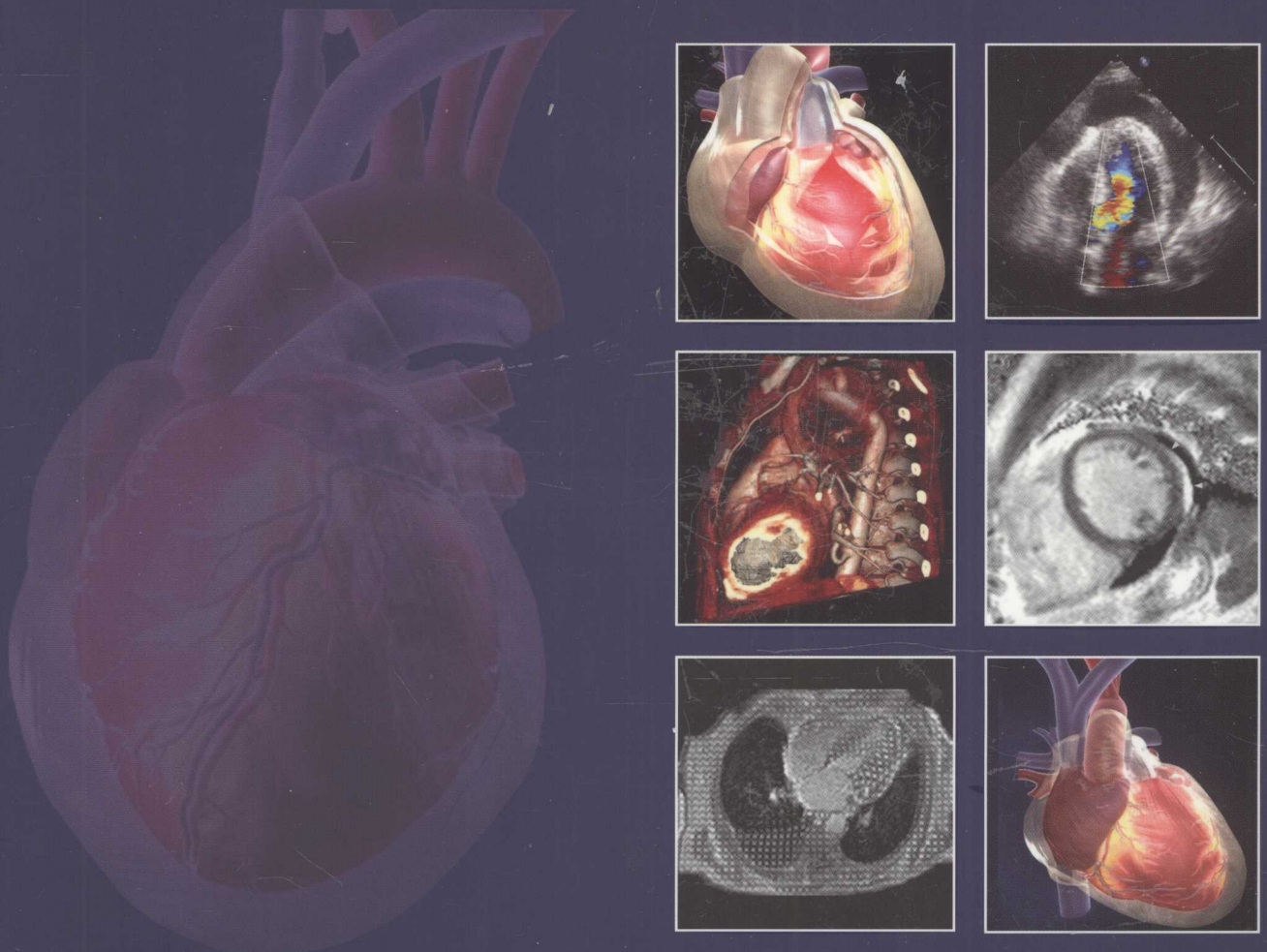
HUTCHISON

PERICARDIAL DISEASES

Clinical Diagnostic Imaging Atlas



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Pericardial Diseases

CLINICAL DIAGNOSTIC IMAGING ATLAS

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PERICARDIAL DISEASES: CLINICAL DIAGNOSTIC IMAGING ATLAS

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To Noel Keith and Cindy Hutchison, for the immeasurable gifts of love and time.

To Bob Chisholm—an inspired and inspiring physician, colleague, and friend, a man of insuppressible alacrity, integrity, and loyalty, and the best cath lab director an echo lab director could ever hope to work with.

Foreword

Diseases of the pericardium represent one of the most intriguing yet demanding areas in clinical cardiology. Understanding the pathophysiology of pericardial disorders, their diagnostic uncertainties, and the associated dilemmas in their treatment continue to challenge all who study the cardiovascular system, from students to experienced cardiologists. Dr. Stuart Hutchison's masterful atlas provides a comprehensive, balanced, and authoritative source covering the full spectrum of diseases of the pericardium. Although the primary focus of *Pericardial Diseases* is the practical application and interpretation of cardiovascular imaging techniques in patients with pericardial disorders, the atlas is also a clinically relevant resource covering the full spectrum of this field, including thoughtful discussions of epidemiology, clinical presentation, physical examination, diagnostic tools, and treatment. The imaging examples from 2-dimensional and Doppler echocardiography and the advanced fields of cardiac magnetic resonance and computed tomography are superbly presented. This scholarly work will be an essential resource for cardiovascular practitioners, cardiothoracic surgeons, researchers, and imaging subspecialists.

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Preface

Pericardial diseases provide ongoing diagnostic and treatment challenges to clinicians. They comprise a considerable breadth of disorders that range from acute to chronic, insidious to fulminant, benign to malignant, self-limited to progressive, symptomatic-only to life-threatening, limited to systemic, consistent to variable, and straightforward to complex. Pericardial diseases are similarly encountered by a wide range of physicians, particularly emergency physicians, cardiologists, cardiac surgeons, trauma surgeons, nephrologists, rheumatologists, immunologists, oncologists, intensivists, and others.

The evaluation of pericardial diseases entails a broad-based approach that begins at the bedside and draws heavily on imaging assessment as well as hemodynamic assessment. The pericardial compressive disorders of constriction and tamponade and their variants involve complex, incompletely established and fascinating interactions of cardiac and pulmonary physiology. Proficiency with the underlying pathophysiology assists immeasurably in the clinical, imaging, and hemodynamic assessment, especially in atypical cases.

The advent of contemporary imaging modalities has brought a new era to the assessment of pericardial diseases that complements the traditional and often historic character of pericardial diseases and rounds out the medical sphere of knowledge of pericardial diseases. As with many classic diseases, learning and discussing the surgical and pathological findings in cases encountered are invaluable in gaining understanding and proficiency. I am indebted to my clinical colleagues, and especially to my surgical and pathology colleagues, for their feedback, knowledge, insights, and willingness to discuss cases and their wonderful collegiality.

My motivation in developing this book and its companion DVD was to integrate contemporary cardiovascular imaging with the overall assessment of pericardial diseases and to emphasize the pathophysiologic basis of the clinical, imaging, and hemodynamic signs and consequences of pericardial diseases. The book provides both traditional chapter presentations of the different pericardial diseases and cases that have been chosen to illustrate significant aspects of the diseases, including the difficulties encountered. The DVD includes dynamic image files to complement the static images within the book.

