A novel approach to determining the cause of pacemaker lead failure

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■ A case of pacemaker lead dysfunction with subsequent removal by a unique transvenous extraction system is presented. This technique allows detailed examination of the extracted pacemaker lead and identification of unsuspected causes of lead failure. Information relative to specific causes of lead failure may result in lead design improvements.

□ INDEX TERMS: PACEMAKER, ARTIFICIAL; EQUIPMENT FAILURE □ CLEVE CLIN J MED 1992; 59:91–92

CHRONIC TRANSVENOUS pacemaker lead extraction has historically been advocated only when life-threatening problems develop which are related to the implanted leads.[1] This approach has been adopted because of the difficulty in removing leads transvenously and the morbidity of thoracotomy, while chronically retained leads have a low incidence of morbidity.[2] Indications for lead extraction are not clear but include extracting infected transvenous lead systems, freeing the subclavian system of obstructive nonfunctional leads, and determining the cause of failure of nonfunctional leads.

Pacemaker lead dysfunction is usually attributed to conductor or insulation failure, lead migration, or excess fibrosis at the lead-endocardial interface. More specific identification of the cause for lead failure is possible when dysfunctional leads are extracted *in toto* and examined in detail. We report a case of lead dysfunction and subsequent extraction by a unique transvenous system which allowed detailed examination of the lead and identification of an unsuspected cause of dysfunction.

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CASE REPORT

A 70-year-old man was admitted to The Cleveland Clinic for coronary artery bypass grafting (CABG) and aortic valve replacement (AVR). Four years earlier, the patient had experienced an episode of bradycardia associated with significant hypotension for which a ventricular demand pacing (VVI) pacemaker was implanted. During the year prior to admission he developed angina and episodes of near-syncope lasting up to 15 minutes.

Cardiac catheterization revealed moderately severe aortic stenosis and significant coronary artery disease. The preoperative pacemaker evaluation showed lack of sensing but satisfactory pacing thresholds and proper lead positioning in the right ventricular apex. The patient underwent successful CABG and AVR.

On the fifth postoperative day, the pulse generator was removed, and the bipolar polyurethane-coated lead (Medtronic model 4002) was tested (*Table*) with intracardiac electrograms and a pacing system analyzer

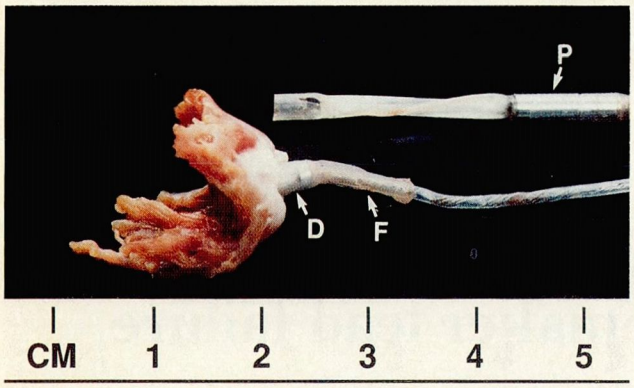

FIGURE. Fibrotic tissue (F) growing under the outer insulation from the distal (D) to the proximal (P) electrode.

TABLE
RESULTS OF TESTING LEADS

Parameters	Old lead	New lead
Pacing characteristics		
Threshold	1.7V	1.0V
Current	20.5mA	0.9mA
Pulse width	0.5ms	0.5ms
Impedance	83Ω (calculated)	859Ω (measured)
Sensing characteristcs		
R-wave amplitude	8.4mV	9.6–11.2 mV
Peak to peak	7.8 mV	9.6 mV
Slew rate	2.6 V/sec	0.88 V/sec

(Medtronic model 5311). This lead was extracted transvenously,[3] employing a locking stylet and a dilator sheath (from the Cook Pacemaker lead extraction system, Cook Pacemaker Corp., Leechburg, Pennsylvania). Some myocardial tissue was avulsed with the lead tip at the time of lead extraction. A new lead (Intermedics model 438-02) was implanted and tested (*Table*). The patient remained hemodynamically and clinically stable but was carefully monitored after he returned to his room in good condition. Sensing and pacing thresholds of the new lead remain satisfactory 14 months after hospital discharge.

Gross examination of the extracted lead showed that fibrous tissue had grown under the insulation at the junction of the insulation and distal electrode (*Figure*). The fibrous tissue had advanced to the proximal electrode under the insulation. In addition, an abundant fibrotic reaction was present at the distal electrode.

DISCUSSION

The low pacing impedance calculated from the preextraction electrical analysis of the old lead (*Table*) suggested deterioration of the insulation as the cause of poor R-wave sensing. Indeed, Medtronic model 4002 leads have been reported to have an abnormally high incidence of insulation failure due to polyurethane degradation. But, in this case, removal of the dysfunctional lead *in toto* revealed an unsuspected probable cause for lead dysfunction—tissue invasion under the lead insulation.

Although no complications resulted from the myocardial avulsion that occurred in this case, hemopericardium, hemothorax, or cardiac tamponade could occur with this procedure. Therefore, patients undergoing lead extraction should be monitored with an arterial line, and two-dimensional echocardiography, pericardiocentesis tools, and surgical backup should be available. The recent addition of telescoping dilator sheaths to this technique has reduced this risk, but the procedure must be done with care.

The locking stylet/dilator sheath system permitted lead extraction *in toto* without a thoracotomy, and it facilitated examination of the lead. Consistent removal of dysfunctional leads with careful lead analysis may result in lead design improvements.

ACKNOWLEDGMENT

We wish to thank Karen L. Chuba for her assistance with the preparation of this manuscript.

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