provides another useful site of generator placement that may be useful in small infants and neonates, patients in whom the need for pacemaker has been decided preoperatively, and those who have erosion of previously placed pectoral pacemaker generators. This space can be created by a thoracotomy or sternotomy approach. We have found this technique safe, rapid, and reproducible, with the advantage of requiring a single incision for both lead and generator placement.

References

Congenital Defects of the Pericardium
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Congenital defects of the pericardium are rare, but when they are reported they are frequently associated with other cardiac lesions. We describe a case of partial pericardial defect found incidentally at surgery for closure of an ostium primum atrial septal defect. Proposed mechanisms of pericardial defect development are discussed and we suggest that associations with congenital and acquired heart disease are mostly circumstantial. (Ann Thorac Surg 2007;83:1552–3) © 2007 by The Society of Thoracic Surgeons

A 27-year-old man presented to his local hospital complaining of left-sided chest pain. He was otherwise asymptomatic and had been previously fit and well. On examination, he was in sinus rhythm with a prominent right ventricular impulse and auscultation revealed fixed splitting of the second heart sound and an ejection systolic murmur. Electrocardiography showed prominent P waves, right axis deviation, and partial right bundle branch block. A large primum atrial septal defect was demonstrated on transthoracic echocardiography. His pain resolved spontaneously and he was referred to our unit.

Transesophageal echocardiography confirmed a primum atrial septal defect unsuitable for percutaneous device closure and he was referred for surgery. At sternotomy, a partial absence of the left pericardium was found, allowing free communication between the heart and the left chest (Fig 1). The pericardium was intact anteriorly but did not extend beyond the reflection of the pericardium with the left pleura. The pericardium reappeared beyond the left pulmonary veins with the phrenic nerve visualized over the remnant. The 2-cm ostium primum defect was closed with a composite velour and autologous pericardial patch; the pericardial defect was left untreated. On routine follow-up in the outpatient clinic, the patient has been found to remain well.

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Fig 1. Incidental finding of a partial left pericardial defect (arrow) with the left chest beyond; opening above due to breach of the pleura during sternotomy.
Comment

The pericardial cavity is formed by subdivision of the embryonic coelom between weeks 5 and 7 of gestation [5]. The septum transversum forms a ventral partition between the superior and inferior coelomic cavities. During the fifth week, broad mesenchymal ridges appear along the lateral body walls in the coronal plane. Under the influence of the common cardinal veins, these pleuropericardial folds grow medially toward each other between the migrating heart and developing lungs, drawing in the phrenic nerves. Fusing with the foregut mesenchyme posteriorly, the pleuropericardial membranes divide the thoracic cavity into a ventral definitive pericardial cavity and two dorsolateral pleural cavities. These compartments remain connected by a pleuropericardial channel on each side, medial to each common cardinal vein. During the sixth week, these channels are obliterated as the common cardinal veins migrate toward the midline; the right common cardinal vein later forms the proximal part of the superior caval vein while the left vein (duct of Cuvier) contributes to the coronary sinus, but mainly degenerates.

Pericardial defects arise from maldevelopment of the pleuropericardial membrane. Almost all of the 200 or so reported cases have occurred on the left. In three-quarters of these, the left pericardium was all but absent with the heart and lung occupying a common serous cavity; in the remainder, the pericardial and left pleural cavities were connected by a foramen of variable diameter forming a partial defect [1]. The most widely accepted hypothesis for the origin of these defects is persistence of the pleuropericardial channel due to premature atrophy of the left common cardinal vein, the size of the defect determined by the timing of its degeneration [1]. Others have suggested that herniation of a lung bud or enlargement of the developing heart may impair closure of the pleuropericardial foramen. A recent article proposed that some defects may result from a traction-induced tear in the pleuropericardial membrane during embryogenesis rather than failure of the foramen to close [6].

Pericardial defects have been described in patients with a range of congenital and acquired heart conditions. As the associations are broad and infrequent, it is unlikely that most of these defects are related to the pathogenic mechanisms of other congenital heart disease. We believe that the finding of pericardial defects in these patients predominately reflects the greater frequency of operative interventions and postmortem examinations, illustrated by the chance finding in our case. However, pericardial defects may form part of a spectrum of abnormal coelomic development affecting the diaphragm, sternum, and anterior abdominal wall, including ectopia cordis and the rare pentalogy of Cantrell. The latter is believed to be due to segmental failure of lateral mesoderm migration and is associated with congenital heart defects, particularly ventricular septal defect and tetralogy of Fallot [7].

Most pericardial defects are asymptomatic and are an unexpected finding at surgery or postmortem, although a partial defect may have an acute presentation. Chest pain, dyspnea, dysrhythmias, syncope, and even sudden death due to cardiac chamber herniation have been reported, and may require prompt diagnosis and emergent intervention [2]. Chronic pain may result from traction on cardiac structures in the absence of the pericardium or even compression of the left anterior descending coronary artery by the rim of a partial defect [8]. In our patient, the defect was small and unlikely to explain the episode of nonspecific chest pain, although its origin remains unclear. Pericardial defects have been diagnosed noninvasively by plain chest roentgenograms, computed tomography, and magnetic resonance imaging [8]. With the widespread use of advanced imaging, nonoperative diagnosis may redefine the prevalence of this condition.

Although the pericardium has several functions, including fixation of the heart in the mediastinum, its integrity is not essential. Asymptomatic complete absence of the pericardium and small defects present no hazard to the patient and require no intervention. Moderate-sized defects are rarely of clinical significance and despite the potential for cardiac chamber herniation, most authors advise leaving incidental defects untreated; in cases diagnosed at surgery for other lesions, subsequent adhesions are likely to restrict cardiac mobility. Therefore intervention should be reserved for symptomatic defects, either primary closure or longitudinal pericardiotomy for partial defects, or pericardioplasty for absent left pericardium [2, 8]. We advocate enlarging partial defects to relieve tension on the pericardial rim and prevent restrictive herniation. Direct incision decreases the risk of patch suture injury to the phrenic nerve, which usually tracks the anterior free edge of the defect but may follow an aberrant course [6].

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References