Venous Obstruction
A Potential Complication of Transvenous Pacemaker Electrodes

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Pervenous endocardial pacing using a self-contained, totally implantable system was popularized in this country by Chardack in 1965 and subsequently has become the established mode of permanent pacing. Although this intervention may be lifesaving, it is not without risk. Occlusion of major vessels incited by the presence of a pacing electrode is an extremely rare occurrence. We describe four patients who had occlusion of the axillary or subclavian veins or of the superior vena cava due to pervenous pacing electrodes. We review the literature of previous observation of venous occlusion and thromboembolic complications due to pervenous pacemakers, describe the clinical features accompanying venous obstruction, and review pathophysiology and proposals for treatment. The use of noninvasive radionuclide venography serves as a useful adjunct in the diagnosis of venous obstruction from pacing catheters.

Pervenous endocardial pacing is an effective means of treating many patients with disturbances of cardiac conduction and rhythm. Because of its relative ease and safety, the pervenous route has become the most common mode of electrode insertion. While this approach entails a smaller surgical risk than the trans-thoracic approach and shares with the latter approach many of the mechanical and electric complications of pacing, several complications (such as electrode perforation and air embolism) are unique to the pervenous route. Vascular obstruction resulting from the chronic residence of the pacing catheter in the venous system is a rarely recognized complication. No instance of thromboembolism was noted among the 305 patients with permanent pacemakers reported by Lagergren et al. Furman et al described their experience with 205 patients who were paced for as long as eight years without overt thromboembolic complications. Scattered reports of venous obstruction and thromboembolism as a complication of pervenous pacing have subsequently appeared. We review these reports, describe four new cases of electrode catheter-induced obstruction, and review the clinical features, radiologic diagnosis, and treatment of this entity.

Case Reports

Case 1
A 57-year-old man with "brady-tachy syndrome" underwent pacemaker insertion (CPI 4250 bipolar electrode) via the left cephalic vein in 1978. Two years later, swelling of his face, neck, arms, and chest developed insidiously, accompanied by exertional chest tightness with a choking sensation, confusion, bandlike pres-

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s sure about his head, and cyanosis of his face. A contrast venogram demonstrated obstruction of the superior vena cava. He was given bedrest and heparin therapy and was asymptomatic when discharged receiving warfarin (Coumadin). The confusion, headaches, and swelling at rest improved, but he continued to experience swelling of the upper torso with arm exercise. Three months later he was readmitted for progressive symptoms. There was edema of the head, neck, arm, chest, and periorbital regions. Numerous dilated veins were visible over his upper chest and shoulders, and left jugular veins were distended to the angle of the jaw. Radionuclide and contrast superior vena cavaogram (Fig 1) demonstrated the previously identified obstruction. The pacing wire was removed without difficulty. He was given continuous intravenous (IV) heparin and later warfarin (Coumadin), with symptomatic improvement. One year later he was symptom-free at rest, but did experience mild arm swelling during vigorous arm exercise.

Case 2
A 49-year-old man with aortic valve disease and cardiomyopathy required a pacemaker for 17 years for complete heart block. During this period his course was punctuated by numerous problems with his pacing system, including marked incisional keloid formation, pocket infection, wire fracture, and generator failure. Epicardial electrodes were inserted and the pervenous electrodes (Medtronic 5818 bipolar lead) were transected and left in situ. Six years later he presented with a one-week history of progressive swelling of his arms and face. His examination was remarkable for edema of the face, conjunctivae, neck, and arms. Marked keloid formation was apparent along his surgical scars (Fig 2). Radionuclide superior vena cavaograms (Fig 3) showed complete obstruction of the superior vena cava, confirmed subsequently by contrast venography. With bedrest and ten days of heparin therapy, the patient's symptoms improved significantly. He was discharged receiving maintenance Coumadin therapy, but was readmitted one month later with exacerbation of facial, neck, and arm swelling. Radionuclide venography again showed obstruction of the superior vena cava. Rest, diuretics, and heparin followed by Coumadin resulted in partial clinical improvement, but mild swelling of his face and arms persisted.

