Permanently Implanted Transvenous Pacemakers

Electrical Measurements of Function

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SUMMARY

Three patients with complete heart block had a high resistance to electrical stimulation of the endocardium through transvenous pacemaker systems. One patient died; one needed reoperation for implantation of epicardial electrodes; and the third required a generator of higher current. The present enthusiasm for permanent transvenous implantation of pacemakers must be tempered somewhat by the knowledge that high endocardial resistance to pacing represents another cause of failure. Simple and rapid methods of determining the current necessary to stimulate the endocardium are available and should be used prior to permanent implantation of the generator. If a substantial margin of safety does not exist, one should consider substituting a pulse generator with greater output or an epicardial electrode system.

Additional Indexing Words:
- Pacemaker failure
- Myocardial stimulation threshold
- Pacemaker testing
- Threshold failure
- Transvenous pacemakers
- Pacemaker testing

A DDITION OF PACEMAKERS to the therapy of complete heart block has initiated a new era in the treatment of a previously malignant cardiac disorder. The use of permanently implanted generators with epicardial wire attachment has become an established surgical approach in spite of the fact that general anesthesia and thoracotomy are necessary in seriously ill patients. The recent availability of permanent transvenous pacemakers has offered the possibility of implantation at lower risk since thoracotomy can be avoided. Overall hospital stay has been reduced and clinical results have been comparable to those in patients with epicardial electrodes.

However, problems still beset the physician caring for patients with transvenous pacemakers. Pulse generator failure can occur. The intracardiac electrode may fracture. The catheter may become displaced, thwarting stimulation. Myocardial perforation may occur, and occasionally the threshold of stimulation at the endocardial surface may rise. The latter problem, that of so-called threshold failure or exit block, has been discussed frequently, although seldom has it been clinically important.

This report deals with three patients in whom high thresholds were probably responsible for the inability to pace. In two patients the endocardial thresholds were measured at surgery. One failure resulted in death of the patient. Attention is drawn to problems of endocardial pacing with the hope that greater attention to detail and precise electrical analysis may prevent these misfortunes.

Methods

Effective low current ventricular capture by transvenous pacing systems can be assured by...
With bipolar catheter systems we believe that at least a 100% margin of safety should be allowed, that is, threshold of less than 2.5 milliampere for a pulse generator of 5 ma maximum output. Adequate allowance must be made since substantial changes in ventricular threshold are expected for several months after implantation.\textsuperscript{5, 12, 13} For a unipolar catheter electrode system,\textsuperscript{**} a threshold of only 1 ma is usually required.\textsuperscript{10} If a high threshold is demonstrated, the catheter must be repositioned and testing should be repeated. If variable thresholds are measured, the catheter is assumed to be improperly positioned. If no satisfactory site can be obtained for low current pacing, one should use a generator with greater output or consider a transthoracic approach with epicardial implantation.

**Report of Cases**

**Case 1**

FB, a 75-year-old man, developed sudden spells of dizziness and unconsciousness 2 months prior to hospitalization. These continued at irregular and variable intervals. After hospitalization, an electrocardiogram showed complete A-V block with idioventricular rhythm. There was no history of ischemic pain or myocardial infarction. Transvenous pacing was instituted via the right antecubital vein and was continued for 3 days. Stimulation was initially successful at an applied current of 6 ma. Over the next 3 days, however, gradually increasing current was necessary. Pacing was continued with the external battery-driven unit\textsuperscript{††} set at approximately 13 ma. Under local anesthesia an incision was made in the right side of the neck, but as preparations were being made to pass the transvenous electrode catheter, the patient became unconscious. The right-sided catheter had not been manipulated. The pacemaker impulse was still seen on the oscilloscopic screen and was unchanged in amplitude, but it was ineffective. The patient failed to respond to closed chest massage and mouth-to-mouth breathing as well as to intravenous injection of isoproterenol and intracardiac injection of epinephrine. Maximum current output of the generator failed to stimulate the heart.

At postmortem examination, the catheter was found to be well impacted at the apex of the right ventricle. Electrical testing showed a catheter resistance of 26 ohms. There was no evidence of infection, clot, or perforation, and no fibrotic reaction was seen around the tip. Sections of the heart from an area near the pacing site (fig. 1)
showed diffuse fibrosis with marked scar replacement of subendocardial tissue. There was severe obstructive coronary disease with patchy areas of muscle loss and fibrosis indicative of prior infarction.

