Comment

Failure of a unipolar permanent pacemaker to capture due to interruption of the electrical circuit from lack of contact of the ground plate with the tissues is rare. Several large series of permanent cardiac pacemakers [1, 2], including a pediatric series with mostly epicardially placed leads [3], make no mention of this as a source of failure to pace. Thorough review of the literature reveals only one previous report of pacemaker failure from surgical emphysema; this occurred in an adult with a transvenously placed pacemaker lead complicated by pneumothorax [4]. Emphasized in that report, and confirmed by our experience, was the value of temporary pacing wires during episodes of unipolar pacemaker failure in pacemaker-dependent patients.

Failure to pace acutely postoperatively is usually due to lead displacement, which requires operative replacement. Pacemaker failure due to pocket emphysema, however, will respond to conservative measures including simple pressure dressings, as we used, or perhaps needle aspiration of the air from the pocket. In some pacemaker centers, a sand bag is placed over the pocket routinely to aid in hemostasis, additionally averting this cause of pacemaker failure. A functioning chest tube in the hemithorax would presumably prevent the accumulation of air in the pocket due to the negative intrapleural pressure.

The somewhat unusual pocket location in this case may have been partially responsible for the pocket emphysema. If the leads had been tunneled to a pocket below the rectus muscle, as is our usual practice, the long tunnel may have prevented a substantial quantity of air from reaching the pocket. The advantage of the axillary pocket is only a single incision is required.

This case and the previously reported one both occurred in the immediate postoperative period with air collecting in the newly formed subcutaneous pocket. A chronically implanted pulse generator has a fibrous capsule around it, so contact between the ground plate of a unipolar pacemaker and tissue cannot be disrupted easily. Therefore, emphysema from any cause should not be a source of failure to pace in a chronically implanted system. When failure to pace occurs in any patient with a newly created pacemaker pocket, pocket emphysema should be considered as a possible cause and investigated by roentgenographic examination.

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"Concealed" Rupture of the Left Ventricle: Successful Surgical Repair

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A 70-year-old woman with a history of angina and hypertension presented with a large anterior infarct complicated by rupture and tamponade. Angiography showed triple-vessel disease and a large anteroapical aneurysm. Operative findings included extensive dissection of the septum and rupture of the right ventricular free wall. The patient survived the operation, which included replacement of the left ventricular free wall, extensive patching of the septum, and plication of the infarcted right ventricle.

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Cardiac rupture occurs in 4% to 24% of deaths after acute myocardial infarction [1]. Rupture may be preceded by extensive intramyocardial dissection with the epicardial and endocardial site of disruption separated by a long serpiginous tract. In the case reported here, extensive dissection of the septum after a large anterior myocardial infarction led to rupture on the surface of the right ventricle (RV).

A 70-year-old woman with a history of angina, hypertension, and previous stroke presented with an evolving anterior myocardial infarction and was treated with streptokinase and heparin infusion. Four days later she experienced syncope associated with hypotension and bradycardia. Signs of acute tamponade developed, and echocardiography showed an obvious pericardial effusion with compression of the RV. A pericardiocentesis and removal of 100 mL of blood resulted in immediate hemodynamic improvement. A cardiac catheterization showed extensive triple-vessel disease, a grade IV ventricle with severe hypokinesis of the lateral wall, and a large anteroapical aneurysm. The patient was transferred to the operating room for an emergency operation. Upon opening of the pericardium, a small amount of clot was removed and no site of active bleeding could be identified. The apical region of the left ventricle was discolored and dyskinetic. An area of subepicardial hemorrhage was noted just to the right of the mid-left anterior descending coronary artery (Fig 1A).

