



Contemporary Surgical Management of Pericardial Disease

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Abstract

Disease of the pericardium, the membranous lining of the heart, can be caused by a variety of different pathologic processes. These include congenital absence, cysts, tumors, inflammation, and effusions. Pericardial derangements can have severe consequences on cardiac function if left untreated and in select cases require surgical correction to prevent cardiovascular collapse. Available surgical therapies range from minimally invasive drainage to median sternotomy. This article reviews common pathologies of the pericardium and their underlying causes. In addition, the diagnostic work-up and contemporary surgical management of pericardial disease is reviewed with detailed analysis of key technical steps.

Keywords: Pericardium; Pericarditis; Pericardial Effusion; Pericardial Tamponade; Cardiac Surgery

Introduction

Successful operations on the pericardium are an achievement of the twentieth century, and in times of antiquity, it was inconceivable to perform pericardiectomy and have a living patient, or escape evil biblical references. The Dead Sea Scrolls (circa 1AD) describes the Wicked Priest as one who “did not circumcise the foreskin of his heart” (Habbakuk 2:16). The pericardium, which envelops the heart and proximal great vessels, consists of an outer fibrous pericardium and inner double-layered sac termed the serous pericardium. The parietal pericardium lines the fibrous layer, with a thin visceral epicardial layer lining the heart [1]. The space between the parietal and visceral layers creates the pericardial cavity which normally contains between 15 mL to 50 mL of fluid in healthy patients. The normal thickness of the pericardium is less than 4 mm, usually between 1 mm to 2 mm in most patients [2]. Although not essential (normal cardiac function can be maintained in its absence), the pericardium functions to provide mechanical protection for the heart and a smooth lubricated surface to decrease friction during myocardial contraction [3]. Surgeons today are most commonly faced with the following pathologies of the pericardium: Congenital absence of the pericardium, pericardial cysts and tumors, pericarditis (acute and chronic), pericardial effusion and tamponade, and constrictive pericarditis.

Pericardial Pathology

Congenital absence of the pericardium

Congenital absence of all or part of the pericardium is rare. Complete absence is generally asymptomatic. Partial defects are generally left-sided and may cause symptoms such as dyspnea or positional chest pain, but are most concerning for risk of cardiac herniation. Chest X-ray will demonstrate rotation and leftward displacement of the heart in both partial and complete absence. Echocardiography demonstrates prominence of right-sided cardiac chambers and abnormal septal motion. Computed Tomography (CT) scanning and Magnetic Resonance Imaging (MRI) will demonstrate rotation and may show presence of lung parenchyma between the aorta and pulmonary artery. Complete absence of the pericardium or partial absence on the right-side do not require treatment, whereas left-sided defects overlying the left ventricle should be intervened upon to decrease the risk of herniation or compression. Surgical options consist of pericardiectomy (via median sternotomy, or thoracotomy/thoracoscopy) or alternatively, repair of the defect with Dacron, Gore-Tex, or bovine pericardium [4].

Pericardial cysts

Pericardial cysts are generally congenital, but may occasionally be inflammatory. Most commonly, they will be found as an incidental mass on chest X-ray. The majority are found at the

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right cardiophrenic and costophrenic angle. Echocardiography, CT or MRI can be utilized to determine cyst character, and fluid density [5]. Asymptomatic cysts with a fluid density consistent with a transudate may be observed. Symptomatic simple cysts may be aspirated and instilled with an ethanol sclerosant, or resected thoracoscopically. Complex cysts which are concerning for malignancy should be excised *via* thoracotomy or thoracoscopy.

Pericardial tumors

Pericardial tumors generally present with concomitant effusions and are most commonly secondary to metastatic disease. Primary tumors include lipomas, leiomyomas and heterotopic tissue, as well as malignant tumors of which mesothelioma is most common. CT and MRI can distinguish cystic disease from solid masses. All solid pericardial masses should be resected unless secondary to metastatic disease with poor prognosis. Mesothelioma is generally aggressive with contiguous spread within the mediastinum and to adjacent structures. Even with aggressive resection, survival is poor [6,7].

