diastolic dimension was associated with increased transplantation risk but not mortality. Short stature was significantly related to death but not transplantation.[5]
- The 5-year incidence rate of sudden cardiac death among children with dilated cardiomyopathy is 3%.[6]
- Tachycardia-induced cardiomyopathy develops slowly and appears reversible, but recurrent tachycardia causes rapid decline in left ventricular function and development of HF. Sudden death is possible.[7]
- Data from Kaiser Permanente indicate that the incidence of peripartum cardiomyopathy is 4.84 per 10 000 live births (95% CI, 3.98–5.83) and is associated with higher maternal and neonatal death rates and worse neonatal outcomes.[8]

Abbreviations Used in Chapter 19

ABC	Aging, Body and Composition
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities Study
BP	blood pressure
CAD	coronary artery disease
CARDIA	Coronary Artery Risk Development in Young Adults Study
CHS	Cardiovascular Health Study
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
EF	ejection fraction
FHS	Framingham Heart Study
HbA_{1c}	hemoglobin A_{1c}
HCM	hypertrophic cardiomyopathy
HF	heart failure
HR	hazard ratio
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
PAR	population attributable risk

Heart Failure

ICD-9 428; ICD-10 I50.

Prevalence

(See Table 19-1 and Chart 19-1.)

- On the basis of data from NHANES 2007–2010, an estimated 5.1 million Americans ≥20 years of age have HF (NHLBI tabulation).
- Projections show that by 2030, the prevalence of HF will increase 25% from 2013 estimates (AHA computation based on methodology described in Heidenreich et al[9]).

Incidence

(See Table 19-1 and Chart 19-2.)

- Data from the NHLBI-sponsored FHS[10] indicate the following:
  - —HF incidence approaches 10 per 1000 population after 65 years of age.
  - —Seventy-five percent of HF cases have antecedent hypertension.
  - —At 40 years of age, the lifetime risk of developing HF for both men and women is 1 in 5. At 80 years of age, remaining lifetime risk for development of new HF

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remains at 20% for men and women, even in the face of a much shorter life expectancy.
—At 40 years of age, the lifetime risk of HF occurring without antecedent MI is 1 in 9 for men and 1 in 6 for women.
—The lifetime risk for people with BP >160/90 mm Hg is double that of those with BP <140/90 mm Hg.

- The annual rates per 1000 population of new HF events for white men are 15.2 for those 65 to 74 years of age, 31.7 for those 75 to 84 years of age, and 65.2 for those ≥85 years of age. For white women in the same age groups, the rates are 8.2, 19.8, and 45.6, respectively. For black men, the rates are 16.9, 25.5, and 50.6,* and for black women, the estimated rates are 14.2, 25.5, and 44.0,* respectively (CHS, NHLBI).[11]
- In MESA, African Americans had the highest risk of developing HF, followed by Hispanic, white, and Chinese Americans (4.6, 3.5, 2.4, and 1.0 per 1000 person-years, respectively). This higher risk reflected differences in the prevalence of hypertension, DM, and socioeconomic status. African Americans had the highest proportion of incident HF not preceded by clinical MI (75%).[12]
- In Olmsted County, Minnesota, the incidence of HF did not decline between 1979 and 2000.[13]
- In the ARIC study of the NHLBI, the age-adjusted incidence rate per 1000 person-years was 3.4 for white women, less than for all other groups, that is, white men (6.0), black women (8.1), and black men (9.1). The 30-day, 1-year, and 5-year case fatality rates after hospitalization for HF were 10.4%, 22%, and 42.3%, respectively. Blacks had a greater 5-year case fatality rate than whites ($P<0.05$). HF incidence rates in black women were more similar to those of men than of white women. The greater HF incidence in blacks than in whites is explained largely by blacks' greater levels of atherosclerotic risk factors.[14]
- Data from Kaiser Permanente indicated an increase in the incidence of HF among the elderly, with the effect being greater in men.[15]
- Data from hospitals in Worcester, MA, indicate that during 2000, the incidence and attack rates for HF were 219 per 100 000 and 897 per 100 000, respectively. HF was more frequent in women and the elderly. The hospital fatality rate was 5.1%.[16]
- In the CARDIA study, HF before 50 years of age was more common among blacks than whites. Hypertension, obesity, and systolic dysfunction are important risk factors that may be targets for prevention.[17]

Mortality

(See Table 19-1.)

- In 2009, HF any-mention mortality was 274 601 (123 661 males and 150 940 females). HF was the underlying cause in 56 410 of those deaths in 2009 (NCHS, NHLBI). Table 19-1 shows the numbers of these deaths that are coded for HF as the underlying cause.
- The 2009 overall any-mention death rate for HF was 82.3. Any-mention death rates were 98.3 for white males, 104.5 for black males, 72.2 for white females, and 79.7 for black females (NCHS, NHLBI).
- One in 9 deaths has HF mentioned on the death certificate (NCHS, NHLBI).
- The number of any-mention deaths attributable to HF was approximately as high in 1995 (287 000) as it was in 2009 (275 000; NCHS, NHLBI).
- Survival after HF diagnosis has improved over time, as shown by data from the FHS[18] and the Olmsted County Study.[13] However, the death rate remains high: ≈50% of people diagnosed with HF will die within 5 years.[13,19]
- In the elderly, data from Kaiser Permanente indicate that survival after the onset of HF has also improved.[15]
- In the CHS, depression and amino-terminal pro-B-type natriuretic peptide were independent risk factors for CVD-related and all-cause mortality.[20]
- Among Medicare beneficiaries, the overall 1-year mortality rate declined slightly over the past decade but remains high.[21] Changes were uneven across states.

Risk Factors

- In the NHLBI-sponsored FHS, hypertension is a common risk factor for HF, followed closely by antecedent MI.[18] B-type natriuretic peptide, urinary albumin-to-creatinine ratio, and elevated serum γ-glutamyl transferase were also identified as risk factors for HF.[22,23]
- In the Framingham Offspring Study, among 2739 participants, increased circulating concentrations of resistin were associated with incident HF independent of prevalent coronary disease, obesity, insulin resistance, and inflammation.[24]
- Among 20 900 male physicians in the Physicians Health Study, the lifetime risk of HF was higher in men with hypertension; healthy lifestyle factors (normal weight, not smoking, regular exercise, moderate alcohol intake, consumption of breakfast cereals, and consumption of fruits and vegetables) were related to lower risk of HF.[25]
- Among 2934 participants in the Health Aging, Body and Composition (ABC) study, the incidence of HF was 13.6 per 1000 person-years. Men and black participants were more likely to develop HF. Coronary disease (PAR 23.9% for white participants, 29.5% for black participants) and uncontrolled BP (PAR 21.3% for white participants, 30.1% for black participants) had the highest PARs in both races. There was a higher overall proportion of HF attributable to modifiable risk factors in black participants than in white participants (67.8% versus 48.9%). Hospitalizations were higher among black participants.[26] Inflammatory markers (interleukin-6 and tumor necrosis factor-α) and serum albumin levels were also associated with HF risk.[27,28]
- In the CHS, baseline cardiac troponin and changes in cardiac troponin levels measured by a sensitive assay were significantly associated with incident HF.[29] Circulating individual and total omega-3 fatty acid concentrations were associated with lower incidence of HF.[30]
- In the ARIC study, white blood cell count, C-reactive protein, albuminuria, HbA_{1c} among individuals without DM, cardiac troponin measured with a sensitive assay,

*Unreliable estimate.

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ventricular premature complexes, and socioeconomic position over the life course were all identified as risk factors for HF.[18,31–35]

Left Ventricular Function

- Data from Olmsted County, Minnesota, indicate that:
  - —Among asymptomatic individuals, the prevalence of left ventricular diastolic dysfunction was 21% for mild diastolic dysfunction and 7% for moderate or severe diastolic dysfunction. The prevalence of systolic dysfunction was 6%. The presence of any left ventricular dysfunction (systolic or diastolic) was associated with an increased risk of developing overt HF, and diastolic dysfunction was predictive of all-cause death.[36] After 4 years of follow-up, the prevalence of diastolic dysfunction increased to 39.2%. Diastolic dysfunction was associated with development of HF during 6 years of subsequent follow-up after adjustment for age, hypertension, DM, and CAD (HR, 1.81; 95% CI, 1.01–3.48).[37]
  - —Among individuals with symptomatic HF, the prevalence of left ventricular diastolic dysfunction was 6% for mild diastolic dysfunction and 75% for moderate or severe diastolic dysfunction.[38] The proportion of people with HF and preserved EF increased over time. Survival improved over time among individuals with reduced EF but not among those with preserved EF.[39]

Hospital Discharges/Ambulatory Care Visits

(See Table 19-1 and Chart 19-3.)

- Hospital discharges for HF were essentially unchanged from 2000 to 2010, with first-listed discharges of 1.008 million and 1.023 million, respectively (NHDS, NHLBI tabulation).
- In 2010, there were 1.801 million physician office visits with a primary diagnosis of HF. In 2009, there were 668 000 ED visits and 293 000 outpatient department visits for HF (NHAMCS, NHLBI tabulation).
- Among 1077 patients with HF in Olmsted County, Minnesota, hospitalizations were common after HF diagnosis, with 83% patients hospitalized at least once and 43% hospitalized at least 4 times. More than one half of all hospitalizations were related to noncardiovascular causes.[40]
- Among Medicare beneficiaries, the overall HF hospitalization rate declined substantially from 1998 to 2008 but at a lower rate for black men.[21] Changes were uneven across states.

Cost

- Projections show that by 2030, the total cost of HF will increase almost 120% to $70 billion from the 2013 estimated total cost of $32 billion (unpublished AHA computation based on methodology described in Heidenreich et al[9]).

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Table 19-1. Heart Failure

Population Group	Prevalence, 2010, Age ≥20 y	Incidence (New Cases), Age ≥45 y	Mortality, 2009, All Ages*	Hospital Discharges, 2010, All Ages
Both sexes	5 100 000 (2.1%)	670 000	56 410	1 023 000
Males	2 700 000 (2.5%)	350 000	23 563 (41.8%)†	501 000
Females	2 400 000 (1.8%)	320 000	32 847 (58.2%)†	522 000
NH white males	2.2%	…	20 815	…
NH white females	1.7%	…	29 372	…
NH black males	4.1%	…	2341	…
NH black females	3.0%	…	2987	…
Mexican American males	1.9%	…	…	…
Mexican American females	1.1%	…	…	…
Asian	…	…	660	…
American Indian/Alaska Native	…	…	235	…

Heart failure includes persons who answered "yes" to the question of ever having congestive heart failure.

NH indicates non-Hispanic; and ellipses (. . .), data not available.

*Mortality data are based on underlying cause and include Hispanics.

†These percentages represent the portion of total mortality attributable to heart failure that is for males vs females.

Sources: Prevalence: National Health and Nutrition Examination Survey 2007–2010 (National Center for Health Statistics) and National Heart, Lung, and Blood Institute. Percentages are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2010 US population estimates. These data are based on self-reports. Incidence: Framingham Heart Study, 1980–2003 from National Heart, Lung, and Blood Institute Incidence and Prevalence Chart Book, 2006. Mortality: Centers for Disease Control and Prevention/National Center for Health Statistics, 2009 Mortality Multiple Cause–US, version July 19, 2012.

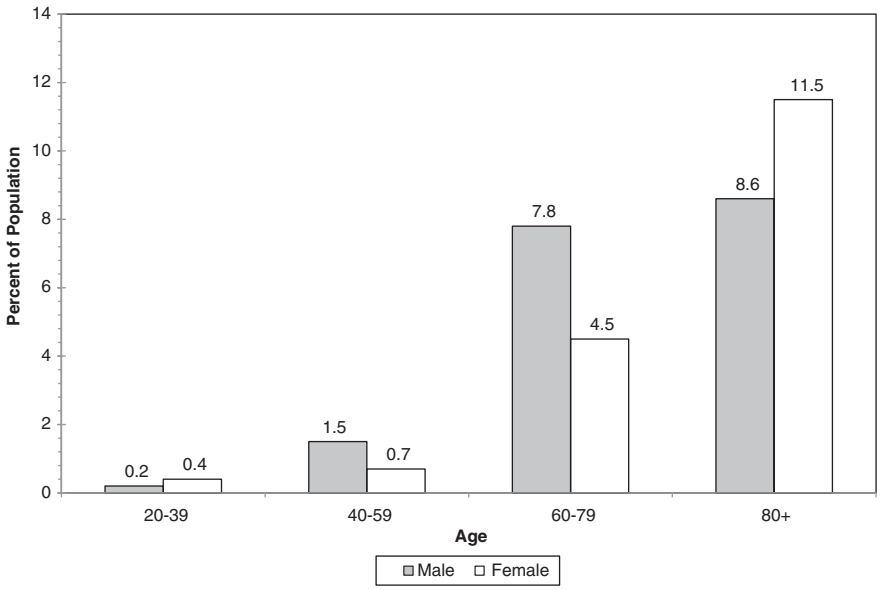

Chart 19-1. Prevalence of heart failure by sex and age (National Health and Nutrition Examination Survey: 2007–2010). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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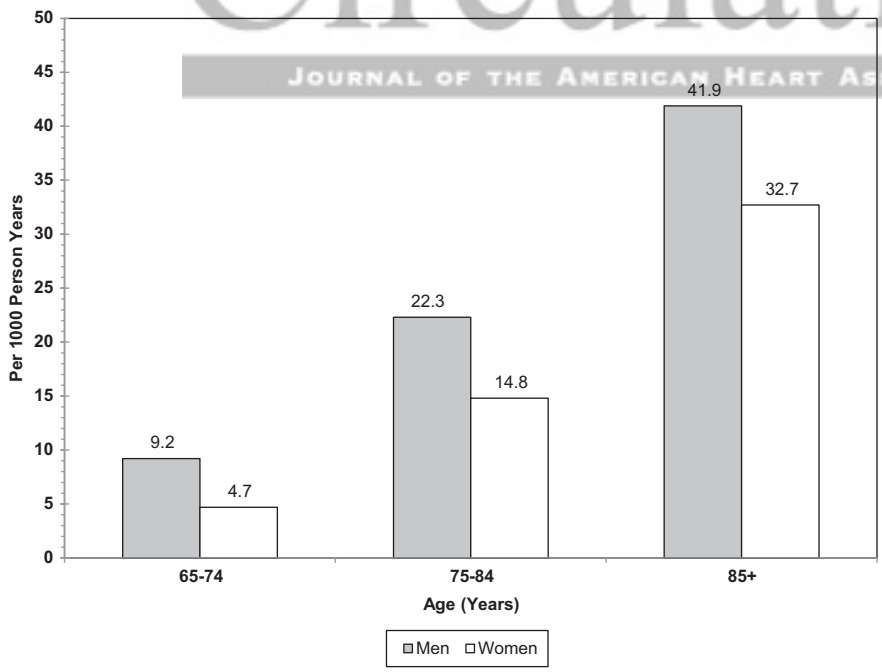

Chart 19-2. Incidence of heart failure (heart failure based on physician review of medical records and strict diagnostic criteria) by age and sex (Framingham Heart Study: 1980–2003). Source: National Heart, Lung, and Blood Institute.

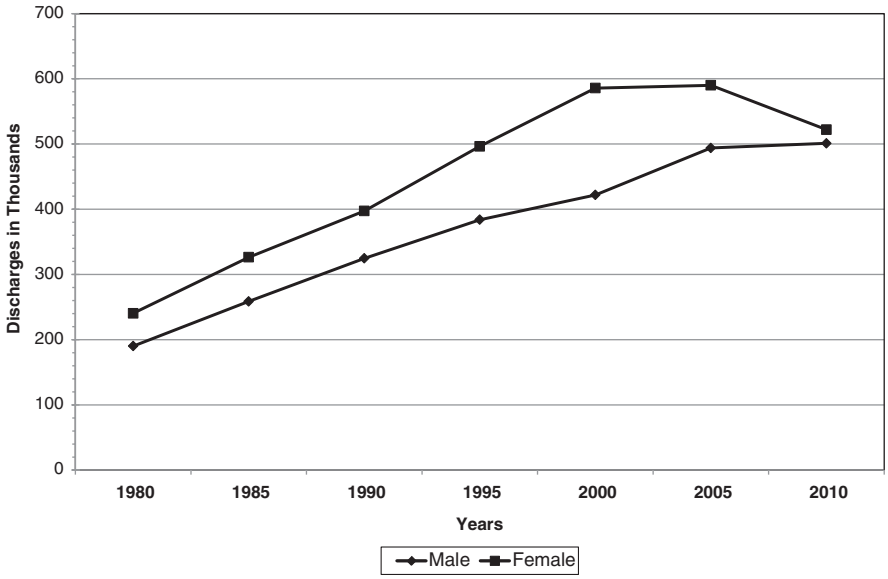

Chart 19-3. Hospital discharges for heart failure by sex (United States: 1980–2010). Note: Hospital discharges include people discharged alive, dead, and status unknown. Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

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20. Valvular, Venous, Aortic, and Peripheral Artery Diseases

See Tables 20-1 and 20-2 and Charts 20-1 through 20-3.

Mortality and any-mention mortality in this section are for 2009. "Mortality" is the number of deaths in 2009 for the given underlying cause based on ICD-10. Prevalence data are for 2006. Hospital discharge data are from the NHDS/NCHS; data include inpatients discharged alive, dead, or status unknown. Hospital discharge data for 2009 are based on ICD-9 codes.

Valvular Heart Disease

(See Table 20-1.)

ICD-9 424; ICD-10 I34 to I38.

Mortality—22 144. Any-mention mortality—45 285. Hospital discharges—85 000.

Two important factors have contributed to the changing epidemiology of valvular heart disease in the United States over the past few decades: aging of the population and the increased ability to diagnose valvular heart disease by echocardiography.

- A large population-based epidemiological study with systematic use of echocardiography on 16 501participants from Olmsted County, Minnesota, showed an overall age-adjusted prevalence of clinically diagnosed (moderate or greater) valvular heart disease of 1.8%.[1]
- Prevalence of any valve disease increased with age[1]:

  — 18 to 44 years: 0.3% (95% CI, 0.2%–0.3%)
  — 45 to 54 years: 0.7% (95% CI, 0.6%–0.9%)
  — 55 to 64 years: 1.6% (95% CI, 1.4%–1.9%)
  — 65 to 75 years: 4.4% (95% CI, 3.9%–4.9%)
  — ≥75 years: 11.7% (95% CI, 11.0%–12.5%)

- Pooled echocardiographic data from 11 911 participants from CARDIA (4351), ARIC (2435), and CHS (5125) demonstrated a similar increase in prevalence with age (Table 20-1)[1]:

  — 18 to 44 years: 0.7% (95% CI, 0.5%–1.0%)
  — 45 to 54 years: 0.4% (95% CI, 0.1%–1.3%)
  — 55 to 64 years: 1.9% (95% CI, 1.2%–2.8%)
  — 65 to 75 years: 8.5% (95% CI, 7.6%–9.4%)
  — ≥75 years: 13.3% (95% CI, 11.7%–15.0%)

- Adjusted to the entire US population, these data suggest that the prevalence of any valve disease is 2.5% (95% CI, 2.2%–2.7%), with no difference between men (2.4% [95% CI, 2.1%–2.8%]) and women (2.5% [95% CI, 2.1%–2.9%]). Within this sample, 0.4% had aortic stenosis, 0.5% had aortic regurgitation, 0.1% had mitral stenosis, and 1.7% had mitral regurgitation.[1]
- In CARDIA, ARIC, and CHS, survival of participants with valve disease was 79% (standard error 2%) at 5 years and 68%

Abbreviations Used in Chapter 20

AAA	abdominal aortic aneurysm
ABI	ankle brachial index
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CI	confidence interval
CKD	chronic kidney disease
CT	computed tomography
CVD	cardiovascular disease
DM	diabetes mellitus
DVT	deep vein thrombosis
ECG	electrocardiogram
FHS	Framingham Heart Study
HD	heart disease
HR	hazard ratio
ICD	*International Classification of Diseases*
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
IE	infective endocarditis
IRAD	International Registry of Acute Aortic Dissection
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
OR	odds ratio
PA	physical activity
PAD	peripheral artery disease
PE	pulmonary embolism
REACH	Reduction of Atherothrombosis for Continued Health
RR	relative risk
SOURCE	United Kingdom SAPIEN Aortic Bioprosthesis European Outcome registry
TAVI	United Kingdom Transcatheter Aortic Valve Implantation registry
TAVR	transcatheter aortic valve replacement
TIA	transient ischemic attack
VTE	venous thromboembolism
VSS	venous stasis syndrome
WHO	World Health Organization
WONDER	Wide-ranging Online Data for Epidemiologic Research

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(1.9%) at 8 years, compared with 93% (0.2%) and 86% (0.4%) in participants without valve disease. In Olmsted County, valve disease was associated with a 75% increased risk of death through 8 years of follow-up (95% CI, 1.61–1.90).

