Benefit of the Steroid Electrode*

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A permanent pacemaker was implanted in an 80-year-old patient with complete heart block. The electrode had to be changed six times (endocardial, five and epicardial, one) due to a progressive rise in threshold with recurrent exit block. A porous, steroid eluting, endocardial electrode was implanted and has given 27 months of excellent service. This lead may be ideal for patients who have a progressive increase in threshold after repeated electrode implantation.

Various electrodes have been designed to obtain low stimulation thresholds and greater sensing amplitudes. The steroid eluting electrode may be the treatment of choice

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for patients with a tendency to develop recurrent exit block from a rise in endocardial stimulation threshold.

CASE REPORT

An 80-year-old woman with degenerative disease of the cardiac conduction system developed symptomatic, complete heart block in 1969. A permanent pacemaker with an endocardial ventricular electrode was then implanted. One year later, this electrode appeared to have an outside contact, and an epicardial electrode was implanted.

The pacemaker functioned normally (the pulse generator was changed in 1975) until 1982 when symptomatic pacemaker exit block was observed. The stimulation threshold rose from 0.5 V at implantation to 5.0 V. A new ventricular endocardial electrode (Medtronic 6972) was implanted with a stimulation threshold of 0.5 V at a pulse width of 0.3 ms.

During the next two years (until August 1984), the patient was hospitalized frequently for recurrent symptomatic pacemaker exit block. The electrodes were changed on six occasions during this period (one epicardial and five endocardial), and progressive exit block occurred. The stimulation threshold rose to 4.5 to 8.5 V from an initial 0.4 to 0.6 V at implantation. The pulse generator was also reprogrammed externally on several occasions using an external programmer by increasing pulse width stepwise from 0.4 to 2 ms, but capture was maintained for a few days only after each change.

In August 1984, an endocardial steroid eluting electrode was implanted with a stimulation threshold of 0.5 V at a pulse width of 0.5 ms. Five months later, the stimulation characteristics were unchanged. Twenty-seven months later, at follow-up, the patient was asymptomatic. Figure 1 shows a chest x-ray film of the patient with several electrodes in her heart.

**DISCUSSION**

Different electrodes have been designed to obtain low stimulation thresholds and greater sensing amplitudes to maintain capture, prolong battery life, and prevent exit block.

Porous steroid-eluting electrodes give stable, lower, long-term thresholds in dogs and man, and improvement in sensing activity with time has been reported. The steroid

**FIGURE 1.** Posteroanterior and lateral views show the multiple epicardial and endocardial electrodes.
electrode (Medtronic 4003) has a straight titanium electrode tip coated with porous platinum. It is hemispherical in shape and has a surface area of 8 mm². The tip contains a silicone rubber pellet in which 1 mg of dexamethasone sodium phosphate has been dissolved; this is eluted within weeks after implant. It also has a multifilary MP-35N nickel alloy conduction coil and polyurethane insulation. The electrode shows a very small increase in pacing threshold during the acute phase. Although the steroid content of the electrode is almost exhausted within weeks after implantation, the low threshold, in our patient, persisted for two years of follow-up. This may be due to the release of steroid during the initial period when the scar tissue forms around the electrode tip.

The antiinflammatory and antifibrotic action of the steroid might reduce the tissue response to irritation during the stabilization. This lowers pacing impedance which permits a greater current density, may persist long after the elution period, and suppress further the local tissue reaction, although direct evidence is lacking. Alternatively, the glucocorticoid steroid may alter membrane receptors, which in heart muscle and blood vessel walls, may potentiate the effects of circulating catecholamines and improve the stimulation threshold. Third, the steroids could alter cellular membrane permeability, and this has been the rationale for systemic administration of steroids to overcome exit block in the postimplant period.

Our patient had several electrodes, epicardial and endocardial, implanted during a two-year period because of recurrent exit block and a rise in stimulation threshold. Evidence of systemic inflammatory disease was not found. An endocardial porous steroid electrode was implanted, and after five months, there were no changes in the stimulation characteristics. After more than two years of follow-up, the patient is still asymptomatic, has excellent pacing, and has benefited from the steroid electrode.

Although the precise mechanism responsible for the action of the steroid eluting electrode is uncertain, it appears to be the treatment of choice for patients who have a tendency to develop recurrent exit block from a rise in endocardial stimulation threshold.

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Malignant Course of a Benign Anomaly: Myocardial Bridging*
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A 35-year-old man, with recent onset angina, developed recurrent episodes of syncope due to ventricular tachycardia. His coronary angiogram showed normal coronary arteries and myocardial bridging of the left anterior descending causing severe systolic milking effect. Extensive invasive and noninvasive investigations did not reveal cardiac pathology other than the myocardial bridging. Electrophysiologic studies, not previously reported in myocardial bridging, demonstrated inducible sustained ventricular tachycardia at a rate of 250 beats/min. The possible relationship between the arrhythmia and the myocardial bridge is suggested. Combined medical treatment with amiodarone and diltiazem proved to be an effective alternative to surgical myotomy of the bridge.

Myocardial bridging is characterized by systolic compression of a portion of the coronary artery by a segment of overlying myocardium. It commonly involves the middle segment of the left anterior descending coronary artery. Symptomatic patients are most frequently middle-aged men with typical or atypical chest pain, both related or unrelated to exercise. The course is usually benign; however, myocardial ischemia, infarction and sudden death have been reported. To the best of our knowledge, there are no previous reports of electrophysiologically documented sustained ventricular tachycardia associated with myocardial bridging.

CASE REPORT
A 35-year-old man, with no history or risk factors for coronary artery disease, was hospitalized due to syncope during a soccer ball game. The syncope was preceded by an episode of squeezing chest pain lasting 30 minutes. On his way to the hospital he had a second syncopal episode. At this time a run of sustained ventricular tachycardia was seen on the monitor. Retrospectively, he recalled having had isolated episodes of similar chest pain a month prior to admission, mainly related to exertion. The admission ECG (Fig 1) was interpreted as apical or anterolateral myocardial infarction; however, a previous routine ECG recorded four years earlier showed an identical pattern. Physical examination, chest roentgenogram and routine laboratory test results were normal. Creatine kinase and glutamic oxaloacetic serum cardiac enzymes were elevated at 928 units (normal <120) and 113 units (normal <40), respectively. Lactic dehydrogenase remained normal in all determinations. A technetium pyrophosphate scan and echocardiogram were normal. A radioisotope angiogram revealed an ejection fraction of 53 percent for the right ventricle and 51 percent for the left ventricle. However, after administration of sublingual nitroglycerin, the left ventricular ejection fraction decreased to 42 percent, while the right ventricle increased to 55 percent. Serial ECG findings remained unchanged and the monitor showed isolated premature ventricular beats. On the fifth hospital day, coinciding with an anxiety-provoking situation, he suddenly became unconscious and pulseless; the monitor showed

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