Pericarditis

Acute pericarditis generally presents with chest pain and a pericardial friction rub on physical examination. EKG may show ST-segment elevation in all leads except AVR and V1. The majority of cases are idiopathic or viral in etiology and managed with high dose anti-inflammatory therapy such as aspirin or other Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), or steroids in refractory cases [8]. Surgical management of acute pericarditis is generally limited to acute bacterial or fungal pericarditis. In acute bacterial pericarditis, the causative organisms are most commonly *Staphylococcus*, *Streptococcus* and gram-negative organisms. These result from either contiguous spread from adjacent structures or bacteremia. In addition to intravenous antibiotics, pericardial drainage is necessary. Catheter based pericardial drainage should be the initial approach with intrapericardial instillation of fibrinolytic agents if a complex effusion is present. Failure of catheter-based drainage or persistence of infection warrants surgical exploration and drainage. Both subxiphoid pericardial drainage and drainage *via* left anterior thoracotomy/thoracoscopy are utilized with success. Median sternotomy is ideally avoided given the risks of subsequent sternal infection [9].

Occasionally patients may present with recurrent or chronic (duration greater than 3 months) episodes of pericarditis refractory to medical management. If these episodes are highly symptomatic, and ideal medical management has failed, pericardiectomy may be indicated. Class IIa recommendations by the European Society of Cardiology support a possible role for pericardiectomy in these cases, but caution that the efficacy of pericardiectomy is questionable and symptoms may often recur after 6 months, reserving surgery for only exceptional cases [10].

Pericardial effusions

Pericardial effusions develop as a result of an imbalance between fluid production and resorption, or secondary to introduction of fluid into the pericardial cavity such as blood. The majority of effusions are secondary to pericarditis, malignancy, infection, trauma, or metabolic disturbances such as uremia or myxedema. Small effusions (less than 250 mL) generally do not result in hemodynamic disturbances, but those which are greater require drainage. Indications for drainage include: Purulent infection, hemodynamic compromise or tamponade, persistence for greater than three months, to aid in diagnosis, and for

progressive enlargement. Pericardiocentesis is sufficient to manage most effusions, but surgical drainage *via* pericardial window or pericardiectomy may be necessary in certain situations. As noted above, bacterial infection refractory to catheter-based drainage and antibiotic therapy necessitates surgical drainage. Malignant effusions are ideally initially managed *via* pericardiocentesis, followed by pleuropericardial drainage for recurrence at which time concomitant pleural effusions may be drained. Effusions secondary to radiation therapy will generally resorb over time, but if symptomatic or recurrent, may require partial pericardiectomy. Uremic effusions should be managed initially with intensification of dialysis with pericardial window or partial pericardiectomy reserved for refractory and recurrent cases [11,12].

Pericardial tamponade may result secondary to an effusion as described, pericardial mass, or from pneumopericardium which is most often a result of air from the lung after chest trauma, bleb rupture, or positive pressure ventilation. Any of the above may induce cardiac compression resulting in hemodynamic compromise. Increased pressure within the pericardium results in decreased right heart filling, followed by decreased pulmonary venous return to the left atrium and subsequent decreases in stroke volume. Diminished diastolic compliance leads to additional decreases in stroke volume and cardiac output. The sum result is arterial hypotension and impaired coronary and systemic perfusion. Physical examination may demonstrate tachycardia, hypotension, jugular venous distension, muffled heart sounds and pulsus paradoxus, which is defined as the decrease in systolic blood pressure of greater than 10 mmHg with inspiration. Richard Lower (1631-1691), a Cornish physiologist, was the first to describe pulsus paradoxus and the mechanism consistent with the clinical state of cardiac tamponade. Hemodynamic monitoring parameters may include elevated central venous pressure, near equalization of central venous, pulmonary arterial and capillary wedge pressure, and decreased cardiac output. These parameters may not be present in post-cardiac surgical patients in whom the pericardium and pleural spaces are open. Echocardiography is a useful adjunct, but transthoracic imaging may be suboptimal in postoperative patients, and transesophageal echocardiography may not always be immediately available. On echocardiography, absence of any chamber collapse has a 92% negative predictive value, and abnormal hepatic venous flow has an 82% positive predictive value. The presence of late right atrial diastolic collapse, early right ventricular diastolic collapse, and left atrial collapse, and ventricular interdependence all support a diagnosis of tamponade [13,14].

