Pericardial Disease: Value of CT and MR Imaging

The pericardium represents an important focus of morbidity and mortality in patients with cardiovascular disease. Fortunately, in recent years knowledge regarding this enigmatic part of the heart and the diagnosis of related diseases has substantially advanced. To a large extent, this can be attributed to the availability of several non-invasive cardiac imaging modalities. Transthoracic echocardiography, which combines structural and physiologic assessment, is the first-line technique for examination of patients suspected of having or known to have pericardial disease; however, cardiac computed tomography (CT) and magnetic resonance (MR) imaging are becoming increasingly popular for the study of this part of the heart. Modern multidetector CT scanners merge acquisition speed and high spatial and contrast resolution, with volumetric scanning to provide excellent anatomic detail of the pericardium. Multidetector CT is by far the modality of choice for depiction of pericardial calcifications. MR imaging is probably the best imaging modality for the acquisition of a comprehensive view of the pericardial abnormalities. MR imaging combines cardiac and pericardial anatomic assessment with tissue characterization and appraisal of the effects of pericardial abnormalities on cardiac performance. This review aims to elucidate the role of the pericardium and its interaction with the remainder of the heart in normal and pathologic conditions. It focuses on the rapidly evolving insights regarding pericardial disease provided by modern imaging modalities, not infrequently necessitating reconsideration of evidence that has thus far been taken for granted.

From the Department of Radiology, Medical Imaging Research Center, University Hospitals Leuven, Herestraat 49, B-3000 Leuven, Belgium (J.B.); and Cardiovascular MRI Unit, Department of Radiological, Oncological and Pathological Sciences, University of Rome La Sapienza, Rome, Italy (M.F.). Received June 6, 2012; revision requested July 16; revision received August 12; accepted October 2; final version accepted October 31. Address correspondence to J.B. (e-mail: jan.bogaert@uz.kuleuven.ac.be).

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Pericardial disease is an important cause of morbidity and mortality in patients with cardiovascular disease. It represents a heterogeneous group of congenital and acquired diseases, with a widely variable clinical manifestation that often necessitates a specific treatment. Besides isolated pericardial disease, the pericardium may be secondarily involved by a large group of organ and systemic diseases, such as infective, autoimmune, and neoplastic processes. Moreover, iatrogenic causes—for example, after cardiac surgery or radiation therapy—represent an important cause of pericardial-related morbidity and mortality. Pericardial disease may be an incidental finding (eg, pericardial cyst) but may also necessitate urgent intervention in case of hemodynamic compromise (eg, acute cardiac tamponade, herniation of cardiac contents through a partial pericardial defect).

The diagnosis of pericardial disease frequently remains clinically challenging, requiring integration of medical history and findings from physical examination, imaging, blood analyses, and, eventually, invasive hemodynamic measurements and/or pericardial biopsy (1–3). The role of transthoracic echocardiography in helping diagnose pericardial disease has been well recognized for almost half a century, allowing both structural assessment and evaluation of the physiologic consequences to the heart (4–8). However, echocardiography may fail to allow adequate evaluation of the pericardium at times. In particular, the diagnostic accuracy of echocardiography is limited in patients with a suboptimal acoustic window (eg, obese subjects, patients with severe chronic obstructive pulmonary disease or skeletal malformations) (9,10). Moreover, transthoracic echocardiography is limited in its ability to aid in recognition of focal effusions, assessment of pericardial thickness, and characterization of tissue (10,11).

Since the late 1970s, computed tomography (CT) and magnetic resonance (MR) imaging have emerged as interesting alternatives to echocardiography for morphologic assessment of the heart and pericardium (Table 1) (12–15). In recent years, improvements in MR imaging hardware and sequence design have further enhanced the diagnostic value of this modality for the study of pericardial disease. For example, fast MR sequences allow one to study cardiac motion and inflow patterns in real time during free breathing, which allows identification of constrictive physiology (15–19). Moreover, delayed (or late) gadolinium-enhanced MR imaging is useful for the detection of pericardial inflammation and for monitoring the effects of medical (anti-inflammatory) treatment (20,21). Cardiac CT with multideector scanners and electrocardiographically (ECG) synchronized data acquisition allows an accurate assessment of the coronary arteries and the remainder of the heart and pericardium, including functional cardiac analysis (22–23).

In this review, we will describe how CT and MR imaging have dramatically shaped our view on imaging of pericardial disease and how these techniques may contribute to the optimization of current patient care.

Pericardial Anatomy and Physiology

“The pericardium is a fibrous bag surrounding the heart and origin of the large blood vessels, but without any direct attachment to the heart itself, having its inner surface lined by a serous membrane.” This definition of the pericardium by R. B. Todd in the Cyclopedia of Anatomy and Physiology from 1835 is still up to date more than 175 years after it was written (26). This thin flask-shaped membrane is macroscopically composed of two layers, the inner serosa (also referred as the visceral pericardium or the epicardium) and the outer fibrosa (also referred as the parietal pericardium) (27,28). The serosa forms a complete sac filled with up to 50 mL of plasmatic ultrafiltrate and is separated from the heart by loose epicardial connective tissue and a single layer of mesothelial cells.