Stuart J. Hutchison, MD

Acknowledgments

My sincere appreciation and gratitude go to Inga Tomas; Simon Abrahmson, MD; Natasha Andjelkovic, PhD; Daniel Bonneau, MD; Robert O. Bonow, MD; Jagdish Butany, MD; John Burgess, MD; Jason Burstein, MD; Warren Cantor, MD; Kanu Chatterjee, MB; Robert Chisholm, MD; the CCU, cardiac ward, cardiac OR, and CVICU nurses; Patrick Disney, MD; Lee Errett, MD; Neil Fam, MD; Matthias Friedrich, MD; Michael Heffernan, MD; Majo Joseph, MD; David Latter, MD; Yves Leclerc, MD; Anne Lenehan; Mat Lotfi, MD; Danny Marcuzzi, MD; David Mazer, MD; Rachel Miller; Abdulelah Mobeirek, MD; Juan Carlos Monge, MD; Ashok Mukherjee, MD; Mark Peterson, MD, PhD; Susan Pioli; Bill Parmley, MD; Geoffrey Puley, MD; Michael Regan; Gail Rudakewich; Myra Rudakewich; Nazmi Said, RVT; Jan-Peter Smedema, MD; Jim Stewart, MD; Bradley Strauss, MD; Subodh Verma, MD; and Andrew Yan, MD.

Stuart J. Hutchison, MD

Abbreviations

Ao, aorta
AV, atrioventricular
BP, blood pressure
bpm, beats per minute
CAD, coronary artery disease
CHF, congestive heart failure
CMR, cardiac magnetic resonance
CO, cardiac output
COPD, chronic obstructive pulmonary disease
CP, constrictive pericarditis
CSPAMM, complementary spatial modulation of magnetization
CT, computed tomography
CVICU, cardiovascular intensive care unit
CVP, central venous pressure
CXR, chest radiography
ECG, electrocardiography; electrocardiogram
ESV, end-systolic volume
GFR, glomerular filtration rate
HR, heart rate
HIV, human immunodeficiency virus
ICD, implantable cardioverter-defibrillator
ICU, intensive care unit
INR, international normalized ratio
IPP, intrapericardial pressure
IRGE, inversion recovery gradient echo
IV, intravenous
IVC, inferior vena cava
IVS, interventricular septum
JVP, jugular venous pressure
LA, left atrium; left atrial
LAA, left atrial appendage
LAO, left anterior oblique
LLPV, left lower pulmonary vein
LUPV, left upper pulmonary vein
LV, left ventricle; left ventricular
LVDP, left ventricular diastolic pressure
LVH, left ventricular hypertrophy
LVOT, left ventricular outflow tract
LVSP, left ventricular systolic pressure
MAP, mean arterial pressure
MPA, main pulmonary artery
MR, magnetic resonance
MRI, magnetic resonance imaging
NPV, negative predictive value
NSAIDs, nonsteroidal anti-inflammatory drugs
NYHA, New York Heart Association
PA, pulmonary artery
PCI, percutaneous coronary intervention
PCR, polymerase chain reaction
PCWP, pulmonary capillary wedge pressure
PEEP, positive end-expiratory pressure
PI, pulmonary insufficiency
PPD, purified protein derivative
PPV, positive predictive value
PTP, pretest probability
RA, right atrium; right atrial
RAA, right atrial appendage
RAO, right anterior oblique
RAP, right atrial pressure
RCA, right coronary artery
RCM, restrictive cardiomyopathy
RLPV, right lower pulmonary vein
RPA, right pulmonary artery
RR, respiratory rate
RUPV, right upper pulmonary vein
RV, right ventricle; right ventricular
RVDP, right ventricular diastolic pressure
RVEDP, right ventricular end-diastolic pressure
RVH, right ventricular hypertrophy
RVOT, right ventricular outflow tract
RVSP, right ventricular systolic pressure
SEM, systolic ejection murmur
SLE, systemic lupus erythematosus
SPAMM, spatial modulation of magnetization
SSFP, steady-state free precession
SV, stroke volume
SVC, superior vena cava
TEE, transesophageal echocardiography
TR, tricuspid regurgitation
TTE, transthoracic echocardiography

Contents

CHAPTER 1

Anatomy and Physiology of the Pericardium

KEY POINTS

- Histologically, the pericardium contains both elastic and collagenous matrices that confer physical properties of compliance and stiffness, respectively. The interior layer of the pericardium is lined by serosal cells. The exterior layer of the pericardium is the predominantly fibrous parietal pericardium, which is 1 mm thick anatomically and 2 mm or less thick by imaging depiction (the limitation of contemporary imaging modalities is illustrated by their inability to depict true anatomic pericardial thickness).
- Anatomically, the pericardial sac surrounds the entire heart other than small areas behind the left atrium, extends up the great vessels and pulmonary veins, and is as simple anteriorly as it is complex posteriorly. Fluid accumulation (effusion) occurs in pericardial recesses. Absence of parietal pericardium allows lung tissue into otherwise excluded recesses, such as between the main pulmonary artery and the ascending aorta, under the left ventricle, and over the dome of the left diaphragm.
- Physiologically, the pericardium limits acute diastolic overfilling to maintain myocardial systolic function. The parietal pericardium imparts an important pressure: volume relationship.
- Pathophysiologically, the pericardium may confer high degrees of ventricular interdependence and may also be responsible for compression of the heart—tamponade, effuso-constriction, and constriction.

The anatomic and histologic features of the pericardium confer its physiologic properties and underlie both its resistance to and susceptibility to disease. Knowledge of pericardial anatomy assists greatly with interpretation of imaging findings and disease detection, and knowledge of pericardial physiology assists immeasurably with understanding of the pathophysiologic manifestations of pericardial diseases.

ANATOMIC COMPONENTS OF THE PERICARDIUM

The components of the pericardium (Fig. 1-1) are the following:

- Visceral (serosal) layer
- Parietal (fibrous) layer
- Pericardial fluid
- Pericardial space and pericardial reflections
- Pericardial attachments

Visceral (Serosal) Layer and the Epicardium

The visceral (serosal) layer of the pericardium is a monolayer of ciliated mesothelial cells that line the entirety of the inside (interior) of the pericardial space; that is, the serosal layer lines the back side of the parietal pericardium and the outside surface of the underlying cardiac chambers and great vessels (Fig. 1-2). It sits on a very thin fibrous layer and a layer of fat of variable thickness. The cilia of the visceral layer increase the surface area available for fluid transport[1] for both production and resorption, and the cilia also reduce friction between opposite layers of the pericardium (Figs. 1-3 and 1-4). The opposing pericardial surfaces move considerable distances over each other through the cardiac cycle. For example, at the atrioventricular groove the pericardial surfaces move 1.5 cm each way over each other through each cardiac cycle. At 105,000 cardiac cycles per day over an 80-year life span, this equates to 92,000 km of movement. In health, the serosal layer has only microscopic thickness and is not even remotely imageable by echocardiography, computed tomography (CT), or cardiac magnetic resonance (CMR) (Fig. 1-5).