Emphasis must be placed on pericardial tamponade as a clinical diagnosis, as the presences of any of the above diagnostic criteria are variable. Patients with tamponade early after cardiothoracic surgery should undergo immediate surgical exploration. In settings outside of the perioperative period, patients presenting with tamponade should be managed with pericardiocentesis and catheter-based drainage. Surgical drainage of the pericardium is reserved for failure of percutaneous techniques, or recurrent tamponade. If percutaneous drainage is not an option, a subxiphoid pericardial window can be created under local anesthetic to avoid the hemodynamic compromise which can occur with induction of general anesthesia [15].

Constrictive pericarditis

Constrictive pericarditis, or pericardial constriction as it may be termed, may be idiopathic or secondary any condition causing pericardial inflammation. Most commonly, the etiology is idiopathic,

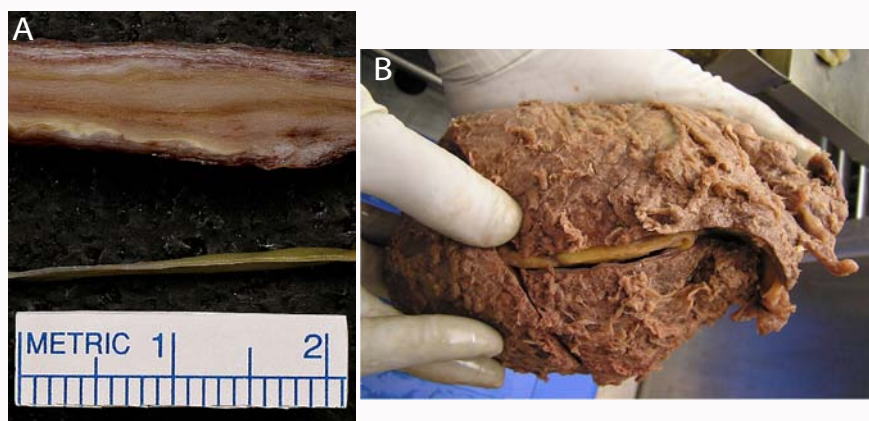


Figure 1: **A)** Thickened, calcified pericardium secondary to constrictive pericarditis relative to normal pericardium (photographs courtesy of William C. Roberts, MD, Baylor Heart and Vascular Institute), and **B)** Gross specimen on autopsy of a heart with constrictive pericarditis (photograph courtesy of Jack H. Lichy, MD, Armed Forces Institute of Pathology and Veterans Affairs Medical Center, Washington, D.C.).

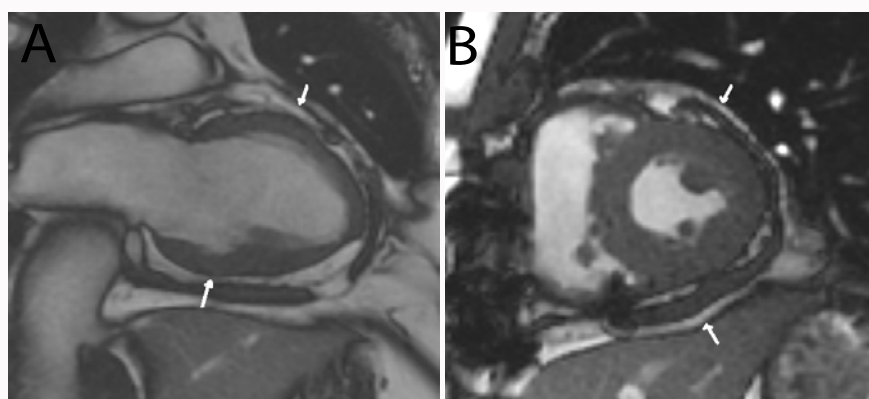


Figure 2: Cardiac Magnetic Resonance Imaging (cMRI) of a thickened pericardium in short axis (**A**) and two-chamber (**B**) in evaluation for constrictive pericarditis. (Arrows depict thickened pericardium) (cMRI images courtesy of Drs. Michael Greenberg and Federico Mordini, Section of Noninvasive Imaging, Veterans Affairs Medical Center, Washington, D.C.).

