Pathology of Sudden Cardiac Death
An Illustrated Guide

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I have chosen to dedicate this book to my wife, Marjorie B. Edwards, and my daughter-in-law, Dr. Terri L. Edwards, for their many encouraging words.
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What do you do if you are a child of the Depression, have studied and collected almost 30,000 cardiac specimens, and are 94 years old? You write a book to share your experience and knowledge – naturally.

This book is largely the work of my father, Jesse E. Edwards. He defined many of the cardiac conditions that we in clinical cardiology deal with everyday, and in doing so he defined the modern specialty of cardiac pathology. His collection of cardiac specimens has become a worldwide teaching and research tool. Each specimen was carefully dissected, clearly described, photographed, and cataloged. Each specimen held a secret, a lesson for those willing to learn. In discovering the secret of each specimen, physicians and scientists, pathologists, radiologists, cardiologists, and surgeons learned not about the dead but more about the living. In my father’s lab, each specimen came alive with the secrets of life and an understanding of disease.

And why does it matter that my father grew up during the Great Depression of the 1920s and ’30s? You see, children of the Depression learned not to waste anything – not a piece of string, not a moment of time, and certainly not the lessons for life itself. For each specimen in the collection was the gift of a family that consented to an autopsy with the hope that something could be learned. My father has looked at each specimen as something special; and whether it was collected last month or 60 years ago, there are still more lessons to be learned, secrets to unravel, and a new generation of physicians to educate. For my father, each specimen is in itself a teachable moment.

This project began with the goal of providing a new generation of cardiac specialists with a look at the many processes resulting in sudden cardiac death. Sudden death is now often, although incorrectly, equated with arrhythmic death. As this book illustrates, a host of derangements can result in cardiac death. The focus on pathology provides clinicians the opportunity to see first hand the primary derangements that we often see nowadays only as reflected sound waves, magnetic images, or lumenograms. Sometimes we fail to see anything until a medical examiner’s report reminds us of the tenuous nature of life itself. As autopsy rates decline, the opportunity to see first hand and understand disease from the cellular, tissue, or organ perspective becomes a rare opportunity. If a picture is worth a thousand words, this book with its more than 800 illustrations provides the reader with an encyclopedic appreciation for the many faces of cardiac disease and the many causes of sudden death. One cannot help but alter one’s clinical practice after seeing the spectrum of pathology resulting in sudden death.

This book in some ways represents the catalog of a collection that will likely never be repeated. While the pathologic processes that we review in this book range from common to unique, the opportunity to see them “in the flesh” is limited.

At 94 years of age most of us are not writing books, but my father is a truly unique individual. His career has spanned the days from before the discovery of antibiotics to the modern-day promise of genomic therapy. He himself has faced many of the challenges of advancing age and benefited from the advances of modern medicine. Through it all, he has maintained a steadfast determination to continue to share his special knowledge and collection so that this precious resource does not go to
waste and the sacred promise that somebody will learn from each autopsy is carried forward everyday.

We are delighted to provide this atlas with the expectation that it will provide new insights into the processes of cardiovascular disease, the care of the patient, and the prevention of premature cardiac death.

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Foreword

Sudden death has plagued mankind from time immemorial. One of the earliest descriptions of a sudden death event was reported in Froissart’s *Chronicles* in the 14th century, with the first autopsy report of sudden cardiac death ascribed to Leonardo de Vinci in the 15th century. In the 21st century, the Center for Disease Control reported that sudden cardiac death accounted for 63 percent of approximately 730,000 cardiac deaths in calendar year 1999. Sudden death has reached epidemic proportions. During the past few decades the major publications on sudden death have emphasized cardiac risk stratification, electrophysiological mechanisms, and drug- and device-related interventions. Somehow the anatomic and pathologic substrates for sudden death, both cardiac and vascular causes, have been neglected as investigators have focused on disordered electrical function of the heart as the *sine qua non* of this disorder. Fortunately, the link between altered structure and function has been reinvigorated with the publication of Edwards’ *Pathology of Sudden Cardiac Death*.

Dr. Jesse Edwards is the pre-eminent cardiovascular pathologist whose contributions span the second half of the 20th century and the beginning of the 21st century. Dr. Edwards is unique in his ability to combine text and detailed photographs of anatomic specimens in case-oriented, clinical-pathological presentations that provide fundamental insight and understanding into the structural and functional alterations of the cardiovascular system involving a spectrum of congenital and acquired cardiac diseases. His first cardiovascular book, *An Atlas of Congenital Anomalies of the Heart and Great Vessels*, has remained a classic ever since it was published in 1954. Now, more than 50 years later, Dr. Edwards provides in his latest atlas a compendium of pathologic, physiologic, electrocardiographic, and clinical findings with photographs and text that offer a broad integrated view into the causes of sudden death.