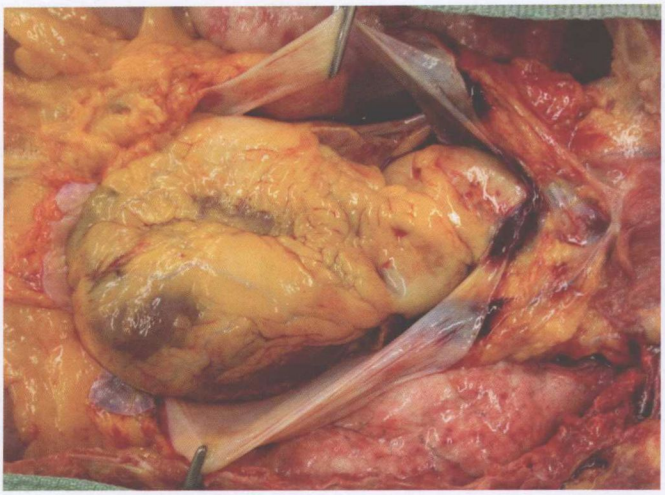

Figure 1-1. This autopsy photograph shows the thin and partially translucent nature of normal parietal pericardium; the invisible thinness of the normal serosal pericardium, whose only visual manifestation is a smooth sheen over the heart; and the epicardial and parietal pericardial fat layers. This view, slightly from the left side, demonstrates that there is less epicardial fat over the left ventricular free wall than there is over the anterior wall of the right ventricle. The parietal pericardial reflection can be seen extending well up the ascending aorta. (Courtesy of Jagdish Butany, MD, FRCPC, Toronto, Canada.)

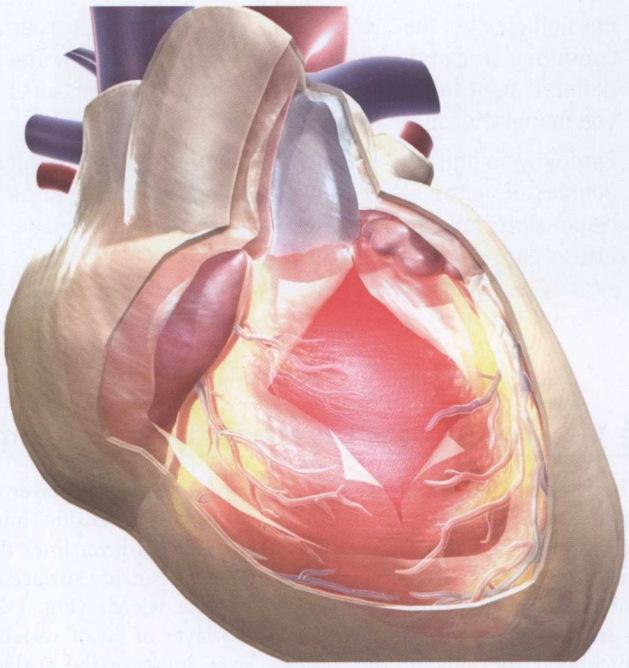

Figure 1-2. Representation of the serosal (visceral) and parietal (fibrous) pericardial layers. The outer thicker parietal pericardial layer extends over the heart chambers and proximal great vessels. The very thin serosal (visceral) layer lines the inside of the parietal layer and the outside of the epicardial layer of the heart, over epicardial coronary arteries and fat.

Because the visceral reflection of the serosal layer lies over the epicardium, it is sometimes referred to as the epicardial layer, but the same layer of ciliated cells also lines the inside of the fibrous parietal pericardium. Epicardium is essentially that which overlies the myocardium up to the serosal layer of pericardium. Epicardium includes variable amounts of fat and is the layer

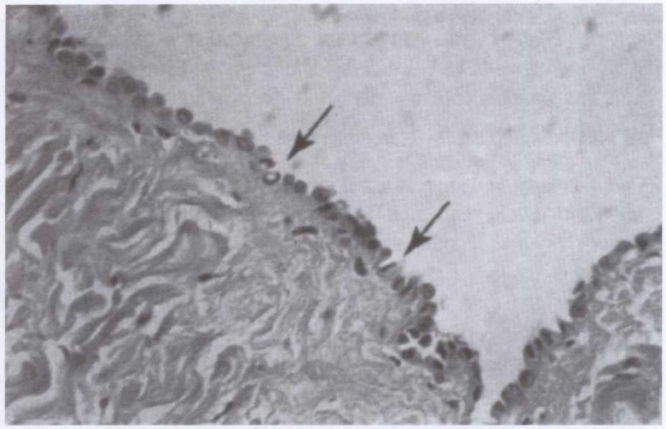

Figure 1-3. The monolayer of serosal cells that lines the entirety of the inside of the pericardial space. The thinness of the layer, in health, is obviously beneath the resolution of any imaging modality other than microscopy. (From Butany J, Woo A: The pericardium and its diseases. In Silver M, Gotlieb AI, Schoen F, eds: Cardiovascular Pathology. Philadelphia, Elsevier, 2001, figure 12-3. Copyright Elsevier, 2001.)

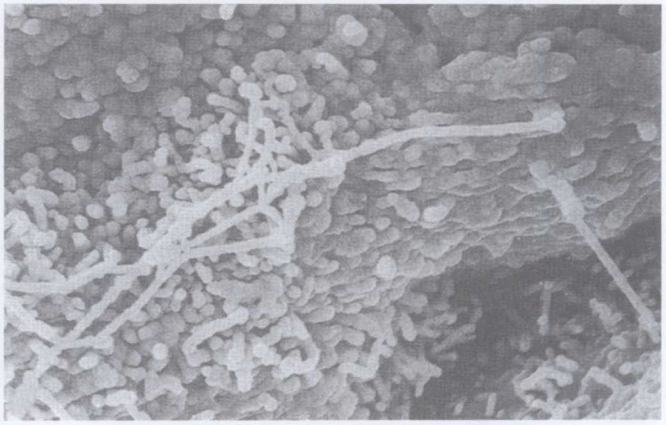

Figure 1-4. Scanning electron micrograph of the delicate multitudinous microvilli arising off of serosal cells. (From Butany J, Woo A: The pericardium and its diseases. In Silver M, Gotlieb AI, Schoen F, eds: Cardiovascular Pathology. Philadelphia, Elsevier, 2001, figure 12-4. Copyright Elsevier, 2001.)

where the "epicardial" coronary arteries and their branches run (Fig. 1-6). Epicardial fat is an important facilitator of parietal pericardial imaging because it confers contrast to the inside surface of the parietal pericardium, which otherwise would be difficult to distinguish from myocardium by CMR, CT, and echocardiography. Most epicardial fat is distributed over the anterior right ventricle and also a short distance around the lateral wall of the left ventricle (Fig. 1-7). Because the presence of epicardial fat is needed to distinguish (contrast) parietal pericardium from myocardium and hence to assist in assessing its thickness, assessment of parietal pericardium over the lateral and posterior left ventricle is more difficult.

Innervation of the epicardium and the overlying serosal pericardium is through sympathetic afferent fibers. Therefore, inflammation of this layer may result in a vague midline visceral pain of anginal nature.

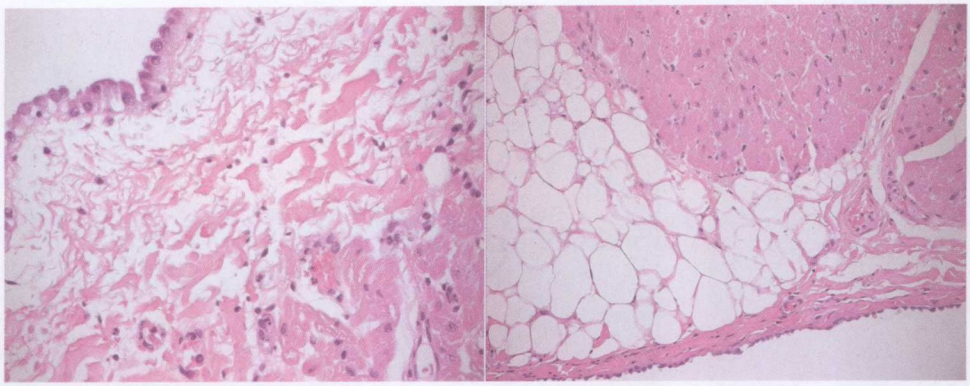

Figure 1-5. The serosal pericardium consists of a single monolayer of palisading cells (LEFT) that lie on a thin layer of connective tissue overlying the epicardial fat and epicardial coronary vessels (RIGHT). The distribution of epicardial fat is variable, as can be seen in the right image. In places, the serosal pericardium and its thin underlying layer of connective tissue lie against the myocardium. In other places, they are offset by fat. (Courtesy of Jagdish Butany, MD, FRCPC, Toronto, Canada.)