post cardiac surgery, post pericarditis, or after mediastinal radiation therapy. In the immunocompromised and patients from developing countries, tuberculosis is a common etiology. The pericardium often becomes thickened and scarred with calcification (Figure 1A, 1B). Giovanni Battista Morgagni (1682-1771), whose work dominated the 18th century with numerous observations, noted pathologic findings of pericardial calcification and compression by hemopericardium. The resulting decrease in the volume of the pericardial sac relative to the heart results in impaired cardiac filling. Patients present with symptoms of volume overload including peripheral edema, hepatomegaly, pleural effusions, ascites, and anasarca. Chest X-ray may demonstrate calcification of the pericardium, often seen on lateral views between the right ventricle and diaphragm. Differentiation from restrictive cardiomyopathy is essential as presenting symptoms may be similar. On history, presence of systemic disorders such as amyloidosis, sarcoidosis, hypereosinophilic syndromes, or endomyocardial fibrosis should raise suspicion for restrictive cardiomyopathy. The most useful studies for diagnosis include echocardiography, CT, MRI and right and left heart catheterization. Table 1 lists key findings on echocardiography and cardiac catheterization in constrictive pericarditis and restrictive cardiomyopathy. CT and MRI may demonstrate pericardial thickening and calcification with minimal effusion, but notably, almost 20% of patients with constrictive pericarditis may have a normal thickness pericardium on imaging

(Figure 2A, 2B) [16,17].

Once a diagnosis of constrictive pericarditis has been confirmed, differentiation must be made between classic chronic constrictive pericarditis and subacute constrictive pericarditis. Subacute constrictive pericarditis occurs weeks to months after the initial insult (such as cardiac surgery) and the transition to the constrictive process may occur slowly over this time period. Symptoms are similar between the two, but calcification is more often absent in the subacute form. The subacute form should be observed for at least 3 months while on appropriate anti-inflammatory therapy as a subset of these patients improve over time and will not require surgery. For those patients who do not respond and those with the classic form, pericardiectomy is indicated. The surgery should be performed before the onset of New York Heart Association (NYHA) class IV heart failure as these patients have a high perioperative mortality [18].