In this setting of an aneurysmal apex, we proceeded

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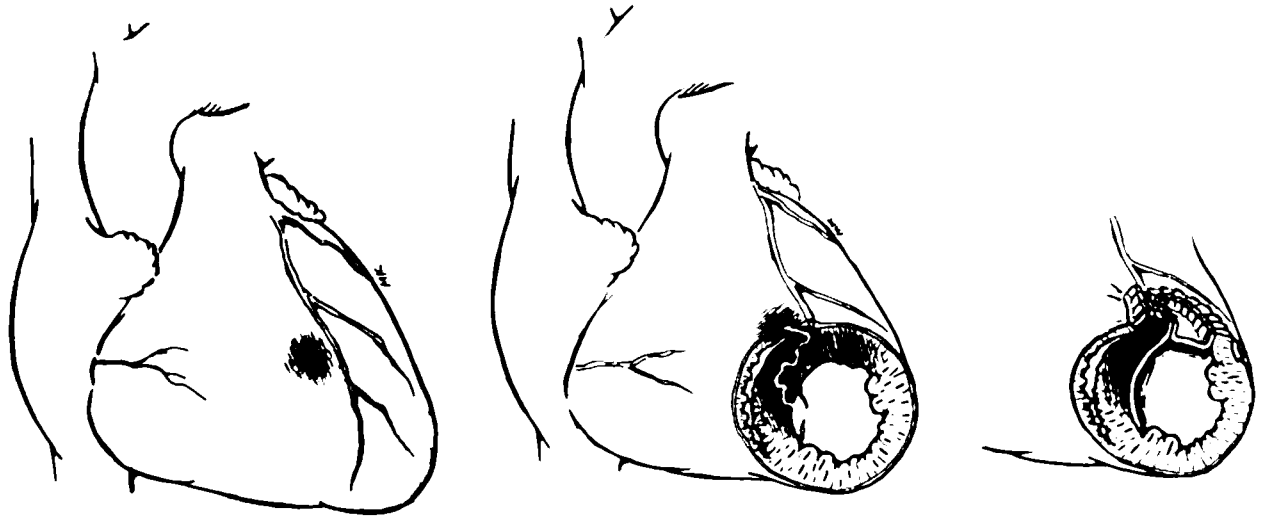

Fig 1. (A) View of the epicardial aspect of the heart as seen at operation. An area of hemorrhage is visible 1 cm to the right of the mid-left anterior descending coronary artery. (B) Extent of infarction (shaded area) *and location of intramural tracts. (C) Extent of repair, including free wall patch to anterior left ventricle, pericardial septoplasty patch, and plication of infarcted right ventricle in anterior repair.*

with infarctectomy. Upon opening of the left ventricle, the septal endocardium was found to be completely disrupted. In the center of the necrotic septum there was an obvious channel, which we believed represented the endocardial aspect of a serpiginous tract extending through the septum and out to the anterior wall of the RV (Fig 1B). Once the anatomy had been defined, the patient was cooled and the heart arrested. We used a large patch of bovine pericardium to cover the entire septum (Fig 1C). A second pericardial patch was used to close the defect in the left ventricular free wall. The two patches as well as what was left of the anterior septum and the hemorrhagic portion of the RV free wall were plicated in the anterior portion of the repair, which was buttressed with felt strips. Bypass grafts were performed to the diagonal, obtuse marginal, and right coronary arteries and the intraaortic balloon pump was inserted for perioperative support. The patient returned to the intensive care unit and made a slow but steady recovery. She was discharged from the hospital 3 weeks later and is alive 4 years postoperatively.

Comment

Factors predisposing to free rupture after myocardial infarction include old age, hypertension, female sex, and thrombolytic or anticoagulation therapy [2]. Most cases of myocardial rupture after acute infarction involve the left ventricular free wall, usually at the junction of the infarct and normal myocardium [3]. From pathologic studies, two types of ventricular rupture have been described, one involving a simple through and through tear and the second or complex type involving a serpiginous tract with extensive intramyocardial dissection before final rupture [4]. Successful repair depends on early diagnosis and rapid surgical intervention before tamponade and irreversible shock or electromechanical dissociation occurs. The need for rapid surgical intervention in these critically ill patients has led some to recommend immediate operative intervention without cardiac catheterization and coronary angiography [5]. In cases with single-vessel disease with preservation of functioh of the surrounding myocardium such an approach is feasible. However, as our case demonstrates, diffuse coronary artery disease may be present and does not preclude the possibility of a successful repair. In such cases appropriate revascularization may be important in achieving a successful outcome. In patients with myocardial rupture we believe that cardiac catheterization and coronary angiography should be performed whenever possible as suggested by Pifarre and associates [6].