Like visceral abdominal fat, the thickness of the epicardial fat is increased in obesity, and there is accumulating evidence that epicardial fat can directly influence coronary atherogenesis, because there is no fibrous layer to impede diffusion to the underlying coronary arteries of the free fatty acids and adipokines that modulate atherothrombosis (29–31). The fat distribution is typically asymmetric, with three to four times more epicardial fat present along the right ventricle (RV) than along the left part of the heart (29). The outer layer, the fibrous pericardium, is attached internally to the epicardium and extends cranially above level of the aortic root (Fig 1). Moreover, this layer is continuous with the deep cervical fascia and is attached to the sternum and the diaphragm by loose ligaments that impede cardiac displacement in the mediastinum. Another anatomic feature of the pericardium is the presence of two major reflections of the visceral layer, which are arranged around two...
complex connected tubes, described as the transverse and oblique sinuses, and include several smaller recesses (Fig 1). These sinuses have a reserve function and may strategically accumulate fluid in the presence of increased fluid content, thus creating pocketlike structures (the so-called pericardial reserve volume) (28,32). The transverse sinus is located behind the ascending aorta and pulmonary trunk and in front of the atria and the superior vena cava. This linear-shaped virtual cavity should not be misinterpreted when filled with fluid as a focal aortic dissection or as an enlarged mediastinal lymph node on CT or MR images (28,32). The oblique sinus is located behind the left atrium and pulmonary trunk and in front of the atri (28).

Despite its relatively simple macroscopic and ultrastructural architecture, the pericardium has a crucial but complex role in ensuring the normal performance of the heart. This role is three fold: mechanical, membranous, and ligamentous. As such, it allows the regulation and maintenance of normal ventricular compliance to minimize ventricular dilatation and to physically protect the heart by means of the production of fluid and surfactants. It also limits the displacement of the heart in the mediastinum (33,34). When surgically incised, the fibrous pericardium spontaneously retracts, indicating that it is under some physical stress. This mild degree of pericardial stretching impedes pathologic overdistention, mainly of the thin-walled cardiac chambers such as the RV and right atrium, and directly influences intracavitary diastolic pressures.

Since the ventricles are surrounded by a relatively inelastic pericardium, the RV and left ventricle (LV) do not act independently, although there is a mutual interaction, a phenomenon defined as “ventricular coupling” or “ventricular interdependence.” During ventricular filling, the position of the interventricular septum is determined by the pressure difference between the RV and LV. In normal loading conditions, LV diastolic pressures slightly exceed RV pressures, which gives the septum a typically convex shape with a slight right-sided bulge. In many pathologic conditions, this shape may be altered. Septal flattening or inversion may occur in conditions with RV volume (eg, atrial septal defect or pressure overload [acute or chronic cor pulmonale]) and in pericardial abnormalities where pericardial pressures are increased (eg, cardiac tamponade) or pericardial compliance is decreased (eg, constractive pericarditis) (27,35). Finally, but importantly, the pericardial sac, having a slightly negative pressure, acts not only as an anatomic barrier but also as an important physiologic intermediary between the heart and the remainder of the chest. As such, it functions as a pressure transducer between the pleural spaces and cardiac chambers. In normal conditions, the intrathoracic respiratory changes are directly transmitted to the cardiac chambers. During inspiration, the intrathoracic pressure decreases, which augments systemic venous return and RV filling, while LV filling is not influenced.
acquisitions, which improve visualization of the pericardial sinuses and recesses and thus reduce the risk of misinterpreting these pericardial structures as enlarged lymph nodes or focal aortic disease (25,28). Although dynamic evaluation of ventricular septal motion to evaluate constrictive physiology is theoretically possible with ECG-synchronized CT, real-time functional imaging is much easier and more accurately assessed by using echocardiography and MR imaging (39).

Comprehensive MR imaging of the pericardium includes (a) morphologic assessment of the heart, pericardium, and surrounding mediastinum; (b) assessment of global and regional LV and RV function; (c) assessment of ventricular coupling by using real-time imaging during free breathing; (d) assessment of ventricular filling during breath holding or free breathing by using real-time imaging; (e) tissue characterization; and (f) evaluation of pericardial mobility and fusion of pericardial layers (Table 2) (40).

Table 2

<table>
<thead>
<tr>
<th>Target</th>
<th>MR Sequences</th>
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<td>Pericardial morphology</td>
<td>T1 weighted, cine</td>
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<td>Pericardial layer and fluid characterization</td>
<td>T1 and T2 weighted, cine, T1-weighted contrast agent enhanced or late gadolinium enhanced</td>
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<td>Pericardial mobility and fusion of pericardial layers</td>
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<td>Cardiac morphology</td>
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<td>Other findings*</td>
<td>T1 weighted, T2 weighted, cine, late gadolinium enhanced</td>
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* Other findings include myocarditis, myocardial infarction, and myocardial infiltrative or storage disease; caval vein size; mediastinal and pulmonary pathologic conditions; and pleural fluid and ascites.

In patients with constrictive pericarditis, however, the pathologic pericardium impedes transmission of intrathoracic pressures, causing typical respiratory-related abnormalities in ventricular filling (8). Assessment of the presence and severity of ventricular coupling, as well as evaluation of the effect of respiration, are crucial in the diagnosis of constrictive pericarditis and its differentiation from restrictive cardiomyopathy (36).

