the wide complexes occur at the end of relatively long P-P intervals. This is poorly consistent with variable degree of pre-excitation. Indeed, the sinus impulse is expected to suffer a tachycardia-dependent delay in the A-V node, but not in the accessory pathway. Therefore, the shortest P-P intervals would have to be associated with a higher degree of pre-excitation, when compared with long P-P intervals. This is because the increased A-V nodal delay occurring at high heart rates enables the impulse conducted through the accessory pathway to reach and depolarize a larger zone of ventricular myocardium.

The association of wide QRS complexes with long P-P intervals suggests the presence of an ectopic ventricular focus synchronous with, and discharging at the same rate of the sinus node. This results in variable degrees of ventricular fusion, depending on the sinus cycle length variations. When the sinus rate decreases, thus, the ectopic focus is able to depolarize part of the ventricles, whereas a slight sinus rate increase enables the sinus node to anticipate the ectopic pacemaker, thereby resulting in a normal QRS complex.

This interpretation is supported by the bottom strip of Figure 1, where, after a critical prolongation of the P-P interval (0.95 s), some pure ectopic QRS complexes, not preceded by P waves, manifest, establishing the ventricular origin of the ectopic rhythm. The concertina effect observed in the initial section of the recording, thus, is due to: (1) fortuitous coincidence between the sinus impulses and the ectopic discharges; and (2) respiratory sinus arrhythmia, with gradual minimal fluctuations of the P-P intervals.

REFERENCE

Total Left Main Coronary Artery Occlusion after Aortic Aneurysm Repair and Valve Replacement

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A 38-year-old woman with complete occlusion of the left main coronary artery secondary to cannulation during aortic valve replacement is presented. The clinical course was characterized by progressive left ventricular dysfunction and congestive heart failure. Recognition of this potential problem when it occurs is important so as to institute therapeutic measures which may interrupt a patient's progressive clinical deterioration.

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CASS = Coronary Artery Surgery Study

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Complete occlusion of the left main coronary artery is seen in only 0.04 percent to 0.06 percent of all patients undergoing coronary arteriography.1,2 Common causes of total left main artery occlusion include coronary atherosclerosis, embolism, thrombosis, and spasm. Recognition of this problem is crucial, because surgery can improve symptoms in patients with this condition.1,2

We report an unusual case of subacute total occlusion of the left main coronary artery in a woman who had undergone aortic valve, ascending, and arch replacement with reimplantation of the coronary arteries. One year after surgery, she developed progressive left ventricular dysfunction, culminating in acute pulmonary edema. The occlusion was postulated to be slow and progressive because of the development of coronary collaterals (seen on angiography) and the patient's one-year clinical course.

CASE REPORT

A 38-year-old woman with a history of pericarditis entered the emergency room with sharp right precordial chest pain associated with back pain. Physical examination revealed blood pressure of 142/60 mm Hg. There was no jugular venous distention. Cardiac auscultation disclosed a grade 2/6 diastolic murmur over the aortic area. The extremities were unremarkable. The electrocardiogram showed a normal sinus rhythm with left axis deviation. The chest x-ray film demonstrated a large thoracic aortic aneurysm. A two-dimensional echocardiogram suggested normal left ventricular function and aortic regurgitation. The patient subsequently underwent aortography, which confirmed dissection of the ascending aorta, with no significant noted.

On May 7, 1987, the patient underwent replacement of the aortic valve and ascending and transverse arch, as well as reimplantation of the coronary arteries and great vessels. The aorta was markedly abnormal, with evidence of calcification, weakness, deep corrugations that abruptly ended at the ascending aorta, and dilatation of the aorta into the middle portion of the transverse aorta arch. The procedure was performed under hypothermic cardiac arrest, with cardioplegia introduced in repeated doses every 20 minutes through the coronary ostia with filling foam aortic canulae. The total ischemic time was 68 minutes. Pathologic examination of the aorta showed atherosclerosis and no evidence of syphilis. Although, after surgery, there was no evidence of myocardial infarction, the postoperative MUGA scan showed a reduction of the ejection fraction to 30 percent; DSA angiography at this time showed a normally functioning aortic valve, with no evidence of pseudoaneurysm formation or extravasation of dye, and normal origins of the vessel.

The patient subsequently did well on a medical regimen consisting of digoxin, furosemide, captopril, and warfarin (Coumadin) until one year following surgery, when she presented to the emergency room with an episode of "flush" pulmonary edema requiring intubation. Her ejection fraction was only 11 percent by MUGA scan. The patient denied any angina pectoris. At this time, catheterization of the right and left heart revealed normal cardiac pressures and pulmonary wedge pressure of 12 mm Hg, with no gradient across the aortic valve and a normal right coronary artery flow. There was total occlusion of the left main coronary artery, and the branches of the left system were filled faintly by a bizarre set of collaterals that appeared to originate within the wall of the aorta and insert into the proximal segment of the main left anterior descending and circumflex arteries (Fig 1). On Aug 9, 1988, the patient underwent valvular replacement of the marginal system using a saphenous graft from the descending thoracic aorta. At the time of surgery, there was no evidence of pseudoaneurysm or suture disruption at the site of the previous repair. The patient had an
uneventful postoperative course and 18 months after coronary revascularization is doing well, with no episodes of recurring congestive heart failure. A repeat MUGA scan showed an improved left ventricular ejection fraction of 14 percent, with improved wall motion and marked normalization of left ventricular size.

**DISCUSSION**

Total occlusion of the left main coronary arteries is extremely rare, with a reported incidence of 0.06 percent in a total of 20,197 patients in the CASS experience. Our patient’s presentation of total occlusion of the left main coronary artery without angina pectoris is rare but has previously been reported, although most patients with this condition initially are seen with severe angina pectoris.