My professional interaction with Dr. Edwards began in 1979 when he, together with Drs. Frank Marcus, Leonard Cobb, and Lewis Kuller, participated as members of the Mortality Committee in our Multicenter Post-infarction Research Study that investigated the factors associated with sudden and non-sudden death during long-term follow-up after survival from acute myocardial infarction. I sat in on several deliberations of this Committee and appreciated Dr. Edwards’ ability to integrate clinical data, functional derangements, and autopsy findings, when available, in the identification of the cause of death. The rationale used in the final mortality classification of each patient served the study well. The findings from that study provided the foundation for several landmark drug and device trials investigating therapies to prevent sudden cardiac death.

It has been said that the safe passage into a harbor is marked by sunken ships. Dr. Edwards’ *Pathology of Sudden Cardiac Death* is an educational masterpiece directed to pathologists, clinical practitioners, and investigative physicians who are responsible for charting the safe passage of patients through the harbor of life.

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By far, cardiovascular disease is the leading primary or contributing cause of death in Western society. Cardiovascular death may be sudden and unexpected or may result from known or suspected disease. This book will review and illustrate the multiplicity of mechanisms that can result in cardiovascular collapse and death.

We have chosen to review the topic of cardiac death in the format of an illustrated guide or atlas since we believe the visual picture provides a lasting impression of the fundamental disease states. The cases reviewed in this book span the spectrum from common and ordinary diseases to rare and unusual conditions. By seeing the different states juxtaposed to each other, it is our hope that the reader will appreciate the many conditions that either directly cause sudden cardiac death or provide the substrate for the development of future lethal cardiac conditions.

It is our hope that the reader can quickly assimilate the many different faces of sudden death presented here. As autopsy rates continue to decline, it is our fear that future generations will not have the opportunity to see first hand the fundamental derangements which lead to cardiac disease and mortality. We have attempted to distill many decades of experience into this volume of work in an effort to insure that the experience is passed on to the next generation of scientists and practitioner.

When considering the many causes of cardiovascular death, we will follow an anatomic classification of the primary disorder. The broad categories include the following: coronary artery disease, including atherosclerotic and nonatherosclerotic diseases; myocardial disease; diseases of the conduction system; hypertension; valvular heart disease; metabolic and infiltrative disorders; tumors; disorders of the great vessels, including pulmonary hypertension; pericardial disease; and diseases of multi-organ systems. The illustrative cases in this text come from the autopsies of patients who died in hospital, or from unexplained death.
Acknowledgments

The photographs in this atlas come predominately from specimens that have been referred from many different pathology laboratories to the Jesse E. Edwards Registry of Cardiovascular Disease at the United Hospital in St. Paul, Minnesota. We wish to thank the families who consented to the postmortem examination for their loved ones in the hope that such an examination could be of service to future generations. We hope this book will be one tangible example of the continuing contributions their loved ones made to the advancement of medicine. The list of physicians who refereed specimens to the Registry is too numerous to mention, but the authors profoundly thank each person who referred material to the Registry.

The authors are grateful to those individuals who processed specimens contributed to the Registry. Among these are Dr. Jack Titus, former director of the Jesse E. Edwards Registry, and Dr. Shannon M. Mackey-Bojack, who is currently leading the efforts of the Registry. Others who contributed significantly to the processing of new specimens include Dr. Karen Kelly, Dr. Susan J. Roe, and Mr. Richard Dykoski.

Mrs. Julie Rooke edited the manuscript to ensure there was a logical order to the many pieces of this project. Her dedication and insightful help were invaluable. Mrs. Jane Jungbauer, former office supervisor of the Registry, participated in the development of the system for specimen and associated material filing and retrieval. Ms. Judith Anderson, current office supervisor, managed the large volume of illustrations and clinical records reviewed for this text. Her knowledge and patience have been deeply appreciated. Ms. Tiffany Dahlberg and Ms. Krista Whitlef also provided outstanding secretarial support for this project.

We wish to acknowledge the support of and encouragement by Mr. Steven Korn of Blackwell Publishing. His enthusiasm for this project allowed it to become a reality. I gratefully acknowledge the expert project management and editorial services provided by Komila Bhat and Fiona Pattison at Blackwell Publishing.