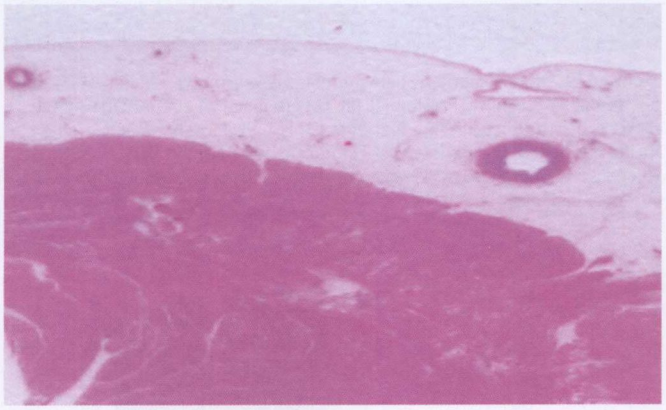

Figure 1-6. The epicardial tissue layer lies on the myocardium, extends up to the serosal pericardium layer, and comprises a highly variable thickness of fat. The epicardial coronary arteries run in this layer, usually nestled and cushioned in fat, as can be seen in this hematoxylin and eosin–stained low-power micrograph. (Courtesy of Gerald Prud'homme, MD, FRCPC, Toronto, Canada.)

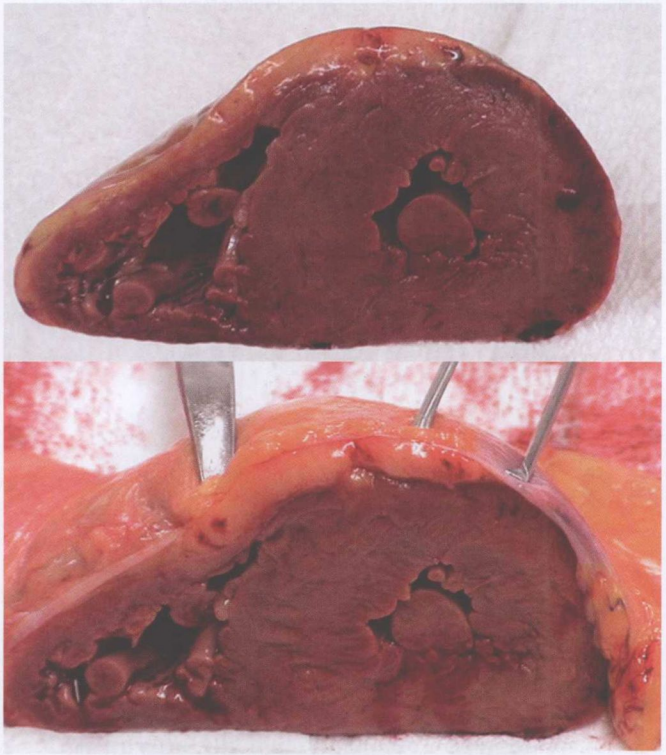

Figure 1-7. TOP, The parietal pericardium and its overlying fat have been removed. Epicardial fat is thick and continuous over the right ventricle and the anterior aspect of the left ventricle, but it has diminished considerably by the mid left ventricular lateral wall. BOTTOM, The parietal pericardium and its fat have been placed on the heart. The parietal pericardium is very thin in health. Fat on the outside of the parietal pericardium (pericardial fat versus epicardial fat) is thickest over the right ventricle and diminishes also over the left ventricular free wall. The outside (parietal) fat layer forms an apron at the diaphragm level. (Courtesy of Gerald Prud'homme, MD, FRCPC, Toronto, Canada.)

Parietal (Fibrous) Layer

The outer layer of pericardium, at the outer limit of the pericardial space, is the fibrous or parietal layer that itself consists of several layers. There are three collagen layers, the fibers of each of which are oriented approximately 120 degrees to each other.[1] The inner collagen layers are interwoven with a small amount of reticulum (elastin fibers). The final inside layer is the lining of mesothelial serosal cells, opposite those overlying the heart chambers and vessels. Because the parietal layer is predominantly collagen, it is tough, constitutes a physical barrier to disease, and has limited stretch (Fig. 1-8). Because it can stretch less than the myocardium, it therefore limits acute myocardial distention and preserves sarcomere architecture and function. The collagen component provides limitation to distention. The lesser elastin component confers only limited compliance to accommodate a finite pericardial volume reserve. The elastin fibers are arranged in a wavy pattern that straightens when stretched, allowing slight lengthening.[1]

The parietal layer is normally at most only about 1 mm thick (Fig. 1-9). Stated "normal" pericardial thickness by advanced imaging techniques depends on the imaging adequacy of the modality. Quite misleadingly, thickened parietal pericardium by CT or CMR was formerly defined as 4 mm or more, far greater than the true pericardial thickness and reflecting former limitations of imaging rather than true anatomic thickness. By modern gated cardiac CT imaging, representation of the thickness of the parietal pericardium is almost anatomically true.

Innervation of the parietal pericardium is achieved mainly from the phrenic nerves, which run over the parietal pericardium and generally generate a discrete, rapid, localized somatic pain—hence, the usual pleuritic nature of pericardial inflammatory diseases.

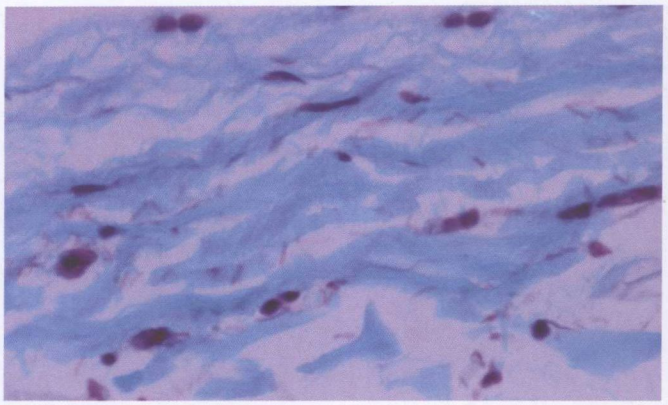

Figure 1-8. Mason trichrome stain of parietal pericardium. The collagen bundles run in planes. (Courtesy of Gerald Prud'homme, MD, FRCPC, Toronto, Canada.)