The output of the pacemaker was subsequently analyzed. There was no change in the duration or configuration of the stimulus and at highest setting, the current generator produced 14 ma.

Case 2

DC, a 22-year-old man, had a Brock procedure for pulmonary stenosis at age 9 years. At age 17, a diagnosis of large ventricular septal defect was made. For closure, cardiopulmonary bypass and hypothermic arrest were used, and the defect was bridged with a Dacron prosthesis. Complete heart block ensued and a myocardial wire was implanted for 1 month. Subsequently, a permanent epicardial pacemaker implant functioned for 3 years. After its pulse generator failed, replacement was followed by infection which spread through the tract into the pericardial space. The entire tract was opened and allowed to heal by secondary intent while the patient was paced for over 3 months with an electrode placed through the left jugular vein. Subsequently, right thoracotomy was performed, and wires were implanted on the right ventricle with the pulse generator inserted into the right upper quadrant. This unit functioned effectively for 9 months until sudden failure again occurred. Exteriorization of the wires and testing showed that neither electrode would consistently transmit an impulse to the heart, implying that both were fractured.

Since the patient had experienced five previous thoracotomies, transvenous pacing through the left subclavian vein was suggested. This was accomplished easily and a pulse generator with 5 ma maximum output* was buried over the left pectoralis muscle. Pacing continued for 7 weeks,

*Medtronic Co., Minneapolis, Minnesota, Model 5870C.

Figure 1

There is a large area of subendocardial fibrosis and hyalinization containing a few isolated clusters of myocardial fibers. Essentially normal muscle bundles are seen elsewhere. This section is taken from the area where the distal stimulating electrode lay.
but again failure occurred. The pocket was opened and the sleeves of the pulse generator were found to be fractured and fluid was present in the cups. The pulse generator was replaced, but the heart would not respond to stimulation. Since x-rays had shown a 10° angulation at the tip of the transvenous catheter, wire fracture was suspected. The catheter was removed and the coiled stainless steel wire was found to be fractured at a point approximately 0.5 cm from the distal platinum electrode. A new bipolar electrode was inserted and pacing ensued. Over the next few days, however, pacing again failed and could not be altered with isoproterenol, ephedrine, or corticosteroids. After 4 days left thoracotomy was performed, and a synchronous pacemaker was implanted.

Prior to epicardial implantation, electrical measurements were obtained. The minimum current necessary to drive the ventricle with the transvenous electrode was 11 ma. This catheter was thought to be well positioned at the ventricular apex because there was evidence of impaction of the tip causing slight flexion with each systole, because ventricular premature beats could be easily produced by slight forward advancement of the catheter, because position was unchanged on x-ray and fluoroscopic examination, and because threshold measurements were consistent. The resistance of the catheter electrode was 20 ohms. When electrodes were placed on the epicardium, stimulation occurred at 0.75 ma. Resistance was 1,000 ohms. Subsequent to surgery, synchronous pacing has continued.

Case 3

MN, a 79-year-old woman, was hospitalized because of intermittent pacing failure. Eighteen months previously a permanent transvenous pacemaker had been inserted because of syncope due to complete heart block. There had been no intervening complications. Generator failure was assumed and surgical removal was performed under local anesthesia. When tested, this unit produced 5.5 ma at 1,000 ohms of resistance. A consistent threshold of 5.8 ma was measured through the transvenous catheter. Attachment of a new generator with 5.6-ma maximal output allowed only intermittent transmission of the impulse. A second generator was adapted to the bipolar catheter with immediate capture. The postoperative course was uneventful.

Discussion

In the past, pacing failures have invariably been traced to mechanical faults arising within the generator-electrode system. Although stimulation thresholds for myocardial tissue have shown substantial rises both immediately after surgery and for many months thereafter, problems of increasing resistance (“threshold failures”) have occurred infrequently.

Low currents are easily capable of stimulating the heart. In dogs, pulses of standard 1 to 2 msec duration will produce cardiac contraction when 0.3 to 2.5 ma are applied at the epicardial surface and when 1 to 4 ma are applied to the endocardium. Similar findings have been noted in man. The current threshold may rise in time, but in man it has seldom exceeded 4 ma.