**CT and MR Imaging Techniques**

For pericardial imaging by means of CT equipped with multidetector technology, use of a high-resolution volumetric acquisition with a section thickness of greater than 3 mm generally yields an excellent anatomic depiction of the pericardium (Fig 2) (15,37,38). Image artifacts related to cardiac motion can be minimized by using ECG-synchronized acquisitions, which improve visualization of the pericardial sinuses and recesses and thus reduce the risk of misinterpreting these pericardial structures as enlarged lymph nodes or focal aortic disease (25,28). Although dynamic evaluation of ventricular septal motion to evaluate constrictive physiology is theoretically possible with ECG-synchronized CT, real-time functional imaging is much easier and more accurately assessed by using echocardiography and MR imaging (39).

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Black-blood T1-weighted spin-echo MR imaging performed with a fast segmented sequence is the best approach for morphologic study of the heart,
pericardium, and mediastinum (41,42). The use of a small field of view and a saturation pulse positioned on the frontal chest wall may improve pericardial visualization. To guarantee complete depiction of the heart, it is advisable to image the heart and pericardium in two perpendicular planes. T2-weighted spin-echo imaging, preferably performed by using a short-tau inversion-recovery sequence (also called triple-inversion spin echo), enables depiction of pericardial fluid and/or edema of the pericardial layers in patients with inflammatory pericarditis. Moreover, concomitant myocardial edema may be identified in patients with an acute coronary syndrome or acute myocarditis (41,43,44). MR imaging after administration of a paramagnetic contrast agent is useful in patients suspected of having pericardial masses or inflammatory pericarditis and to depict concomitant myocardial pathologic conditions (eg, myocarditis) and is of great value in depicting persistent chronic inflammation in patients with constrictive pericarditis (3,20,21,41,44).

Although T1-weighted spin-echo sequences can be used for contrast-enhanced imaging, we recommend use of late (or delayed) gadolinium-enhanced inversion-recovery MR imaging, as has been proposed for imaging myocardial infarct and viability (20,45–47). Cine MR imaging performed with balanced steady-state free precession (SSFP) gradient-echo sequences is the currently preferred technique for quantifying global and regional cardiac systolic function and is well suited to rule out associated RV or LV dysfunction (40,41). The high spatial and temporal resolutions of this sequence can be applied in new applications, such as dynamic evaluation of the rigidity of the pericardial layers in patients with constrictive pericarditis. Fast cine sequences enable real-time evaluation of dynamic fast-changing physiologic events such as ventricular coupling (17). MR tagging techniques are valuable in the detection of both fibrotic adhesion of pericardial layers and myocardial involvement in constrictive pericarditis (48). Assessment of diastolic heart function, usually accomplished by means of Doppler echocardiography, can also be achieved by using velocity-encoded or phase-contrast MR sequences (49,50). Analysis of pulmonary and/or systemic venous patterns in combination with cardiac inflow patterns through the atrioventricular valves can yield findings that are classic for restrictive cardiac filling. Real-time acquisition during free breathing allows one to identify constrictive physiology (19).

## Congenital Pericardial Disease

### Pericardial Cyst and Diverticulum

Pericardial cyst and pericardial diverticulum are rare congenital abnormalities that occur in approximately one in 100,000 individuals and account for 13%–17% of all mediastinal cysts (60). A pericardial cyst is a benign unicocular mass of celomic origin. These cysts are usually incidental findings at chest radiography or transthoracic echocardiography, although symptoms may occur in patients with cardiac compression (Fig 4; Movie 1 [online]) (33,61). A pericardial cyst appears on CT and MR images as an encapsulated fluid-filled structure without internal septa or nodules that generally is directly attached to the pericardium but rarely can be attached by contours and is surrounded by a variable amount of epicardial fat tissue (Figs 2, 3). Typically, the pericardium is best visualized along the RV, while, because of a sparseness of epicardial fat and the vicinity of pulmonary parenchyma, the pericardium is often difficult to discern along the lateral and posterior LV wall (15,41). Normal pericardial thickness ranges from 1.2 to 1.7 mm on MR images and from 0.7 to 2.0 mm on CT images (25,32,38,51–54). These values slightly overestimate those obtained in anatomic cadaveric studies (ie, 0.4–1.0 mm) (55,56). Differences can be explained by the intrinsically limited spatial and temporal resolutions of CT and MR imaging, which do not allow one to fully discriminate between pericardial layers and fluid component. Pericardial sinuses and their recesses are frequently depicted on CT and MR images (14,28,37,57–59). Their visualization on CT images can be improved by using an ECG-synchronized acquisition and thin-section scanning (28,37). Large recesses are depicted in up to 45% of patients, and smaller recesses are shown in nearly 20% (37). Most recesses are linear when not filled with fluid and hand-shaped as the fluid content increases. However, they may also present as crescent, triangle, spindle, ovoid, hemisphere, or irregular shapes (37).

## Imaging of Normal Pericardium

The normal pericardium appears on CT and MR images as a thin curvilinear structure that follows the myocardial
a peduncle. Most commonly, it is found in the right cardiophrenic angle (70% of cases), and less frequently in the left cardiophrenic angle (20% of cases) (62). Pericardial diverticulum is a focal outpouching of the pericardial sac, which can be differentiated from a congenital cyst by its presence of direct communication with the pericardial cavity, usually identified by changes in size related to body position (60).