The authors were fortunate for the availability and willingness of Mr. Jack Baldwin to apply his expertise in helping coordinate the development of the manuscript and illustrations, as well as his unfailing good humor, constant support, and guidance. His participation added a pearl to the creation of this manuscript.

I would like to thank Dr. Arthur J. Moss, an admired friend for many years, for providing his thoughtful foreword to the atlas and for his many contributions to the field of cardiology and the prevention of sudden cardiac death.

I wish to acknowledge the specific talents and expertise of Drs. Leonard Schloff, Raymond Gibbons, Allen Brown and Mr. Brad King. Without their help this book would not have been possible. United Hospital Foundation of St. Paul and Mayo Foundation provided the support, environment and necessary infrastructure to allow this project to proceed.

My daughter, Mrs. Ellen Villa, an editor at the Washington Post, provided guidance with the development of this manuscript throughout the process.

I wish to thank the following physicians who made suggestions or corrections throughout the compilation of this manuscript. They are Dr. Kenneth Jue, Dr. Milton Hurwitz, Dr. Zev Vlodaver, and Dr. Howard Burchell. Mr. Robert Benassi contributed to this publication by having prepared numerous line drawings that are frequently used in this atlas. He is a valued friend and colleague.

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Atherosclerotic heart disease and its complications continue to be the leading causes of sudden cardiac death in Western society. Cross sections of normal and atherosclerotic coronary arteries are shown in this chapter. The atherosclerotic lesions tend to be eccentric and are made up of accumulations of lipids and fibrous tissue, as well as inflammatory cells. Plaque rupture or localized hemorrhage into the atheroma facing the lumen is the usual precursor to coronary thrombosis. The fibrous tissue separating the site of hemorrhage from the lumen may perforate and lead to thrombosis on the luminal side of the lesion. Once the clot has formed, the thrombus may occlude the lumen of the artery. The thrombus may, with time, undergo organization as it becomes replaced by fibrous tissue and, ultimately, the lumen may become recanalized.

While atherosclerosis is by far the dominant cause of coronary obstruction, there are less common situations that may cause obstruction of the coronary arteries. Such lesions are primarily of the aorta, as, for example, narrowing of a coronary artery and its ostia as it lies adjacent to the beginning of a proximal aortic dissection. Also, in certain cases of trauma to the chest, a resulting laceration may cause obstruction of a coronary artery.

Pathology of coronary atherosclerosis

Cross sections of coronary arteries afflicted with atherosclerosis, two types of lesions are seen: one is purely fibrous, and the other is a pairing of lipid pools and walling fibrous tissue (together these form a “parite”) [1]. The combination of collagen and lipid together may be termed collipid. In established atherosclerosis, an episodic character to the development of the atheroma is strongly supported. Intimal hemorrhages rarely narrow the lumen but may underlie formation of occlusive thrombi.

Pathologically, calcification in atherosclerosis is a sign of age of the lesion but, by itself, has no direct bearing on the severity of luminal obstruction. The location and shape of the arterial lumen varies in segments with severe atherosclerosis. The lumen may be central or eccentric. In about one quarter of obstructed segments, the lumen appears eccentric and slit-like in prepared histologic sections. Study of distribution of atherosclerotic lesions in all segments of the coronary tree indicates that the segment of the right coronary artery between the marginal vessel and the posterior descending artery is the most commonly involved by atherosclerosis. Second to this site is the proximal half of the left anterior descending coronary artery.

Atherosclerotic aneurysm of coronary arteries

Aneurysm formation affecting the coronary arteries may be caused by coronary atherosclerosis or by an active inflammation of the coronary arteries. The latter subject will be covered in Chapter 2. Aneurysms of coronary arteries secondary to atherosclerosis are characterized by an unusual thickening of the arterial intima of the arteries associated with dilatation of the media. The atherosclerotic aneurysm tends to be isolated and solitary, in contrast to inflammatory causes of aneurysms where there are frequently multiple aneurysms. The external width of the artery is greatly increased in coronary aneurysms, but the lumen is often very narrow. In classical atherosclerosis without aneurysm formation, the effect of the atheromatous deposits causes the lumen to be narrowed; in atherosclerotic aneurysms the lumen is also narrowed but, in contrast, the effect on the media is one of dilatation, and hence formation of the aneurysm.
**Sudden death as a first indication of coronary atherosclerosis**

Sudden cardiac death may be the first sign of coronary atherosclerosis. While some patients have experienced an emotional or physical traumatic event as a trigger, most cases of sudden death due to coronary artery disease do not have such a clearly inciting antecedent event, and sudden death during sleep is not unusual.