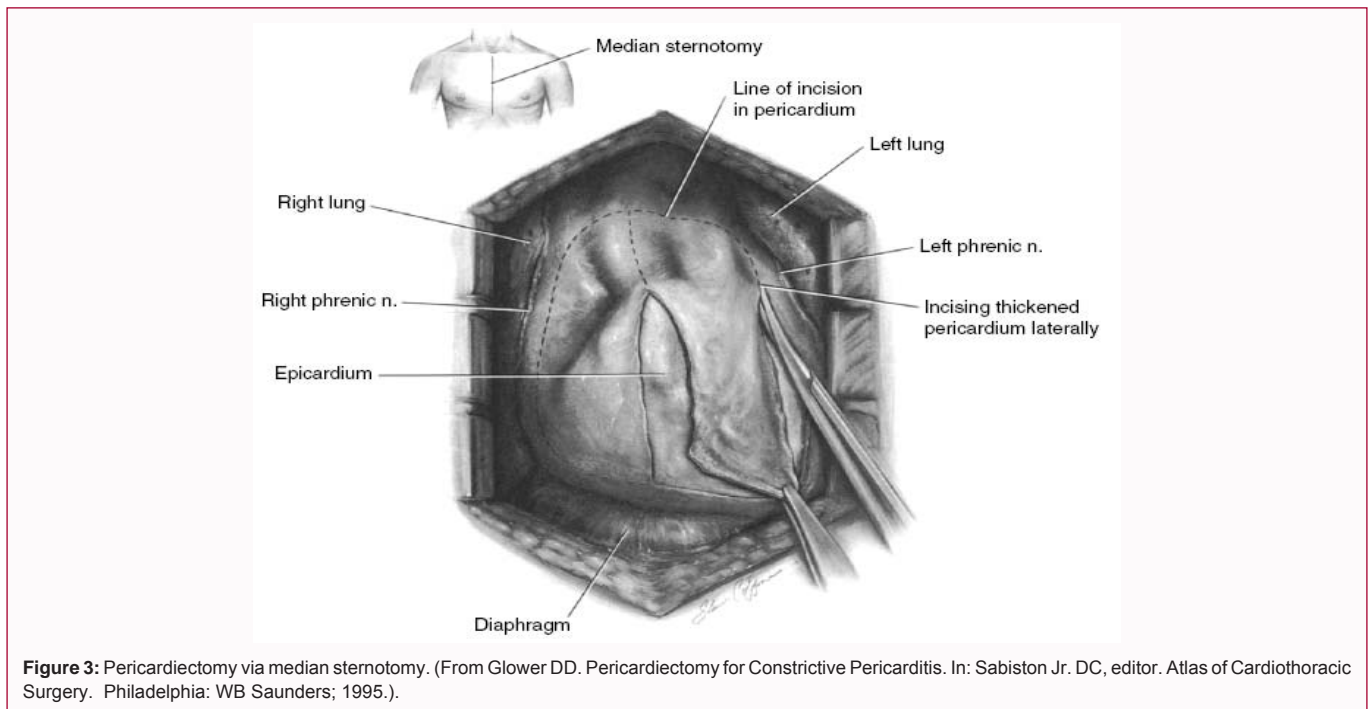
Surgical Techniques

Pericardiocentesis

Pericardiocentesis is indicated for pericardial effusions in which etiology remains undefined, those refractory to medical management, those highly suspicious for purulent infection, tuberculosis, or malignancy, and tamponade outside of the perioperative setting. The patient is placed supine and anesthesia is provided with local agents.

Table 1: Characteristics of constrictive pericarditis vs. restrictive cardiomyopathy.

Constrictive Pericarditis	Restrictive Cardiomyopathy
Echocardiography	
Thickened pericardium	Normal pericardium
Abnormal ventricular septal motion	Normal ventricular septal motion
Dilation and decrease or absence of IVC/hepatic vein collapse	
Restrictive mitral and tricuspid inflow velocities with respiratory variation	No respiratory variation of mitral inflow velocities
Preserved or increased mitral annular early diastolic velocity	Decreased mitral annular early diastolic velocity
Increased hepatic vein flow reversal with expiration	Increased hepatic vein flow reversal with inspiration
Cardiac Catheterization	
Increased atrial pressures	Increased atrial pressures
Equalization of end-diastolic pressure	Equalization of end-diastolic pressure
Dip and plateau diastolic pressure tracing ("Square root sign")	Dip and plateau diastolic pressure tracing ("Square root sign")
Enhanced ventricular interdependence	No ventricular interdependence
Respiratory variation of right and left ventricular filling	
Discordant change in right and left ventricular systolic pressure with respiration	Concordant change in right and left ventricular systolic pressure with respiration



Transthoracic echocardiography or fluoroscopy are useful adjuncts to select point of entry. A long spinal needle is inserted into the pericardial space starting to the left of the xiphoid process inferior to the left costal margin and advanced towards the left mid scapula. Alternative points of entry may be selected based on imaging. Attachment of an EKG electrode to the needle can be useful to avoid myocardial puncture by monitoring for negative deflection of the QRS complex which indicates myocardial contact. Upon aspiration of pericardial fluid, a guidewire is passed into the pericardial space. Injection of agitated saline can help confirm location if echocardiography is available. Utilizing a Seldinger technique, a percutaneous drainage catheter may be left in place. Attempts should be made to drain all fluid during placement with the drain left in place for 48 h to 72 h or until drainage is less than 30 mL/24 h. If pericardial biopsy is necessary, a dilator sheath may be passed over

a wire during initial entry followed by usage of a biopptome under echocardiographic guidance. Rates of complication range from 5% to 50%, depending upon the presence of coagulopathy and use of image guidance. Simple aspiration is associated with recurrence rates of over 50%, with significant reduction if a catheter is left in place [19].