**Pericardial Defect**
A congenital defect of the pericardium is an extremely rare anomaly that occurs as a consequence of premature atrophy of the cardinal veins supplying the pleuropericardial folds during embryogenesis, with subsequent failure of the membrane to develop. The extent of the defect, likely determined by the timing of vascular degeneration, ranges from partial to complete absence of the pericardial sac (63,64). Partial defects (large ones are more common than small ones) are far more frequent than total defects, and the left side is more commonly affected than the right side (Fig 5) (25,33,63). Although patients are often asymptomatic, symptoms may arise when cardiac structures are transiently entrapped or incarcerated in the defect (65). Herniation of the left atrial appendage through a small defect may lead to infarction of the appendage, or the left coronary artery might be compressed, leading to ischemia especially during exercise (66).

In about one-third of cases, pericardial defects are associated with other congenital abnormalities such as bronchogenic cysts, ventricular septal defect, patent ductus arteriosus, or diaphragmatic hernia. On chest radiographs, a left-sided pericardial defect causes typical levodisplacement of the heart and aortic knob, with the trachea remaining at the midline. The problem in CT and MR imaging of left-sided pericardial defects is the inability of these modalities to visualize the pericardium in this part of the heart. Therefore, the diagnosis of left-sided defects usually relies on other signs, such as an abnormal position of cardiac structures with excessive levorotation or cardiac indentation at the location of the defect (Fig 5) (67,68). Since herniation is often intermittent in time, positional changes to the left lateral decubitus position, for example, can be helpful in diagnosing pericardial defects. A functional examination may also be helpful in establishing the diagnosis of congenital pericardial defect (69).

**Acquired Pericardial Disease**

**Pericardial Effusion**
Abnormal accumulation of pericardial fluid may occur in patients with heart failure, renal and liver insufficiency, inflammation, infection, neoplastic disease, trauma, and myocardial infarction (39). Imaging is often necessary to confirm the presence, severity, and extent of fluid; to rule out pericardial inflammation; to determine the hemodynamic effect on the heart; and ultimately to guide pericardiocentesis.

Although transthoracic echocardiography is the preferred first-line modality for this evaluation, CT and MR imaging often allow for a more comprehensive...
relaxation (higher signal intensity) and cell content, increase the rate of T1 images. Exudates, having high protein intensity on T1-weighted MR images and dates typically manifest with low signal intensity on T2-weighted images. Exudates, having high protein intensity on T1-weighted MR images and dates typically manifest with low signal intensity on T2-weighted images. If the CT attenuation value is greater than that of water, then an effusion is more likely to attenuating attenuation values on CT images and signal intensity on MR images. If the CT attenuation value is greater than that of water, then an effusion is more likely to be due to hemopericardium, malignancy, purulent exudates, or hypothyroid-related effusion (25). Low-attenuation pericardial effusions have been reported in cases of chylopericardium (15). Transudates typically manifest with low signal intensity on T1-weighted MR images and with high signal intensity on T2-weighted images. Exudates, having high protein and cell content, increase the rate of T1 relaxation (higher signal intensity) and may lead to cardiac chamber compression, compromising diastolic filling and causing a reduction of cardiac output with subsequent hypotension, tachycardia, and potentially progression to cardiogenic shock (71). This condition can be triggered by multiple causes, including trauma, inflammation, aortic dissection, neoplastic involvement of the pericardial space, acute myocardial infarction, and cardiac surgery (71,72). Symptoms may arise acutely or more chronically and are determined by the absolute volume of fluid, the speed of fluid accumulation, and the physical characteristics (ie, elasticity) of the pericardium and epicardium.

Since pericardial layers have a limited capacity for stretching, the fast accumulation of a relatively limited amount of fluid can cause a substantial elevation in pericardial pressures, which can potentially be lethal minutes after onset (Fig 7). In contrast, a slow accumulation of a considerable amount of fluid (up to 2 L or more) may have relatively unremarkable effects on ventricular filling.

The diagnosis of pericardial tamponade is a clinical one that is usually confirmed with echocardiography. Typical features are diastolic collapse of the RV free wall, indicating that the pericardial pressure exceeds ventricular pressures; right atrial compression during early systole; exaggerated respiratory variation in mitral and tricuspid inflow as a consequence of increased ventricular coupling; and distention of the inferior vena cava and hepatic veins with diastolic flow reversal (10,41). However, the radiologist should be familiar with the appearance on CT and MR images, although their role in the diagnosis of acute cardiac tamponade is usually limited. Patients with tamponade usually have a large pericardial effusion (or, as already mentioned, other contents such as air or blood) at the time of presentation. Typical findings of tamponade include flattening or inversion of the right atrial or RV wall, with compression of these chambers; inversion of the interventricular septum; distention of the superior vena cava and inferior vena cava; and reflux.