Lecomte and associates [2] reported on the autopsies in cases of sudden death occurring immediately after experiencing emotional stress. Forty-three cases were studied, which included 29 males and 14 females. In 20 cases death occurred during the stressful event; in the other 23 cases, death occurred within two hours of the event. Ninety percent of the patients had no known antecedent clinical history of cardiovascular disease. In spite of this, evidence of previous myocardial infarction was present in 92% of the patients with corresponding coronary atherosclerosis. Cardiomegaly was present in most of the subjects. Acute coronary thrombosis was found in only 8 of 43 cases, suggesting in this series that most of the subjects suffered an arrhythmic death, in the setting of prior silent myocardial infarction.

**Myocardial infarction**

In patients with coronary atherosclerotic disease, the major clinical risk relates to the development of acute myocardial infarction [3]. There are two frequently used categories of acute myocardial infarction: subendocardial (non-Q wave) infarction and transmural (Q wave) infarction. Subendocardial infarction is frequently associated with coronary atherosclerosis, but usually not acute coronary thrombosis. “Subendocardial” refers to the distribution of the infarction, which tends not to involve the full thickness of the myocardium. While early survival after subendocardial infarction is greater than that observed in transmural infarction, by one year, survival is similar in the two groups. Patients with prior myocardial scarring, transmural or subendocardial, are at risk of ventricular arrhythmias and sudden death. Low ejection fraction (less than 35%) dramatically compounds the risk of sudden death in patients with coronary artery disease.

The second category of acute myocardial infarction is the transmural (Q wave) infarct. The infarct tends to involve almost the full thickness of the involved segments of myocardium. Frequently, preserved myocardium is found near the endocardium of involved segments. Patients with transmural myocardial infarction have a higher risk of developing cardiogenic shock; this may be due to conduction disturbances or valvular malfunction (insufficiency of atrioventricular valves) or simply related to the mass of myocardium lost. In the early stages of acute myocardial infarction, certain pathologic changes may be observed. In early transmural infarction it is common that acute fibrinous pericarditis is seen pathologically as the visceral pericardium becomes inflamed and is covered by a layer of fibrin. Clinically, this pericarditis may present after a silent transmural myocardial infarction and be the only clinical sign of underlying coronary artery disease; the phenomenon is frequently followed by either organization of the fibrin with adhesions or, more commonly, complete healing of the process. Unusually, the fibrin of the associated pericarditis may organize with granulation tissue that may lead to hemorrhagic exudation in the pericardium, a condition that might be confused with rupture of the left ventricle [4]. Rarely the hemorrhagic pericarditis may progress to frank cardiac tamponade.

Histologically, in the early stages of infarction there may be seen the so-called contraction bands, areas of poor staining of the cytoplasm and disorganization. In about 24 hours the site of infarction may be characterized by interstitial leukocytes, namely neutrophils. Associated with the leukocytic infiltration, the infarct has a tendency for early disruption and fragmentation of the myocytes. By the end of the first week the affected myocytes have lost nuclei and are fragmented. The second week is characterized by further fragmentation and disappearance of myocytes. At the end of the first month, the process of postinfarctive removal of infarcted tissue may be nearly completed and scar formation continues.

The gross pictures of acute myocardial infarction reflect the age of the infarct. In the earliest stage there is no recognizable alteration. By one day, the infarcted tissue differs slightly from the normal. During the first week the muscle that is infarcted shows a progressively different quality than the normal areas. At about one week there is a depression
between the normal muscle and the adjacent infarct; this depression reflects the early removal of myocardial fibers, leaving only the stroma (capillaries and interstitial tissue). As time goes on, the width of the depression increases and the infarcted muscle yet to be removed is visible as a pale yellow zone. By one month most infarcts will show removal of the infarcted tissue, which will have become replaced by connective tissue. At first the connective tissue is pink and later, as collagen is added, the color becomes gray. At this stage the infarct may be called “healed” by scarring. At the level of the scar the myocardium is thinner than elsewhere.

Nonarhythmic complications of acute myocardial infarction
Beyond arrhythmic complications, patients with acute myocardial infarction may die from mechanical complications. This subject is well reviewed by Prieto and associates [3]. Large infarctions result in hypotension with both systolic and diastolic dysfunction resulting clinically in an increase in pulmonary capillary wedge pressures. Right ventricular infarction, as well as several forms of rupture of the heart, may lead to lethal nonarhythmic complications.

