CASE REPORTS

The Pacemaker-Twiddler's Syndrome:

A New Complication of Implantable Transvenous Pacemakers

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THE implantation of transvenous pacemakers has become a well-established procedure. This has led to the recognition of cardiac pacing abnormalities which include failure to pace, intermittent pacing, and a change in cardiac rate or rhythm which indicates potential failure of the pacing system. Malfunction in the pacing system may occur at one of the following sites: pulse generator, lead(s) and electrode(s), and the heart itself. Table I is a modification of the schema originally proposed by Furman *et al.*[1] and by Gerst *et al.*[2] for epicardial pacing systems, extended to include pacing abnormalities in transvenous systems.

TABLE I.—SITES IN PACING SYSTEM WHERE MALFUNCTION MAY OCCUR

1. *Pulse generator*
   (a) battery decay
   (b) pack component failure
   (c) internal short circuits
2. *Lead(s) and electrode(s)*
   (a) lead fracture
   (b) electrode fracture
   (c) electrode dislocation
      —epicardial
      —endocardial
      —perforation (endocardial)
3. *Heart*
   (a) elevated stimulation threshold
   (b) elevated electrical resistance

Electrode dislocation in transvenous systems can produce bizarre pacing abnormalities. Perforation of the myocardium can bring about failure to pace together with diaphragmatic and epigastric muscle stimulation. Dislocation of the electrode into the superior vena cava can result in failure to pace as well as phrenic nerve stimulation.[3] Retraction of the electrode into the neck brings about failure to pace and also phrenic nerve and brachial plexus stimulation. We were not aware of this latter combination as a clinical entity until our experience with one patient whose case we report here. We have since learned from the manufacturer that this complication is occasionally seen. To our knowledge, what can be termed the pacemaker-twiddler's syndrome has not previously been reported in the literature.

Mrs. A.S., a 79-year-old widow, was well until April 1965, when she suffered an acute posterior myocardial infarct and subsequently developed intermittent complete heart block. With appropriate medical therapy she was asymptomatic until February 1967, when she developed frequent Stokes-Adams attacks with complete loss of consciousness for several seconds at a time. A continuous electrocardiographic record revealed first-degree heart block, a ventricular rate of 50 per minute and periods of asystole lasting several seconds associated with clinically evident Stokes-Adams attacks.

A Ventricor III* transvenous implantable "standby" pacemaker was inserted via the right external jugular vein on April 10, 1967. The pulse generator was positioned deep to the pectoralis major muscle in the right upper chest and only a light dressing was applied. On April 15, failure to pace occurred and perforation by the electrode was suggested by the position of its tip on the chest radiograph (Fig. 1). Pacemaking was resumed under the influence of continuous intravenous isoproterenol for 11 days while the patient was treated for an episode of congestive heart failure and pneumonitis. When isoproterenol was discontinued, failure of pacemaking again was evident.

On April 26, by retracting one inch of the lead in the neck, the electrode tip was repositioned within the right ventricular cavity and resecured to the external jugular vein by a single non-absorbable (Mersilene) ligature as before. Pacing was satisfactory and the patient was asymptomatic for five weeks at home. During this interval she noted that the implanted pulse generator appeared to be more prominent at some times than at others. This caused

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Canad. Med. Ass. J.
Aug. 24 and 31, 1968, vol. 99