Figure 5: Congenital absence of pericardium. (a) Axial and (b) coronal T1-weighted spin-echo MR images (two heart beats/43, 90° flip angle, 1.4 × 2.0-mm in-plane resolution) show pronounced left-sided rotation and displacement of the heart, filling anterior portion of left hemithorax. The pericardial defect as such is usually difficult to visualize, but large defects such as this can be suspected on the basis of the abnormal location of the heart in the absence of other predisposing factors. (Images courtesy of Luigi Lovato, MD, S. Orsola Malpighi Hospital, Bologna, Italy.)

Cardiac Tamponade
Cardiac tamponade may be a life-threatening condition that requires urgent therapy. It is caused by a pericardial accumulation of fluid, blood, pus, or gas that increases pericardial pressure and
include iatrogenic posttraumatic pericarditis after radiation therapy for breast cancer and mediastinal tumors and cardiac interventions such as cardiac surgery, percutaneous coronary interventions, pacemaker insertion, and catheter ablation (78). Pericarditis may occur early after infarction in approximately 10% of patients and is typically of contrast material into the azygos vein and inferior vena cava on contrast-enhanced CT images (Movie 2 [online]) (72). Finally, cardiac tamponade should be differentiated from effusive-constrictive pericarditis. In these patients with a pericardial effusion at the time of presentation, complaints are caused by a pathologic noncompliant pericardium rather than by the effusion itself. Thus, care should be taken to look for features of constriction, which can occur transiently in the resolution phase, after pericardiocentesis or with organized effusions (35).

**Inflammatory Pericarditis**

Inflammation of the pericardium (pericarditis or inflammatory pericarditis) manifests in many clinical settings and has a wide range of causes (35). Although the true incidence and prevalence of pericarditis are difficult to measure, a prevalence of 1% in autopsy studies suggests that pericarditis may frequently be subclinical (35). In North America and Western Europe, most cases of acute pericarditis are idiopathic (80%-85%) (73). These cases are generally presumed to be viral. Major nonidiopathic causes include tuberculosis, neoplasia, and systemic (generally autoimmune) disease. Each of these specific causes accounts for about 5% of unsellected cases of pericarditis. A high prevalence of tuberculosis-related pericarditis is reported in developing countries, and this disease accounts for up to 90% of cases when the disease is associated with human immunodeficiency virus infection (74–77). In developed countries, leading causes of pericarditis include iatrogenic posttraumatic pericarditis after radiation therapy for breast cancer and mediastinal tumors and cardiac interventions such as cardiac surgery, percutaneous coronary interventions, pacemaker insertion, and catheter ablation (78). Pericarditis may occur early after infarction in approximately 10% of patients and is typically

**Figure 6**

Malignant pericardial effusion in patient with primary pulmonary adenocarcinoma. (a) Frontal chest radiograph shows massive cardiomegaly with retrocardiac opacity in right hemithorax (arrows). (b) Axial contrast-enhanced CT image confirms presence of large pericardial effusion. A centrally excavated mass in right lower lobe (arrow) and bilateral (paraneoplastic) pulmonary emboli (arrowheads) are also present. (c) Coronal contrast enhanced CT image shows extent of the pericardial effusion. Histologic examination of a pericardial biopsy showed presence of malignant cells, likely metastases from pulmonary adenocarcinoma.

**Figure 7**

Acute cardiac tamponade due to ruptured aortic dissection. (a) Axial non-ECG-gated arterial phase CT image shows intimal flap in the ascending aorta extending into the aortic arch, corresponding to Stanford type A aortic dissection. No pericardial effusion is present. (b) Axial CT image acquired 60 seconds after contrast agent administration shows large high-attenuation pericardial effusion corresponding to massive hemopericardium. Weakening of aortic wall with subsequent rupture has led to massive extravasation of iodinated contrast agent to the pericardial sac. The patient died immediately after CT angiography. (Image courtesy of G. F. Gualdi, MD, and C. Valentini, MD, DEA Policlinico Umberto I, Rome, Italy.)
found in patients with transmural infarction ("epistemocardiac pericarditis") (Fig 8) (79). This condition should be differentiated from late postinfarction pericarditis (Dressler syndrome). Whereas acute postinfarction pericarditis has a close temporal relation with the acute event due to the pericardial spread of infarct-related inflammation, Dressler syndrome has an autoimmune origin without a close temporal relation with myocardial infarction.

The manifestation of pericarditis can be acute, but it may manifest in subacute, recurrent, and chronic forms as well. The diagnosis of acute pericarditis is usually clinically suspected on the basis of a combination of typical and often severe chest pain, pericardial rub at physical examination, the presence of widespread ST elevation on the ECG trace. Diagnosis of acute pericarditis is supported by elevation of serologic markers of inflammation with or without evidence of infection (35). Symptoms are mainly related to the severity of pericardial inflammation. Histologically, the inflamed pericardial layers are composed of a highly vascularized granulation tissue with fibrin deposition that may cause fibrinous adhesion of the pericardial layers (20). Although a variable amount of pericardial fluid is usually present, "dry pericarditis" (also known as pericarditis sicca) may found as well and is thought to produce the typical pericardial friction rub on auscultation. The natural history of acute pericarditis is commonly benign, and most patients respond favorably to nonsteroidal anti-inflammatory drugs, although recurrent bouts of pericardial pain are found in up to 30% of patients. Pericarditis may progress to chronic sclerosing pericarditis, which is characterized by fibroblasts and collagen deposition and ultimately leads to end-stage chronic fibrosing pericarditis, which consists of a stiff pericardium that constricts the heart (constrictive pericarditis).