Rupture of the heart
Among patients who die from acute myocardial infarction there is a subset of those that develop rupture of the heart [5]. Rupture of the heart is not an unusual finding in medical examiner cases of sudden unexplained cardiac death. There are three anatomical locations for cardiac rupture. The most common type of rupture is that of the free wall, leading to a communication between the left ventricle and the pericardial space. Free wall rupture may be further subdivided according to location: anterior (area of left anterior descending coronary artery), lateral (territory of left circumflex coronary artery), and posterior (territory of right coronary artery or circumflex). The anterolateral region represents the most common location for free wall rupture. In cases of rupture of the free wall, the accumulation of blood in the pericardium occurs rapidly resulting in cardiac tamponade with sudden collapse and death. Early identification of impending rupture may prompt appropriate imaging and potential surgical intervention. Of all the cases of rupture of the heart, about 85% occur in the free wall. In other cases, rupture of the ventricular septum or left ventricular papillary muscles may occur. Rupture of the ventricular septum has been classified as occurring in either a simple or complex course between the two ventricles [6]. The simple course is characterized by a direct connection between the ventricles. The complex course involves a serpiginous connection between the right and left ventricle, making surgical repair difficult.

In rupture of a papillary muscle, usually multiple heads of a single papillary muscle rupture leading to severe mitral incompetence. Uncommonly, one or only several heads of the papillary muscles are involved in the rupture [7]. In some cases early replacement or repair of the mitral valve may be life saving. In those cases of rupture of the papillary muscle in which a restricted number of heads have been torn, surgical approaches may be delayed [5]. Trauma may cause papillary muscle rupture, as in a case described by Farmery and associates [8]. Additionally, with rupture of a papillary muscle, the laceration of the myocardium may go through the thickness of the left ventricular wall. In such a case [9], rupture of the papillary muscle leads to hemopericardium. Without infarction, blunt trauma such as a deceleration injury from a steering wheel may cause rupture of a left or right ventricular papillary muscle [10,11].

Varying states of healed myocardial infarction
The authors have decided to illustrate several cases of healed myocardial infarction. One group may be subdivided into healed myocardial infarction with either true or false aneurysm. True aneurysm involves an aneurysmal part of the left ventricle with continuity of the myocardium. In a false aneurysm the process is brought about by contained rupture at the site of myocardial infarction [12]. In such cases the site of rupture is devoid of myocardial tissue but instead is made up of scar tissue. The formation of a false aneurysm involves rupture of the left ventricle with retention of some of the hemorrhage by the surrounding pericardium and inflammatory tissue. In the classical case of false aneurysm there is a pocket of blood derived from the left ventricle held in place by the reaction, which may be called the wall of the false aneurysm. In some cases, the
wall of the false aneurysm may rupture leading to hemorrhage into the pericardium [13].

Among exceptional cases of false aneurysm is de Boer’s report [14], indicating a left ventricular false aneurysm complicating staphylococcal-isolated pericarditis. In a case reported by Lee and Spencer [15] a false aneurysm of the left ventricle was caused by a tumor. And in the case reported by Dubel and associates [16] a huge false aneurysm in the posterior wall of the left ventricle followed resection of a true aneurysm in the same location.