- Echocardiography data from FHS of 1696 men and 1893 women (mean [standard deviation] age 54 [10] years) were used to assess the prevalence of valvular regurgitation. Mitral regurgitation or tricuspid regurgitation of more than or equal to mild severity was seen in 19.0% and 14.8% of men and 19.1% and 18.4% of women, respectively. Aortic regurgitation of more than or equal to trace severity was present in 13.0% of men and 8.5% of women.[3]

Aortic Valve Disorders

ICD-9 424.1; ICD-10 I35.

Mortality—14 703 Any-mention mortality—29 730. Hospital discharges—55 000.

- The prevalence of moderate or severe aortic stenosis in patients ≥75 years old is 2.8% (95% CI, 2.1%–3.7%), and the prevalence of moderate or severe aortic regurgitation in patients ≥75 years is 2.0% (95% CI, 1.4%–2.7%).[1]
- Congenitally malformed bicuspid or unicuspid valve appeared to be at least as common (56%) as so-called degenerative tricuspid aortic valve disease (45%; 1% undetermined) in a large (n=932 patients aged 26–91 years) single-center experience of patients who presented with isolated aortic valve replacement.[4]
- Aortic regurgitation prevalence in patients presenting for aortic valve replacement suggests that cusp prolapse (type II aortic regurgitation) is a common cause of aortic regurgitation, on the basis of data from a large study from a single center in Belgium (n=163 patients; mean [standard deviation] age 58 [14] years)[5]:
  - Type I: enlargement of the aortic root with normal cusps (25.2%)
  - Type II: cusp prolapse or fenestration (38.0%)
  - Type III: poor cusp tissue quality or quantity (36.8%)
- In MESA participants aged 45 to 84 years (n=5880), aortic valve calcium was quantified with serial CT images. During a mean follow-up of 2.4 years, 210 (4.1%) of the 5142 participants with no aortic valve calcium had a mean incidence rate of progression of 1.7% per year, which increased with age. Incident aortic valve calcium was associated with several conventional cardiovascular risk factors, including age, male sex, BMI, and smoking.[6]
- Approximately 50% of patients with severe aortic stenosis are referred for cardiothoracic surgery, and ≈40% undergo aortic valve replacement according to data from 10 US centers of various sizes and geographic distribution. Reasons for not undergoing aortic valve replacement included high perioperative risk, age, lack of symptoms, and patient/family refusal.[7]

Aortic Valve Interventions

- Lipid-lowering therapy does not appear to reduce aortic stenosis progression on the basis of any echocardiographic measures of aortic stenosis, as reported by a meta-analysis of 4 randomized controlled trials by Teo and colleagues.[8]
- Transcatheter aortic valve replacement (TAVR) has emerged as an innovative technology for treatment of aortic stenosis in patients at high risk for perioperative complications.
  - Data from the UK Transcatheter Aortic Valve Implantation (TAVI) Registry on 877 patients (through December 2009) with 100% follow-up demonstrate survival rates of 92.9% at 30 days, 78.6% at 1 year, and 73.7% at 2 years.[9]
  - Safety of TAVR appeared to be improving from 2008–2009 to 2009–2010 in the United Kingdom SAPIEN Aortic Bioprosthesis European Outcome (SOURCE) Registry, with lower rates of >2+ aortic regurgitation (4.5% versus 2.1%, *P*=0.01) and conversion to open surgery (3.7% versus 1.5%, *P*=0.03) between the respective study periods. Pacemaker implantation (7.7% versus 6.7%), stroke (2.4% versus 2.6%), and 30-day mortality rates (10.8% versus 10.7%) did not change.[10]
  - Thirty-day stroke/TIA rates appear higher with TAVR than with surgical aortic valve replacement (5.5% versus 2.4%, *P*=0.04).[11]

Mitral Valve Disorders

ICD-9 424.0; ICD-10 I34.

Mortality—2280. Any-mention mortality—5250. Hospital discharges—22 000.

Prevalence

(See Table 20-1.)

- In pooled data from CARDIA, ARIC, and CHS, mitral valve disease was the most common valvular lesion. At least moderate mitral regurgitation occurred at a frequency of 1.7% as adjusted to the US adult population of 2000, increasing from 0.5% in participants aged 18 to 44 years to 9.3% in participants aged ≥75 years.[1]
- A systematic review by de Marchena and colleagues[12] found that in the US population, the prevalence of mitral regurgitation according to Carpentier's functional classification system was as follows:
  - Type I (congenital mitral regurgitation and endocarditis): <20 per 1 million
  - Type II (myxomatous mitral regurgitation): 15 000 per 1 million
  - Type IIIa (rheumatic HD, systemic lupus erythematosus, antiphospholipid syndrome): 10 520 per 1 million
  - Type IIIb (ischemic mitral regurgitation, left ventricular dysfunction, dilated cardiomyopathy): 23 250 per 1 million
- Data from the Framingham Offspring Study demonstrated that the prevalence of classic (1.3%) and nonclassic (1.1%) mitral valve prolapse was similar. The average degree of mitral regurgitation in both types of mitral valve prolapse was trace or mild. The frequencies of chest pain, dyspnea, and ECG abnormalities were similar among participants with and without prolapse.[13]

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Pulmonary Valve Disorders
ICD-9 424.3; ICD-10 I37.

Mortality—14. Any-mention mortality—35.

Tricuspid Valve Disorders
ICD-9 424.2; ICD-10 I36.

Mortality—12. Any-mention mortality—95.

Rheumatic Fever/Rheumatic HD
(See Table 20-2 and Chart 20-1.)

ICD-9 390 to 398; ICD-10 I00 to I09.

Mortality—3234. Any-mention mortality—6085. Hospital discharges—20 000.

- Rheumatic HD is uncommon in high-income countries such as the United States but remains endemic in Africa, Asia, and the Pacific, affecting >15 million individuals and causing 233 000 deaths annually.[14]
- The reported prevalence of rheumatic HD is increasing in all regions of the world except Europe.[15]
- Recent echocardiography-based screening studies in schoolchildren have demonstrated rheumatic HD prevalence rates ranging from 14.8 (95% CI, 7.3–22.3) per 1000 (Uganda)[16] to 20.4 (95% CI, 16.9–23.9) per 1000 in northern India[17] to 21.5 (95% CI, 16.8–26.2) per 1000 in Cambodia and 30.4 (95% CI, 23.2–37.6) per 1000 (Mozambique).[18]
  - Echocardiography reveals a 3- to 10-fold higher prevalence of rheumatic HD than clinical examination[16,18]
- Acute rheumatic fever incidence is decreasing in all WHO regions except for the Americas, where it appears to be increasing slightly, and the Western Pacific, where it appears to be increasing steadily.[15]
- In 1950, ≈15 000 Americans (adjusted for changes in ICD codes) died of rheumatic fever/rheumatic HD compared with ≈3100 annually in the present era (NCHS/NHLBI).
- The 2009 overall death rate for rheumatic fever/rheumatic HD was 1.1 per 100 000. Death rates varied across race/ethnic groups but were generally low: white, 1.2 per 100 000; black or African American, 0.7 per 100 000; Asian or Pacific Islander, 0.6 per 100 000; American Indian or Alaska Native, 0.6 per 100 000; and Hispanic or Latino origin individuals, 0.4 per 100 000.[19]

Bacterial Endocarditis
ICD-9 421.0; ICD-10 I33.0.

Mortality—1119. Any-mention mortality—2289. Hospital discharges—34 000, primary plus secondary diagnoses.

- The 2007 AHA guidelines on prevention of infective endocarditis (IE)[15] state that IE is thought to result from the following sequence of events: (1) Formation of nonbacterial thrombotic endocarditis on the surface of a cardiac valve or elsewhere that endothelial damage occurs; (2) bacteremia; and (3) adherence of the bacteria in the bloodstream to nonbacterial thrombotic endocarditis and proliferation of bacteria within a vegetation. Viridans group streptococci are part of the normal skin, oral, respiratory, and gastrointestinal tract flora, and they cause ≥50% of cases of community-acquired native valve IE not associated with intravenous drug use.[20]
- Although the absolute risk for acquiring IE from a dental procedure is impossible to measure precisely, the best available estimates are as follows: If dental treatment causes 1% of all cases of viridans group streptococcal IE annually in the United States, the overall risk in the general population is estimated to be as low as 1 case of IE per 14 million dental procedures. The estimated absolute risk rates for acquiring IE from a dental procedure in patients with underlying cardiac conditions are as follows[21]:

  —Mitral valve prolapse: 1 per 1.1 million procedures
  —CHD: 1 per 475 000
  —Rheumatic HD: 1 per 142 000
  —Presence of a prosthetic cardiac valve: 1 per 114 000
  —Previous IE: 1 per 95 000 dental procedures

- Cessation of antibiotic prophylaxis for IE before dental procedures has not led to a change in pediatric cases of endocarditis. Using 2003 to 2010 data from 37 centers in the Pediatric Health Information Systems Database, Pasquali and colleagues[22] did not demonstrate a significant difference in the number of IE hospitalizations after the guidelines were implemented in 2007 (1.6% difference after versus before guideline implementation; 95% CI, −6.4% to 10.3%; P=0.7).
- Cardiac device IE appears to be present in 6.4% (95% CI, 5.5%–7.4%) of patients with definite IE, according to data from the International Collaboration on Endocarditis–Prospective Cohort Study (2000–2006). Nearly half (45.8%; 95% CI, 38.3%–53.4%) of such cases are associated with healthcare-associated infection. In-hospital and 1-year mortality rates for these patients were 14.7% (26/177; 95% CI, 9.8%–20.8%) and 23.2% (41/177; 95% CI, 17.2%–30.1%), respectively.[23]

Endocarditis, Valve Unspecified
ICD-9 424.9; ICD-10 I38.

Mortality—5135. Any-mention mortality—10 414.

Venous Thromboembolism Epidemiology (Including Deep Vein Thrombosis and Pulmonary Embolism)[24]

Pulmonary Embolism

ICD-9 415.1; ICD-10 I26.
Mortality—7040. Any-mention mortality—28 404. Hospital discharges—186 000.

Deep Vein Thrombosis

ICD-9 451.1; ICD-10 I80.2.

Mortality—2452. Any-mention mortality—12 439.

Incidence

- Venous thromboembolism (VTE) consists of deep vein thrombosis (DVT; typically involving deep veins of the leg or pelvis) and its complication, pulmonary embolism (PE).

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- The average annual incidence of VTE among whites is 108 per 100 000 person-years, with ≈250 000 incident cases occurring annually among US whites.
- VTE incidence appears to be similar or higher among African Americans and lower among Asian Americans and Native Americans than among whites.
- After adjustment for the different age and sex distribution of African Americans, VTE incidence is ≈78 per 100 000, which suggests that 27 000 incident VTE cases occur annually among US blacks.
- VTE incidence has not changed significantly over the past 25 years.
- Incidence rates increase exponentially with age for both men and women and for both DVT and PE.
- Incidence rates are higher in women during childbearing years, whereas incidence rates after age 45 years are higher in men.
- PE accounts for an increasing proportion of VTE with increasing age in both sexes.
- VTE event type (DVT versus PE) has a common familial background and shared genetic susceptibility.[25]

Survival

- Observed survival after VTE is significantly worse than expected survival for age and sex, and survival after PE is much worse than after DVT alone.
- For almost one quarter of PE patients, the initial clinical presentation is sudden death.
- Thirty-day VTE survival is 74.8% (DVT alone, 96.2%; PE with or without DVT, 59.1%).[26]
- PE is an independent predictor of reduced survival for up to 3 months.
- Because most PE deaths are sudden and usually attributed to underlying disease (eg, cancer, other chronic heart, lung, or renal disease), secular trends in VTE survival are confounded by autopsy rates.

Recurrence

- VTE is a chronic disease with episodic recurrence; ≈30% of patients develop recurrence within the next 10 years.
- The hazard of recurrence varies with the time since the incident event and is highest within the first 6 to 12 months.
- Independent predictors of early (within 180 days) recurrence include active cancer, proportion of time spent taking heparin with a heparin level ≥0.2 anti-Xa U/mL, and proportion of time spent taking warfarin with an international normalized ratio ≥2. Two-week case fatality for recurrent DVT alone and recurrent PE with or without DVT is 2% and 11%, respectively.[27]
- Independent predictors of long-term recurrence include age, BMI, neurological disease with leg paresis, active cancer, lupus anticoagulant or antiphospholipid antibody, antithrombin, protein C or protein S deficiency, and persistently increased plasma fibrin D-dimer.
- Idiopathic incident VTE, incident PE, and male sex may predict a higher risk of recurrence, but reports are conflicting.[28–30]
- Oncology patients with incidentally discovered PE have similarly high rates of recurrence and reduced survival compared with oncology patients with symptomatic PE.[31]

Complications

- VTE complications include venous stasis syndrome (also called postthrombotic syndrome) and venous ulcer, as well as chronic thromboembolic pulmonary hypertension.
- The 20-year cumulative incidence of venous stasis syndrome and venous ulcer after proximal DVT is ≈40% and 3.7%, respectively.
- The incidence of chronic thromboembolic pulmonary hypertension is 6.5 per million person-years; ≈1400 incident cases occur annually among US whites.

Risk Factors

- Independent VTE risk factors include increasing patient age, surgery, trauma/fracture, hospital or nursing home confinement, active cancer, central vein catheterization or transvenous pacemaker, prior superficial vein thrombosis, infection, varicose veins, and neurological disease with leg paresis, and among women, use of oral contraceptives, pregnancy/postpartum period, and hormone therapy.[32]
- Together, these risk factors account for >75% of all incident VTE that occurs in the community.
- Compared with residents in the community, hospitalized residents have a >130-fold higher VTE incidence (71 versus 9605 per 100 000 person-years).[33]
- Hospitalization and nursing home residence together account for almost 60% of incident VTE events that occur in the community.
- Among patients hospitalized for acute medical illness, independent risk factors for VTE include prior VTE, thrombophilia, cancer, age >60 years, leg paralysis, immobilization ≥7 days, and admission to an intensive care unit or coronary care unit.[34]
- Among cancer patients beginning chemotherapy, tumor site, BMI, hemoglobin, platelet and white blood cell count, and plasma D-dimer and soluble P-selectin levels are predictors of VTE in the next 6 months.[35]
- Pregnancy-associated VTE incidence is 200 per 100 000 woman-years; compared with nonpregnant women of childbearing age, the RR is increased ≈4-fold.
- VTE risk during the postpartum period is ≈5-fold higher than during pregnancy.
- VTE is highly heritable and follows a complex mode of inheritance that involves environmental interaction.
- Inherited thrombophilias (eg, inherited antithrombin, protein C, or protein S deficiency; factor V Leiden; prothrombin G20210A; ABO blood type non-O) interact with such clinical risk factors (ie, environmental "exposures") as oral contraceptives, pregnancy, hormone therapy, and surgery to compound VTE risk.
- Similarly, genetic interaction compounds the risk of incident and recurrent VTE.

Arteries, Diseases of

ICD-9 440 to 448; ICD-10 I70 to I78. Includes PAD.

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Penetrating Aortic Ulcers

- A single-center evaluation of 388 penetrating aortic ulcers found on CT angiography (2003–2009) demonstrated penetrating aortic ulcers in the aortic arch (6.8%), descending thoracic aorta (61.2%), and abdominal aorta (29.7%). Nearly 2 of every 3 penetrating aortic ulcers (57.7%) did not have a saccular aneurysm or intramural hematoma, whereas ≈1 in 4 (27.8%) had associated saccular aneurysms, and ≈1 in 7 (14.4%) had an associated intramural hematoma. Rupture was present in ≈1 in 25 (4.1%) penetrating aortic ulcers.[36]

Aortic Aneurysm

ICD-9 441; ICD-10 I71.

Mortality—10 597. Any-mention mortality—17 215. Hospital discharges—64 000.

- Although the definition varies somewhat by age and body surface area, generally an AAA is considered to be present when the anteroposterior diameter of the aorta reaches 3.0 cm.[37]
- The prevalence of AAAs that are 2.9 to 4.9 cm in diameter ranges from 1.3% in men 45 to 54 years of age to 12.5% in men 75 to 84 years of age. For women, the prevalence ranges from 0% in the youngest to 5.2% in the oldest age groups.[37]
- Between 1997 and 2009, age-adjusted mortality attributable to AAA declined from 40.4 to 25.7 per 100 000 in England and Wales and from 30.1 to 20.8 per 100 000 population in Scotland.[38]
- A meta-analysis of 15 475 individuals from 18 studies on small AAAs (3.0–5.4 cm) demonstrated that mean aneurysm growth rate was 2.21 mm per year and was independent of age and sex. Growth rates were higher in smokers (by 0.35 mm/y) and lower in patients with DM (by 0.51 mm/y).[39]
- Rupture rates range from 0.71 to 11.03 per 1000 person-years, with higher rupture rates in smokers (pooled HR, 2.02; 95% CI, 1.33–3.06) and women (pooled HR, 3.76; 95% CI, 2.58–5.47).[39]
- Data from the International Registry of Acute Aortic Dissection (IRAD) suggest that patients with acute type B aortic dissection have heterogenous in-hospital outcomes. In-hospital mortality in patients with and without complications (such as mesenteric ischemia, renal failure, limb ischemia, or refractory pain) was 20.0% and 6.1%, respectively. In complicated patients, in-hospital mortality associated with surgical and endovascular repair was 28.6% and 10.1% (P=0.006), respectively.[40]

AAA Treatment

- After multivariable adjustment, Medicare patients who underwent open AAA repair had higher risk of all-cause mortality (HR, 1.24; 95% CI, 1.05–1.47) and AAA-related mortality (HR, 4.37; 95% CI, 2.51–7.66) at 1 year than patients who underwent endovascular repair.[41]

Peripheral Artery Disease

(See Charts 20-2 and 20-3.)

ICD-9: 440.20 to 440.24, 440.30 to 440.32, 440.4, 440.9, 443.9, 445.02; ICD-10: I70.2, I70.9, I73.9, I74.3, I74.4.

Mortality—13 893. Any-mention mortality—64 469. Hospital discharges—146 000.

- PAD affects ≈8.5 million Americans aged ≥40 years and is associated with significant morbidity and mortality.[42] PAD prevalence is higher in older individuals and appears to disproportionately affect African Americans.[42]
- The age- and sex-standardized prevalence (standard error) of PAD in the United States is 4.6% (0.3%) based on data from NHANES 1999–2004. The highest prevalence of PAD was observed among elderly people, non-Hispanic blacks, and women. In a multivariable age-, sex-, and race/ethnicity-adjusted regression model, hypertension, DM, CKD, and smoking were associated with incident PAD (P≤0.05 for each).[43]
- In the general population, only ≈10% of people with PAD have the classic symptom of intermittent claudication. Approximately 40% do not complain of leg pain, whereas the remaining 50% have a variety of leg symptoms different from classic claudication.[44,45] Data from NHANES 1999–2002 suggest that up to two thirds of US adults with PAD who are ≥40 years old are asymptomatic, with one fourth having severe PAD (ABI <0.7).[46] In an older, disabled population of women, as many as two thirds of individuals with PAD had no exertional leg symptoms.[47]
- The risk factors for PAD are similar but not identical to those for CHD. DM and cigarette smoking are stronger risk factors for PAD than for CHD.[37] ORs for associations of DM and smoking with symptomatic PAD are ≈3.0 to 4.0. Most studies suggest that the prevalence of PAD is similar in men and women.[48]
- Pooled data from 11 studies in 6 countries found that PAD (defined by ABI <0.9) is a marker for systemic atherosclerotic disease. The pooled age-, sex-, risk factor–, and CVD-adjusted RR for all-cause death was 1.60 (95% CI, 1.32–1.95), the RR for cardiovascular mortality was 1.96 (95% CI, 1.46–2.64), the RR for CHD was 1.45 (95% CI, 1.08–1.93), and the RR for stroke was 1.35 (95% CI, 1.10–1.65).[49]
- A 2008 meta-analysis of 24 955 men and 23 339 women demonstrated that the association of the ABI with mortality has a reverse J-shaped distribution in which participants with an ABI of 1.11 to 1.40 are at lowest risk for mortality.[50] Low ABI (≤0.9) carried a 3-fold (RR, 3.33; 95% CI, 2.74–4.06) risk of all-cause death compared with men with normal ABI (1.11–1.40) and a similar risk in women (RR, 2.71; 95% CI, 2.03–3.62).[50]
- Among 508 patients (449 men) identified from 2 vascular laboratories in San Diego, CA, a decline in ABI of >0.15 within a 10-year period was associated with a subsequent increased risk of all-cause mortality (RR, 2.4; 95% CI, 1.2–4.8) and CVD mortality (RR, 2.8; 95% CI, 1.3–6.0) at 3 years' follow-up.[51]
- Among 440 patients with PAD, male sex and smoking were more associated with aortoiliac (proximal) disease than with infrailiac (distal) disease. In addition, aortoiliac disease was associated with an increased risk of mortality or cardiovascular events compared with infrailiac disease (adjusted HR, 3.28; 95% CI, 1.87–5.75).[52]
- People with PAD have impaired function and quality of life. This is true even for people who do not report leg symptoms. Furthermore, patients with PAD, including those

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who are asymptomatic, experience a significant decline in lower-extremity functioning over time.[53–55]

- Among patients with established PAD, higher PA levels during daily life are associated with better overall survival rate, a lower risk of death because of CVD, and slower rates of functional decline.[56,57] In addition, better 6-minute walk performance and faster walking speed are associated with lower rates of all-cause mortality, cardiovascular mortality, and mobility loss.[58,59]
- A cross-sectional, population-based telephone survey of >2500 adults ≥50 years of age, with oversampling of blacks and Hispanics, found that 26% expressed familiarity with PAD. Of these, half were not aware that DM and smoking increase the risk of PAD. One in 4 knew that PAD is associated with increased risk of MI and stroke, and only 14% were aware that PAD could lead to amputation. All knowledge domains were lower in individuals with lower income and education levels.[60]

Interventions

- Data from the Reduction of Atherothrombosis for Continued Health (REACH) Registry of 8273 PAD participants suggest that only 70% of PAD patients receive lipid-lowering therapy and only 82% receive antiplatelet therapy for secondary CVD prevention.[61]
- A 2011 systematic review evaluated lower-extremity aerobic exercise against usual care and demonstrated a range of benefits, including the following[62]:
  - —Increased claudication time by 71 seconds (79%) to 918 seconds (422%).
  - —Increased claudication distance by 15 m (5.6%) to 232 m (200%).
  - —Increased walking distance/time by 67% to 101% after 40 minutes of walking 2 to 3 times per week.