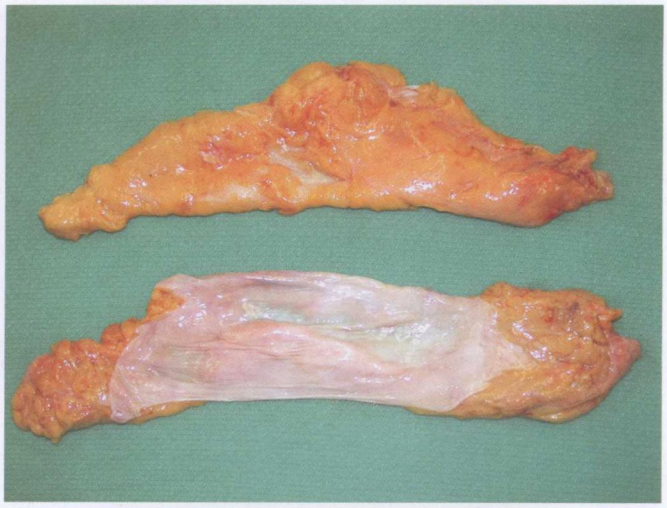

Figure 1-9. Normal parietal pericardium, viewed on its outside (TOP) and on its inside (BOTTOM). Pericardial fat covers much of the outside of the parietal pericardium in many patients. It is only loosely attached. The inside surface of the parietal pericardium is fat free and very smooth. Its thickness is only at most 1 mm. (Courtesy of Jagdish Butany, MD, FRCPC, Toronto, Canada.)

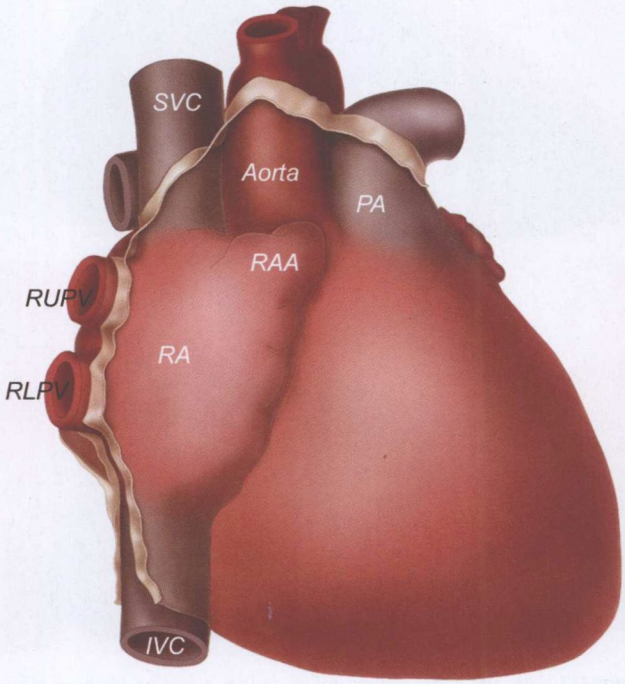

Figure 1-10. The pericardial space extends several centimeters up the great vessels; thus, rupture of the ascending aorta (as with acute dissection) will cause tamponade, and tamponade will compress the right atrium (RA), the right ventricle, and also the first several centimeters of the superior vena cava (SVC) and the inferior vena cava (IVC). PA, pulmonary artery; RAA, right atrial appendage; RLPV, right lower pulmonary vein; RUPV, right upper pulmonary vein.

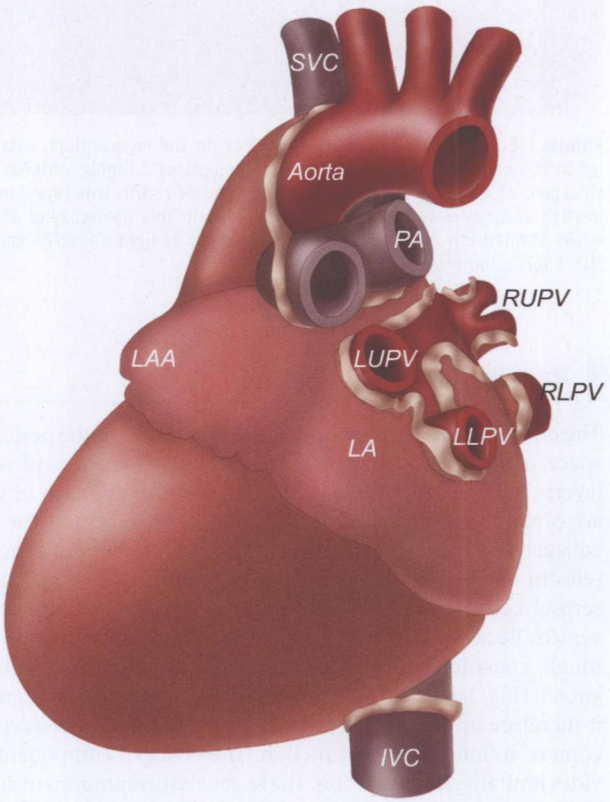

Figure 1-11. The pericardial space extends posteriorly over most of the left atrium (LA), other than between the upper pulmonary veins. Hence, fluid or clot may compress the left atrium or pulmonary veins. IVC, inferior vena cava; LAA, left atrial appendage; LLPV, left lower pulmonary vein; LUPV, left upper pulmonary vein; PA, pulmonary artery; RLPV, right lower pulmonary vein; RUPV, right upper pulmonary vein; SVC, superior vena cava.

Pericardial Fluid

Between the visceral and the parietal pericardium normally resides 15 to 50 mL of an ultrafiltrate "lubricating fluid," produced by the serosal cells, in both health and disease states. It is transparent and minimally "straw" colored. This amount of fluid is minimally visible by 2-dimensional echocardiography or appears as only a scant fluid layer, best seen in ventricular systole when reduction of ventricular volume and dimension allows separation of the pericardial layers and renders the appearance of the fluid layer more obvious. There is continuous flux of production and resorption of the fluid; therefore, the net amount reflects the