Although echocardiography is considered the modality of choice if additional imaging is needed in the diagnosis of pericarditis or to guide diagnostic or therapeutic procedures (pericardiocentesis or pericardial biopsy), the value of CT and MR imaging is probably underestimated owing to the ability of improved lesion visualization and characterization with these modalities. Imaging findings depend on the pathologic substrate. On CT images, pericardial layers are thickened and show diffuse enhancement after contrast material administration. The attenuation of pericardial fluid may be similar to that of water or may be increased in case of exudative or purulent forms. Signs of cardiac tamponade may be present. In chronic forms of pericarditis, pericardial layers tend to be irregularly thickened and effusions may be loculated owing the presence of adhesions (20).

Similar morphologic findings can be shown on MR images when a combination of T1-weighted and bright-blood sequences is used (Fig 9, Movie 3 [online]). T2-weighted short tau inversion-recovery spin-echo MR imaging is helpful for visualizing edema of the inflamed pericardial layers, while contrast-enhanced MR studies—in particular those obtained with the inversion-recovery gradient-echo sequences—are useful in depicting pericardial inflammation, as has been recently shown in several publications (Figs 8, 9) (3,20,21). Pericardial enhancement reflects inflammation at histologic examination and is correlated with elevated inflammatory markers. Contrast-enhanced MR imaging, moreover, allows evaluation of the extent of inflammation into the surrounding fat and adjacent myocardial tissue.

Because acute pericarditis is a main differential diagnosis of acute chest pain,
as is observed in about 5% of patients admitted to emergency department, the radiologist should be familiar with this entity since an increasing number of cardiac CT scans are performed in the emergency setting to rule out significant coronary artery disease, or to triple rule out, in addition, aortic dissection and pulmonary embolism (73,80); alternatively, these patients undergo MR imaging primarily to exclude ischemia-related myocardial damage (77).

Acute pericarditis is often accompanied by some degree of myocarditis. In clinical practice, both pericarditis and myocarditis may coexist because they share common etiologic agents, mainly cardiotropic viruses (73). However, they are rarely present with equivalent intensity, giving rise to clinical syndromes that are mainly pericarditic or myocarditic. The term myocarditis indicates a primarily myocarditic syndrome, while perimyocarditis indicates a primarily myocarditic syndrome. Recognition of associated myocarditis may be clinically relevant, and is a negative prognostic predictor in patients with pericarditis, often requiring hospitalization and a full etiologic search (75,81). Myocardial involvement can be suspected in the presence of atypical ECG changes associated with transient regional and global wall motion abnormalities and increase in the levels of cardiac enzymes.

MR imaging has today become the reference for diagnosis and follow-up of myocarditis (82,83). This technique allows appreciation of the spread of inflammation throughout the heart, assessment of disease activity, and evaluation of the functional implications of the myocardial involvement. In particular functional myocardial abnormalities may not be prominent and, therefore, often not recognized at echocardiography (44). Although patterns of myocardial enhancement similar to those seen on MR images have been described for CT images in patients with myocarditis, the role of CT in this diagnosis is currently undefined (84).

Constrictive Pericarditis

Constrictive pericarditis reflects a condition in which the compliance of the pericardium is decreased, which may result in impaired ventricular filling, severe diastolic dysfunction, and right heart failure (33,35). Although subacute and even acute forms of pericardial constriction have been described, this disease usually clinically manifests years after an initial pathologic trigger. The risk of constrictive pericarditis after acute pericarditis is relatively low in viral or idiopathic acute pericarditis (< 0.5%) but is relatively frequent in purulent and tuberculous pericarditis, particularly in patients with an incessant course and large pericardial effusions (85). The spectrum of causes of pericardial constriction has shifted over time from infectious causes (particularly tuberculous) to postirradiation and postoperative forms, which have now become the most frequent causes of the disease (86,87).

The diagnosis of constrictive pericarditis, even in the modern era, remains challenging. In clinical practice today, a combined approach is used to evaluate the morphologic pericardial (and cardiac) abnormalities in conjunction with assessment of the functional and hemodynamic consequences. Although this is commonly achieved by using a multimodality imaging approach, comprehensive MR imaging offers an almost complete appreciation of constrictive pericarditis, with exception of demonstration of...
pericardial calcifications. First, all other causes of (right) heart failure (eg, pulmonary hypertension, severe tricuspid insufficiency, myocardial infarction) should be excluded. Second, it should be determined whether the pericardium is causing constriction, thereby impeding cardiac filling. Third, the optimal treatment needs to be determined (eg, pericardial stripping vs medical treatment) (40). In this workup, which is tailored to each patient’s specific needs, the differentiation of constrictive pericarditis from restrictive cardiomyopathy is crucial.

Although, to our knowledge, there are no prospective studies in which medical and surgical management are compared, pericardiectomy is the accepted treatment to improve patient hemodynamics. Long-term survival after pericardiectomy is related to the underlying cause. In particular, postoperative survival in postirradiation constrictive pericarditis is poor (86). It is also important to mention that a number of patients with constrictive pericarditis may not benefit from pericardiectomy because of associated myocardial compliance abnormalities (ie, restrictive cardiomyopathy), myocardial atrophy after prolonged constriction, or other myocardial processes, thus emphasizing the need for a complete evaluation of the heart and pericardium (88).

