Suppression of Demand Pacemakers by Inactive Pacemaker Electrodes

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SUMMARY

Ventricular inhibited cardiac pacemakers were suppressed by intimate contact of the tip of the active pacemaker electrode with that of the inactive pacemaker electrode in two patients. This contact apparently was sensed as myocardial electrical activity and resulted in variable suppression of pacemaker emission. There was no interference with R-wave sensing function. The threshold for stimulation was unaffected. There was no loss of capture as tested with the magnet-controlled continuous mode of stimulation. This type of pacemaker failure can be remedied simply by withdrawing the tips of the electrodes from intimate contact.

Additional Indexing Words:
Electrode interference Pacemaker failure Pacemaker inhibition
Ventricular inhibited pacemaker

It is well known that demand cardiac pacemakers, of both the ventricular inhibited and ventricular synchronous type, are subject to electrical interference. Ventricular inhibited units sense extraneous electrical activity as of cardiac origin, and pacemaker emission is temporarily suppressed. Ventricular synchronous units respond to external electrical stimuli of adequate strength by extra pacemaker emissions. A new type of interference has been documented in our experience, presumably electromechanical in origin, as illustrated by the two cases described herein. In both cases inactive temporary transvenous bipolar electrodes, disconnected from all electrical sources, and properly insulated, but left adjacent to the permanent bipolar electrodes, with intimate contact of the electrode tips, caused suppression of the demand inhibited cardiac pacemakers connected to the permanent electrode.

Report of Cases

Case 1

A 70-year-old man presented with atrial fibrillation with a ventricular response averaging 40 beats/min and cardiac failure unresponsive to diuretic therapy. A temporary transvenous bipolar "semifloating" pacemaker electrode (Elecath, 4F, 100 cm) was inserted percutaneously into the right internal jugular vein and positioned at the apex of the right ventricle. R-wave sensing was above 16 mv, and the threshold for ventricular stimulation was less than 1 v, tested by the American Optical Corporation's demand pacer (battery operated, catalog no. 262002). After 8 days of pacemaker stimulation at a rate of 70, cardiac function had improved, and permanent pacemaker implantation was performed.

A bipolar electrode (Chardack thin-diameter catheter electrode, 9-10F tip, 58 cm long [American Optical Corporation, catalog no. 283101]), was inserted under fluoroscopic control into the apex of the right ventricle via the left internal jugular vein. Its tip lay adjacent to the temporary pacemaker catheter. X-ray views confirmed the position (fig. 1).

The pacemaker electrode was then loosely secured in position with 2-0 silk sutures. Using the AO Demand Pacer the R-wave sensing level was found to be above 16 mv and the threshold
for stimulation was less than 1 v. Additional sutures then secured the permanent electrode more firmly. The permanent electrode was then connected to a bipolar demand pacer (American Optical Corporation Cardio-Care Bipolar demand pacer, catalog no. 281003), demand rate 70 to 75 pulses/min. Erratic suppression of the demand unit was then recorded (fig. 2A). The malfunction was attributed to a defect in the pacemaker, and five different units readily available were inserted, but with each the same variable suppression was documented. By use of the magnet for conversion of the pacemaker to continuous-mode stimulation, proper competitive pacemaker function and capture were documented (fig. 2B).

Electrocardiographic recordings of leads I, II, and III failed to reveal any focus of electrical activity not seen in lead V1. Nonetheless, 100 mg of lidocaine was administered intravenously in an attempt to abolish concealed ectopic ventricular activity. There was no change in the erratic suppression of the demand pacemaker, although there was elimination of the escape beats and premature ventricular beats previously seen (fig. 2C).

Throughout all this time a search was made for possible sources of improper grounding and none was found. The connections from the patient to the temporary external demand pacemaker had proper insulation, were firmly secured, and had been disconnected from the external demand pacemaker unit. The temporary pacemaker electrode was thus completely isolated from any sources of electrical activity.

At this point it was suspected that the close proximity of the tip of the temporary electrode to that of the permanent electrode might have been the cause of the suppression phenomenon noted with the five different implantable pacemakers used. Accordingly, the temporary electrode was withdrawn approximately 2 inches. There was immediate documentation of proper pacemaker function at a rate of 72 pulses/min (fig. 2D). Recovery of the patient was uneventful. Proper pacemaker function continued throughout the remainder of the hospitalization and on follow-up 1 month later.