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Table 20-1. Pooled Prevalence of Valvular Heart Disease From CARDIA, ARIC, and CHS Cohorts

	Age, y					*P* Value for Trend	Frequency Adjusted to 2000 US Adult Population
	18–44	45–54	55–64	65–74	≥75		
Participants, n	4351	696	1240	3879	1745	…	209 128 094
Male, n (%)	1959 (45)	258 (37)	415 (33)	1586 (41)	826 (47)	…	100 994 367 (48)
Mitral regurgitation (n=449), n (%)	23 (0.5)	1 (0.1)	12 (1.0)	250 (6.4)	163 (9.3)	<0.0001	1.7% (95% CI, 1.5%–1.9%)
Mitral stenosis (n=15), n (%)	0 (0)	1 (0.1)	3 (0.2)	7 (0.2)	4 (0.2)	0.006	0.1% (95% CI, 0.02%–0.2%)
Aortic regurgitation (n=90), n (%)	10 (0.2)	1 (0.1)	8 (0.7)	37 (1.0)	34 (2.0)	<0.0001	0.5% (95% CI, 0.3%–0.6%)
Aortic stenosis (n=102), n (%)	1 (0.02)	1 (0.1)	2 (0.2)	50 (1.3)	48 (2.8)	<0.0001	0.4% (95% CI, 0.3%–0.5%)
Any valve disease, n (%)							
Overall (n=615)	31 (0.7)	3 (0.4)	23 (1.9)	328 (8.5)	230 (13.2)	<0.0001	2.5% (95% CI, 2.2%–2.7%)
Women (n=356)	19 (0.8)	1 (0.2)	13 (1.6)	208 (9.1)	115 (12.6)	<0.0001	2.4% (95% CI, 2.1%–2.8%)
Men (n=259)	12 (0.6)	2 (0.8)	10 (2.4)	120 (7.6)	115 (14.0)	<0.0001	2.5% (95% CI, 2.1%–2.9%)

CARDIA indicates Coronary Artery Risk Development in Young Adults; ARIC, Atherosclerosis Risk in Communities study; CHS, Cardiovascular Health Study; and CI, confidence interval.

Source: Reprinted from Nkomo et al[1] with permission of the publisher.

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Table 20-2. Rheumatic Fever/Rheumatic Heart Disease

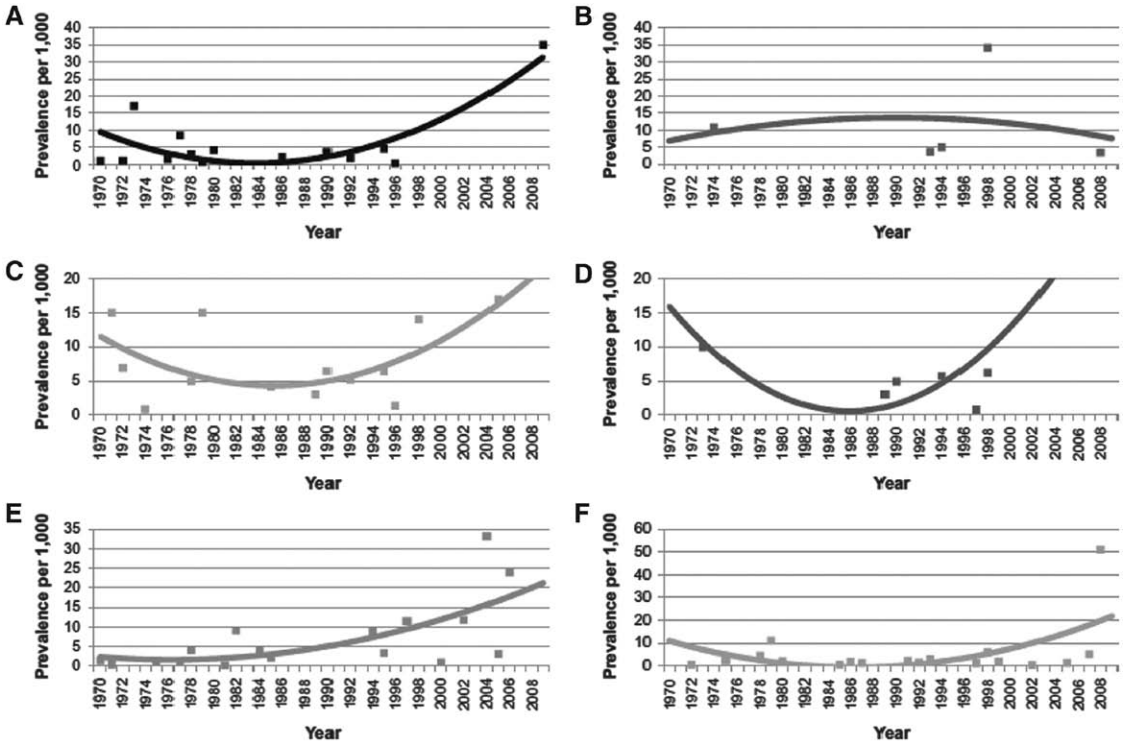
Population Group	Mortality, 2009: All Ages*	Hospital Discharges, 2010: All Ages
Both sexes	3234	20 000
Males	1071 (33.1%)†	5000
Females	2163 (66.9%)†	15 000
NH white males	946	…
NH white females	1903	…
NH black males	89	…
NH black females	178	…
Asian	98	…
American Indian/ Alaska Native	20	…

NH indicates non-Hispanic; ellipses (…), data not available.

*Mortality data include Hispanics.

†These percentages represent the portion of total mortality that is for males vs females.

Sources: Mortality: Centers for Disease Control and Prevention/National Center for Health Statistics, 2009 Mortality Multiple Cause–US, version July 19, 2012; data represent underlying cause of death only. Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics, and National Heart, Lung, and Blood Institute; data include those inpatients discharged alive, dead, or of unknown status.



Source: Reprinted from Seckeler et al[15] with permission of the publisher.

Chart 20-1. Rheumatic heart disease prevalence trends per 1000 people for each World Health Organization region: **A**, The Americas; **B**, Europe; **C**, Africa; **D**, Eastern Mediterranean; **E**, Western Pacific; and **F**, Southeast Asia. Reprinted from Seckeler et al[15] with permission of the publisher.

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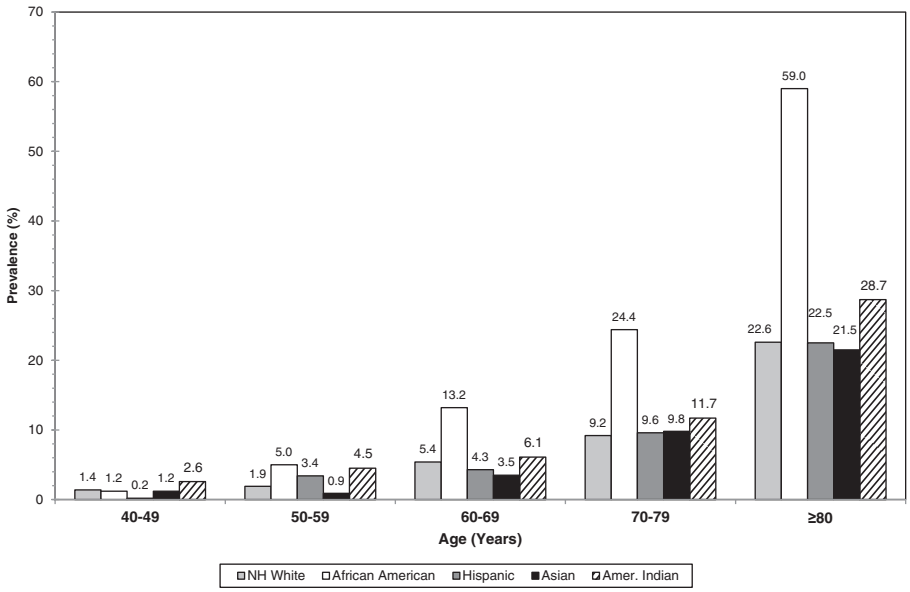

Chart 20-2. Peripheral artery disease prevalence estimates in males by age and ethnicity. NH indicates non-Hispanic; Amer., American. Source: Reprinted from Allison et al[42] with permission of the publisher.

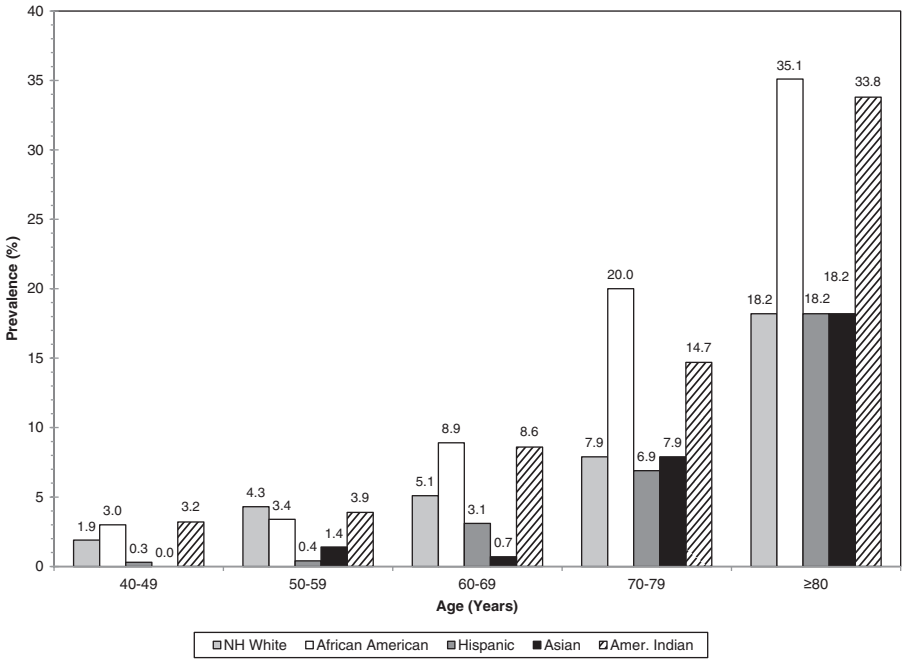

Chart 20-3. Peripheral artery disease prevalence estimates in females by age and ethnicity. NH indicates non-Hispanic; Amer., American. Source: Reprinted from Allison et al[42] with permission of the publisher.

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21. Quality of Care

See Tables 21-1 through 21-13.

The Institute of Medicine defines quality of care as "the degree to which health services for individuals and populations increase the likelihood of desired health outcomes and are consistent with current professional knowledge."[1] The Institute of Medicine has defined 6 specific domains for improving health care, including care that is safe, effective, patient-centered, timely, efficient, and equitable.

In the following sections, data on quality of care will be presented based on the 6 domains of quality as defined by the Institute of Medicine. This is intended to highlight current care and to stimulate efforts to improve the quality of cardiovascular care nationally. Where possible, data are reported from recently published literature or standardized quality indicators from quality-improvement registries (ie, those consistent with the methods for quality performance measures endorsed by the ACC and the AHA).[2] Additional data on aspects of quality of care, such as adherence to ACC/AHA clinical practice guidelines, are also included to provide a spectrum of quality-of-care data. The data selected are meant to provide examples of the current quality of care as reflected by the Institute of Medicine domains and are not meant to be comprehensive given the sheer number of publications yearly.

- The *safety* domain has been defined as avoiding injuries to patients from the care that is intended to help them. The following are several publications that have focused on safety issues:
  - In a small, single-center study conducted over a 2-month period in the cardiac care unit of a tertiary center, Rahim et al[3] demonstrated that iatrogenic adverse events were

Abbreviations Used in Chapter 21

ACC	American College of Cardiology	HF	heart failure
ACEI	angiotensin-converting enzyme inhibitor	HIQR	Hospital Inpatient Quality Reporting
ACS	acute coronary syndrome	HR	hazard ratio
ACTION	Acute Coronary Treatment and Intervention Outcomes Network	IV	intravenous
ACS	acute coronary syndrome	LDL	low-density lipoprotein
AED	automated external defibrillator	LV	left ventricular
AF	atrial fibrillation	LVEF	left ventricular ejection fraction
AHA	American Heart Association	LVSD	left ventricular systolic dysfunction
AMA-PCPI	American Medical Association–Physician Consortium for Performance Improvement	MI	myocardial infarction
AMI	acute myocardial infarction	N/A	not available or not applicable
ARB	angiotensin receptor blocker	NCDR	National Cardiovascular Data Registry
BMI	body mass index	NHANES	National Health and Nutrition Examination Survey
BP	blood pressure	NM	not measured
CABG	coronary artery bypass grafting	NSTEMI	non–ST-elevation myocardial infarction
CAD	coronary artery disease	OR	odds ratio
CHF	congestive heart failure	PAD	peripheral artery disease
CI	confidence interval	PCI	percutaneous coronary intervention
COURAGE	Clinical Outcomes Utilizing Revascularization and AGgressive drug Evaluation trial	PINNACLE	Practice Innovation and Clinical Excellence
CPR	cardiopulmonary resuscitation	RR	relative risk
CRUSADE	Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines	SBP	systolic blood pressure
D2B	Door-to-Balloon Alliance	SCD-HeFT	Sudden Cardiac Death in Heart Failure Trial
DM	diabetes mellitus	SD	standard deviation
DVT	deep vein thrombosis	STEMI	ST-elevation myocardial infarction
		TAVR	transcatheter aortic valve replacement
ED	emergency department	tPA	tissue-type plasminogen activator
EF	ejection fraction	UFH	unfractionated heparin
EMS	emergency medical services	VF	ventricular fibrillation
GWTG	Get With The Guidelines	VHA	Veterans Health Administration
GWTG-HF	Get With The Guidelines–Heart Failure	VT	ventricular tachycardia
HbA_{1c}	hemoglobin A_{1c}		

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common (99 of 194 patients), of which bleeding (27%) was the most common preventable iatrogenic adverse event.

— Using the NCDR (National Cardiovascular Data Registry) CathPCI Registry, Tsai et al[4] found that almost one fourth of dialysis patients undergoing PCI (n=22 778) received a contraindicated antithrombotic agent, specifically enoxaparin, eptifibatide, or both. Patients who received a contraindicated antithrombotic agent had an increased risk of in-hospital bleeding (OR, 1.63; 95% CI, 1.35–1.98) and a trend toward increased mortality (OR, 1.15; 95% CI, 0.97–1.36).[4]

— Using data from the ACTION Registry-GWTG, Mathews and colleagues developed a contemporary model to stratify in-hospital bleeding risk for patients after STEMI and NSTEMI.[5] The 12 factors associated with major bleeding in the model were heart rate, baseline hemoglobin, female sex, baseline serum creatinine, age, electrocardiographic changes, HF or shock, DM, PAD, body weight, SBP, and home warfarin use. The risk model discriminated well in the derivation (C statistic=0.73) and validation (C statistic=0.71) cohorts, and the risk score for major bleeding corresponded well with observed bleeding.[5]

— In a random sample of medical and surgical long-term care adult patients in Massachusetts hospitals, López et al[6] assessed the association between disclosure of an adverse event and patients' perception of quality of care. Overall, only 40% of adverse events were disclosed. Higher quality ratings were associated with disclosure of an adverse event. Conversely, lower patient perception of quality of care was associated with events that were preventable and with events that caused discomfort.[6]

— Using prospective propensity-matched cohort analysis of 7 newly introduced cardiovascular devices, Resnic et al[7] showed the feasibility of automated prospective surveillance to identify low-frequency safety signals in a cardiovascular registry. In this study, 3 of the 21 safety alerts triggered sustained alerts in 2 implantable devices.[7]

- *Effective care* has been defined as providing services based on scientific knowledge to all who could benefit and refraining from providing services to those not likely to benefit. It also encompasses monitoring results of the care provided and using them to improve care for all patients.[1] There are many quality-improvement registries that have been developed for inpatient cardiovascular/stroke care, and the data on these are provided in subsequent tables. Similar efforts are under way for quality-of-care registries in the outpatient setting. In 2011, the AHA published a policy statement for expanding the applications of existing and future clinical registries.[8] This statement discusses recommendations on ensuring high-quality data, linking clinical registries with supplemental data, integrating registries with electronic health records, safeguarding privacy, securing adequate funding, and developing a business model to initiate and sustain these registries.

— In the CRUSADE registry, 1 in 10 patients (10.3%) had a documented contraindication to reperfusion. Primary reasons for contraindications were identified as absence of an ischemic indication (53.8%), bleeding risk (16.7%), patient-related reasons (25.3%), and other (4.2%). Conversely, 7.2% of patients with STEMI without a reperfusion contraindication did not have reperfusion therapy administered, and this was associated with greater in-hospital mortality.[9]

— According to data from NHANES 1988–1994 and 1999–2008, rates of hypertension have increased from 23.9% in 1988–1994 to 29.0% in 2007–2008, and hypertension control has increased from 27.3% in 1988–1994 to 50.1% in 2007–2008. In addition, among patients with hypertension, BP has decreased from 143.0/80.4 to 135.2/74.1 mm Hg.[10]

— Weintraub et al[11] reported results from a comparative effectiveness study of PCI versus CABG using observational data among patients ≥65 years of age with 2- or 3-vessel CAD without AMI. Their results showed that at 1 year, there was no significant difference in adjusted mortality between groups (6.24% in the CABG group versus 6.55% in the PCI group). At 4 years, there was lower mortality in the CABG group than in the PCI group (16.4% versus 20.8%; RR, 0.79; 95% CI, 0.76–0.82).[11]

— Appel et al[12] reported results of a randomized controlled trial comparing the effectiveness of 2 behavioral weight loss interventions with controls. The interventions included either remote weight loss intervention (delivered through the telephone, a study-specific Web site, and e-mail) or in-person support (individual and group sessions along with the 3 means of remote support). At 24 months, the mean change in weight from baseline was −0.8 kg in the control group, −4.6 kg in the group with remote support only ($P<0.001$ for comparison with the control group), and −5.1 kg in the group receiving in-person support ($P<0.001$ for comparison with the control group). The change in weight from baseline did not differ significantly between the 2 intervention groups at the end of the trial.