Morphologic abnormalities.—The typical morphologic manifestation of constrictive pericarditis is that of generalized thickening of the pericardium with or without calcifications. Abnormalities are usually most pronounced over the right heart (RV and anterior atrioventricular groove), and pericardial delineation is often irregular (Fig 10). The underlying cardiac cavities may be constricted by the abnormal pericardium, having a flattened or tubular-shaped appearance (Fig 10). Indirectly, as a result of the increased cardiac filling pressures, unilateral or bilateral atrial enlargement, dilatation of caval and hepatic veins, pleural effusion, and ascites are encountered. These abnormalities may be easily recognized on CT images. The thickened fibrotic and/or calcified pericardium has low signal intensity on T1-weighted and T2-weighted spin-echo MR images and at cine imaging. In the end stage of chronic fibrosing forms of constrictive pericarditis there is no enhancement after contrast material administration, whereas pericardial enhancement is suggestive of residual inflammation (3,20,21). Differentiation between pericardial thickening and effusion is usually straightforward on MR images.

Although pericardial thickness is traditionally used as an important criterion for constrictive pericarditis (ie, pericardial thickness ≤ 2 mm is normal, > 4 mm is suggestive of pericardial constriction in patients with the appropriate clinical presentation, and > 5–6 mm is highly specific for constriction [52–54]), this concept has been increasingly challenged and should be put in perspective with regard to recent findings. Although fibrosis with or without calcification is nearly always present at pathologic examination (96%) in cases of pericardial constriction, the maximal pericardial thickness shows a wide range (1–17 mm; mean, 4 mm) with up to 20% of patients showing a normal (≤2-mm) thickness (Fig II, Movie 4 [online]) (54,74).

The findings from two recent studies (3,21) indicate that evolution toward an end-stage irreversible chronic fibrosing pericarditis is characterized by a thinning of the chronically inflamed pericardium. In the study by Feng et al (3), pericardial thickness was significantly less in patients with persistent constrictive pericarditis than in those with reversible constrictive pericarditis (2 mm ± 1 vs 4 mm ± 1; P < .001). Similar results were reported by Zurick et al (21), who found a thinner pericardium in patients with end-stage constrictive pericarditis than in those with evidence of persistent chronic inflammation (3.1 mm ± 1.7 vs 4.6 mm ± 1.4; P = .021). As mentioned previously, both studies showed the value of contrast-enhanced MR imaging in depicting residual inflammation in patients with constrictive pericarditis (20). These patients have “reversible” or “transient” forms of constrictive pericarditis that respond to optimized anti-inflammatory treatment. This underscores the fact that not all patients with constrictive pericarditis need to undergo pericardiectomy (89). It should also be noted that the degree of pericardial thickening is only weakly related to the degree of cardiac constriction (35).

Pericardial calcifications are considered an important sign of constrictive...
Functional and hemodynamic consequences.—Encasement of the heart by a noncompliant, rigid pericardium leads to (a) dissociation between intracardiac and intrathoracic pressure, which isolates the heart from normal respiratory changes in intrathoracic pressure; (b) increased ventricular coupling; and (c) increased cardiac filling pressures with pressure equalization in all four cardiac chambers. Although the hemodynamic consequences are assessed in clinical practice by means of echocardiography and cardiac catheterization, MR imaging has great potential, because information regarding pericardial-cardiac morphology and tissue characteristics can be merged with functional hemodynamic information (8,35,75). Phase-contrast MR imaging of the tricuspid valve in flow shows a restrictive filling pattern of enhanced early filling and decreased or absent late filling, depending on the degree of pericardial constriction and increased filling pressures. Also, flow in the inferior vena cava shows restrictive physiology with diminished or absent forward—or even reversed—systolic flow, increased early diastolic forward flow, and late reversed flow. Constrictive complications such as the intramyocardial extent of the fibrocalcific process, which may hamper the success of a pericardiectomy (Fig 12; Movies 5, 6 [online]). Preoperative CT may be useful to provide a detailed depiction of both the severity of thickening and the presence and location of calcifications, thus allowing better surgical planning and stratification of procedural risk (2). Pericardial calcifications, however, are less common these days than in the past, which is probably related to the decrease in rates of tuberculosis and the increase in iatrogenic causes of constriction. The authors of two recent studies (74,90) reported pericardial calcifications in 27% and 28% of patients with histologically confirmed constrictive pericarditis. Since tuberculosis was ruled out in nearly all these patient populations, these results suggest that the occurrence of calcification should be considered a nonspecific response to chronic inflammation (90).

Finally, patients clinically suspected of having constrictive pericarditis may show atypical manifestation of pericardial abnormalities such as effusive-constrictive pericarditis manifesting with a combination of symptoms related to tamponade and constriction (91,92). Other atypical forms include localized and occult forms of constrictive pericarditis (93).
pericarditis, in contrast to restrictive cardiomyopathy, is typically characterized by a strong respiratory-related variation in cardiac filling (ie, enhanced RV filling on inspiration, enhanced LV filling on expiration). Real-time phase-contrast MR imaging is an attractive alternative to Doppler echocardiography to assess the effects of respiration on cardiac filling (19).