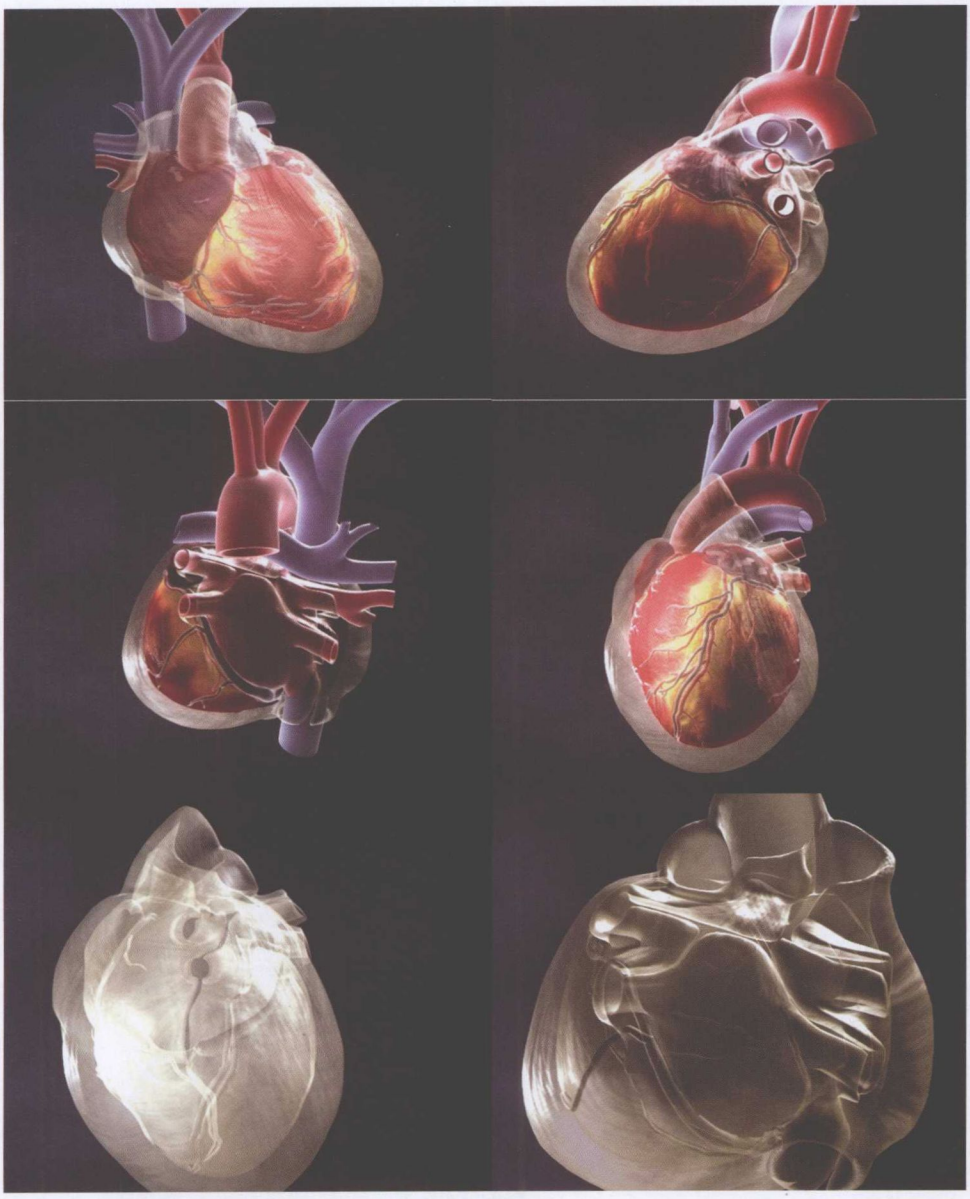

Figure 1-12. The pericardium is not a "round" container but one with a complex shape that envelops both cardiac chambers and all of the great vessels with "sleeves." The pericardial space, here exemplified by a pericardial effusion, is therefore complex in shape, especially in the posterior aspect of the heart.

balance of production and removal. In many states of increased production, such as pericarditis, there is no accumulation because removal is as rapid as fluid formation is. The composition of "normal" pericardial fluid is of less protein than serum but more albumin, as albumin is more readily transported than other larger proteins are. The fluid has a high phospholipid content, which is believed to confer the lubricant quality. The fluid equalizes pericardial pressure onto the underlying heart chambers.

Pericardial Space

The continuity of the pericardial layers (visceral and parietal) establishes the pericardial space, a space lined on its interior side (against the heart chambers and against the parietal pericardium of the outside) by the thin monolayer of serosal cells. On the outside, the same monolayer of serosal cells and also a restraining and protective layer of fibrous tissue (parietal pericardium) are present.

The parietal pericardium and space cover the proximal parts of all of the great vessels (Fig. 1-10) and all the cardiac chambers other than a limited part of the posterior left atrium (Fig. 1-11). Thus, rupture of the ascending aorta, as commonly occurs with type A aortic dissection, leads to pericardial tamponade, whereas rupture of the descending aorta, which is extrapericardial, generally leads to intrapleural exsanguination. The pericardial space is as complex posteriorly, where it runs up the venous and arterial sleeves, as it is simple anteriorly, where it covers the cardiac chambers (Fig. 1-12).

The *pericardial reflections*, where the visceral pericardium folds back on itself to line the outer fibrous pericardium, extend to the first arch branch vessel of the aorta, to the pulmonary artery bifurcation, up (and down) the cavae (Fig. 1-13) a few centimeters, and up the pulmonary veins a few centimeters (Figs. 1-14 to 1-16). The cohesion of the connective tissue at the sites of reflection is variable: the fibrous parietal pericardium is securely attached to the aorta and pulmonary arteries but imperfectly attached to the pulmonary veins. This fact underlies the extension

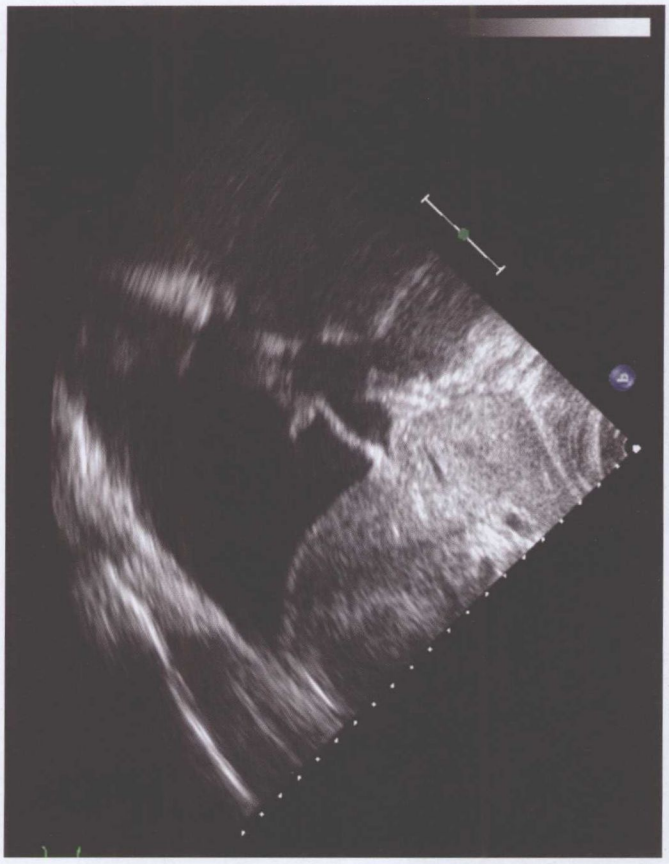

Figure 1-13. This transthoracic echocardiographic image reveals a large right pleural effusion over the liver and also the supradiaphragmatic portion of IVC as it enters into the right atrium. There is normally 2 or 3 cm of supradiaphragmatic IVC above the diaphragm. It is largely intrapericardial and occasionally is a site of localized compression. The supradiaphragmatic IVC, when visible on the lateral chest radiograph, is a useful yardstick to gauge posterior left ventricular dilation (the rule of Rigler).

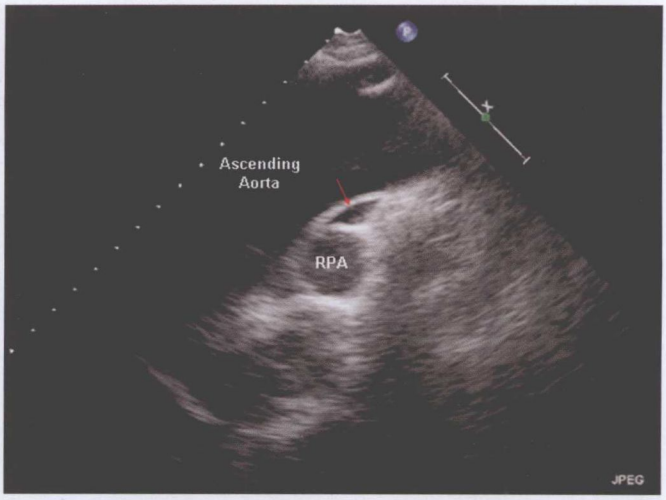

Figure 1-14. There is a small amount of pericardial fluid posterior to the ascending aorta *(arrow)* over the right pulmonary artery (RPA), at the most superior extent of the pericardial cavity recesses.