— Choudhry et al[13] reported results of a cluster randomized trial that evaluated the impact of eliminating out-of-pocket costs (full prescription coverage) on medication adherence and cardiovascular outcomes in patients discharged after MI. Compared with the usual prescription coverage, rates of adherence to statins, β-blockers, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers were on average 4% to 6% higher in the full-coverage group. There was no significant difference in the primary outcome (first major vascular event or revascularization) between the 2 groups (17.6 per 100 person years in the full-coverage group versus 18.8 in the usual-coverage group; HR, 0.93; 95% CI, 0.82–1.04). The rates of secondary outcomes of total major vascular events or revascularization were significantly reduced in the full-coverage group (21.5 versus 23.3; HR, 0.89; 95% CI, 0.90–0.99), as was the rate of first major vascular event (11 versus 12.8; HR, 0.86; 95% CI, 0.74–0.99). The elimination of copayments did not increase total spending, although patient costs were reduced for drugs and other services.

— Data from the ACC PINNACLE (Practice Innovation and Clinical Excellence) outpatient registry[14] of patients with CAD (n=38 775) showed that 77.8% of the patients

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(30 160) were prescribed statins, 2042 (5.3%) were treated only with nonstatin lipid-lowering medications, and 6573 (17%) were not taking any lipid-lowering medication. Lack of medical insurance (RR, 0.94; 95% CI, 0.89–1.00) was associated with a lower likelihood of statin treatment, whereas male sex (RR, 1.10; 95% CI, 1.07–1.13), coexisting hypertension (RR, 1.07; 95% CI, 1.02–1.12), prior CABG (RR, 1.09; 95% CI, 1.05–1.14), and prior PCI (RR, 1.11; 95% CI, 1.06–1.16) were associated with a higher likelihood of statin treatment.

— In patients recently hospitalized with HF, a randomized clinical trial did not show improvement in the primary end point (readmission for any reason or death of any cause within 180 days after enrollment) or the secondary end points (hospitalization for HF, number of days in the hospital, and number of hospitalizations) with the use of telemonitoring.[15] Similar results were seen in a randomized clinical trial of remote telemedical management in patients with chronic HF.[16]

— Heisler et al[17] reported results of a prospective, multisite, cluster randomized trial that evaluated the effectiveness of a pharmacist-led intervention that targeted medication management and adherence counseling to improve BP control in patients with DM in 2 high-performing integrated healthcare systems. Although the mean SBP of patients in the intervention arm was 2.4-mm Hg lower (95% CI, –3.4 to –1.5; $P<0.001$) immediately after the intervention than that of patients in the control arm, the mean SBP decrease from 6 months before to 6 months after the intervention (primary outcome) was similar in magnitude (≈9 mm Hg) in both arms.[17]

— Outcome measures of 30-day mortality and 30-day readmission after hospitalization for AMI or HF have been developed that adjust for patient mix (eg, comorbidities) so that comparisons can be made across hospitals.[18–21] Using national Medicare data from July 2006 through June 2009, the median (10th, 90th percentile) hospital risk-standardized mortality rate was 16% (13.9%, 17.8%) for AMI and 10.8% (8.9%, 12.7%) for HF. The median risk-standardized readmission rate was 19.9% (18.5%, 21.2%) for AMI and 24.5% (22.2%, 27.1%) for HF. Distinct regional patterns were seen for both measures and both conditions.[18]

— A study of 30 947 patients admitted with ischemic strokes showed that admission to a designated stroke center compared with admission to a nondesignated hospital was associated with more frequent use of thrombolytic therapy (4.8% versus 1.7%, $P<0.001$), and lower 30-day all-cause mortality (10.1% versus 12.5%, $P<0.001$).[22]

— A study of 458 hospitals participating in the Society of Thoracic Surgeons National Cardiac Database showed that an intervention of receiving quality-improvement educational material designed to influence the prescription rates of 4 medication classes (aspirin, β-blockers, lipid-lowering therapy, and angiotensin-converting enzyme inhibitors) after CABG discharge in addition to site-specific feedback reports led to a significant improvement in adherence for all 4 secondary prevention medications at the intervention sites compared with the control sites.[23]

— In 2011, the ACC Foundation/AHA/American Medical Association–Physician Consortium for Performance Improvement (AMA-PCPI) published a joint report on performance measures for CAD and hypertension.[24] The 9 performance measures for CAD care included BP control, lipid control, symptom and activity assessment, symptom management, tobacco use (screening, cessation, and intervention), antiplatelet therapy, β-blocker therapy, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker therapy, and cardiac rehabilitation patient referral from an outpatient setting. For hypertension care, the performance measures included BP control. This set was an update to the 2005 ACC Foundation/AHA performance measures for CAD and hypertension and included modifications to 7 of the 2005 performance measures. Screening for DM was retired from the CAD set published in 2005, whereas symptom management and cardiac rehabilitation referral were added to the 2011 CAD set. Similarly, the ACC Foundation/AHA/AMA-PCPI published a report on performance measures for HF,[25] which was an update to the 2005 report.[26] Eight measures from the 2005 report were retired, β-blocker use in patients with HF was expanded as a performance measure for the inpatient setting, symptom management and counseling about implantable cardioverter-defibrillators were added as new quality metrics, and patient education was changed from a performance measure to a quality metric.

— Inpatient ACS, HF, and stroke quality-of-care measures data, including trends in care data, where available from national registries, are given in Tables 21-1 through 21-6.

— Selected outpatient quality-of-care measures from the National Committee for Quality Assurance for 2010 appear in Table 21-7.

— Quality-of-care measures for patients who had out-of hospital cardiac arrest and were enrolled in the Resuscitation Outcomes Consortium cardiac arrest registry in 2010 (Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2012) are given in Table 21-8.

— Quality-of-care measures for patients who had in-hospital cardiac arrest and were enrolled in the AHA's GWTG-Resuscitation quality-improvement project in 2011 (GWTG-Resuscitation Investigators, unpublished data, June 20, 2012) are given in Table 21-9.

- *Patient-centered care* has been defined as the provision of care that is respectful of and responsive to individual patient preferences, needs, and values and that ensures that patient values guide all clinical decisions. Dimensions of patient-centered care include the following: (1) Respect for patients' values, preferences, and expressed needs; (2) coordination and integration of care; (3) information, communication, and education; (4) physical comfort; (5) emotional support; and (6) involvement of family and friends. Studies that focused on some of these aspects of patient-centered care are highlighted below.

— The COURAGE trial,[27] which investigated a strategy of PCI plus optimal medical therapy versus optimal medical therapy alone, demonstrated that both groups had significant improvement in health status

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during follow-up. By 3 months, health status scores had increased in the PCI group compared with the medical therapy group, to 76±24 versus 72±23 for physical limitation (P=0.004), 77±28 versus 73±27 for angina stability (P=0.002), 85±22 versus 80±23 for angina frequency (P<0.001), 92±12 versus 90±14 for treatment satisfaction (P<0.001), and 73±22 versus 68±23 for quality of life (P<0.001). The PCI plus optimal medical therapy group had a small but significant incremental benefit compared with the optimal medical therapy group early on, but this benefit disappeared by 36 months.

— In the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT)[28] of single-lead implantable cardioverter-defibrillator versus amiodarone for moderately symptomatic HF, patients with implantable cardioverter-defibrillators had improvement in quality of life compared with medical therapy patients at 3 and 12 months but not at 30 months. Implantable cardioverter-defibrillator shocks in the month preceding a scheduled assessment were associated with a decrease in quality of life in multiple domains. The authors concluded that the presence of a single-lead implantable cardioverter-defibrillator was not associated with any detectably adverse quality of life during 30 months of follow-up.

— Peikes et al[29] reported on 15 care-coordination programs as part of a Medicare demonstration project for patients with CHF, CAD, DM, and other conditions. Thirteen of the 15 programs did not show a difference in hospitalization rates, and none of the programs demonstrated net savings. The interventions tested varied significantly, but the majority of the interventions included patient education to improve adherence to medication, diet, exercise, and self-care regimens and improving care coordination through various approaches. These programs overall had favorable effects on none of the adherence measures and only a few of the many quality-of-care indicators examined. The authors concluded that programs with substantial in-person contact that target moderately to severely ill patients can be cost-neutral and improve some aspects of care.

— Hernandez et al[30] showed that patients with outpatient follow-up within 7 days of discharge for an HF hospitalization were less likely to be readmitted within 30 days in the GWTG-HF registry of patients who were ≥65 years of age. The median length of stay was 4 days (interquartile range, 2–6 days), and 21.3% of patients were readmitted within 30 days. At the hospital level, the median percentage of patients who had early follow-up after discharge from the index hospitalization was 38.3% (interquartile range, 32.4%–44.5%).

— Smolderen et al[31] assessed whether health insurance status affects decisions to seek care for AMI. Uninsured and insured patients with financial concerns were more likely to delay seeking care during AMI and had prehospital delays of >6 hours (48.6% of uninsured patients and 44.6% of insured patients with financial concerns compared with 39.3% of insured patients without financial concerns). Lack of health insurance and financial concerns about accessing care among those with health insurance were each associated with delays in seeking emergency care for AMI.

— Using a cohort (n=192) nested within a randomized trial at a university-affiliated ambulatory practice, Murray et al[32] demonstrated that refill adherence of <40% was associated with a 3-fold higher incidence of HF hospitalization than refill adherence of ≥80% (P=0.002). In multivariable analysis, prescription label–reading skills were associated with a lower incidence of HF-specific emergency care (incidence rate ratio, 0.76; 95% CI, 0.19–0.69), and participants with adequate health literacy had a lower risk of HF hospitalization (incidence rate ratio, 0.34; 95% CI, 0.15–0.76).

— Reynolds et al[33] reported results on health-related quality of life after TAVR in inoperable patients with severe aortic stenosis compared with those receiving standard therapy. Health-related quality of life was assessed at baseline and at 1, 6, and 12 months with the Kansas City Cardiomyopathy Questionnaire and the 12-item Short Form-12 General Health Survey. Although the Kansas City Cardiomyopathy Questionnaire summary scores improved in both groups, the extent of improvement was greater in the TAVR group than in the standard-care group at 1 month (mean between-group difference, 13 points; 95% CI, 8–19), with larger benefits at 6 months (mean difference, 21 points; 95% CI, 15–27 points) and 12 months (mean difference, 26 points; 95% CI, 19–33). At 12 months, TAVR patients also reported higher physical and mental health scores on the 12-item Short Form-12 General Health Survey, with a mean difference of 5.7 and 6.4 points, respectively (P<0.001 for both comparisons) compared with standard care.[33]

— In 2012, the AHA published a scientific statement on decision making in advanced HF. This statement discusses the clinical trajectory of HF, importance and process of shared decision making in advanced HF, timing of discussion, discussion on outcomes beyond survival (ie, major adverse events, symptom burden, functional limitations, loss of independence, quality of life, and obligations for caregivers), discussions regarding end-of-life issues, and assessment and integration of emotional readiness of the patient and family in these discussions.[34]

— In 2012, the ACC Foundation published a policy statement on patient-centered care in cardiovascular medicine. This policy statement discusses and provides recommendations on topics such as enhanced clinician-patient communication, health literacy, clinician-directed patient education, assessment of patient-centered outcomes, process of shared decision making, collaborative care planning and goal setting, and patient empowerment and self-management. The policy statement also discusses newer paradigms and challenges in patient-centered care, such as the impact of technology, complexity of care strategies with self-care, a systemic approach to episodic care, and barriers to patient-centered care.[35]

- The *timely care* domain relates to reducing waits and sometimes harmful delays for both those who receive and those who give care. Timeliness is an important characteristic of any service and is a legitimate and valued focus of improvement in health care and other industries.

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— Data from the CRUSADE national quality-improvement initiative showed that median delay from onset of symptoms to hospital presentation for patients presenting with NSTEMI was 2.6 hours and was significantly associated with in-hospital mortality but did not change over time from 2001 to 2006.[36]
— Bradley et al[37] demonstrated that participation in the Door-to-Balloon (D2B) Alliance led to a reduction in door-to-balloon time to within 90 minutes for patients with STEMI. By March 2008, >75% of patients had door-to-balloon times of ≤90 minutes compared with only approximately one fourth of patients in April 2005.
— Using data between 2005 and 2007 from the NCDR CathPCI Registry, Wang et al[38] demonstrated that among STEMI patients, only 10% of the transfer patients received PCI within 90 minutes (versus 63% for direct-arrival patients; $P<0.0001$).
— Glickman et al[39] showed that a year-long implementation of standardized protocols as part of a statewide regionalization program was associated with a significant improvement in median door-in–door-out times among 436 STEMI patients who presented at non-PCI hospitals who required transfer (before intervention: 97 minutes, interquartile range 56–160 minutes; after intervention: 58 minutes, interquartile range 35–90 minutes; $P<0.0001$).
— Data on time to reperfusion for STEMI or ischemic stroke are provided from national registries in Table 21-10.

- *Efficiency* has been defined as avoiding waste, in particular waste of equipment, supplies, ideas, and energy. In an efficient healthcare system, resources are used to get the best value for the money spent.

— The AHA and ACC have jointly developed a scientific statement that outlines standards for measures to be used for public reporting of efficiency in health care. The group identified 4 standards important to the development of any efficiency performance measure, including (1) integration of quality and cost, (2) valid cost measurement and analysis, (3) no or minimal incentive to provide poor-quality care, and (4) no or proper attribution of the measure. In the statement, 4 examples were provided of hospital-based efficiency measures, as well as information on how each of the measures fared within the 4 domains recommended. The examples were length of stay, 30-day readmission, hospitalization costs, and nonrecommended imaging tests.[40]
— At an urban, tertiary care, academic medical center ED, elements of departmental work flow were redesigned to streamline patient throughput before implementation of a fully integrated ED information system with patient tracking, computerized charting and order entry, and direct access to patient historical data from the hospital data repository. Increasing the clinical information available at the bedside and improving departmental work flow through ED information system implementation and process redesign led to decreased patient throughput times and improved ED efficiency (eg, the length of stay for all patients [from arrival to time patient left the ED] decreased by 1.94 hours, from 6.69 [n=508] before the intervention to 4.75 [n=691] hours after the intervention; $P<0.001$).[41]
— Himmelstein et al[42] analyzed whether more-computerized hospitals had lower costs of care or administration or better quality, to address a common belief that computerization improves healthcare quality, reduces costs, and increases administrative efficiency. They found that hospitals that increased computerization faster had more rapid administrative cost increases ($P=0.0001$); however, higher overall computerization scores correlated weakly with better quality scores for AMI ($r=0.07$, $P=0.003$) but not for HF, pneumonia, or the 3 conditions combined. In multivariate analyses, more-computerized hospitals had slightly better quality. The authors concluded that hospital computing might modestly improve process measures of quality but does not reduce administrative or overall costs.
— In a retrospective cohort study of cases (111 707) submitted to the NCDR ICD [implantable cardioverter-defibrillator] Registry between January 1, 2006, and June 30, 2009, 25 145 (22.5%) received non–evidence-based implantable cardioverter-defibrillator therapy. Patients who received non–evidence-based implantable cardioverter-defibrillator therapy compared with those who received evidence-based implantable cardioverter-defibrillator therapy had a significantly higher risk of in-hospital death (0.57% versus 0.18%, $P<0.001$) and any postprocedure complication (3.23% versus 2.41%, $P<0.001$).[43]
— In a multicenter study of patients within the NCDR undergoing PCI, Chan et al[44] reported results of the appropriateness of PCI for both acute and nonacute indications. Among patients undergoing PCI for acute indications (71.1% of the cohort), 98.5% of the procedures were classified as appropriate, 0.3% as uncertain, and 1.1% as inappropriate. Among patients undergoing PCI for nonacute indications (28.9% of the cohort), 50.4% of the procedures were classified as appropriate, 38% as uncertain, and 11.6% as inappropriate. There was also substantial variation for inappropriate nonacute PCI across hospitals (median hospital rate 10.8%; interquartile range 6.0%–16.7%).

- *Equitable care* means the provision of care that does not vary in quality because of personal characteristics such as sex, ethnicity, geographic location, and socioeconomic status. The aim of equity is to secure the benefits of quality health care for all the people of the United States. With regard to equity in caregiving, all individuals rightly expect to be treated fairly by local institutions, including healthcare organizations.

— Chan et al[45] demonstrated that rates of survival to discharge were lower for black patients (25.2%) than for white patients (37.4%) after in-hospital cardiac arrest. Lower rates of survival to discharge for blacks reflected lower rates of both successful resuscitation (55.8% versus 67.4%) and postresuscitation survival (45.2% versus 55.5%). Adjustment for the hospital site at which patients received care explained a substantial portion of the racial differences in successful resuscitation (adjusted RR, 0.92; 95% CI, 0.88–0.96; $P<0.001$)

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and eliminated the racial differences in postresuscitation survival (adjusted RR, 0.99; 95% CI, 0.92–1.06; *P*=0.68). The authors concluded that much of the racial difference was associated with the hospital center in which black patients received care.

— Kapoor et al[46] evaluated 99 058 HF admissions from 244 sites between January 2005 and September 2009. Patients were grouped on the basis of payer status (private/health maintenance organization, no insurance, Medicare, or Medicaid). Compared with private/health maintenance organization group, the other 3 groups were less likely to receive evidence-based therapies (β-blockers, implantable cardioverter-defibrillators, anticoagulation for AF, angiotensin-converting enzyme inhibitors, or angiotensin receptor blockers) and had longer hospital stays. Higher adjusted rates of in-hospital mortality were also seen in patients with Medicaid (OR, 1.22; 95% CI, 1.06–1.41) and in patients with reduced EF and no insurance (OR, 1.61; 95% CI, 1.15–2.25).

— Cohen et al[47] demonstrated that among hospitals engaged in a national quality monitoring and improvement program, evidence-based care for AMI appeared to improve over time for patients irrespective of race/ethnicity, and differences in care by race/ethnicity care were reduced or eliminated. They analyzed 142 593 patients with AMI (121 528 whites, 10 882 blacks, and 10 183 Hispanics) at 443 hospitals participating in the GWTG-CAD program. Overall, defect-free care was 80.9% for whites, 79.5% for Hispanics (adjusted OR versus whites, 1.00; 95% CI, 0.94–1.06; *P*=0.94), and 77.7% for blacks (adjusted OR versus whites, 0.93; 95% CI, 0.87–0.98; *P*=0.01). A significant gap in defect-free care was observed for blacks during the first half of the study but was no longer present during the remainder of the study. Overall, progressive improvements in defect-free care were observed regardless of race/ethnic groups.

— Thomas et al[48] analyzed data among hospitals that voluntarily participated in the AHA's GWTG-HF program from January 2005 through December 2008. Relative to white patients, Hispanic and black patients hospitalized with HF were significantly younger (median age 78, 63, and 64 years, respectively) but had lower EFs (mean EF 41.1%, 38.8%, and 35.7%, respectively) with a higher prevalence of DM (40.2%, 55.7%, and 43.8%, respectively) and hypertension (70.6%, 78.4%, and 82.8%, respectively). The provision of guideline-based care was comparable for white, black, and Hispanic patients. Black (1.7%) and Hispanic (2.4%) patients had lower in-hospital mortality than white patients (3.5%). Improvement in adherence to all-or-none HF measures increased annually from year 1 to year 3 for all 3 racial/ethnic groups.[48]

— Al-Khatib et al[49] analyzed implantable cardioverter-defibrillator use for primary prevention among 11 880 patients with a history of HF, left ventricular EF<35%, and age >65 years enrolled in the GWTG-HF registry from January 2005 through December 2009. From 2005 to 2007, overall implantable cardioverter-defibrillator use increased from 30.2% to 42.4% and then remained unchanged in 2008 to 2009. After adjustment for confounders, implantable cardioverter-defibrillator use increased significantly in the overall study population during 2005 to 2007 (OR, 1.28; 95% CI, 1.11–1.48 per year; *P*=0.0008) and in black women (OR, 1.82; 95% CI, 1.28–2.58 per year; *P*=0.0008), white women (OR, 1.30; 95% CI, 1.06–1.59 per year; *P*=0.010), black men (OR, 1.54; 95% CI, 1.19–1.99 per year; *P*=0.0009), and white men (OR, 1.25; 95% CI, 1.06–1.48 per year; *P*=0.0072). The increase in implantable cardioverter-defibrillator use was greatest among blacks. They concluded that although previously described racial disparities in the use of implantable cardioverter-defibrillators were no longer present in their study by the end of the study period, a sex difference in their use persisted.[49]

— GWTG data by race, sex, and ethnicity are provided in Tables 21-11 through 21-13.