**Infarction of the right ventricle**

Right ventricular infarction has many ramifications. These depend, to some extent, upon the relative proportion of the right and left ventricles involved in the infarction. In the classical case, right ventricle contraction has been eliminated from contributing to the circulation. The right ventricle then acts in a nonpulsatile conduit between the right atrium and the pulmonary arteries. Generally, patients with right ventricular infarction are older than average and exhibit extensive coronary atherosclerosis [17,18].
Figure 1
(a) Epicardial coronary artery supplies subjacent myocardium. An epicardial coronary artery sends a branch, which penetrates the subjacent myocardium. This illustration portrays the source of blood for the myocardium. (b) Endarterectomy of atherosclerotic coronary artery as viewed in gross specimen.
Figure 2 Histology of atherosclerotic coronary arteries.
(a) A classical atherosclerotic lesion. The main substance is lipoid; this is covered by a fibrous cap. The lumen is eccentric and narrow. (b) Atherosclerosis is partly composed of lipoid and partly fibrous tissue. The lumen is narrowed and eccentric. (c) An eccentric atherosclerotic lesion, composed of pairs of fibrous tissue and lipoid. The lumen is markedly narrowed and eccentric. (d) Atherosclerotic lesion with early thrombosis. The fibrous cap over the atheroma has broken. Secondary thrombosis paves the way for ultimate luminal obstruction. (e) Atherosclerotic coronary artery. In the upper half there is a break in the fibrous cap beneath which is thrombus. (f) In the upper zone of the atheroma a break has resulted in a channel containing thrombotic material.
Figure 3 Coronary atherosclerosis, thrombosis, and recanalization.
(a) Atherosclerotic coronary artery. The lumen contains a recent thrombus. (b) In the upper region of the artery an old break is evident. Through this an organized thrombus in the lumen has communicated with the underlying substance of an atheroma. (c) Atherosclerotic coronary artery contains an organized thrombus. The level of this section is above the classical break in the fibrous cap. (d) The lumen contains an organized thrombus. The section of this preparation is away from the site of rupture of the fibrous cap. (e) Organized thrombus showing beginnings of recanalization. (f) Organized thrombus recanalized and represented by multiple channels.
Figure 4 Aneurysm of coronary arteries secondary to atherosclerosis.
Extensive atherosclerosis; the involved coronary artery is widened and associated with deposits of atheromatous material. The lumen of the artery is narrowed. (a) Aneurysmal right coronary artery. (b) Photomicrograph of aneurysmal part of right coronary artery. (c) Cross section of the coronary artery with widening of the wall and deposits of atheromatous material. The lumen (Lu) is marked by Narrowed. (d) Gross view of the involved coronary artery with an Anureysm (An) involving the proximal portion of the artery. Male, 61 years.
Reprinted with permission from Kalke and Edwards [157].
Figure 5 Pericardial exudates and pulmonary edema in acute myocardial infarction.
(a) External view of the epicardium in a case of recent myocardial infarction with sudden death showing fibrinous exudation. Male, 66 years. (b) Cross section of the ventricles revealing a large anterior acute myocardial infarct, surrounded by A which is fibrinous pericardial exudate. (c) Photomicrograph of pericardium in acute fibrinous pericarditis. Male, 66 years. (d) Photomicrograph of lung showing congestion and edema, with fluid in the alveolar spaces.
Figure 6 Examples of acute myocardial infarction.
(a) A large anteroseptal myocardial infarction (arrows). The zone of infarction is localized and may be classified as subendocardial. (b) Cross section of ventricular portion of heart showing discoloration of acute transmural inferior wall myocardial infarction (arrows) leading to sudden death. Female, 48 years. (c) Massive inferior myocardial infarction extending into the right ventricle. Specimen viewed in the following manner: inferior below; anterior above. Male, 54 years. (d) Anterior view of the heart showing extensive acute transmural myocardial infarction. Male, 72 years.
Figure 7 Further examples of acute transmural myocardial infarction.
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Figure 9 Healing stages of acute myocardial infarction.
(a) Interior of the left ventricle demonstrating myocardial scarring and endocardial thickening in a case of acute myocardial infarction about 2-3 weeks old. (b) Histologic section from a. The section shows a zone of removed myocardium from an infarct that occurred 2–3 weeks earlier sudden death. (c) Histologic appearance of acute myocardial infarction about 9 days old. Some elements of necrotic myocardium are evident; others have been removed. Female, 76 years. (d) Myocardial infarct about 3 months old showing loss of myocardial tissue with remaining vascular stroma.
Figure 10 Healed acute myocardial infarction.
(a) Healed transmural anteroseptal myocardial infarction showing thinning and scarring of the anterior wall. Male, 60 years. (b) Healed subendocardial anterior myocardial infarction. Male, 65 years. (c) Close-up view of healing anterior transmural myocardial infarction. (d) Close-up view of an old, healed myocardial infarction. The site of infarction is narrower than in the surrounding myocardium. The infarcted area is partly calcified. Male, 71 years.
Figure 11 Variations in healed myocardial infarction.
(a) Internal view of the left ventricle. Major ventricular dilatation and thinning of the left ventricular wall due to prior myocardial infarction and subsequent remodeling. (b) Healed circumferential myocardial infarction. There is marked left ventricular dilatation. (c) Healed posterolateral myocardial infarction of left ventricle showing marked thinning of the lateral wall of the left ventricle compared with the ventricular septum. (d) Healed lateral wall and septal infarction and acute inferior wall myocardial infarction with rupture of the left ventricle. Ventricular rupture in a case of previously healed myocardial infarction is unusual. Male, 61 years.