Dislodgment of Pacemaker Electrode Simulating Focal Motor Seizure

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The unusual finding of dislodgment of the electrode of a pacemaker in a patient caused repetitive twitching of the right leg. Initial confusion with focal motor seizure resulted in the administration of antiepileptic medication. Malposition of the electrode was confirmed by appropriate roentgenographic studies, and subsequent repositioning terminated the episode.

The complications of transvenous cardiac pacing are frequently secondary to electrical stimulation of non-cardiac sites. Previous reports have documented diaphragmatic contraction in synchrony with discharge of a pacemaker, direct stimulation of the phrenic nerve, and muscular twitching at the site of implantation of a unipolar pulse generator. Migration of electrodes in association with myocardial perforation has resulted in direct contractions of intercostal muscles. In addition, neuromuscular complications of cardiac pacing include rhythmic contractions of the shoulder girdle and sensory precipitated epilepsy.

In this clinical report, we describe the unusual finding of dislodgment and migration of an electrode simulating focal seizure activity in the leg. To our knowledge, previous documentation of this phenomenon in the literature is limited to a single case report.

**CASE REPORT**

An 80-year-old man came to a neurologist's office complaining of sudden onset of a repetitive twitching of his right leg for the preceding 18 hours. Twitching began while the patient was sleeping, and he was able to obtain only brief periods of remission by leaning far forward or lying on his left side. He denied headache, paresthesia, weakness, nausea, vomiting, blurred vision, dizziness, palpitations, or pain in the chest.

One month prior to this complaint, the patient had undergone implantation of a permanent transvenous demand pacemaker (Cordis Omni Stalcor Lambda 100A) using unipolar percutaneous electrode via left internal jugular approach. Evidence of paroxysmal atrial fibrillation and bradyarrhythmia coincident with subjective dizziness had been documented on a continuous electrocardiographic monitor (Holter monitor). Electrocardiograms obtained after insertion had demonstrated sensing and capture, with a chest x-ray film showing excellent position of the electrode in the right ventricular apex. The patient had been subsequently discharged while asymptomatic and receiving digoxin (0.25 mg daily).

On physical examination, the blood pressure was 140/90 mm Hg, the pulse rate was 74 beats per minute and regular, the respiratory rate was 14/min, and the oral temperature was 36.9°C (98.4°F). The patient appeared mildly anxious but well oriented and had obvious rhythmic clonic spasms of the right leg. The neck was supple; no carotid bruits were heard. Cardiovascular examination revealed a minimally displaced apical impulse to the left of the midclavicular line, with a regular rhythm except for rare extrasystoles. A repetitive clonic spasm at a rate of 70/min was noted, involving the proximal musculature of the right thigh. With each contraction, there was a slight internal rotation of the extremity, with some spread of the muscular twitching to involve the lower abdominal wall. No fasciculations, sustained spasm, or other lateralizing neurologic finding was detected.

The presumptive diagnosis was focal motor seizure, possibly on an embolic basis; and the patient was admitted to Hahmemann Medical College Hospital, Philadelphia. The results of routine laboratory studies (including blood cell count, levels of electrolytes, and blood glucose and creatinine levels) were normal, and phenytoin sodium (900 mg) and phenobarbital (100 mg) were administered parenterally without effect. Shortly thereafter, close examination while an ECG was obtained on admission (Fig 1) revealed synchrony.

**Figure 1.** Pacing spikes demonstrate lack of sensing or ventricular capture. Underlying rhythm is sinus, with rate of 74 impulses per minute.
between nonsensing, noncapture paced complexes and the movements of the patient’s leg. A chest x-ray film (Fig 2) and abdominal flat plate (Fig 3) demonstrated dislodgment and migration of the electrode of the pacemaker to the level of S-1 in the right iliac vein. Adjustment of the rate of the demand pacemaker from 70 to 80 impulses per minute by an external unit over the battery caused a coincident increase in the frequency of twitching of the right leg.

Subsequent repositioning of the electrode resulted in “cure” of the focal motor activity. Distribution of muscular innervation was most consistent with stimulation of the obturator nerve through the iliac vein or body of the psoas muscle.

**DISCUSSION**

A number of excellent reviews describe the complications and malfunctions of the cardiac pacemaker. Insertion and positioning of electrodes may be followed by infection of the wound, hematoma, or induction of ventricular ectopic beats. A cervical venous cutdown or subclavian approach enhances the risk of pulmonary air embolism or pneumothorax. Later morbidity, often weeks or months after implantation of the pacemaker, may be caused by myocardial perforation or dislocation and subsequent migration of the electrode to extracardiac sites.

Neuromuscular symptoms often predominate when noncardiac structures are subjected to rhythmic electrical stimulation. Dislodgment or malposition of the electrode resulting in twitching of thoracic structures such as the intercostal muscles, diaphragm, or phrenic nerve offers little diagnostic difficulty; however, dislocation and distant migration, as in our patient, may result in symptoms that are less than obvious.

Embolization, both cerebral and peripheral, occurs in a significant percentage of patients with the sick sinus syndrome. Rubenstein et al noted a 24 percent incidence of cerebral embolization in their series of patients with bradyarrhythmias and episodic supraventricular tachyarrhythmias. These authors hypothesized that stasis in the atria, with the associated tachyarrhythmias, promoted development of mural thrombi. Accordingly, as most of these patients require transvenous pacing for the management of bradyarrhythmias, localized muscular twitching in an extremity on the basis of displacement of the electrode could easily be mistaken for focal motor seizure secondary to a cerebral event. Therefore, before considering therapy with antiepileptic medication in patients with this clinical symptom, the position of the electrode should be carefully checked.

**REFERENCES**