Case 3
A 63-year-old man with symptomatic carotid sinus hypersensitivity required pacemaker insertion (Medtronic 5842 bipolar) in 1972. Eight years later he awakened with dusky discoloration and

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painful swelling of his left arm and hand. One month earlier he had undergone his most recent pacemaker revision, involving removal of a right-sided fractured pervenous electrode and the insertion, following a difficult surgical dissection, of the left-sided pacing electrode via a tributary of the left subclavian vein. His examination was remarkable for left arm and hand edema, cyanosis, and dilated superficial vessels at the shoulder. There was no palpable venous cord or tenderness. The clinical diagnosis was left subclavian vein thrombosis, and the patient was treated with seven days of continuous IV heparin, bedrest, and left arm elevation. The edema and discoloration resolved rapidly, and he was discharged receiving Coumadin. Six months later he remained free of symptoms.

CASE 4

A 67-year-old woman was admitted with crushing chest pain. An acute anterior wall myocardial infarction was diagnosed. Two months after discharge, she was brought to the emergency room following three syncopal events occurring in quick succession, and was found to be in complete heart block, with an escape ventricular rate of 28, for which a permanent puvvenous pacemaker (CPI 4130 unipolar lead) was inserted via the right cephalic vein. Three weeks later, the patient noted the sudden onset of right hand swelling, which progressed over 24 hours to include the entire arm. The arm was edematous, cyanotic, and diffusely tender, with a firm venous cord palpable in the axilla. There was a prominent superficial venous pattern over the right shoulder and chest (Fig 4), and the region was warmer than the contralateral side. The clinical diagnosis of right axillary vein thrombosis was made, and the patient was treated with continuous IV heparin, arm elevation, and rest. There was striking improvement within the first 24 hours, and after one week Coumadin therapy was begun. An exacerbation of her symptoms required reint introduction of heparin therapy for five additional days before she was discharged, asymptomatic and receiving Coumadin. Symptoms of venous obstruction did not recur; she died of a myocardial infarction three years later. Autopsy showed no evidence of fibrosis or thrombus formation about the pacing vein.

FIGURE 1. Case 1: Bilateral contrast venography compared with radionuclide venograms injected selectively via antecubital veins. Right arm injection demonstrates innominate vein stenosis as it enters superior vena cava. Selective left injection reveals developed collateral circulation via azygous system. Note near-complete vena cava obstruction with faint appearance of injectate in right heart chambers.

FIGURE 2. Case 2: Marked keloid formation in previous pacemaker implantation sites on infrared photograph. Note subtle increase in superficial vascular pattern across anterior chest.
Venous Obstruction (Fritz et al)

**DISCUSSION**

The first thromboembolic complication of pervenous pacing was presented by Prozan et al in 1968. They described a patient who died of multiple pulmonary emboli. Autopsy showed the source to be a clot on a permanent pacing catheter as it coursed through the right atrium and ventricle. Reynolds et al described a patient who had convincing clinical and laboratory evidence of acute pulmonary embolism during the insertion of temporary pervenous electrode along a malfunctioning permanent electrode. Subsequently, an organizing clot was recovered from the tip of the temporary electrode. Since the patient had been anticoagulated in the interim, the authors presumed that the clot had been sheared from the permanent electrode during the insertion of the temporary pacing catheter. Sidd et al described a case of fatal pulmonary embolism from a large clot originating from a redundant loop of pervenous pacing catheter as it prolapsed through the pulmonic valve.

More recent reports have described extracardiac venous obstruction arising from pacing electrodes with or without pulmonary emboli. Superior vena cava syndrome complicating pervenous electrodes has also been described in the absence of right heart thrombi or pulmonary emboli. A tight fibrotic stenosis around pacing catheters in the superior vena cava has been reported in two patients with superior vena cava syndrome. Although one of these patients had a small thrombus at the electrode tip, no thrombi were found at the site of stenosis in either patient.