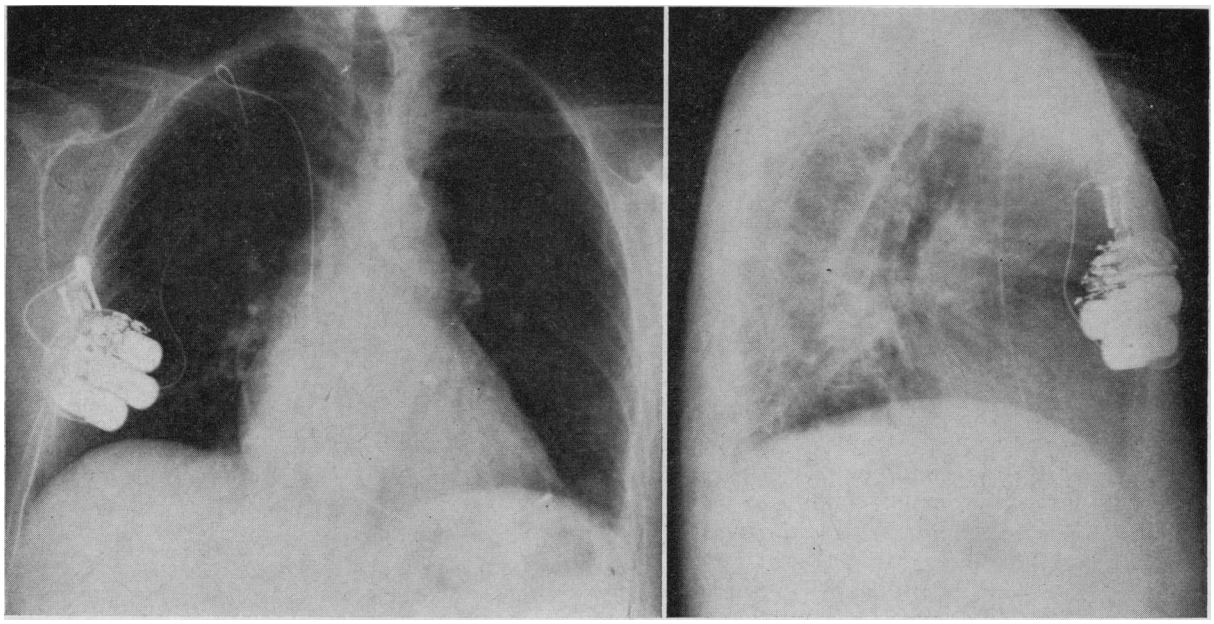

Fig. 1.—Radiograph of chest showing original transvenous lead in position; failure to pace possibly due to perforation.

her no discomfort, and the prominence usually disappeared spontaneously or after manipulation by the patient.

The patient was seen again on June 2, 1967, several hours after the sudden onset of irregular epigastric jerking movements, occasional spasms of involuntary inspiration, and regular involuntary supinating movements of the right forearm and hand; the latter movements were present only when the patient was in the sitting position. The electrocardiogram showed failure to pace and first-degree heart block with a ventricular rate of 50 per minute. The patient was not having Stokes-Adams attacks. Because she was not examined initially in the sitting position, the significant arm movements were not appreciated and the erroneous diagnosis of catheter perforation was made. The chest radiograph (Fig. 2) clearly revealed that the pulse generator had rotated several times on its long axis, coiling the lead wire around the boot and retracting the electrode tip into the neck adjacent to the phrenic nerve and brachial plexus.

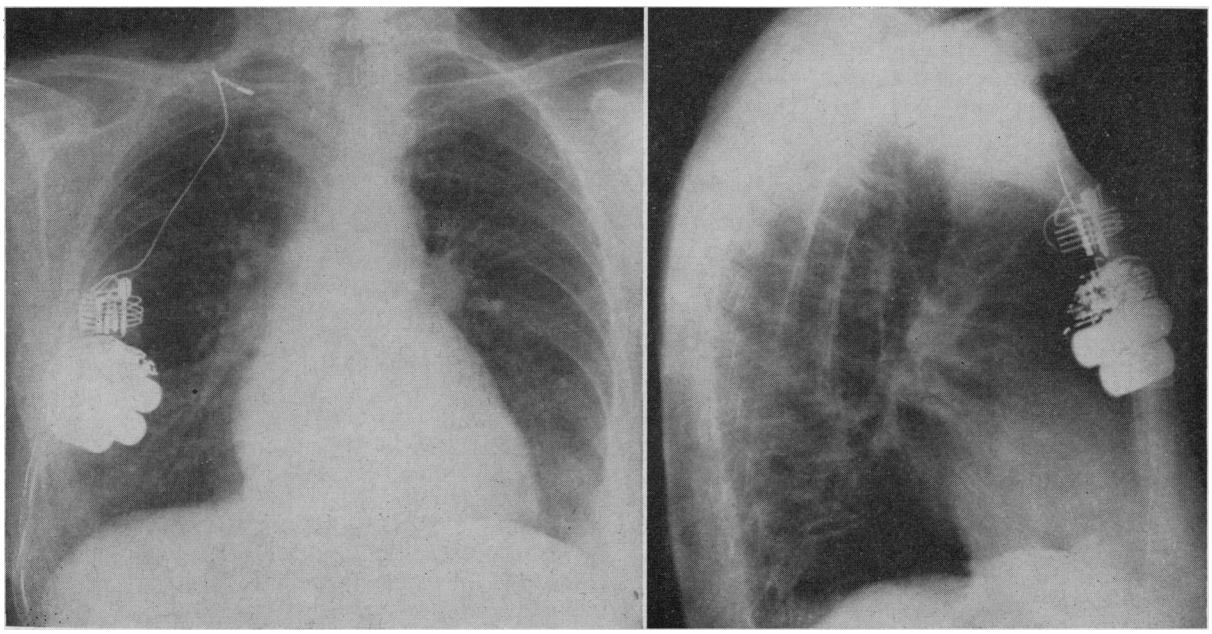

Fig. 2.—Radiograph of chest showing transvenous lead coiled around pacemaker boot and electrode retracted into the neck.