Pericardial window

Creation of a pericardial window is indicated for recurrent tamponade, recurrent moderate to large effusions which are symptomatic and refractory to medical management, and in the setting of trauma. Additionally, they may be created to obtain pericardial tissue for biopsy. The most commonly employed techniques are the subxiphoid and transpleural approach [20].

For the subxiphoid approach, local anesthetic is preferred in cases of large effusions with hemodynamic compromise as induction of

general anesthesia may result in hemodynamic collapse; otherwise, general anesthesia is acceptable. A vertical 6 cm to 8 cm incision is started at the cephalad aspect of the xiphoid process and dissection is carried down to the xiphoid and Linea Alba. The Linea Alba is divided and the xiphoid can be resected to facilitate exposure or retracted superiorly. Blunt dissection is carried out until the pericardium is visualized. The pericardium is then grasped with a tissue clamp or forceps and opened. A suction trap is useful to obtain fluid for analysis. A 4 cm piece of pericardium should be excised under direct vision. Digital inspection for loculations should precede advancement of suction catheters. After clearing any loculations and drainage of all fluid/clot, the window may be extended to open into one or both pleural spaces. A right-angled chest tube or small-bore flexible drainage catheter should be placed on the diaphragmatic surface and a pleural tube if the pleural space has been entered. All tubes should be brought out through separate incisions from the initial point of entry. The Linea Alba should be closed, followed by remaining soft tissue. Drains should remain in place until drainage is less than 100 cc/24 h. Mortality rates are low after a subxiphoid approach (1%) with recurrent effusions occurring in 10% to 25% of patients [21].

The transpleural approach may be undertaken *via* left anterior thoracotomy or Video Assisted Thoracoscopy (VATS) from either side. Additional cardiopulmonary reserve is required with this approach as general anesthesia and single-lung ventilation are needed. The VATS approach may be preferable in patients with concomitant pleural effusions as these may be addressed at the time of surgery. For the open approach, the patient is placed supine with the left side elevated 30 degrees to 45 degrees. A standard 5 cm to 10 cm left anterior thoracotomy is created in the 4th or 5th interspace taking care to avoid the left internal mammary artery. The pericardium is incised 1 cm to 2 cm anterior to the phrenic nerve and a 4 cm specimen excised. Pericardial drain placement is unnecessary as pleural tube placement is sufficient. The VATS approach may be performed using 2 ports to 3 ports. The initial port should be placed

posteriorly along the posterior axillary line in the 5th interspace with one or two additional ports placed along the anterior axillary line in the 4th and 6th interspaces. Alternatively, two ports may be used with a retracting grasper passed parallel to the thoracoscope through the same port. Perioperative mortality rates are similar to the subxiphoid approach, and rates of recurrent effusion may be lower (5%) than the subxiphoid approach, although this remains controversial [22].

Pericardiectomy

Pericardiectomy is indicated for constrictive pericarditis, and may be indicated for selected patients with recurrent tamponade, frequent highly symptomatic recurrence of pericarditis refractory to medical management, and failure of fibrinolytics in bacterial purulent pericarditis. The most common approach is *via* median sternotomy, but left anterolateral thoracotomy and bilateral thoracotomies have been used [23].