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Table 21-1. Acute Coronary Syndrome Quality-of-Care Measures, 2011

Quality-of-Care Measure	VHA*	National Data From HIQR Program†	ACTION-GWTG STEMI‡	ACTION-GWTG NSTEMI‡
Aspirin within 24 h of admission	99	99	98.4	97.1
Aspirin at discharge	99	98.8	98.9	97.9
β-Blockers within 24 h of admission, among AMI and angina patients	96	R	NM	NM
β-Blockers at discharge	99	98.6	97.7	96.1
Lipid-lowering medication at discharge§	98	96.8	99	98.1
Lipid therapy at discharge if LDL cholesterol >130 mg/dL	97	NM	NM	NM
ARB/ACEI at discharge for patients with LVEF <40%	95	96.8	90.4	85.7
ACEI at discharge for AMI patients	NM	NM	72.9	60.8
ARB at discharge for AMI patients	NM	NM	8.1	12.4
Adult smoking cessation advice/counseling	100	99.7	98.7	98.1
Cardiac rehabilitation referral for AMI patients	NM	NM	83.2	72

Values are percentages.

VHA indicates Veterans Health Administration; HIQR, Hospital Inpatient Quality Reporting; ACTION-GWTG, Acute Coronary Treatment and Intervention Outcomes Registry–Get With The Guidelines; STEMI, ST-elevation myocardial infarction; NSTEMI, non–ST-elevation myocardial infarction; AMI, acute myocardial infarction; R, retired in 2009; NM, not measured; LDL, low-density lipoprotein; ARB, angiotensin receptor blocker; ACEI, angiotensin-converting enzyme inhibitor; and LVEF, left ventricular ejection fraction.

*VHA: AMI patients.

†HIQR Program includes data from all payers, including Medicare and Medicaid.

‡ACTION Registry: STEMI and NSTEMI patients are reported separately. Patients must be admitted with acute ischemic symptoms within the previous 24 hours, typically reflected by a primary diagnosis of STEMI or NSTEMI. Patients who are admitted for any other clinical condition are not eligible.

§Denotes statin use at discharge. Use of nonstatin lipid-lowering agent was 9.7% for STEMI patients and 12.6% for NSTEMI patients in the ACTION registry.

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Table 21-2. HF Quality-of-Care Measures, 2011

Quality-of-Care Measure	National Data From HIQR Program*	AHA GWTG-HF	VHA
LVEF assessment	98.3	99.2	100
ARB/ACEI at discharge for patients with LVSD	95.3	95.4	96
Complete discharge instructions	91	93.5	97
Adult smoking cessation advice/counseling	98.9	99.2	99
β-Blockers at discharge for patients with LVSD, no contraindications	NM	96.2	NM
Anticoagulation for atrial fibrillation or atrial flutter, no contraindications	NM	75.4	95

Values are percentages.

In the GWTG registry, mechanical ventilation was required in 1.1% of patients. In-hospital mortality rate was 3.4%, and mean length of hospital stay was 5.7 days (median 4.0 days).

HF indicates heart failure; HIQR, Hospital Inpatient Quality Reporting; AHA GWTG-HF, American Heart Association's Get With The Guidelines–Heart Failure; VHA, Veterans Health Administration; LVEF, left ventricular ejection fraction; ARB/ACEI, angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; LVSD, left ventricular systolic dysfunction; and NM, not measured.

*HIQR Program includes data from all payers, including Medicare and Medicaid.

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Table 21-3. Time Trends in GWTG-ACS Quality-of-Care Measures, 2006–2011

Quality-of-Care Measure	2006	2007	2008	2009	2010*	2011*
Aspirin within 24 h of admission	94.7	92.8	91.2	90.9	97	97.6
Aspirin at discharge	94.4	95.8	94.9	95.5	98	98.3
β-Blockers at discharge	92.8	94.6	94.5	94.9	96	96.7
Lipid-lowering medication at discharge	84.5	85.6	81.6	86.8	92†	98.4†
Lipid therapy at discharge if LDL cholesterol >100 mg/dL	89.1	90.7	91.9	92.5	NM	NM
ARB/ACEI at discharge for patients with LVEF <40%	87.3	91.1	91.9	91.9	86	87.8
Adult smoking cessation advice/counseling	94.3	97.4	98.4	98.4	98	98.4
Cardiac rehabilitation referral for AMI patients	71.1	63.6	52.0	49.1	75	76.5

Values are percentages.

In the ACTION registry (Acute Coronary Treatment and Intervention Outcomes Registry), the unadjusted in-hospital mortality rate for 2011 was 4.6% (95% confidence interval, 4.4%–4.7%); excludes transfer-out patients). The American Heart Association's (AHA's) GWTG-CAD has now merged into the ACTION registry.

GWTG-ACS indicates Get With The Guidelines–Acute Coronary Syndrome; LDL, low-density lipoprotein; NM, not measured; ARB/ACEI, angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; LVEF, left ventricular ejection fraction; and AMI, acute myocardial infarction.

*Measures from 2006–2009 are from the AHA's GWTG-CAD (Coronary Artery Disease) registry. 2010/2011 measures are from the AHA's ACTION registry (Acute Coronary Treatment and Intervention Outcomes Registry.

†Represents statin use.

Table 21-4. Time Trends in GWTG-HF Quality-of-Care Measures, 2006–2011

Quality-of-Care Measure	2006	2007	2008	2009	2010	2011
LVEF assessment*	93.8	96.2	96.8	98.2	98	99.2
ARB/ACEI at discharge for patients with LVSD*	85.5	89.1	91.6	93.0	94.2	95.4
Complete discharge instructions†	78.8	84.8	88.5	90.9	93.3	93.5
Postdischarge appointment (new for 2011)*	…	…	…	…	…	13.3
Adult smoking cessation advice/counseling†	90.8	94.7	97.1	97.6	99.3	99.2
β-Blockers at discharge for patients with LVSD, no contraindications†	89.9	90.2	92.5	92.7	94.8	96.2
Evidence-based specific β-blockers*	67.7	58.9	54.1	45.2	48.4	58.4
Anticoagulation for atrial fibrillation or atrial flutter, no contraindications	62.9	61.6	60.7	68.9	70.2	75.4

Values are percentages.

In the GWTG registry, mechanical ventilation was required in 1.1% of patients. In-hospital mortality was 3.4%, and mean length of hospital stay was 5.7 days (median 4.0 days).

GWTG-HF indicates Get With The Guidelines–Heart Failure; LVEF, left ventricular ejection fraction; ARB/ACEI, angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; and LVSD, left ventricular systolic dysfunction.

*Indicates the 4 key achievement measures targeted in GWTG-HF. The composite quality-of-care measure for 2011 was 57.6%. The composite quality-of-care measure indicates performance on the provision of several elements of care. It is computed by summing the numerators for each key achievement measure across the population of interest to create a composite numerator (all the care that was given), summing the denominators for each measure to form a composite denominator (all the care that should have been given), and reporting the ratio (the percentage of all the needed care that was given).

†Indicates historical key achievement measures in GWTG-HF. The composite quality-of-care measure for 2011 for the historical key achievement measures was 82.3%.

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Table 21-5. Time Trends in GWTG-Stroke Quality-of-Care Measures, 2006–2011

Quality-of-Care Measure	2006	2007	2008	2009	2010	2011
IV tPA in patients who arrived ≤2 h after symptom onset, treated ≤3 h*	55.8	60.2	63.9	73.1	76.2	78.3
IV tPA in patients who arrived <3.5 h after symptom onset, treated ≤4.5 h†	…	…	…	…	42.5	57.9
IV tPA door-to-needle time ≤60 min	22.5	24.9	25.9	28.0	29.5	33.8
Thrombolytic complications: IV tPA and life-threatening, serious systemic hemorrhage	20.8	17.3	16.1	15.1	13.1	15.7
Antithrombotics <48 h after admission*	94.8	95.8	96.0	96.2	96.3	96.7
DVT prophylaxis by second hospital day*	85.3	88.9	92.2	92.7	92.2	93.5
Antithrombotics at discharge*	94.1	95.1	97.0	97.8	97.7	98.1
Anticoagulation for atrial fibrillation at discharge*	88.2	89.5	93.1	93.5	93.5	93.1
Therapy at discharge if LDL cholesterol >100 mg/dL or LDL cholesterol not measured or on therapy at admission*	70.3	76.3	82.1	86.2	88.1	89.8
Counseling for smoking cessation*	86.1	92.2	94.3	96.2	96.7	97.0
Lifestyle changes recommended for BMI >25 kg/m^2	42.5	45.7	51.7	57.3	57.8	57.8
Composite quality-of-care measure	85.9	88.9	91.7	93.3	93.7	94.4

Values are percentages.

In-hospital mortality for the 2011 patient population was 6.3% percent, and mean length of hospital stay was 5.3 days (median 4.0 days).

GWTG-Stroke indicates Get With The Guidelines–Stroke; IV, intravenous; tPA, tissue-type plasminogen activator; DVT, deep venous thrombosis; LDL, low-density lipoprotein; and BMI, body mass index.

*Indicates the 7 key achievement measures targeted in GWTG-Stroke.

†New quality measure subsequent to the European Cooperative Acute Stroke Study III.

Table 21-6. Additional ACTION-GWTG Quality-of-Care Metrics for ACS Care, 2011

Quality Metrics	Overall	STEMI	NSTEMI
ECG within 10 min of arrival	62.4	74.2	57.1
Aspirin within 24 h of arrival	97.6	98.4	97.1
Any anticoagulant use*	93.2	95.4	91.7
Dosing error			
UFH dose	48.6	47.5	49.4
Enoxaparin dose	11.8	10.6	11.9
Glycoprotein IIb/IIIa inhibitor dose	7.2	7.4	7.0
Aspirin at discharge	98.3	98.9	97.9
Prescribed statins on discharge	98.4	99	98.1
Adult smoking cessation advice/ counseling	98.4	98.7	98.1
Cardiac rehabilitation referral	76.5	83.2	72
In-hospital mortality† (95% CI)	4.6 (4.4–4.7)	5.9 (5.7–6.2)	3.7(3.5–3.8)

Values are percentages.

ACTION-GWTG indicates Acute Coronary Treatment and Intervention Outcomes Registry–Get With The Guidelines; ACS, acute coronary syndrome; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non–ST-segment elevation myocardial infarction; ECG, electrocardiogram; UFH, unfractionated heparin; and CI, confidence interval.

*Includes UFH, low-molecular-weight heparin, or direct thrombin inhibitor use.

†Excludes transfer-out patients.

Table 21-7. National Committee for Quality Assurance Health Plan Employer Data and Information Set Measures of Care, 2010

	Commercial, %	Medicare, %	Medicaid, %
AMI			
β-Blocker persistence*	75.5	83.1	76.3
Cholesterol management for patients with cardiovascular disease			
Cholesterol screening	88.9	88.5	82.0
LDL cholesterol control (<100 mg/dL)	59.9	56.7	42.8
Hypertension			
BP <140/90 mm Hg	63.4	61.9	55.6
DM			
HbA_{1c} testing	89.9	90.4	82.0
HbA_{1c} >9.0%	27.3	25.9	44.0
Eye examination performed	57.7	64.6	53.1
LDL cholesterol screening	85.6	87.8	74.7
LDL cholesterol <100 mg/dL	47.7	52.1	34.6
Monitoring nephropathy	83.6	89.2	77.7
BP <140/90 mm Hg	65.7	62.3	60.4
Advising smokers and tobacco users to quit	76.7	77.9	73.6
BMI percentile assessment in children and adolescents	35.2	N/A	37.3
Nutrition counseling (children and adolescents)	37.4	N/A	45.6
Counseling for physical activity (children and adolescents)	35.3	N/A	36.7
BMI assessment for adults	40.7	50.4	42.2
Physical activity discussion in older adults (65 and older)	N/A	52.3	N/A

Values are percentages.

AMI indicates acute myocardial infarction; LDL, low-density lipoprotein; BP, blood pressure; DM, diabetes mellitus; HbA_{1c}, hemoglobin A_{1c}; BMI, body mass index; and N/A, not available or not applicable.

*β-Blocker persistence: Received persistent β-blocker treatment for 6 months after AMI hospital discharge.

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Table 21-8. Quality of Care for EMS-Treated Out-of-Hospital Cardiac Arrest

	Overall	Adults	Children
Bystander CPR, mean (95% CI), %	40.1 (38.9–41.3)	40.1 (38.9–41.4)	55.9 (49.1–62.7)
Shocked by AED before EMS, mean (95% CI), %	1.8 (1.5–2.2)	1.9 (1.5–2.2)	1.0 (0–2.3)
Chest compression fraction during first 5 min of EMS CPR, mean (SD)*	0.79 (0.22)	0.79 (0.22)	0.80 (0.19)
Compression depth, mean (SD)*	39.2 (14.9)	39.3 (14.9)	31.9 (14.2)
Preshock pause duration, mean (SD)†	15.4 (24.3)	15.4 (24.4)	13 (N/A)
Time to first EMS defibrillator applied, mean (SD), min	7.4 (9.0)	7.4 (9.2)	6.7 (3.2)

EMS indicates emergency medical services; CPR, cardiopulmonary resuscitation; CI, confidence interval; AED, automated external defibrillator; and SD, standard deviation.

*During first 10 minutes of resuscitation.

†Up to and including first 6 shocks.

Table 21-9. Quality of Care for In-Hospital Cardiac Arrest

	Overall
Time to first shock within 3 min*	92.1% (91.0%–93.2%)
Hypothermia after resuscitation maintained between 32°C and 34°C	35.6% (30.5%–40.7%)

*Among those with a first recorded rhythm of ventricular fibrillation, pulseless ventricular tachycardia, or shockable by automated external defibrillator.

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Table 21-10. Timely Reperfusion for ACS and Stroke, 2011

Quality-of-Care Measure	VHA* (for STEMI) or GWTG-Stroke (for Stroke)	National Data From HIQR Program†	ACTION-GWTG STEMI‡
STEMI			
tPA within 30 min	50§	58.1	46.8§
PCI within 90 min	69	92.5	94.2
Stroke			
IV tPA in patients who arrived <2 h after symptom onset, treated ≤3 h	78.3	N/A	N/A
IV tPA in patients who arrived <3.5 h after symptom onset, treated ≤4.5 h	57.9	N/A	N/A
IV tPA door-to-needle time ≤60 min	33.8	N/A	N/A

Values are percentages.

ACS indicates acute coronary syndrome; VHA, Veterans Health Administration; STEMI, ST-segment–elevation myocardial infarction; GWTG-Stroke, Get With The Guidelines–Stroke; HIQR, Hospital Inpatient Quality Reporting; ACTION-GWTG, Acute Coronary Treatment and Intervention Outcomes Registry–Get With The Guidelines; tPA, tissue-type plasminogen activator; PCI, percutaneous coronary intervention; IV, intravenous; and N/A, not applicable.

*VHA: acute myocardial infarction patients.

†HIQR Program includes data from all payers, including Medicare and Medicaid.

‡ACTION Registry: STEMI and NSTEMI patients are reported separately. Patients must be admitted with acute ischemic symptoms within the previous 24 hours, typically reflected by a primary diagnosis of STEMI or NSTEMI. Patients who are admitted for any other clinical condition are not eligible.

§Indicates low number.

Table 21-11. Quality of Care by Race/Ethnicity and Sex in the ACTION Registry, 2011

Quality-of-Care Measure	White	Black	Other	Men	Women
Aspirin at admission	97.7	97.2	96.6	97.9	97.1
Aspirin at discharge	98.4	97.8	97.9	98.6	97.8
β-Blockers at discharge	96.8	96.2	96.4	97.1	96.0
Time to PCI <90 min for STEMI patients	94.5	92.2	93.0	94.6	93.1
ARB/ACEI at discharge for patients with LVEF <40%	87.8	87.6	86.5	87.9	87.5
Statins at discharge	98.4	98.2	98.5	98.7	97.8

Values are percentages.

ACTION indicates Acute Coronary Treatment and Intervention Outcomes Network; PCI, percutaneous coronary intervention; STEMI, ST-segment–elevation myocardial infarction; ARB/ACEI angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; and LVEF, left ventricular ejection fraction.

Table 21-12. Quality of Care by Race/Ethnicity and Sex in the GWTG-HF Program

Quality-of-Care Measure	White	Black	Hispanic	Men	Women
Postdischarge appointment (new for 2011)*	14.4	16.6	14.8	15.0	13.1
Complete set of discharge instructions†	93.3	94.2	91.5	93.9	93.0
Measure of LV function *	99.4	99.5	98.8	99.3	99.1
ACEI or ARB at discharge for patients with LVSD, no contraindications*	95.0	96.5	94.1	95.3	95.4
Smoking cessation counseling, current smokers†	99.1	99.3	97.8	99.1	99.1
Evidence-based specific β-blockers*	57.8	62.9	64.5	61.9	60.6
β-Blockers at discharge for patients with LVSD, no contraindications†	96.6	96.7	95.0	96.5	96.2
Hydralazine/nitrates at discharge for patients with LVSD, no contraindications	…	13.0	…	12.7‡	12.1‡
Anticoagulation for atrial fibrillation or atrial flutter, no contraindications	77.7	73.2	72.2	77.1	73.7
Composite quality-of-care measure	57.5	61.4	59.6	59.7	56.6
Composite quality-of-care measure (historical measures)	91.5	91.7	88.2	90.8	91.6

Values are percentages.

GWTG-HF indicates Get With The Guidelines–Heart Failure; LV, left ventricular; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; and LVSD, left ventricular systolic dysfunction.

*Indicates the 5 key achievement measures targeted in GWTG-HF.

†Indicates historical key achievement measures in GWTG-HF.

‡For black patients only.

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Table 21-13. Quality of Care by Race/Ethnicity and Sex in the GWTG-Stroke Program

Quality-of-Care Measure	White	Black	Hispanic	Male	Female
IV tPA in patients who arrived ≤2 h after symptom onset, treated ≤3 h*	77.9	78.7	78.4	78.7	78.0
IV tPA in patients who arrived <3.5 h after symptom onset, treated ≤4.5 h	57.4	58.8	61.7	58.5	57.4
IV tPA door-to-needle time ≤60 min	33.7	33.8	34.6	35.4	32.3
Thrombolytic complications: IV tPA and life-threatening, serious systemic hemorrhage	14.2	18.8	20.6	15.1	14.7
Antithrombotics <48 h after admission*	96.9	96.3	96.1	97.0	96.4
DVT prophylaxis by second hospital day*	93.5	93.8	92.9	93.6	93.4
Antithrombotics at discharge*	98.3	97.6	97.3	98.3	97.9
Anticoagulation for atrial fibrillation at discharge*	93.3	93.2	91.6	93.5	92.8
Therapy at discharge if LDL>100 mg/dL or LDL not measured or on therapy at admission*	89.4	91.0	90.5	91.2	88.5
Counseling for smoking cessation*	97.2	97.0	96.5	97.0	97.1
Lifestyle changes recommended for BMI >25 kg/m^2	57.6	57.6	61.6	58.0	57.5
Composite quality-of-care measure	94.4	94.6	93.9	94.9	93.9

Values are percentages.

GWTG-Stroke indicates Get With The Guidelines–Stroke; IV, intravenous; tPA, tissue-type plasminogen activator; DVT, deep vein thrombosis; LDL, low-density lipoprotein; and BMI, body mass index.

*Indicates the 7 key performance measures targeted in GWTG-Stroke.

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22. Medical Procedures

See Tables 22-1 and 22-2 and Charts 22-1 through 22-4.

Trends in Operations and Procedures

(See Charts 22-1 and 22-2.)

- The total number of inpatient cardiovascular operations and procedures increased 28%, from 5 939 000 in 2000 to 7 588 000 in 2010 (NHLBI computation based on NCHS annual data). Data from the NHDS were examined for trends from 1990 to 2004 for use of PCI and CABG and in-hospital mortality rate attributable to PCI and CABG by sex.[1]
  - — Discharge rates (per 10 000 population) for PCI increased 58%, from 37.2 in 1990 to 1992 to 59.2 in 2002 to 2004.
  - — Discharge rates for CABG increased from 34.1 in 1990 to 1992 to 38.6 in 1996 to 1998, then declined to 25.2 in 2002 to 2004.
  - — In 1990 to 1992, discharge rates for CABG were 53.5 for males and 18.1 for females; these rates increased through 1996 to 1998, then declined to 38.8 and 13.6, respectively, in 2002 to 2004. The magnitude of these declines decreased by age decile and were essentially flat for both men and women ≥75 years of age.
  - — PCI discharge rates increased from 54.5 for males and 23.0 for females to 83.0 and 38.7, respectively, over the 15-year time interval. In 2002 to 2004, discharge rates for men and women 65 to 74 years of age were 135.1 and 64.0, respectively. For those ≥75 years of age, the rates were 128.7 and 69.0, respectively.
  - — In-hospital mortality rate (deaths per 100 CABG discharges) declined from 4.3 to 3.5 between 1990 to 1992 and 2002 to 2004 despite an increase in Charlson comorbidity index. The mortality rate declined in all age and sex subsets, but especially in women.
- Data from the Acute Care Tracker database were used to estimate the population-based rates per 100 000 population for PCI and CABG procedures from 2002 to 2005, standardized to the 2005 US population[2]:
  - — Adjusted for age and sex, the overall rate for coronary revascularization declined from 382 to 358 per 100 000. PCI rates during hospitalization increased from 264 to 267 per 100 000, whereas CABG rates declined from 121 to 94.
- Data from men and women enrolled in Medicare from 1992 to 2001 suggest that efforts to eliminate racial disparities in the use of high-cost cardiovascular procedures (PCI, CABG, and carotid endarterectomy) were unsuccessful.[3]
  - — In 1992, among women, the age-standardized rates of carotid endarterectomy were 1.59 per 1000 enrollees for whites and 0.64 per 1000 enrollees for blacks. By 2002, the rates were 2.42 per 1000 enrollees among white women and 1.15 per 1000 enrollees among black women. For men, the difference in rates between whites and blacks remained the same. In 1992, the rates were 3.13 per 1000 enrollees among white men and 0.82 per 1000 enrollees among black men; in 2001, the rates were 4.42 and 1.44, respectively.