Thrombosis of other great veins had also been reported. Floyd and Mahaley describe a patient who illustrates the potential hazard of using the internal jugular route. Their patient had insidious neurologic symptoms and papilledema following electrode inser-
tation via the right internal jugular vein which was ligated. This patient was found to have thrombotic occlusion of major dura sinuses.

The use of cephalic veins for the introduction of the pacing catheter is often advantageous. There is a decreased risk of air embolism, dissection and fixation are relatively easy, and only one incision is required for both venous access and creation of a generator pocket. However, thrombosis of the axillary, subclavian, and innominate veins has been described using this approach as well.14-16

Clinical Manifestations

Patients with superior vena cava obstruction usually experience the gradual development of edema and cyanosis of the face, neck, or upper extremities. At times this is accompanied by headache, cough, dysphagia, somnolence, and dyspnea, with symptoms usually exacerbated by bending forward. Physical findings may include edema of the drainage territory of the superior vena cava; at times this edema is pitting. Cyanosis of the face, neck, and chest is common. Retinal venous engorgement may often be appreciated. Dilated superficial veins on the chest and arms may appear as a result of either the venous obstruction or enhanced flow through collateral channels. The jugular venous pressure is elevated and may vary paradoxically with respiration. When obstruction is severe and symptomatic, the diagnosis can usually be made at the bedside, although the exact site of obstruction and the adequacy and location of collateral vessels may not be established. Our patients manifested the relatively classic clinical description and also shared with other patients who have pacemaker-induced superior vena cava obstruction the long latent period (greater than 12 months) between catheter insertion and development of symptoms. Although the number of cases is small, such a long latency may differ from that noted with subclavian-axillary thrombosis, which usually becomes manifest within several months following pacemaker insertion.

Radiologic Diagnosis

Contrast venography has been the mainstay of the radiologic assessment of these syndromes. Sequential x-ray films obtained during contrast injections into antecubital veins allow identification of the site of obstruction, delineation of collateral vessels, and frequently helps discern whether an obstruction is intravascular or extrinsic.17

With improved technique and resolution of the gamma camera, radionuclide venography has become an effective means of evaluating venous obstruction.18,19 Radioisotope cavoangiography has been used to diagnose malignant superior vena caval obstruction, but has not, to our knowledge, heretofore been reported in venous obstruction arising from a pacing catheter. The technique utilizes 5 mCi of 99mTc sulfur colloid injected into both antecubital veins. Multiple images of the venous system are then obtained using a gamma camera. We demonstrate that this method accurately identifies the site and extent of obstruction as well as collateral vessels. Anatomic findings correlate well with those obtained by contrast venography. While the resolution using such a technique is not as high as that with contrast venography, the low volume and low pressure injection of radioisotope causes less venous irritation and is better tolerated by the patient. The ease and reproducibility of this technique permit serial examinations to be performed with little morbidity.

Isotope uptake in the umbilical vein has been cited20 as a sign of collateral flow around an obstructed SVC. The absence of this finding in our patients emphasizes the insensitivity of this sign (as pointed out by Hattner and Shames20) and probably results from effective flow via other well-developed collateral channels.

Pathophysiology

The relative infrequency with which symptomatic venous obstruction has been reported has not allowed for an extensive study of pathophysiologic events. The pathogenesis of venous obstruction therefore remains speculative. The presence of a foreign body frequently incites a significant inflammatory and subsequent fibrous tissue reaction along the catheter route, as well as promoting stagnant venous flow which may predispose to thrombosis.15

The relatively small cephalic vein used frequently for transvenous lead insertion may act as a blind pouch, from which thrombi may propagate into the subclavian and axillary veins. In the setting of elevated right-sided pressures, venous stasis may be enhanced and predispose to increased clotting. In addition, the electrode may limit elasticity and mobility of the subclavian vein, making it more susceptible to compression as it passes through the thoracic outlet.