Pericardiectomy *via* median sternotomy has been our preferred approach (Figure 3). After induction of general anesthesia, a transesophageal echocardiography probe is positioned and a pulmonary arterial catheter placed. The groins are prepped and exposed in case Cardiopulmonary Bypass (CPB) is urgently required as central cannulation can be difficult if extensive adhesions are present. We prefer to perform the procedure without the use of cardiopulmonary bypass as bleeding from the epicardial surface can be extensive in the setting of anti-coagulation. Nevertheless, we have CPB available for all cases. Cardioplegic arrest is not necessary and the procedure can be performed on CPB with a beating heart. CPB may be most helpful in patients with a prior sternotomy, extensive pericardial calcification, and in patients where there is difficulty with epicardial dissection as CPB will reduce intracardiac pressure. A full median sternotomy is performed and the sternum is partially opened with a retractor. Care should be taken to ensure the innominate vein is not densely adhered as wide opening of the retractor may result in significant bleeding if not freed. Adhesions to the inner table of the

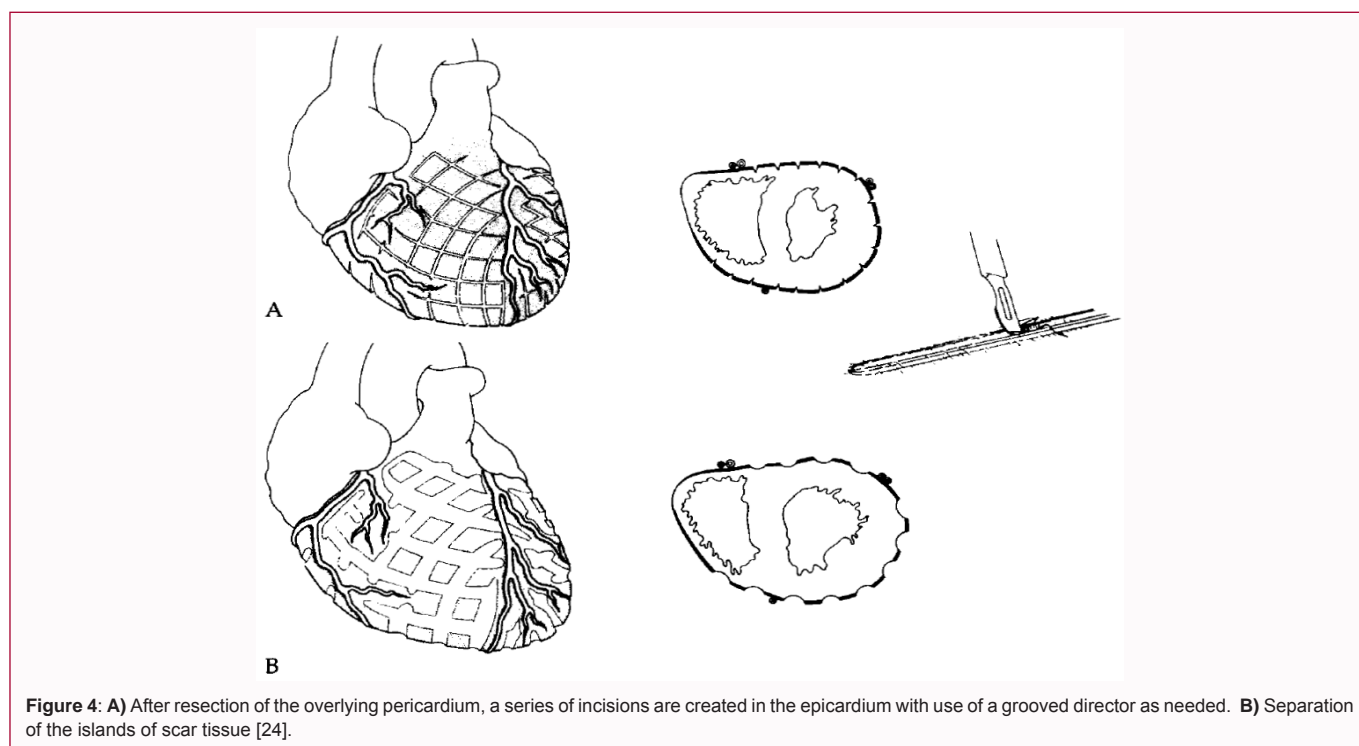


Figure 4: A) After resection of the overlying pericardium, a series of incisions are created in the epicardium with use of a grooved director as needed. B) Separation of the islands of scar tissue [24].