In 1968 Cobbs and associates [7] operated on a patient with no electrocardiographic evidence of acute infarction who presented with tamponade. In this patient, bleeding at the apex of the RV was successfully controlled with suture of the tear without the aid of cardiopulmonary bypass. Padro and colleagues [8] reported successful control of bleeding from subacute cardiac rupture in 13 patients in whom the defect was reinforced with a Dacron patch applied to the epicardial surface with surgical glue. Their patients were operated on without cardiac catheterization or revascularization, and cardiopulmonary bypass was needed in only 1 case.

This case report illustrates successful surgical repair of a complex myocardial rupture after extensive anterior myocardial infarction. Preoperative echocardiographic examination confirmed the diagnosis of pericardial effusion and tamponade but did not identify the site of perforation or of tract formation. In our patient, who had a large anterior myocardial infarction, tamponade resulted from bleeding at an RV epicardial site associated with an extensive dissecting intramural tract that originated on the left side

of the necrotic septum. Simple suture closure of the epicardial defect as reported by Cobbs and associates or reinforcement of the area with an external patch as advocated by Padro and colleagues might have led to rupture in another area with a fatal result in this case. We achieved long-term patient survival using an aggressive surgical approach that included revascularization, extensive infarctectomy, and replacement of the left ventricular free wall as well as patching of the infarcted perforated septum and plication of the infarcted RV in the repair.

In spite of the patient's extensive infarction and coronary artery disease, successful repair was followed by long-term survival (4 years). Attempts to salvage such critically ill patients seem worthwhile.

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INVITED COMMENTARY

This case report of "concealed" rupture of the left ventricle by Komeda and Mickleborough is remarkable and of interest to cardiac surgeons for several reasons. First, the imaginative repair used by the authors could not be planned in advance and needed to be created out of "whole cloth" because of the unusual anatomy of the anterior wall and septal defect. Second, the complementary construction of coronary bypass grafts is supported by the patient's excellent long-term survival and is consistent with the advantage in longevity conferred by complementary bypass grafting with other types of mechanical complications of myocardial infarction [1].

The serpiginous nature of the tract within the myocardium probably accounts for the patient's survival and prevention of free rupture into the pericardial cavity. Although I agree that in cases like the one reported by Komeda and Mickleborough there is a definite advantage to having performed preoperative coronary arteriography, many patients with myocardial rupture will not be sufficiently stable hemodynamically to allow this type of angiographic investigation. In such instances, the emergent demands of this myocardial catastrophe must be balanced and take precedence over longer term concerns. It is safe to say, I think, that the majority of patients with myocardial rupture will be treated best by immediate sternotomy, relief of tamponade, and control of the myocardial site of rupture.

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Fracture of the Internal Lumen of a Datascope Percor Stat-DL Balloon, Resulting in Stroke

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Intraaortic balloon counterpulsation was used to wean a 71-year-old man from cardiopulmonary bypass. Thirty-six hours after insertion, the internal lumen of the Datascope balloon fractured, releasing an unknown quantity of helium into the patient's circulation. Left-sided hemiparesis developed, secondary to a right hemispheric infarction.

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Intraaortic balloon counterpulsation was first used by Kantrowitz and colleagues in 1968 to support a 45-year-old woman in cardiogenic shock [1]. Since this original description, the intraaortic balloon has benefited countless patients, and it is now used in more than 70,000 patients annually. The intraaortic balloon, however, has been associated with significant morbidity and mortality [2, 3]. In this report, we describe a patient who suffered an embolic episode that resulted from a spontaneous fracture of the internal lumen of a Datascope Percor DL Balloon

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