Several reports25,26 have demonstrated fibrous encapsulation of the pacing wire, particularly at the vena caval-right atrial junction. In a pathologic study, Huang24 describes evidence of thrombus formation at the implantation site and along the electrode occurring early (within four to five days) after implantation. Within two weeks there is a proliferative response of fibroblasts and granulation tissue, which frequently results in endothelialization along the electrode. This tissue reaction and fibrous encapsulation may in part be protective against thrombus formation.25 However, if an overwhelming tissue reaction ensues, the fibrosis itself may produce a significant obstruction or create an environment of impaired venous flow, which may promote thrombus formation. Rubbing of the catheter...
against vessels or the endocardial wall may also incite local release of clotting factors which may contribute to thrombus formation, which occurs as an early inciting event and also later after significant obstruction has taken place. From these studies it appears that the processes of thrombus formation and fibrous tissue reaction both contribute to venous obstruction.

**Treatment**

The apparent infrequency of venous obstruction arising from pacing catheters has not permitted a thorough or objective evaluation of treatment modalities. Consequently, opinions regarding therapy are varied and founded on scant data. The development with time of collateral channels may be a major determinant of resolution of symptoms. The collateral routes which may develop after superior vena caval obstruction may include: internal mammary, vertebral, ayzygous, and lateral thoracic routes. Although collateral development will allow eventual decompression of the obstructed circulation, it is slow, does not alleviate the obstruction per se, nor does it prevent possible further propagation of the thrombus or alter the tissue fibrotic process. Furthermore, collateral drainage may not be adequate to permit decompression.

Conservative, symptomatic modes of therapy, such as inactivity, elevation, or compressive bandages, have a role in the treatment of subclavian-axillary thrombosis. Swinton et al. reported a significant reduction of arm edema using conservative measures through one week of therapy.

The role of anticoagulant therapy remains speculative, although the use of anticoagulants is intuitively appealing, when one considers the risk of further propagation of thrombus or embolization. The use of heparin in three of our four patients has generally been followed by gratifying improvement. While we know of no controlled trial for the use of anticoagulants in this situation, the symptomatic rebound and subsequent improvement in case 4 with the discontinuation of re-institution of heparin suggests a salutary effect. Williams et al. describes a favorable response to streptokinase in one patient with thrombotic SVC obstruction. In patients with a more fibrotic tissue response to the pacing catheter (as suggested by case 2 and those described by Faulletti et al.), the use of anticoagulant therapy has not produced such favorable clinical improvement, and may indeed reflect the underlying etiology of obstruction.

The question of whether one should remove the pacing catheter is likewise unsettled. As described above, the pacing catheter in our case 1 was removed with apparent safety. A technique of prolonged, continuous traction has been proposed for removal of entrapped electrodes. Yet severe complications have been reported with forceful and prolonged traction due to the tight union which pacing catheters established with the tricuspid valve and right ventricle. Such complications have included invagination of the right ventricular wall, lethal hemopericardium, tear of the tricuspid valve, and sustained ventricular arrhythmias. We therefore do not advocate routine extractions of the pacing electrode catheter.

Occasionally, when immediate decompression appears mandatory, surgical intervention may be considered. Thrombectomy is most feasible in the acute stage, especially if the thrombus is short. Autogenous saphenous vein bypass grafts from the ayzygous vein to the inferior vena cava have been reported to decompress successfully the superior vena cava and have remained patent over the long term.

**Conclusions**

We report four cases of venous occlusion involving the superior vena cava and the subclavian-axillary system as a complication of permanent venous pacing. A literature review of these entities is also presented. The use of radioisotope imaging, not previously described in the setting of pacemaker-induced venous thrombosis, is introduced as a safe, accurate, and reproducible means of accurately relieving symptoms, allowing collateral vessels to form while relieving venous obstruction, and preventing potentially disastrous hemodynamic and embolic events.

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