sternum are freed next. The parietal pericardium is incised sharply at the diaphragmatic surface. Ideally, a plane between the epicardium and parietal pericardium should be identified to minimize bleeding. If the appropriate plane cannot be readily identified, a scalpel should be used to incise the parietal pericardium until bulging myocardium is visible. With the assistant placing gentle vertical traction on the pericardium, a flap should be started at this point and raised. The dissection plane should be carried out sharply with use of Metzenbaum scissors freeing the left ventricle first to avoid sudden volume overload of the right ventricle and ensuing right ventricular failure. The dissection should be carried 1cm anterior to the phrenic nerve. Identification of the phrenic nerve may be difficult, and require pleural opening to facilitate identification of its course. After freeing the left ventricle and diaphragmatic aspect of the pericardium, dissection is carried to the contralateral side 1 cm anterior to the phrenic nerve. Subsequent to freeing the ventricles, the atria and cavae are freed if there is constriction present. The superior extent of dissection is carried out to the base of the great vessels. Once freed, the pericardial flaps should be excised with use of electrocautery to minimize bleeding. Patches of pericardium may be left in place if dissection is difficult to avoid myocardial injury and bleeding. Epicardial calcium plaques should be removed and this may be facilitated with use of freer elevators and rongeurs. Freeing of the pericardium over the ventricles provides the greatest hemodynamic benefit. Aggressive attempts to free atria may not be warranted if dissection is difficult given the limited benefit. Pericardiectomy posterior to the phrenic nerves has been described, but the necessity for cardiopulmonary bypass, added risks of bleeding and unclear hemodynamic benefits limit its application. Chest tubes should be left in place as there may be postoperative epicardial bleeding and associated pleural effusions drained.

Pericardiectomy can be performed *via* left anterior thoracotomy, providing adequate exposure of the left ventricle, but limited right ventricular and atrial exposure. The patient is positioned with the left chest elevated 45 degrees. The left groin should be prepped for peripheral cannulation in case CPB is needed. An anterolateral thoracotomy is created in the 5th interspace. The lung should be mobilized cephalad and this may be facilitated by releasing the inferior pulmonary ligament. A linear incision is extended from the pulmonary artery to the diaphragm, 1 cm anterior to the phrenic nerve. The pericardium is dissected free from the left ventricle to the furthest extent visible over the right ventricle. Visualization of the pericardium posterior to the phrenic nerve may allow for its resection, taking care to leave a 1 cm width of pericardium around the nerve.

A small subset of patients may demonstrate continued pericardial constriction physiology (lack of improvement in central venous, pulmonary arterial, and pulmonary capillary wedge pressure) after pericardiectomy. This can be due to a constricting layer of visceral pericardium/epicardium. The “Waffle” procedure as originally described by Heimbecker and colleagues in 1983 involves creation of a series of longitudinal and transverse incisions in the epicardium leaving 1 cm² islands of scar tissue (resembling a waffle pattern) [24]. A grooved director is placed deep to the epicardial layer taking care to avoid injury to the coronary arteries which are at significant risk, followed by incision of the tissue above the director with a scalpel. This is carried out over the entire right and left ventricular surface (Figure 4A, 4B).

Postoperatively, patients may suffer from a low cardiac output syndrome requiring inotropic or mechanical support but myocardial

ischemia secondary to inadvertent coronary injury should be ruled out as well. Perioperative mortality rates range from 5% to 14%, with the lowest rates for NYHA class I and II patients (1%), 10% for class III, and 46% for class IV. Most patients (60% to 90%) experience an improvement in hemodynamics and NYHA functional class [25].

Conclusion

Pathology of the pericardium can range from pericardial masses to inflammation and pericardial effusion. These different disease processes can result in a restrictive or obstructive physiology, preventing normal cardiac function and in certain cases cardiovascular collapse. It is imperative to recognize these processes and manage them appropriately. Management can involve surgical intervention, and the various indications and technical approaches have been reviewed in fastidious detail above. As our knowledge of cardiac physiology continues to expand, future research on the long-term clinical benefits of surgical correction of pericardial disease should be a top priority in the field of cardiothoracic surgery.

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