Abbreviations Used in Chapter 22

CABG	coronary artery bypass graft
CHF	congestive heart failure
D2B	Door-to-Balloon Alliance
GWTG-CAD	Get With The Guidelines–Coronary Artery Disease
HD	heart disease
HPLHS	hypoplastic left heart syndrome
ICD-9-CM	*International Classification of Diseases, 9th Revision, Clinical Modification*
NCHS	National Center for Health Statistics
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
PCI	percutaneous coronary intervention
PTCA	percutaneous transluminal coronary angioplasty
STEMI	ST-segment–elevation myocardial infarction
TOF	tetralogy of Fallot
VSD	ventricular septal defect

Cardiac Catheterization and PCI

- From 2000 to 2010, the number of cardiac catheterizations decreased slightly, from 1 221 000 to 1 029 000 annually (NHDS, NHLBI tabulation).
- In 2010, an estimated 492 000 patients underwent PCI (previously referred to as percutaneous transluminal coronary angioplasty, or PTCA) procedures in the United States (NHDS, NHLBI tabulation).
- In 2010, ≈67% of PCI procedures were performed on men, and ≈51% were performed on people ≥65 years of age (NHDS, NHLBI tabulation).
- In-hospital death rates for PCI have remained stable, although comorbidities increased for patients who received the procedure.[1]
- In 2010, ≈75% of stents implanted during PCI were drug-eluting stents compared with 25% that were bare-metal stents (NHDS, NHLBI computation).
- In a study of nontransferred patients with STEMI treated with primary PCI from July 2006 to March 2008, there was significant improvement over time in the percentage of patients receiving PCI within 90 minutes, from 54.1% from July to September 2006 to 74.1% from January to March 2008, among hospitals participating in the GWTG-CAD program. This improvement was seen whether or not hospitals joined the D2B Alliance during that period.[4]
- The rate of any cardiac stent procedure rose by 61% from 1999 to 2006, then declined by 27% between 2006 and 2009.[5]

Cardiac Open Heart Surgery

The NHDS (NCHS) estimates that in 2010, in the United States, 219 000 patients underwent a total of 397 000 coronary artery bypass procedures (defined by procedure codes). CABG volumes have declined nationally since 1998. Risk-adjusted mortality for CABG has declined significantly over the past decade.

- Data from the Society of Thoracic Surgeons' National Adult Cardiac Database, which voluntarily collects data

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from ≈80% of all hospitals that perform CABG in the United States, indicate that a total of 158 008 procedures involved CABG in 2010.[6]

- Data from the Society of Thoracic Surgeons' National Adult Cardiac Database document a 50% decline in the risk-adjusted mortality rate despite a significant increase in preoperative surgical risk.[7]

Congenital Heart Surgery, 1998 to 2002 (From Society of Thoracic Surgeons)

There were 103 664 procedures performed from July 2006 to June 2010. The in-hospital mortality rate was 3.2% in 2010. The 5 most common diagnoses were the following: patent ductus arteriosus (7.4%); HPLHS (6.9%); VSD, type 2 (6.3%); cardiac, other (5.3%); and TOF (4.9%).[8]

Congenital Heart Surgery, 1998 to 2002 (From Society of Thoracic Surgeons)

There were 16 920 procedures performed from 1998 to 2002 at 18 centers. In 2002, there were 4208 procedures performed. The in-hospital mortality rate ranged from 5.7% in 1998 to 4.3% in 2002. Of these procedures, ≈46% were performed in children >1 year old, ≈32% in infants between 29 days and 1 year of age, and ≈22% in neonates (<29 days old). The conditions for which these procedures were most commonly performed were the following: patent ductus arteriosus (6.5%), VSD (6.4%), and TOF (6.0%).

Heart Transplantations

(See Charts 22-3 and 22-4.)

In 2011, 2322 heart transplantations were performed in the United States (Chart 22-3). There are 246 transplant hospitals in the United States, 128 of which performed heart transplantations (based on Organ Procurement and Transplantation Network data as of April 24, 2012).

- Of the recipients in 2011, 68.7% were male, and 65.8% were white; 20.9% were black, whereas 9.6% were Hispanic. Heart transplantations by recipient age are shown in Chart 22-4.
- As of April 18, 2012, for transplants that occurred between 2007 and 2008, the 1-year survival rate was 89.4% for males and 87.8% for females; the 5-year rates between 2003 and 2008 were 76.1% for males and 72.6% for females; and the 10-year rates between 1998 and 2008 were 57.0% for males and 54.7% for females. The 1-, 5-, and 10-year survival rates for white cardiac transplant patients were 89.5%, 77.4%, and 58.8%, respectively. For black patients, they were 85.7%, 65.8%, and 44.8%, respectively. For Hispanic patients, they were 91.5%, 76.2%, and 62.5%, respectively.
- As of April 24, 2012, 3151 patients were on the transplant waiting list for a heart transplant, and 52 patients were on the list for a heart/lung transplant.

Cardiovascular Healthcare Expenditures

An analysis of claims and enrollment data from the Continuous Medicare History Sample and from physician claims from 1995 to 2004 was used to evaluate the conditions that contributed to the most expensive 5% of Medicare beneficiaries.[8]

- Ischemic HD, CHF, and cerebrovascular disease, respectively, constituted 13.8%, 5.9%, and 5.7% of the conditions of all beneficiaries in 2004. In patients in the top 5% overall for all expenditures, the respective figures were 39.1%, 32.7%, and 22.3% for these cardiovascular conditions.

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Table 22-1. 2010 National Healthcare Cost and Utilization Project Statistics: Mean Hospital Charges, In-Hospital Death Rates, and Mean Length of Stay for Various Cardiovascular Procedures

Procedure	Mean Hospital Charges, $	In-Hospital Death Rate, %	Mean Length of Stay, d	ICD-9 CM Procedure Codes
Total vascular and cardiac surgery and procedures	71 330	3.00	6.2	35–39, 00.50–00.51, 00.53–00.55, 00.61-00.66
Cardiac revascularization (bypass)	133 247	1.52	8.9	36.1–36.3
PCI	67 086	1.08	3.2	00.66
Cardiac catheterization	39 264	0.87	3.7	37.21–37.23
Pacemakers	65 538	1.12	5.0	37.7–37.8, 00.50, 00.53
Implantable defibrillators	146 941	0.62	5.4	37.94–37.99, 00.51, 00.54
Endarterectomy	35 189	0.22	2.6	38.12
Valves	186 622	4.22	10.9	35.1–35.2, 35.99
Heart transplantation	536 947	3.95	37.0	37.51

ICD-9-CM indicates *International Classification of Diseases, Clinical Modification, 9th Revision*; PCI, percutaneous coronary intervention.

Data derived from the Agency for Healthcare Research and Quality, Healthcare Cost and Utilization Project Nationwide Inpatient Sample, 2010.[9]

Table 22-2. Estimated* Inpatient Cardiovascular Operations, Procedures, and Patient Data by Sex and Age: United States, 2010 (in Thousands)

		Sex			Age, y			
Operation/Procedure/Patients	ICD-9-CM Procedure Codes	All	Male	Female	<15	15–44	45–64	≥65
Valves	35.1, 35.2, 35.99	106	64	42	4†	5†	32	65
Angioplasty	36.0, 0.66	955	642	313	...	44	421	489
PCI (patients)	36.06, 36.07, 0.66	492	330	162	...	23	216	253
PCI	0.66	500	334	166	...	23	220	257
PCI with stents	36.06, 36.07	454	308	146	...	21	201	233
Cardiac revascularization‡	36.1–36.3	397	298	99	...	9†	157	231
Cardiac revascularization (patients)	36.1–36.3	219	164	55	...	5†	86	128
Cardiac catheterization	37.21–37.23	1029	638	391	7†	64	456	502
Pacemakers	37.7, 37.8, 00.50, 00.53	370	196	174	3†	6†	57	305
Pacemaker devices	37.8, 00.53	159	81	78	1†	3†	20	135
Pacemaker leads	37.7, 00.50	212	115	96	1†	3†	36	171
Implantable defibrillators	37.94–37.99, 00.51, 00.54	97	71	26	...	8†	31	58
Endarterectomy	38.12	100	55	45	...	...	29	71
Total vascular and cardiac surgery and procedures§‖	35–39, 00.50–00.51, 00.53–00.55, 00.61–00.66	7588	4397	3191	310	681	2706	3891

These data do not reflect any procedures performed on an outpatient basis. Many more procedures are being performed on an outpatient basis. Some of the lower numbers in this table compared with 2006 probably reflect this trend. Data include procedures performed on newborn infants.

ICD-9-CM indicates *International Classification of Diseases, Clinical Modification, 9th Revision*; PCI, percutaneous coronary intervention; and ellipses (. . .), data not available.

*Breakdowns are not available for some procedures, so entries for some categories do not add to totals. These data include codes for which the estimated number of procedures is <5000. Categories with such small numbers are considered unreliable by the National Center for Health Statistics and in some cases may have been omitted.

†Estimate should be used with caution because it may be unreliable or does not meet standards of reliability or precision.

‡Because ≥1 procedure codes are required to describe the specific bypass procedure performed, it is impossible from these (mixed) data to determine the average number of grafts per patient.

§Totals include procedures not shown here.

‖This estimate includes angioplasty and stent insertions for noncoronary arteries.

Data derived from the National Hospital Discharge Survey/National Center for Health Statistics, 2010. Estimates are based on a sample of inpatient records from short-stay hospitals in the United States.

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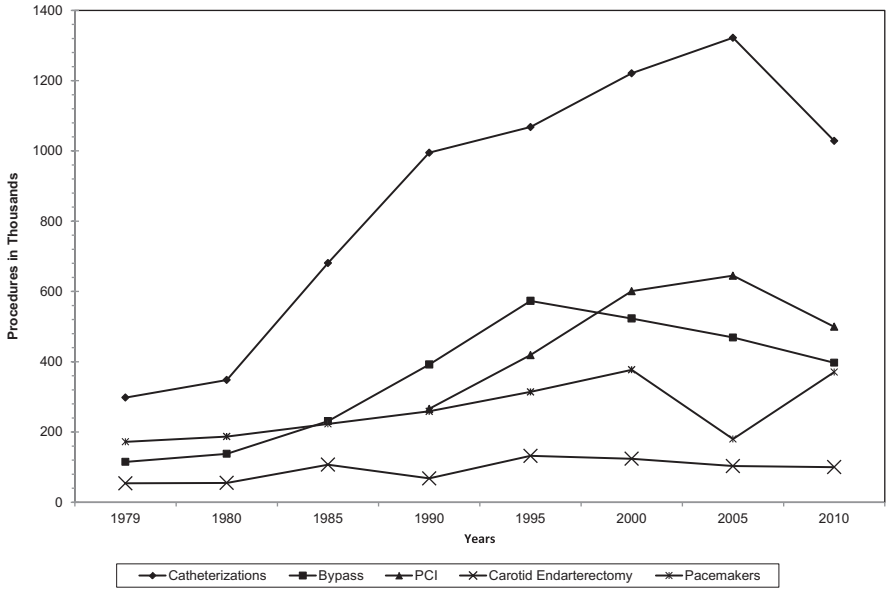

Chart 22-1. Trends in cardiovascular procedures, United States: 1979 to 2010. PCI indicates percutaneous coronary intervention. Note: Inpatient procedures only. Source: National Hospital Discharge Survey, National Center for Health Statistics, and National Heart, Lung, and Blood Institute.

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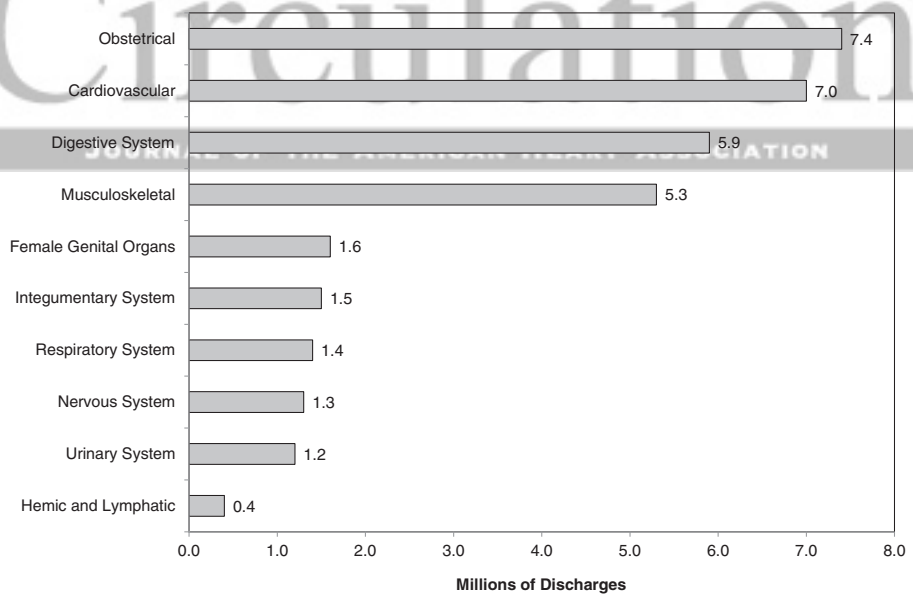

Chart 22-2. Number of surgical procedures in the 10 leading diagnostic groups, United States: 2010. Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

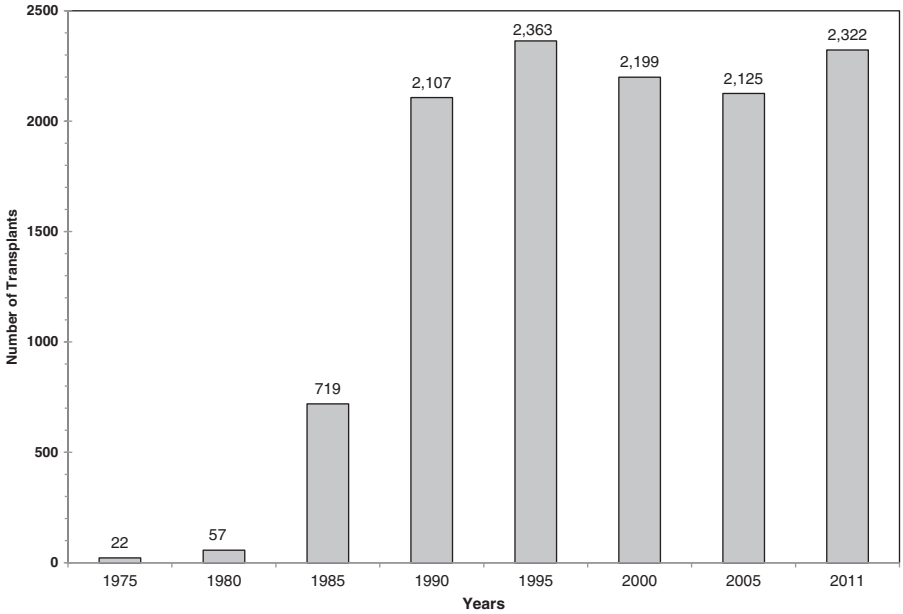

Chart 22-3. Trends in heart transplantations (United Network for Organ Sharing: 1975–2011). Source: United Network for Organ Sharing, scientific registry data.

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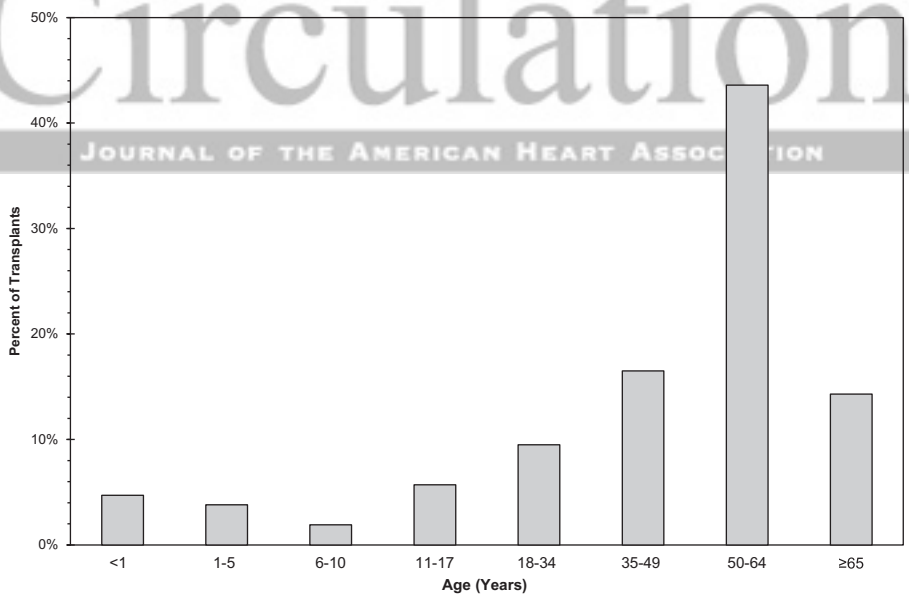

Chart 22-4. Heart transplantations in the United States by recipient age, 2011. Source: Organ Procurement and Transplantation Network data as of April 13, 2012.

23. Economic Cost of Cardiovascular Disease

See Tables 23-1 and 23-2 and Charts 23-1 through 23-4.

The annual direct and indirect cost of CVD and stroke in the United States is an estimated $312.6 billion (Table 23-1; Chart 23-1). This figure includes $192.1 billion in expenditures (direct costs, which include the cost of physicians and other professionals, hospital services, prescribed medication, and home health care, but not the cost of nursing home care) and $120.5 billion in lost future productivity attributed to premature CVD and stroke mortality in 2009 (indirect costs).

The direct costs for CVD and stroke are the healthcare expenditures for 2009 available on the Web site of the nationally representative MEPS of the Agency for Healthcare Research and Quality.[1] Details on the advantages or disadvantages of using MEPS data are provided in the Heart Disease and Stroke Statistics–2011 Update.[2] Indirect mortality costs are estimated for 2009 by multiplying the number of deaths that year attributable to CVD and strokes, in age and sex groups, by estimates of the present value of lifetime earnings for those age and sex groups as of 2009. Mortality data are from the National Vital Statistics System of the NCHS.[3] The present values of lifetime earnings are unpublished estimates furnished by the Institute on Health and Aging, University of California at San Francisco, by Wendy Max, PhD, on April 25, 2012. Those estimates have a 3% discount rate, the recommended percentage.[4] The discount rate removes the effect of inflation in income over the lifetime of earnings.

The indirect costs exclude lost productivity costs attributable to CVD and stroke illness during 2009 among workers, people keeping house, people in institutions, and people unable to work. Those morbidity costs were substantial in very old studies, but an adequate update could not be made.

Most Costly Diseases

(See Table 23-2 and Chart 23-2.)

CVD and stroke accounted for 15% of total health expenditures in 2009, more than any major diagnostic group.[1,5] That is also the case for indirect mortality costs. By way of comparison, CVD total direct and indirect costs shown in Table 23-1 are higher than the official National Cancer Institute estimates for cancer and benign neoplasms in 2008, which were cited as $228 billion total ($93 billion in direct costs, $19 billion in indirect morbidity costs, and $116 billion in indirect mortality costs).[6]

Abbreviations Used in Chapter 23

AHA	American Heart Association
CHD	coronary heart disease
CHF	congestive heart failure
COPD	chronic obstructive pulmonary disease
CVD	cardiovascular disease
GI	gastrointestinal
HBP	high blood pressure
HD	heart disease
HF	heart failure
MEPS	Medical Expenditure Panel Survey
NCHS	National Center for Health Statistics

Table 23-2 shows direct and indirect costs for CVD by sex and by 2 broad age groups. Chart 23-2 shows total direct costs for the 14 leading chronic diseases in the MEPS list. HD is the most costly condition.[1]

Projections

(See Charts 23-3 and 23-4.)