Causes of left main coronary occlusion, other than atherosclerosis, include coronary embolization and coronary thrombosis and spasm; however, total left main occlusion may occur following aortic valve replacement.

Although long recognized as a late complication of coronary perfusion in cardiac surgery, coronary ostial stenosis and occlusion is less prominent because of improved surgical materials and methods. It is believed that mechanical irritation of the left main coronary artery results in intimal damage with secondary fibrosis and stenosis. It is postulated that the initial pathology began with intraoperative cannulation of the left main coronary artery. The progressive slow obliteration of the lumen caused deterioration in left ventricular function as a result of chronic ischemia and infarction. The collaterals at angiography support the chronic nature of the occlusion, and the patient’s clinical course and serial MUGA scans demonstrate the progressive effects. This is unlike acute occlusion of the left main coronary artery, which usually presents with acute anterior myocardial infarction.

In a patient who has undergone aortic valve replacement or aortic aneurysm repair with reimplantation of the coronary arteries, or where the coronary arteries are cannulated for cardioplegia or perfusion, left main coronary artery occlusion should be suspected when congestive heart failure or progressive left ventricular deterioration become evident in the absence of overt angina pectoris. Recognition of this problem is important, since surgery may be beneficial in some patients. Occlusion of the left main coronary artery need not be acute, and intraoperative cannulation may be the initial insult. The sooner a chronic progressive left main artery occlusion is recognized, the higher the likelihood that surgery may salvage left ventricular function or improve prognosis.

**REFERENCES**

8 Trimble AS, Bigelow WG, Wige ED, Silver MD. Coronary ostial stenosis: a late complication of coronary perfusion in open...
Cardiac Tamponade and Contralateral Hemothorax After Subclavian Vein Catheterization*  
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A patient developed life-threatening cardiac tamponade and contralateral hemothorax after insertion of a subclavian catheter in the operating room. Contrast was infused through the catheter, demonstrating its malposition in the pericardial space. Contrast infusion was valuable in evaluating this complication of central line placement.  
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Central venous catheterization has become an increasingly common procedure in the care of critically ill and chronically ill patients. Although the reported rate of major complications has varied from 0.4 percent to as high as 11.1 percent, the value of central venous access has led to ever broader indications for its use in hemodynamic monitoring, parenteral nutrition, and fluid infusion. In addition, subclavian vein catheterization has emerged as a rapid means for establishing temporary access for hemodialysis. When the need for central access is elective, internists often refer their patients for surgical line placement for the obvious benefits of enhanced safety and sterility. We describe a patient who developed cardiac tamponade and contralateral hemothorax following placement of a subclavian vein dialysis catheter. This life-threatening complication occurred despite placement in the controlled setting of an operating room.  

CASE REPORT  
A 48-year-old man was admitted with progressive exertional dyspnea and chest discomfort. There was a history of hypertension and chronic renal failure due to membranous glomerulonephropathy, but the patient had been noncompliant with medications and was lost to follow-up for several years. Physical examination was notable for a blood pressure of 170/115 mm Hg and an S4 gallop. Laboratory test results revealed the following values: blood urea nitrogen, 136 mg/dl; creatinine, 29 mg/dl; potassium, 4.9 mEq/L; bicarbonate 16 mEq/L; and hemoglobin, 6.2 g/dl. A right subclavian vein catheter was placed percutaneously, and the patient received dialysis on two consecutive days without difficulty. On the third hospital day, the patient underwent creation of a left forearm arteriovenous fistula in the operating room. At the same time, the preexisting subclavian catheter was replaced over a guidewire for continued access until maturation of the fistula. A postoperative chest x-ray film confirmed catheter position in the right atrium without pneumothorax or effusion. After returning to the ward, the patient complained of pleuritic chest pain. The blood pressure was 80/60 mm Hg, heart rate, 96 beats per minute, and physical exam revealed clear lungs, jugular venous distension, and distant heart sounds. Repeat chest x-ray film demonstrated a widened mediastinum and a new left subpulmonic effusion. Since the suspected mediastinal hematoma and hemothorax were thought insufficient to explain the patient's hypotension, a 2-D echocardiogram was requested. This revealed a moderate-sized anterior and posterior pericardial effusion with diastolic right atrial collapse and respiroplastic changes in transverse velocity, all consistent with cardiac tamponade. In an attempt to localize the tip of the new subclavian catheter, contrast material was infused and a chest x-ray film taken (Fig 1). Contrast filled the pericardial space, dramatically locating the catheter tip, and proving the cause of the cardiac tamponade. The patient promptly underwent mediastinal exploration, at which time the catheter tip was found in the sulcus between the aorta and superior vena cava. The catheter was removed and the surgeon withdrew 300 ml of serosanguinous fluid and clot from the pericardial sac. One liter of blood was then aspirated from the left pleural space. Examination of the left superior mediastinal pleural reflection in search of the cause of the hemothorax was unrevealing. The patient recovered uneventfully with no reaccumulation of pleural or pericardial fluid. Postoperatively, it was learned that the line placement had been complicated by repeated kinking of the catheter and guidewire, requiring multiple punctures.  

DISCUSSION  
Numerous major complications of central venous catheterization have been reported, often resulting from vascular laceration or embolization, extravascular catheter migration, thrombosis, or infection. These are thoroughly reviewed elsewhere. While both cardiac tamponade and contralateral hemothorax have been previously described as compli-

FIGURE 1. Although the catheter tip is projected over the superior vena cava, contrast infusion fills the pericardial space, proving that the catheter is within the pericardium.