As a consequence, manufacturers have specifically designed their pulse generator systems to exceed these demands. The majority of available units allow either 5 or 10 ma output. In two of the cases reported herein, these outputs were greatly exceeded by threshold values of 11 ma in one and in excess of 14 ma in the other.

We believe that in these two patients increased threshold and resistance to stimulation were caused by myocardial fibrosis. In the first one there was a rapid increase in endocardial threshold, and at autopsy microscopic sections showed extensive subendocardial fibrosis in the region where stimulation had occurred. In the second patient there were a variety of technical problems. However, after their correction, a high current threshold was still measured at the endocardial surface. The minimum threshold level of 11 ma was consistently found, and this suggested that regardless of location its relation to the endocardium was stable. Although no pathological material was available in the second case, myocardial fibrosis again may be inferred since a number of processes known to produce it were present. These included severe pulmonary valvular stenosis, a left-to-right shunt of large volume at the ventricular level, surgically induced pulmonary valvular insufficiency and extensive inflammatory disease.
within the pericardial space. The cause of threshold rise in the third patient is unexplained.

It should be clearly understood that myocardial fibrosis is not the only reason for the changes described. Other causes of increased current demand must be excluded before a diagnosis of threshold failure can be made. Faults such as poor electrode stabilization and wire fracture can change current need. These represent correctible dysfunctions which can be solved without recourse to a trans-thoracic procedure.

The technique of catheter electrode insertion requires an intimate contact between the stimulating body and the endocardial surface. An apparent rise in the stimulation threshold can be mimicked by improper positioning and mechanical instability of the electrode. Fluoroscopy allows only a gross appreciation of catheter position. Impaction of the electrode beneath the trabeculae carneae in contact with excitable tissue must be assured before the need for high current will suggest myocardial fibrosis. Stable thresholds less than 50% of pulse generator output imply adequate placement of a bipolar electrode and adequate current reserve. In the chronic implant, fracture of the catheter wire may also increase voltage demand. This fault may be suspected roentgenographically, but positive identification cannot be made until the electrode is exteriorized and tested.

Electrical testing, therefore, is essential for analysis of generator and electrode function both before the completion of surgery and in cases in which stimulation has failed months or years later. The additional time required for these measurements will be justified if a faulty electrical system, an unstable catheter position, or a high threshold can be identified before closure of the incision. It is no longer sufficient to consider pacing failure a function of generator deterioration or electrode fracture alone.

The technique described is by no means the only one available to physicians for making such measurements. The type of testing equipment used will depend upon the nature of the pacing system employed. When permanent transvenous pacemakers are coupled to induction coils and stimulation is produced either by external battery units or by radio-frequency transmitters, the signals may be progressively retarded until threshold is measured.7,20 One manufacturer* of implanted battery-driven pulse generators distributes clip-on resistors which can attenuate the output to 0.7 or 1.5 ma for gross assessment of threshold prior to implantation of a unipolar stimulating electrode. Sowton9 has described a manually operated magnetic reed switch that can reduce the generator output by 50%. All of these techniques have merit, and the choice of one or another depends upon the pacing unit employed. The major point to be stressed is that such measurement should be made in all patients whenever possible since a high resistance to electrical stimulation of the heart may be an important cause of pacemaker failure.

The exact frequency of this complication is unknown, since the technique is new and only a few such failures have been reported.8,14 However, a recent technical memorandum (Cordis Corporation, February, 1967) implies that in a substantial number of patients with transvenous systems requiring reoperation for pacing failure, a rise in threshold has been responsible. We have measured thresholds in two other patients in whom pulse generators have failed and have found values of 2.8 and 3.7 ma. Because of our unfortunate experiences and the potentially lethal nature of this complication, we believe that ideally a pulse generator of 10 ma output at physiological resistances should be chosen. This should in most cases allow an adequate reserve.

We do not believe that our findings should discourage transvenous implantation which remains an important palliative therapy for the elderly patient with complete heart block. However, as more pacemakers are inserted into patients with severe coronary disease and with the various forms of cardiomyopathy, myocardial fibrosis and increasing electrical

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*Cordis Corp., Miami, Florida.

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resistance to stimulation should be expected and sought.

References

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