The AHA developed methodology to project future costs of care for HBP, CHD, HF, stroke, and all other CVD.[7] By 2030, 40.8% of the US population is projected to have some form of CVD. Between 2013 and 2030, total direct medical costs of CVD are projected to increase from $320 billion to $818 billion. Indirect costs (attributable to lost productivity) for all CVDs are estimated to increase from $203 billion in 2013 to $308 billion in 2030, an increase of 52%. These data indicate that CVD prevalence and costs are projected to increase substantially. It is important to underscore that differences exist between these estimates and those stated above. These apparent discrepancies largely reflect methodological differences and emphasize that the importance of cost projections resides in the documentation of profoundly adverse trends, which constitute an urgent call to action and must be reversed, rather than in the calculation of precise numbers.

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Table 23-1. Estimated Direct and Indirect Costs (in Billions of Dollars) of CVD and Stroke: United States, 2009

	Heart Disease*	Stroke	Hypertensive Disease†	Other Circulatory Conditions	Total CVD
Direct costs‡					
Hospital inpatient stays	56.3	10.7	5.9	13.2	86.1
Hospital emergency room visits	5.6	0.6	1.3	0.8	8.3
Hospital outpatient or office-based provider visits	21.4	4.5	14.1	6.7	46.7
Home health care	7.4	6.0	4.8	1.0	19.2
Prescribed medicines	8.5	1.0	21.4	0.9	31.8
Total expenditures	99.2	22.8	47.5	22.6	192.1
Indirect costs§					
Lost productivity/mortality‖	96.0	15.8	3.5	5.2	120.5
Grand totals	195.2	38.6	51.0	27.8	312.6

Numbers do not add to total because of rounding.

CVD indicates cardiovascular disease.

*This category includes coronary heart disease, heart failure, part of hypertensive disease, cardiac dysrhythmias, rheumatic heart disease, cardiomyopathy, pulmonary heart disease, and other or ill-defined heart diseases.

†Costs attributable to hypertensive disease are limited to hypertension without heart disease.

‡Medical Expenditure Panel Survey healthcare expenditures are estimates of direct payments for care of a patient with the given disease provided during the year, including out-of-pocket payments and payments by private insurance, Medicaid, Medicare, and other sources. Payments for over-the-counter drugs are not included. These estimates of direct costs do not include payments attributed to comorbidities. Total CVD costs are the sum of costs for the 4 diseases but with some duplication.

§The Statistics Committee agreed to suspend presenting estimates of lost productivity attributable to morbidity until a better estimating method can be developed.

‖Lost future earnings of persons who died in 2009, discounted at 3%.

Sources: Estimates from the Household Component of the Medical Expenditure Panel Survey of the Agency for Healthcare Research and Quality for direct costs (2009).[1] Indirect mortality costs are based on 2009 counts of deaths by the National Center for Health Statistics and an estimated present value of lifetime earnings furnished for 2009 by Wendy Max (Institute for Health and Aging, University of California, San Francisco, April 25, 2012).

All estimates prepared by Michael Mussolino, National Heart, Lung, and Blood Institute.

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Table 23-2. Costs of Total CVD in Billions of Dollars by Age and Sex: United States, 2009

		Sex		Age	
	Total	Male	Female	<65 y	≥65 y
Direct	192.1	93.7	98.4	87.7	104.4
Indirect mortality	120.5	88.6	31.9	103.7	16.8
Total	312.6	182.3	130.3	191.4	121.2

Numbers may not add to total because of rounding.

CVD indicates cardiovascular diseases and stroke.

Source: Medical Expenditure Panel Survey, 2009 (direct costs) and mortality data from the National Center for Health Statistics and present value of lifetime earnings from the Institute for Health and Aging, University of California, San Francisco (indirect costs).

All estimates prepared by Michael Mussolino, National Heart, Lung, and Blood Institute.

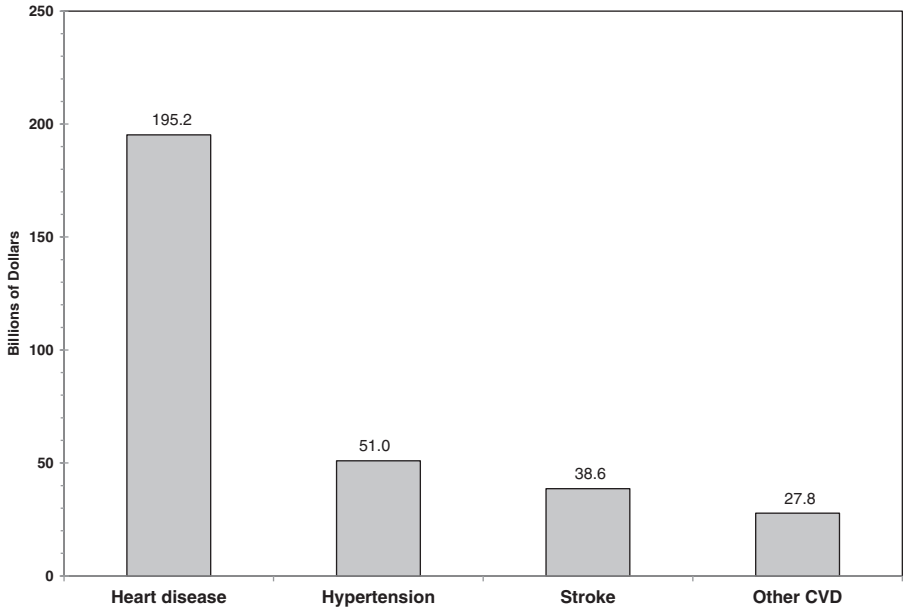

Chart 23-1. Direct and indirect costs of cardiovascular disease (CVD) and stroke (in billions of dollars), United States, 2009. Source: Prepared by the National Heart, Lung, and Blood Institute.[1,3]

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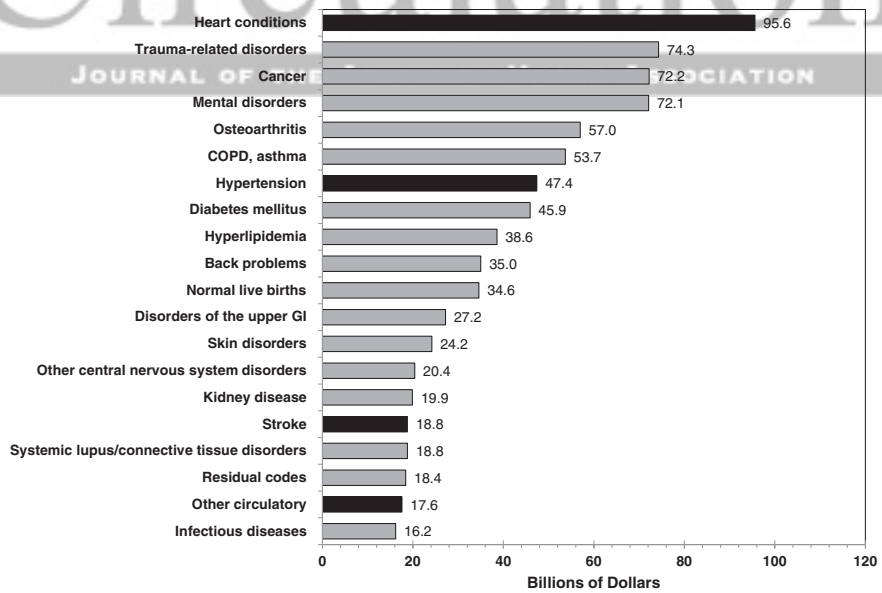

Chart 23-2. The 20 leading diagnoses for direct health expenditures, United States, 2008 (in billions of dollars). COPD indicates chronic obstructive pulmonary disease; GI, gastrointestinal. Source: National Heart, Lung, and Blood Institute; estimates are from the Medical Expenditure Panel Survey, Agency for Healthcare Research and Quality, and exclude nursing home costs.

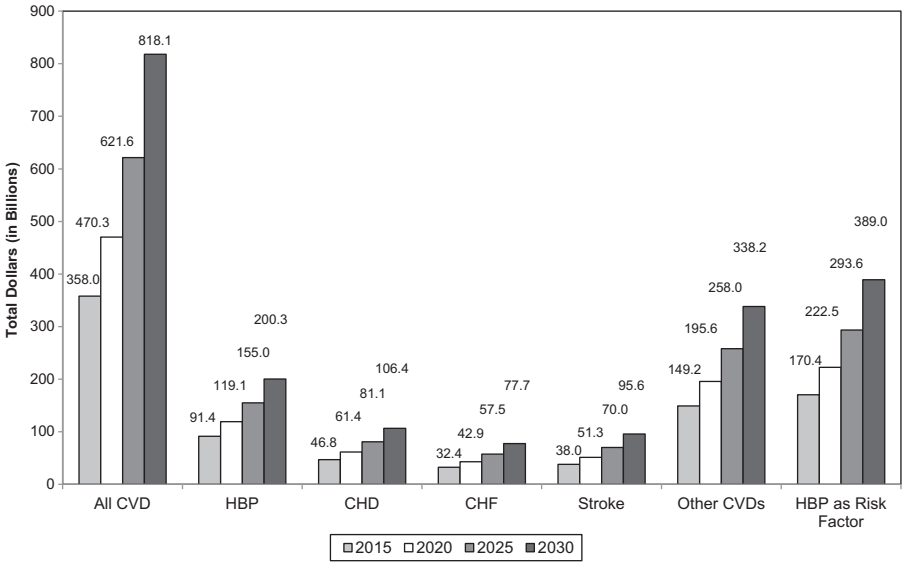

Chart 23-3. Projected total costs of cardiovascular disease (CVD), 2015–2030 (2010 $ in billions) in the United States. HBP indicates high blood pressure; CHD, coronary heart disease; and CHF, congestive heart failure. Unpublished data tabulated by the American Heart Association using methods described in Heidenreich et al[7].

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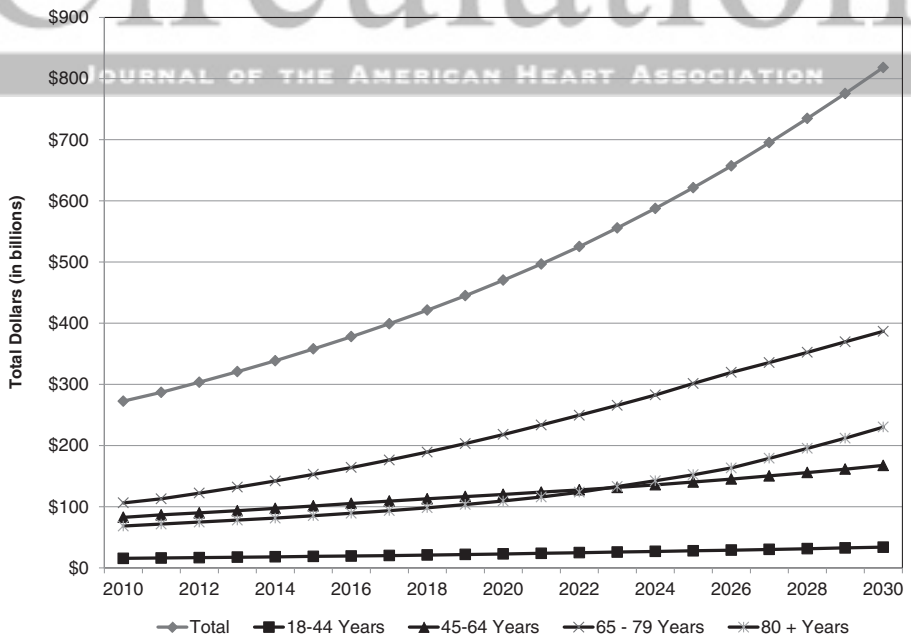

Chart 23-4. Projected total (direct and indirect) costs of total cardiovascular disease by age (2010 $ in billions). Unpublished data tabulated by the American Heart Association using methods described in Heidenreich et al[7].

24. At-a-Glance Summary Tables

See Tables 24-1 through 24-4.

Sources: See the following summary tables and charts for complete details:

- Smoking—Table 3-1
- Physical activity —Table 4-1
- Overweight/obesity—Table 6-1; Chart 6-1
- Blood cholesterol—Table 8-1
- High blood pressure—Table 9-1
- Diabetes mellitus—Table 10-1
- Total cardiovascular diseases—Table 13-1
- Stroke—Table 14-1
- Congenital heart defects—Table 15-1
- Coronary heart disease—Table 18-1
- Heart failure—Table 19-1

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Table 24-1. Males and CVD: At-a-Glance Table

Diseases and Risk Factors	Both Sexes	Total Males	White Males	Black Males	Mexican American Males
Smoking					
Prevalence, 2011*	43.8 M (19.0%)	24.1 M (21.3%)	22.8%	23.3%	16.2%†
PA‡					
Prevalence, 2011*	21.0%	24.9%	26.2%	25.9%	19.0%†
Overweight and obesity					
Prevalence, 2010					
Overweight and obesity, BMI >25.0 kg/m^2§	154.7 M (68.2%)	79.9 M (72.9%)	73.1%	68.7%	81.3%
Obesity, BMI >30.0 kg/m^2§	78.4 M (34.6%)	36.8 M (33.6%)	33.8%	37.9%	36.0%
Blood cholesterol					
Prevalence, 2010					
Total cholesterol >200 mg/dL§	98.9 M (43.4%)	45.3 M (41.3%)	40.5%	38.6%	48.1%
Total cholesterol >240 mg/dL§	31.9 M (13.8%)	14.0 M (12.7%)	12.3%	10.8%	15.2%
LDL cholesterol >130 mg/dL§	71.0 M (31.1%)	35.2 M (31.9%)	30.1%	33.1%	39.9%
HDL cholesterol <40 mg/dL§	48.7 M (21.8%)	34.6 M (31.8%)	33.1%	20.3%	34.2%
HBP					
Prevalence, 2010§	77.9 M (33.0%)	37.2 M (33.6%)	33.4%	42.6%	30.1%
Mortality, 2009‖	61 762	27 668	20 286	6574	N/A
DM					
Prevalence, 2010					
Physician-diagnosed DM§	19.7 M (8.3%)	9.6 M (8.7%)	7.7%	13.5%	11.4%
Undiagnosed DM§	8.2 M (3.5%)	5.3 M (4.7%)	4.5%	4.8%	6.6%
Prediabetes§	87.3 M (38.2%)	50.7 M (46.0%)	47.7%	35.7%	47.0%
Incidence, diagnosed DM§	1.9 M	N/A	N/A	N/A	N/A
Mortality, 2009‖	68 705	35 054	28 205	5488	N/A
Total CVD					
Prevalence, 2010§	83.6 M (35.3%)	40.7 M (36.7%)	36.6%	44.4%	33.4%
Mortality, 2009‖	787 931	386 436	329 565	46 334	N/A
Stroke					
Prevalence, 2010§	6.8 M (2.8%)	3.0 M (2.6%)	2.4%	4.3%	2.3%
New and recurrent strokes‖	795.0 K	370.0 K	325.0 K	45.0 K	N/A
Mortality, 2009‖	128 842	52 073	43 190	6962	N/A
CHD					
Prevalence, CHD, 2010§	15.4 M (6.4%)	8.8 M (7.9%)	8.2%	6.8%	6.7%
Prevalence, MI, 2010§	7.6 M (2.9%)	5.0 M (4.2%)	4.4%	3.9%	3.6%
Prevalence, AP, 2010§	7.8 M (3.2%)	3.7 M (3.3%)	3.3%	2.4%	3.4%
New and recurrent CHD¶#	915.0 K	535.0 K	465.0 K	65.0 K	N/A
New and recurrent MI#	715.0 K	410.0 K	N/A	N/A	N/A
Incidence, AP (stable angina)**	500.0 K	320.0 K	N/A	N/A	N/A
Mortality, 2009, CHD‖	386 324	210 069	183 453	21 051	N/A
Mortality, 2009, MI‖	125 464	68 814	60 316	6717	N/A
HF					
Prevalence, 2010§	5.1 M (2.1%)	2.7 M (2.5%)	2.5%	4.1%	1.9%
Mortality, 2009‖	56 410	23 563	20 815	2341	N/A

CVD indicates cardiovascular disease; M, millions; PA, physical activity; LDL, low-density lipoprotein; HDL, high-density lipoprotein; BMI, body mass index; HBP, high blood pressure; N/A, data not available; DM, diabetes mellitus; K, thousands; CHD, coronary heart disease (includes heart attack, angina pectoris chest pain, or both); MI, myocardial infarction (heart attack); AP, angina pectoris (chest pain); and HF, heart failure.

*Age ≥18 years (National Health Interview Survey).

†All Hispanic (National Health Interview Survey).

‡Met 2008 full Federal PA guidelines for adults.

§Age >20 years.

‖All ages.

¶New and recurrent MI and fatal CHD.

#Age ≥35 years.

**Age ≥45 years.

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Table 24-2. Females and CVD: At-a-Glance Table

Diseases and Risk Factors	Both Sexes	Total Females	White Females	Black Females	Mexican American Females
Smoking					
Prevalence, 2011*	43.8 M (19.0%)	19.7 M (16.7%)	19.7%	15.1%	8.3%†
PA‡					
Prevalence, 2011*	21.0%	17.1%	20.0%	11.3%	11.5%†
Overweight and obesity					
Prevalence, 2010					
Overweight and obesity, BMI >25.0 kg/m^2§	154.7 M (68.2%)	74.8 M (63.7%)	60.2%	79.9%	78.2%
Obesity, BMI >30.0 kg/m^2§	78.4 M (34.6%)	41.6 M (35.6%)	32.5%	53.9%	44.8%
Blood cholesterol					
Prevalence, 2010					
Total cholesterol >200 mg/dL§	98.9 M (43.4%)	53.6 M (44.9%)	45.8%	40.7%	44.7%
Total cholesterol >240 mg/dL§	31.9 M (13.8%)	17.9 M (14.7%)	15.6%	11.7%	13.5%
LDL cholesterol >130 mg/dL§	71.0 M (31.1%)	35.8 M (30.0%)	29.3%	31.2%	30.4%
HDL cholesterol <40 mg/dL§	48.7 M (21.8%)	14.1 M (12.3%)	12.4%	10.2%	15.1%
HBP					
Prevalence, 2010§	77.9 M (33.0%)	40.7 M (32.2%)	30.7%	47.0%	28.8%
Mortality, 2009‖	61 762	34 094	26 201	6951	N/A
DM					
Prevalence, 2010					
Physician-diagnosed DM§	19.7 M (8.3%)	10.1 M (7.9%)	6.2%	15.4%	12.0%
Undiagnosed DM§	8.2 M (3.5%)	2.9 M (2.3%)	1.8%	2.9%	4.7%
Prediabetes§	87.3 M (38.2%)	33.6 M (30.5%)	30.0%	29.0%	31.9%
Incidence, diagnosed DM§	1.9 M	N/A	N/A	N/A	N/A
Mortality, 2009‖	68 705	33 651	25 908	6472	N/A
Total CVD					
Prevalence, 2010§	83.6 M (35.3%)	42.9 M (34.0%)	32.4%	48.9%	30.7%
Mortality, 2009‖	787 931	401 495	343 955	48 070	N/A
Stroke					
Prevalence, 2010§	6.8 M (2.8%)	3.8 M (3.0%)	2.9%	4.7%	1.4%
New and recurrent strokes‖	795.0 K	425.0 K	365.0 K	60.0 K	N/A
Mortality, 2009‖	128 842	76 769	65 574	8916	N/A
CHD					
Prevalence, CHD, 2010§	15.4 M (6.4%)	6.6 M (5.1%)	4.6%	7.1%	5.3%
Prevalence, MI, 2010§	7.6 M (2.9%)	2.6 M (1.7%)	1.5%	2.3%	1.7%
Prevalence, AP, 2010§	7.8 M (3.2%)	4.1 M (3.2%)	2.8%	5.4%	3.3%
New and recurrent CHD¶#	915.0 K	380.0 K	325.0 K	60.0 K	N/A
New and recurrent MI#	715.0 K	305.0 K	N/A	N/A	N/A
Incidence, AP (stable angina)**	500.0 K	180.0 K	N/A	N/A	N/A
Mortality, 2009, CHD‖	386 324	176 255	152 785	19 470	N/A
Mortality, 2009, MI‖	125 464	56 650	48 802	6567	N/A
HF					
Prevalence, 2010§	5.1 M (2.1%)	2.4 M (1.8%)	1.8%	3.0%	1.1%
Mortality, 2009‖	56 410	32 847	29 372	2987	N/A

CVD indicates cardiovascular disease; M, millions; PA, physical activity; LDL, low-density lipoprotein; HDL, high-density lipoprotein; BMI, body mass index; HBP, high blood pressure; N/A, data not available; DM, diabetes mellitus; K, thousands; CHD, coronary heart disease (includes heart attack, angina pectoris chest pain, or both); MI, myocardial infarction (heart attack); AP, angina pectoris (chest pain); and HF, heart failure.