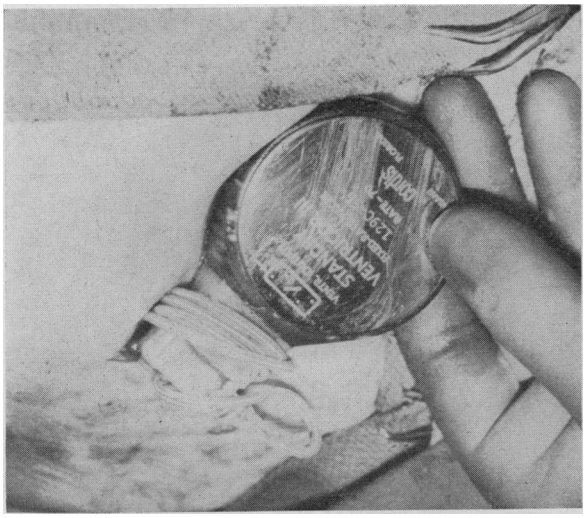

Fig. 3.—Pulse generator at time of removal showing lead coiled around boot and right-angle take-off from pacer boss.

On June 9, the transvenous unit was removed and the coiled lead was visualized (Fig. 3). The subpectoral pocket had enlarged sufficiently to permit free rotation of the pulse generator within a small bath of clear fluid. A Ventricor III "standby" pacemaker with epicardial leads was inserted through a left thoracotomy and the patient resumed normal pacing. Although the surface of the left ventricle was somewhat stained by an old hematoma, no definite point of perforation of either ventricle and no residual hemopericardium were identified at the time of thoracotomy. A single Mersilene ligature, still firmly attached to the lead, suggested that the external jugular vein had been disrupted by the traction. The patient continued to pace well and was discharged from hospital on June 18, 1967.

DISCUSSION

Transvenous leads for long-term pacing were developed as an alternative to epicardial leads for patients in whom thoracotomy or general anesthesia was contraindicated. Once positioned, the endocardial electrode will continue to pace reliably only if it remains stable in its original position. After the first few weeks, collagenous ingrowth may be relied upon to fix the electrode tip in position. Initially, it is important to minimize the chances of electrode dislocation by preventing traction on the lead. The pacemaker twiddler's syndrome represents a new clinical entity in which lead traction is the result of a capstan effect produced by a rotating pulse generator within a pocket which has become too capacious. Rotation can occur either spontaneously or as the result of repeated twiddling on the part of the patient, as in our case. It is apparent that an extreme degree of lead retraction is possible, with consequent failure of pacing action.

Viewed in retrospect, this complication seems to be the logical sequel to a number of predisposing factors.

Firstly, inadequate fixation of a transvenous lead at the site of introduction can be followed by dislocation. Particular care must be taken to resecure the lead if it becomes necessary to reposition the electrode tip within the right ventricular cavity. It is suggested that multiple ligatures of non-absorbable material be used at this site to provide greater stability.

Secondly, a potential capstan mechanism is provided by pulse generators having a right-angle plug between the lead and the pacer boss (Fig. 3). With this system it is particularly important to prevent rotational movement of the pulse generator, since this results in traction on the lead. Theoretically, pacemakers with straight take-offs should permit rotation until lead fracture occurs, and the problem of traction is unlikely to arise. Nevertheless, we would like to emphasize that the right-angle take-off is a definite advantage and permits implantation of a more compact unit. Efforts should therefore be directed to the prevention of rotational movement of the pulse generator.

Thirdly, care must be taken to prevent enlargement of the subpectoral pocket. There appears to be a natural tendency for fluid accumulation and pulse generator movement gradually to enlarge the pocket in the initial stages of healing. The unit should be implanted in the plane between the pectoralis major and minor muscles to avoid the loose subcutaneous tissue present in older patients. The pocket should be a tight fit initially, and should be plicated if necessary once the pulse generator is in position. The use of a simple pouch of polypropylene mesh[5] to enclose the pulse generator has been suggested as a means of promoting better tissue fixation. Perhaps most important is the use of a firm compression dressing for five to seven days postoperatively. In the case presented, only a light dressing was used and because of movement of the inserted unit as well as fluid accumulation, this permitted enlargement of the initially dry pocket.

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