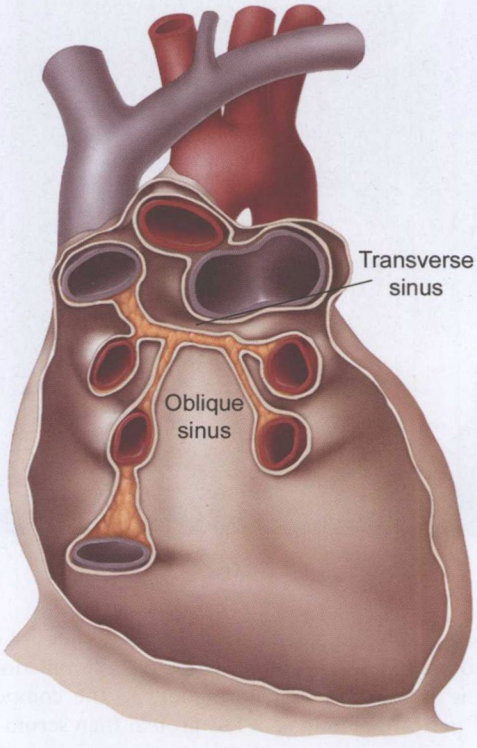

Figure 1-15. The posterior parietal pericardial anatomy is complex. The pericardial space does extend behind the left atrium and also along the pulmonary veins. The pericardial space also surrounds the proximal aorta and pulmonary artery. The cul-de-sac of pericardial space between the pulmonary veins is the oblique sinus; the passageway under the aorta and pulmonary artery is the transverse sinus, through which saphenous vein grafts to the circumflex territory are sometimes passed.

of mediastinal air into the pericardial space through the pulmonary venous reflections and the secondary indirect development of pneumopericardium (Fig. 1-17).

The parietal pericardium and space therefore cover a more extensive field than is usually anticipated. The sleeve-like extension of the parietal pericardium over the great arteries is significant for multiple reasons: the demise of type A dissection is usually rupture of the ascending aorta into the pericardial space, pericardial fluid collection can be anticipated to occur around the great vessels, and the parietal pericardium normally excludes lung parenchyma from anatomic recesses between the aorta and main pulmonary artery.

Pericardial Attachments

Ligamentous attachments of the parietal pericardium tether it, and the underlying heart, stably in place in the chest, which is important given the weight and inertia of the heart. Pericardial-sternal ligaments attaching to the sternum, pericardial-diaphragmatic ligaments attaching to the diaphragm (the most extensive ligaments), and pericardial-vertebral ligaments attaching to the vertebrae hold the heart in place when acceleration-deceleration forces occur within the chest (Fig. 1-18). The heart is well anchored by these ligaments within the chest and is moved little by anteroposterior acceleration forces. Conversely, the descending aorta is poorly anchored within the chest and is subject to motion and distortion by acceleration forces, and it is therefore subject to motion-related injury. Congenital deficiency

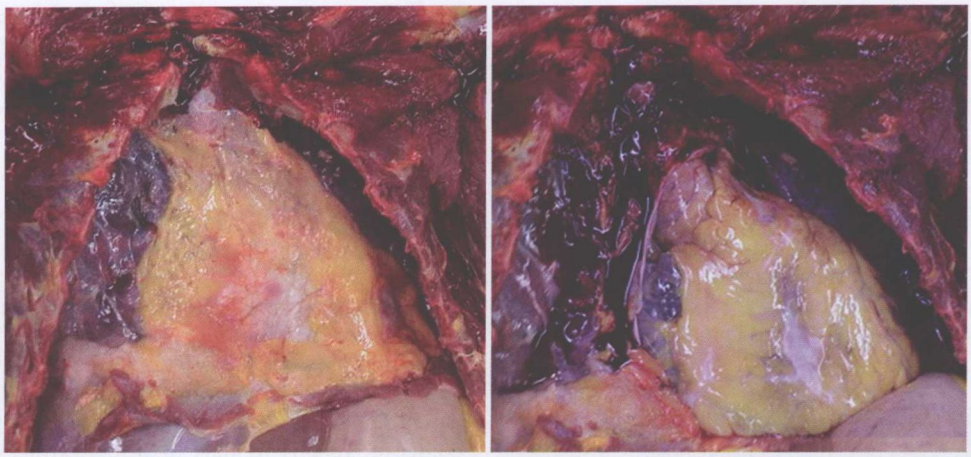

Figure 1-16. These autopsy photographs demonstrate the wider extent of pericardial coverage of the heart than is often appreciated. TOP, With the chest opened, there is abundant fat anterior and exterior to the parietal pericardium and the far superior extension of the pericardium well up the great vessels into the mediastinum. As well, there is a copious "skirt" of fat at the diaphragm level on either side of the heart, often visible on the posteroanterior chest radiograph. BOTTOM, After the anterior parietal pericardium had been excised, its free edge can be seen beside the right atrial appendage and across the ascending aorta. The free edge has folded over onto itself, making it look thicker than it truly is. The superior reflection of the parietal pericardium is higher than the location of the excision across the ascending aorta. The anterior epicardial fat over the right ventricle is obvious. Thus, anterior to the right ventricle, there is normally a fat plane beneath (epicardial fat) and exterior to (pericardial fat) the parietal pericardium. (Courtesy of Gerald Prud'homme, MD, FRCPC, Toronto, Canada.)

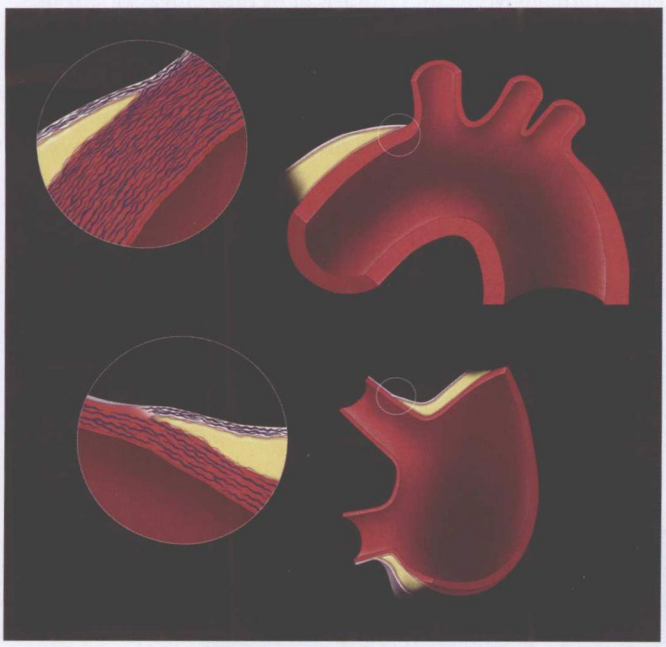

Figure 1-17. Pericardial reflections and connective tissue integrity. TOP, Pericardial reflection at the ascending aorta. BOTTOM, Pericardial reflection at the pulmonary veins. The reflection of the pericardium over the great vessels, such as the aorta, involves incorporation of the collagen connective tissue layers of the parietal pericardium into those of the aortic wall; hence, there is continuity of connective tissue and an effective barrier. The serosal layer of the inside of the parietal pericardium reflects back over the outside surface of the vessel. The reflection of the pericardium at the pulmonary veins occurs without interweaving of the collagen layers of the parietal pericardium with those of the pulmonary veins; hence, this represents a possible weak site of pericardial anatomy and a potential weakness of the barrier. In fact, air tracking along the intrapulmonary perivascular sheaths of pulmonary veins (that resulted from pneumomediastinum) can gain entry into the pericardial space at this site (resulting in pneumopericardium).