*Age >18 years (National Health Interview Survey).

†All Hispanic (National Health Interview Survey)

‡Met 2008 full Federal PA guidelines for adults.

§Age >20 years.

‖All ages.

¶New and recurrent MI and fatal CHD.

#Age >35 years.

**Age >45 years.

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Table 24-3. Race/Ethnicity and CVD: At-a-Glance Table

Diseases and Risk Factors	Both Sexes	Whites		Blacks		Mexican Americans		Hispanics/ Latinos		Asians: Both Sexes	American Indian/ Alaska Native: Both Sexes
		Males	Females	Males	Females	Males	Females	Males	Females		
Smoking											
Prevalence, 2011*	43.8 M (19.0%)	22.8%	19.7%	23.3%	15.1%	12.3%		16.2%	8.3%	9.6%	26.7%
PA†											
Prevalence, 2011*	21.0%	21.7%		17.8%		15.4%		15.4%		16.7%	17.0%
Overweight and obesity											
Prevalence, 2010											
Overweight and obesity, BMI >25.0 kg/m²‡	154.7 M (68.2%)	73.1%	60.2%	68.7%	79.9%	81.3%	78.2%	N/A	N/A	N/A	N/A
Overweight and obesity, BMI >30.0 kg/m²‡	78.4 M (34.6%)	33.8%	32.5%	37.9%	53.9%	36.0%	44.8%	N/A	N/A	N/A	N/A
Blood cholesterol											
Prevalence, 2010											
Total cholesterol >200 mg/dL‡	98.9 M (43.4%)	40.5%	45.8%	38.6%	40.7%	48.1%	44.7%	N/A	N/A	N/A	N/A
Total cholesterol >240 mg/dL‡	31.9 M (13.8%)	12.3%	15.6%	10.8%	11.7%	15.2%	13.5%	N/A	N/A	N/A	N/A
LDL cholesterol >130 mg/dL‡	71.0 M (31.1%)	30.1%	29.3%	33.1%	31.2%	39.9%	30.4%	N/A	N/A	N/A	N/A
HDL cholesterol <40 mg/dL‡	48.7 M (21.8%)	33.1%	12.4%	20.3%	10.2%	34.2%	15.1%	N/A	N/A	N/A	N/A
HBP											
Prevalence, 2010‡	77.9 M (33.0%)	33.4%	30.7%	42.6%	47.0%	30.1%	28.8%	22.2%*		18.7*	25.8%*
Mortality, 2009§	61 762	20 286	26 201	6574	6951	N/A	N/A	N/A	N/A	N/A	N/A
DM											
Prevalence, 2010											
Physician-diagnosed DM‡	19.7 M (8.3%)	7.7%	6.2%	13.5%	15.4%	11.4%	12.0%	N/A	N/A	N/A	N/A
Undiagnosed DM‡	8.2 M (3.5%)	4.5%	1.8%	4.8%	2.9%	6.6%	4.7%	N/A	N/A	N/A	N/A
Prediabetes‡	87.3 M (38.2%)	47.7%	30.0%	35.7%	29.0%	47.0%	31.9%	N/A	N/A	N/A	N/A
Incidence, diagnosed DM‡	1.9 M	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, 2009§	68 705	28 205	25 908	5488	6472	N/A	N/A	N/A	N/A	N/A	N/A
Total CVD											
Prevalence, 2010‡	83.6 M (35.3%)	36.6%	32.4%	44.4%	48.9%	33.4%	30.7%	N/A	N/A	N/A	N/A
Mortality, 2009§	787 931	329 565	343 955	46 334	48 070	N/A	N/A	N/A	N/A	N/A	N/A
Stroke											
Prevalence, 2010‡	6.8 M (2.8%)	2.4%	2.9%	4.3%	4.7%	2.3%	1.4%	2.8%*		2.7%*	4.6%‖
New and recurrent strokes§	795.0 K	325.0 K	365.0 K	45.0 K	60.0 K	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, 2009§	128 842	43 190	65 574	6962	8916	N/A	N/A	N/A	N/A	N/A	N/A
CHD											
Prevalence, CHD, 2010‡	15.4 M (6.4%)	8.2%	4.6%	6.8%	7.1%	6.7%	5.3%	N/A	N/A	N/A	N/A
Prevalence, MI, 2010‡	7.6 M (2.9%)	4.4%	1.5%	3.9%	2.3%	3.6%	1.7%	N/A	N/A	N/A	N/A
Prevalence, AP, 2010‡	7.8 M (3.2%)	3.3%	2.8%	2.4%	5.4%	3.4%	3.3%	N/A	N/A	N/A	N/A
New and recurrent CHD¶#	915.0 K	465.0 K	325.0 K	65.0 K	60.0 K	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, CHD, 2009§	386 324	183 453	152 785	21 051	19 470	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, MI, 2009§	125 464	60 316	48 802	6717	6567	N/A	N/A	N/A	N/A	N/A	N/A
HF											
Prevalence, 2010‡	5.1 M (2.1%)	2.5%	1.8%	4.1%	3.0%	1.9%	1.1%	N/A	N/A	N/A	N/A
Mortality, 2009§	56 410	20 815	29 372	2341	2987	N/A	N/A	N/A	N/A	N/A	N/A

CVD indicates cardiovascular disease; M, millions; PA, physical activity; N/A, data not available; LDL, low-density lipoprotein; HDL, high-density lipoprotein; BMI, body mass index; HBP, high blood pressure; DM, diabetes mellitus; K, thousands; CHD, coronary heart disease (includes heart attack, angina pectoris chest pain, or both); MI, myocardial infarction (heart attack); AP, angina pectoris (chest pain); and HF, heart failure.

*Age >18 years (National Health Interview Survey).

†Met 2008 full Federal PA guidelines for adults.

‡Age >20 years.

§All ages.

‖Figure not considered reliable.

¶New and recurrent MI and fatal CHD.

#Age >35 years.

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Table 24-4. Children, Youth, and CVD: At-a-Glance Table

				NH Whites		NH Blacks		Mexican Americans	
Diseases and Risk Factors	Both Sexes	Total Males	Total Females	Males	Females	Males	Females	Males	Females
Smoking, %									
High school students, grades 9–12									
Current cigarette smoking, 2011	18.1	19.9	16.1	21.5	18.9	13.7	7.4	19.5*	15.2*
Current cigar smoking, 2011	13.1	17.8	8.0	19.0	7.5	15.1	8.5	17.2*	9.1*
PA†									
Prevalence, grades 9–12, 2011‡									
Met currently recommended levels of PA, %	49.5	59.9	38.5	62.1	42.6	57.1	31.9	57.1*	33.0*
Overweight and obesity									
Prevalence, 2010									
Children and adolescents, ages 2–19 y, overweight or obese	23.9 M (31.8%)	12.7 M (33.0%)	11.2 M (30.4%)	30.1%	25.6%	36.9%	41.3%	40.5%	38.2%
Children and adolescents, age 2–19 y, obese‡	12.7 M (16.9%)	7.2 M (18.6%)	5.5 M (15.0%)	16.1%	11.7%	24.3%	24.3%	24.0%	18.2%
Blood cholesterol, mg/dL, 2010									
Mean total cholesterol									
Ages 4–11 y	161.9	162.3	161.5	160.9	161.6	165.2	157.9	159.6	160.7
Ages 12–19 y	158.2	156.1	160.3	156.8	161.1	154.1	160.6	157.8	158.0
Mean HDL cholesterol									
Ages 4–11 y	53.6	55.1	51.9	53.9	51.4	59.9	55.3	53.5	50.5
Ages 12–19 y	51.4	49.2	53.6	48.4	53.0	53.9	55.4	47.5	53.3
Mean LDL cholesterol									
Ages 12–19 y	89.5	88.6	90.5	90.4	90.9	85.8	91.8	90.6	87.1
Congenital cardiovascular defects									
Mortality, 2009§	3189	1754	1435	1370	1086	304	268	N/A	N/A

CVD indicates cardiovascular disease; NH, non-Hispanic; PA, physical activity; HDL, high-density lipoprotein; LDL, low-density lipoprotein; M, millions; and N/A, data not available. Overweight indicates a body mass index in the 95th percentile of the Centers for Disease Control and Prevention 2000 growth chart.

*Hispanic.

†Regular leisure-time PA.

‡Eaton DK, Kann L, Kinchen S, Shanklin S, Flint KH, Hawkins J, Harris WA, Lowry R, McManus T, Chyen D, Whittle L, Lim C, Wechsler H; Centers for Disease Control and Prevention. Youth risk behavior surveillance: United States, 2011. *MMWR Surveill Summ*. 2012;61:1–162.

§All ages.

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25. Glossary

- *Age-adjusted rates*—Used mainly to compare the rates of ≥2 communities or population groups or the nation as a whole over time. The American Heart Association (AHA) uses a standard population (2000), so these rates are not affected by changes or differences in the age composition of the population. Unless otherwise noted, all death rates in this publication are age adjusted per 100 000 population and are based on underlying cause of death.
- *Agency for Healthcare Research and Quality*—A part of the US Department of Health and Human Services, this is the lead agency charged with supporting research designed to improve the quality of health care, reduce the cost of health care, improve patient safety, decrease the number of medical errors, and broaden access to essential services. The Agency for Healthcare Research and Quality sponsors and conducts research that provides evidence-based information on healthcare outcomes, quality, cost, use, and access. The information helps healthcare decision makers (patients, clinicians, health system leaders, and policy makers) make more informed decisions and improve the quality of healthcare services. The Agency for Healthcare Research and Quality conducts the Medical Expenditure Panel Survey (MEPS; ongoing).
- *Bacterial endocarditis*—An infection of the heart's inner lining (endocardium) or of the heart valves. The bacteria that most often cause endocarditis are streptococci, staphylococci, and enterococci.
- *Body mass index (BMI)*—A mathematical formula to assess body weight relative to height. The measure correlates highly with body fat. It is calculated as weight in kilograms divided by the square of the height in meters (kg/m^2).
- *Centers for Disease Control and Prevention/National Center for Health Statistics (CDC/NCHS)*—CDC is an agency within the US Department of Health and Human Services. The CDC conducts the Behavioral Risk Factor Surveillance System (BRFSS), an ongoing survey. The CDC/NCHS conducts or has conducted these surveys (among others):
  - National Health Examination Survey (NHES I, 1960–1962; NHES II, 1963–1965; NHES III, 1966–1970)
  - National Health and Nutrition Examination Survey I (NHANES I; 1971–1975)
  - National Health and Nutrition Examination Survey II (NHANES II; 1976–1980)
  - National Health and Nutrition Examination Survey III (NHANES III; 1988–1994)
  - National Health and Nutrition Examination Survey (NHANES; 1999 to …) (ongoing)
  - National Health Interview Survey (NHIS) (ongoing)
  - National Hospital Discharge Survey (NHDS) (1965–2010)
  - National Ambulatory Medical Care Survey (NAMCS) (ongoing)
  - National Hospital Ambulatory Medical Care Survey (NHAMCS) (ongoing)
  - National Nursing Home Survey (periodic)
  - National Home and Hospice Care Survey (periodic)
- *Centers for Medicare & Medicaid Services, formerly Health Care Financing Administration*—The federal agency that administers the Medicare, Medicaid, and Child Health Insurance programs.
- *Comparability ratio*—Provided by the NCHS to allow time-trend analysis from one *International Classification of Diseases* (ICD) revision to another. It compensates for the "shifting" of deaths from one causal code number to another. Its application to mortality based on one ICD revision means that mortality is "comparability modified" to be more comparable to mortality coded to the other ICD revision.
- *Coronary heart disease (CHD) (ICD-10 codes I20–I25)*—This category includes acute myocardial infarction (I21–I22), other acute ischemic (coronary) heart disease (I24), angina pectoris (I20), atherosclerotic cardiovascular disease (I25.0), and all other forms of chronic ischemic CHD (I25.1–I25.9).
- *Death rate*—The relative frequency with which death occurs within some specified interval of time in a population. National death rates are computed per 100 000 population. Dividing the total number of deaths by the total population gives a crude death rate for the total population. Rates calculated within specific subgroups, such as age-specific or sex-specific rates, are often more meaningful and informative. They allow well-defined subgroups of the total population to be examined. Unless otherwise stated, all death rates in this publication are age adjusted and are per 100 000 population.
- *Diseases of the circulatory system (ICD codes I00-I99)*—Included as part of what the AHA calls "cardiovascular disease." ("Total cardiovascular disease" in this Glossary.)
- *Diseases of the heart*—Classification the NCHS uses in compiling the leading causes of death. Includes acute rheumatic fever/chronic rheumatic heart diseases (I00–I09), hypertensive heart disease (I11), hypertensive heart and renal disease (I13), CHD (I20-I25), pulmonary heart disease and diseases of pulmonary circulation (I26–I28), heart failure (I50), and other forms of heart disease (I29–I49, I50.1–I51). "Diseases of the heart" are not equivalent to "total cardiovascular disease," which the AHA prefers to use to describe the leading causes of death.
- *Health Care Financing Administration*—See Centers for Medicare & Medicaid Services.
- *Hispanic origin*—In US government statistics, "Hispanic" includes people who trace their ancestry to Mexico, Puerto Rico, Cuba, Spain, the Spanish-speaking countries of Central or South America, the Dominican Republic, or other Spanish cultures, regardless of race. It does not include people from Brazil, Guyana, Suriname, Trinidad, Belize, or Portugal, because Spanish is not the first language in those countries. Most of the data in this update are for Mexican Americans or Mexicans, as reported by government agencies or specific studies. In many cases, data for all Hispanics are more difficult to obtain.
- *Hospital discharges*—The number of inpatients (including newborn infants) discharged from short-stay hospitals for whom some type of disease was the first-listed diagnosis. Discharges include those discharged alive, dead, or "status unknown."
- *International Classification of Diseases (ICD) codes*—A classification system in standard use in the United States. The *International Classification of Diseases* is published by the World Health Organization. This system is reviewed

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and revised approximately every 10 to 20 years to ensure its continued flexibility and feasibility. The 10th revision (ICD-10) began with the release of 1999 final mortality data. The ICD revisions can cause considerable change in the number of deaths reported for a given disease. The NCHS provides "comparability ratios" to compensate for the "shifting" of deaths from one ICD code to another. To compare the number or rate of deaths with that of an earlier year, the "comparability-modified" number or rate is used.

- *Incidence*—An estimate of the number of new cases of a disease that develop in a population, usually in a 1-year period. For some statistics, new and recurrent attacks, or cases, are combined. The incidence of a specific disease is estimated by multiplying the incidence rates reported in community- or hospital-based studies by the US population. The rates in this report change only when new data are available; they are not computed annually.
- *Major cardiovascular diseases*—Disease classification commonly reported by the NCHS; represents ICD codes I00 to I78. The AHA does not use "major cardiovascular diseases" for any calculations. See "Total cardiovascular disease" in this Glossary.
- *Metabolic syndrome*—The metabolic syndrome is defined* as the presence of any 3 of the following 5 diagnostic measures: Elevated waist circumference (≥102 cm in men or ≥88 cm in women), elevated triglycerides (≥150 mg/dL [1.7 mmol/L] or drug treatment for elevated triglycerides), reduced high-density lipoprotein (HDL) cholesterol (<40 mg/dL [0.9 mmol/L] in men, <50 mg/dL [1.1 mmol/L] in women, or drug treatment for reduced HDL cholesterol), elevated blood pressure (≥130 mm Hg systolic blood pressure, ≥85 mm Hg diastolic blood pressure, or drug treatment for hypertension), and elevated fasting glucose (≥100 mg/dL or drug treatment for elevated glucose).
- *Morbidity*—Incidence and prevalence rates are both measures of morbidity (ie, measures of various effects of disease on a population).
- *Mortality*—Mortality data for states can be obtained from the NCHS Web site (http://cdc.gov/nchs/), by direct communication with the CDC/NCHS, or from the AHA on request. The total number of deaths attributable to a given disease in a population during a specific interval of time, usually a year, are reported. These data are compiled from death certificates and sent by state health agencies to the NCHS. The process of verifying and tabulating the data takes ≈2 years.
- *National Heart, Lung, and Blood Institute (NHLBI)*—An institute in the National Institutes of Health in the US Department of Health and Human Services. The NHLBI conducts such studies as the following:
  - — Framingham Heart Study (FHS; 1948 to …) (ongoing)
  - — Honolulu Heart Program (HHP) (1965–1997)
  - — Cardiovascular Health Study (CHS; 1988 to …) (ongoing)
  - — Atherosclerosis Risk in Communities (ARIC) study (1985 to …) (ongoing)
  - — Strong Heart Study (SHS) (1989–1992, 1991–1998)
  - — The NHLBI also published reports of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure and the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III).
- *National Institute of Neurological Disorders and Stroke (NINDS)*—An institute in the National Institutes of Health of the US Department of Health and Human Services. The NINDS sponsors and conducts research studies such as these:
  - — Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS)
  - — Rochester (Minnesota) Stroke Epidemiology Project
  - — Northern Manhattan Study (NOMAS)
  - — Brain Attack Surveillance in Corpus Christi (BASIC) Project
- *Physical activity*—Any bodily movement produced by the contraction of skeletal muscle that increases energy expenditure above a basal level.
- *Physical fitness*—The ability to perform daily tasks with vigor and alertness, without undue fatigue, and with ample energy to enjoy leisure-time pursuits and respond to emergencies. Physical fitness includes a number of components consisting of cardiorespiratory endurance (aerobic power), skeletal muscle endurance, skeletal muscle strength, skeletal muscle power, flexibility, balance, speed of movement, reaction time, and body composition.
- *Prevalence*—An estimate of the total number of cases of a disease existing in a population during a specified period. Prevalence is sometimes expressed as a percentage of population. Rates for specific diseases are calculated from periodic health examination surveys that government agencies conduct. Annual changes in prevalence as reported in this statistical update reflect changes in the population size. Changes in rates can be evaluated only by comparing prevalence rates estimated from surveys conducted in different years. Note: In the data tables, which are located in the different disease and risk factor categories, if the percentages shown are age adjusted, they will not add to the total.
- *Race and Hispanic origin*—Race and Hispanic origin are reported separately on death certificates. In this publication, unless otherwise specified, deaths of people of Hispanic origin are included in the totals for whites, blacks, American Indians or Alaska Natives, and Asian or Pacific Islanders according to the race listed on the decedent's death certificate. Data for Hispanic people include all people of Hispanic origin of any race. See "Hispanic origin" in this Glossary.
- *Stroke (ICD-10 codes I60–I69)*—This category includes subarachnoid hemorrhage (I60); intracerebral hemorrhage (I61); other nontraumatic intracranial hemorrhage (I62); cerebral infarction (I63); stroke, not specified as hemorrhage or infarction (I64); occlusion and stenosis of precerebral arteries not resulting in cerebral infarction (I65); occlusion and stenosis of cerebral arteries not resulting in cerebral infarction (I66); other cerebrovascular diseases (I67);

*According to criteria established by the American Heart Association/National Heart, Lung, and Blood Institute and published in *Circulation* (*Circulation*. 2005;112:2735–2752).

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cerebrovascular disorders in diseases classified elsewhere (I68); and sequelae of cerebrovascular disease (I69).

- *Total cardiovascular disease (ICD-10 codes I00–I99, Q20–Q28)*—This category includes rheumatic fever/rheumatic heart disease (I00–I09); hypertensive diseases (I10–I15); ischemic (coronary) heart disease (I20–I25); pulmonary heart disease and diseases of pulmonary circulation (I26–I28); other forms of heart disease (I30–I52); cerebrovascular disease (stroke) (I60–I69); atherosclerosis (I70); other diseases of arteries, arterioles, and capillaries (I71–I79); diseases of veins, lymphatics, and lymph nodes not classified elsewhere (I80–I89); and other and unspecified disorders of the circulatory system (I95–I99). When data are available, we include congenital cardiovascular defects (Q20–Q28).
- *Underlying cause of death or any-mention cause of death*—These terms are used by the NCHS when defining mortality. Underlying cause of death is defined by the World Health Organization as "the disease or injury which initiated the train of events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury." Contributing cause of death would be any other disease or condition that the decedent may also have had and that was reported on the death certificate.

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