The lack of pericardial stretch in constrictive pericarditis leads to increased ventricular coupling, which is characterized by septal flattening or inversion (“septal bounce”) at early diastolic ventricular filling (Movie 3 [online]) (16,36,94,95). Because of the dissociation between intrathoracic and intracardiac pressure, this pattern is strongly influenced by respiration (35,95). Septal abnormalities are most pronounced at the onset of inspiration and rapidly fade away, while at onset of expiration an opposite (right-sided) septal shift occurs. Abnormalities are most pronounced in the basal septum, leading to an S-like septal motion on a horizontal long-axis view (Movie 3 [online]).

We exploited the clinical potential of novel real-time cine sequences to study the effects of free breathing on ventricular coupling (17). Patients with constrictive pericarditis showed the typical respiratory pattern of septal abnormalities, while those with restrictive cardiomyopathy showed a pattern similar to that of healthy volunteers (Figs 11, 13; Movie 4 [online]). Quantification of the total septal excursion between inspiration and expiration was very helpful in differentiating between constrictive pericarditis and restrictive cardiomyopathy (Fig 13) (18). Moreover, patients with inflammatory pericarditis also often show increased septal excursion, most likely related to the decreased compliance of the inflamed pericardial layers. The added value of real-time imaging during free breathing to evaluate the hemodynamic effect of the pericardium on cardiac filling has become accepted as an essential part of an MR examination (96,97).

This sequence, which takes approximately 10 seconds of extra measurement time, is of great help in clinical routine practice. Two extreme examples illustrate its usefulness. On the one hand, a pericardium may have a normal or near-normal thickness but cause severe constriction and thus lead to marked increase of ventricular coupling, while on the other hand a thick pericardium may not be constrictive and can show normal ventricular coupling at real-time cine imaging. Without this essential information one risks wrongly categorizing patients. Finally, MR tagging enables one to better appreciate the fibrotic fusion of the pericardial layers and the extent of the fibrocalcific process into the underlying myocardium (Fig 14; Movies 7, 8 [online]) (48). Whereas in normal pericardium, tag lines rapidly become discontinuous during the cardiac cycle due to shear motion of the inner and outer pericardial layer, persistence of these tag lines is indicative of fibrotic fusion.

**Pericardial (Pseudo)-Masses**

Pericardial masses represent a heterogeneous group of cystic lesions, hematomas, complex organized effusions, masslike structures, and primary and secondary malignancies that affect or involve the pericardium. CT and MR imaging are often necessary in the diagnostic workup, because they provide an accurate description of the pericardial abnormalities and the relationship to the surrounding structures and facilitate understanding of the underlying cause, establishment of the diagnosis and differential diagnosis, and assessment of complications such as cardiac tamponade (Fig 15, Movie 8 [online]). Because of the superior anatomic detail, these techniques allow one to easily differentiate “true” pericardial masses from “pseudo” masses—for example, the presence of abundant epicardial fat, which was originally misinterpreted at transthoracic echocardiography (Fig 14).

While primary pericardial tumors are extremely rare, secondary involvement has been described in up to 10%–12% of patients (Fig 16) (98–100). Primary tumors are more frequently malignant and include mesothelioma, angiosarcoma, liposarcoma, and lymphoma. Benign tumors include fibroma, teratoma, hemangioma, and lipoma and are extremely rare. Malignant mesothelioma is the most common primary lesion and is usually characterized by the presence of hemorrhagic effusion with pericardial nodular lesions or plaques (Fig 17).
is needed to establish the definitive diagnosis.

Other entities that have a masslike appearance are pericardial hematoma and pericardial gossypiboma. The CT and MR imaging characteristics of a pericardial hematoma depend on the age of the collection (41,100). The possibility of a pericardial gossypiboma or foreign body granuloma (eg, surgical sponge) should always be considered in patients with a history of cardiac surgery. A final entity to be mentioned is pericardial fat necrosis. This benign entity of unknown cause typically manifests with sudden onset of chest pain, mimicking other diseases such as myocardial infarction and pulmonary embolism. The typical CT appearance is a lesion of fat attenuation surrounded by the increased attenuation of the anterior mediastinal paracardiac fat adjacent to the pericardium (102).

**Conclusion**

Although pericardial disease often represents a diagnostic challenge, CT and MR imaging have notably contributed in the demonstration of pericardial pathologic conditions and greatly improved our understanding of this enigmatic part of the heart. Since pericardial diseases have substantial morbidity and...
mortality, both techniques have an increasingly important role in decision making, particularly in determination of the optimal treatment for patients with constrictive pericarditis.

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References


Figure 17: Pericardial mesothelioma in a 42-year-old man with history of pericardial effusion (presumably postviral) complicated by constriction. CT scans were obtained without ECG gating. (a) Before pericardiectomy, diffuse pericardial thickening can be seen (arrows). (b, c) Four years later, the patient presented with symptoms of right heart failure similar to those before surgery. Follow-up CT scans show several nodular masses diffusely spread around in the pericardial sac (arrows). Biopsy showed epithelial mesothelioma. (Image courtesy of Scott H. Yang, MD, PhD, Santa Rosa, Calif.)