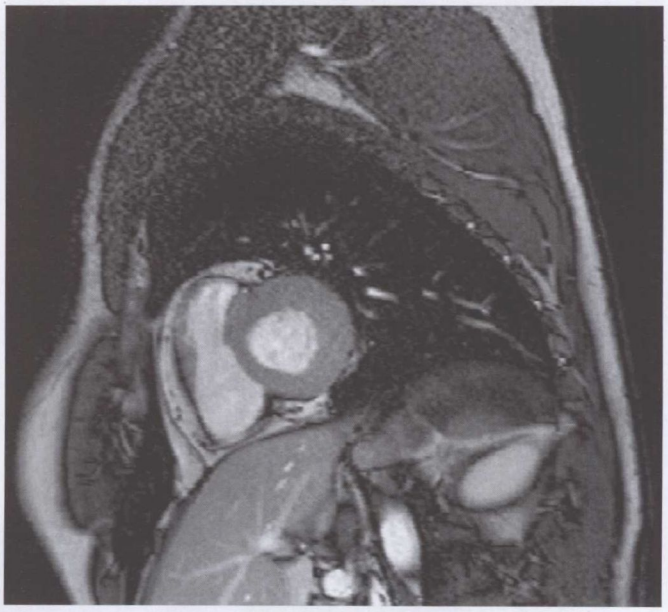

Figure 1-18. On this steady-state free precession CMR image, the fat planes over the right side of the heart (epicardial and pericardial) are sufficiently distinct for the insertion of the parietal pericardium into the diaphragm to be seen. There is little epicardial or pericardial (outside) fat over the left ventricular free wall.

(absence) of the pericardium removes restraining forces on the heart, and accordingly the heart shifts toward the defect. Because absence of the left pericardium is the most common variant of congenital absence, leftward shift of the heart is the usual. Absence of parietal pericardium allows lung tissue into otherwise excluded recesses, such as between the main pulmonary artery and the ascending aorta, under the left ventricle, and over the dome of the left diaphragm. These are reliable imaging details by chest radiography, CT, and CMR to detect the absence of pericardium.

Fat Planes

The parietal pericardium is difficult to image because its imaging characteristics (relaxation times on CMR, attenuation coefficient on CT scanning) are too similar to those of the underlying atrial and ventricular myocardium. It is the presence of adjacent fat planes on either side of the parietal pericardium (when both are present), which have very different CT and CMR characteristics and therefore appearance, that enables clear depiction of the parietal pericardium. Epicardial fat (under the parietal pericardium) and overlying (outside, parietal) fat are far more likely over the right ventricular free wall than over the left ventricular free wall or over either atrium. Adjacent fat planes that "sandwich" the parietal pericardium are requisite to imaging of the parietal pericardium (Fig. 1-19).

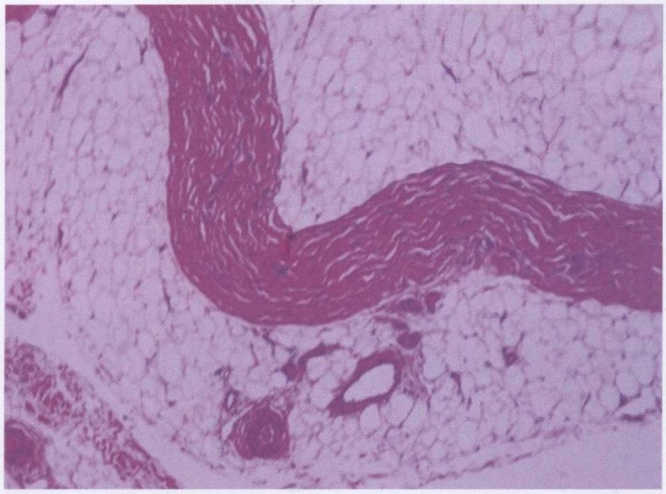

Figure 1-19. Hematoxylin and eosin–stained low-power micrograph. The collagenous parietal pericardium consists principally of bundles of collagen. In certain areas of the heart, particularly over the right ventricle, there is likely to be an underlying "epicardial" fat plane and an overlying parietal (outside) fat layer. The "fat sandwich" is required to image the pericardium by CMR and by CT scanning; otherwise, without the contrast with fat, the parietal pericardium cannot be distinguished from myocardium or adjacent exterior tissues. (Courtesy of Gerald Prud'homme, MD, FRCPC, Toronto, Canada.)

Other Anatomic and Physiologic Considerations

The internal thoracic arteries confer blood supply to most of the parietal pericardium, and their proximity to the pericardium (their avoidance) is relevant for percutaneous pericardial drainage procedures. The aorta supplies small branches to the posterior pericardium. The internal thoracic and azygos venous system confer the venous drainage to the pericardial space (Fig. 1-20). As the azygos vein drains into the superior vena cava, central venous pressure elevation increases the forces (hydrostatic pressure) that favor pericardial fluid accumulation.

The anterior mediastinal lymph nodes and the internal thoracic duct supply the lymphatic drainage but unfortunately also establish proximity and continuity of the lymph system to the pericardium. Therefore, malignant involvement of anterior mediastinal nodes is particularly likely to extend to the pericardium, either through lymphatic channels or by direct invasive spread.

Mechanical receptors and chemoreceptors use sympathetic afferent fibers to mediate reflexes and pain. The left and right phrenic nerves run over the lateral surface of the parietal pericardium; their presence is relevant to surgical procedures, particularly pericardiectomy, in which they must be left intact to preserve diaphragmatic function. Innervation of the pericardium also involves the left recurrent laryngeal nerve, the vagus nerve, and the esophageal plexus.[1]

A very important relation of the pericardial space, for imaging purposes, is the retrocardiac descending thoracic aorta. The pericardial space extends between the left atrium and the descending aorta, whereas the left pleural space does not extend between the descending aorta and the left atrium but rather extends posteriorly behind the descending aorta.

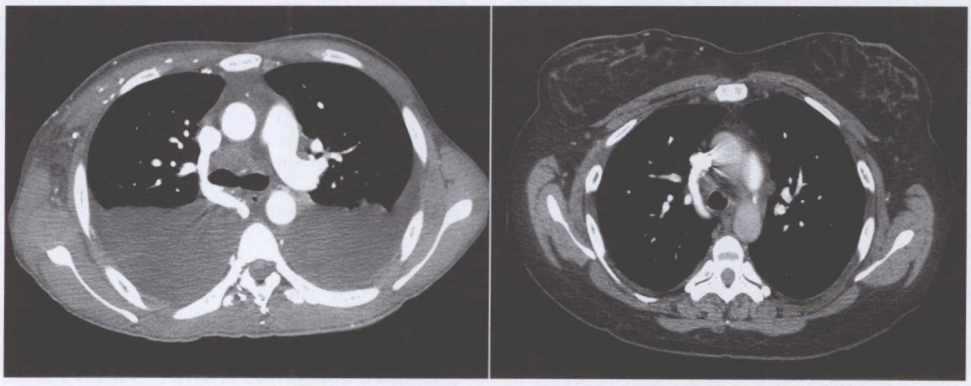

Figure 1-20. Azygos vein and pericardial disease. The azygos vein drains the pericardium, among many other structures, and empties into the SVC above the right pulmonary artery. Pericardial diseases that compress the heart, such as constriction or even tamponade, result in increased central venous pressure and dilation of the cavae and their branches. The left image (contrast-enhanced CT) depicts the dilated SVC and azygos vein of a patient with a severe case of pericardial constriction. The azygos vein is remarkably dilated, consistent with the central venous pressure of nearly 30 mm Hg. As the pleural cavities are drained by the azygos and hemiazygos veins, it is not surprising that there are bilateral pleural effusions due to the increased hydrostatic pressure. The right image, also a contrast-enhanced CT image, depicts a moderately dilated azygos vein in a patient with a case of moderate constriction but without pleural effusions.