Case 2
A 64-year-old man developed syncope with variable heart block including 1° heart block, bifascicular block, episodes of Mobitz type II 2° block, and A-V dissociation with nodal rhythm. A temporary transvenous pacemaker was inserted, as in case 1. Ten days later, after stabilization on a drug program using procainamide and digitalis, a permanent pacemaker electrode was inserted via the left cephalic vein. It was positioned in the apex of the right ventricle with its tip adjacent to that of the temporary electrode. The R-wave sensing level and threshold for stimulation were measured, as in case 1, with an external demand pacemaker unit (American Optical Corporation) connected to the permanent electrode. In the course of this testing intermittent suppression of the external unit was recorded (fig. 3). Again the temporary electrode was fully insulated and had been disconnected from its external unit. In this case, the permanent electrode had not yet been firmly secured in position. Minor adjustment of the electrode position with separation of the catheter tips of the two electrodes resulted in immediate cessation of suppression. An implantable unit was then connected, and proper function documented. Recovery of the patient was uneventful.

Discussion
Demand cardiac pacemakers offer a degree of safety from electrical competition not available with continuous rate pacemakers. They also are more complex and have been found to be subject to interference phenomena from internal and external sources. Possible internal sources of interference include triggering from physiologic sources other than the QRS complex, such as a high-amplitude T wave, or a focus of concealed myocardial electrical activity as suggested by Bilitch and
Case 1. Electrocardiograms, lead V₁, 25 mm/sec. (A) Suppression of pacemaker emission is evident after the first paced beat which is followed by a pause longer than the escape interval of the unit (approximately 0.84 sec). After an escape beat and a premature ventricular beat there is an appropriate interval and then pacemaker firing is resumed at the proper rate, followed by further pacemaker suppression without any evidence of interfering electrical spikes. (B) Proper magnet-controlled continuous-mode pacemaker function at a rate of 80/min. There is no pacemaker suppression by spontaneous beats or other electrical influences. (C) Strip recorded after administration of a 100-mg bolus of lidocaine showing continued variable suppression of pacemaker emission. No escape beats and no premature beats are evident. (D) Strip recorded immediately after withdrawing the tip of the temporary electrode from intimate contact with that of the permanent electrode. There is proper pacemaker emission at a rate of 72.

Case 2. Electrocardiogram, lead V₁, 25 mm/sec. Variable suppression of pacemaker emission is evident after the first four paced beats (the pacemaker spikes are not as clearly evident as in case 1). The pause is followed by an escape beat and then proper pacemaker function is resumed.

*Figure 2*

*Figure 3*
The variable suppression with unequal and irregular intervals ruled against sensing of physiologic electrical activity. Likewise, the failure of lidocaine to alter the suppression ruled against a concealed electrical focus.

External sources of electrical interference with demand pacemaker function include automobile ignition systems, electrocautery units, radiofrequency transmitters, radar transmitters, physiotherapy diathermy units, electric razors, and microwave ovens.

None of the aforementioned devices was a factor in our two cases. This directed attention to the electrode systems themselves.

It is known that temporary pacemaker electrode systems with faulty electrodes, loose contacts, or excessive electrode mobility can inhibit demand pacemaker function. Again, it is stressed that in our cases no defects were found nor loose connectors identified in the temporary system, and the temporary system was inactive during the episodes of suppression.

The two cases of this report document a new source of electromechanical pacemaker interference, which probably is similar in mechanism to that described in systems with only a single electrode by Furman and coworkers and by Lasseter and associates. However, in our cases there was probably a change in electrical resistance associated with intermittent contact of the two bipolar catheter tips. These events were then sensed as myocardial activity with resultant suppression of emission of pacemaker beats. This occurred both with implanted demand units (case 1) and with the external demand unit (case 2). This suppression of pacemaker emission can be reproduced by connecting a unit to an electrode placed in saline. Pacemaker emission also is variably suppressed by rubbing an inactive electrode against the tip of the active electrode. This suppression is evident with R-wave sensing levels up to 8 mv.

This type of pacemaker malfunction can cause great concern at the time of implantation if the source is not recognized. Ordinarily, one would be hesitant to move a permanent electrode once a position with excellent R-wave sensing and a low threshold for stimulation has been achieved. Likewise, with malfunction of the permanent pacemaker, one is hesitant to remove a well-positioned temporary electrode. Since this type of pacemaker suppression is caused by intimate contact of the temporary and permanent electrode tips, it is evident that one or the other must be repositioned. In case 1, the catheter had been secured to the vein before the malfunction was recognized. Accordingly, after R-wave sensing and threshold values had been found excellent, and proper function of the implanted unit was documented with the magnet-controlled continuous mode of stimulation, it was felt better to remove the temporary electrode catheter. In case 2, the permanent electrode was not yet firmly tied at the vein junction, and the minor repositioning of it eliminated the suppression.

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