INHIBITION OF DEMAND PACEMAKERS by musculoskeletal potentials has been reported to occur only with unipolar pacing systems. Mymin et al., however, described one case of apparent suppression of a bipolar unit by myopotentials but gave no details about the mechanism of interference. Peter et al. recently described transient inhibition of a unipolar demand pacemaker during deep respiration and suggested, without direct proof, that the pacemaker was suppressed by myopotentials associated with respiration.

We describe in this report transient inhibition of a bipolar demand pacemaker by diaphragmatic myopotentials. This unusual type of interference became apparent only with active contraction of the diaphragm such as deep respiration, straining, Valsalva maneuver, coughing, sneezing, and laughing.

**Case Report**

A 61-year-old male was admitted to the Genesee Hospital on 11/6/70 with recurrent syncope and dizziness for several months. The electrocardiogram (ECG) showed normal sinus rhythm, left anterior hemiblock, and complete right bundle branch block. Continuous monitoring revealed Mobitz type II atrioventricular (A-V) block soon complicated by prolonged asystole with a Stokes-Adams attack only a few hours after admission. A permanent transvenous pacemaker was implanted using a Medtronic demand pulse generator (model 5842) and a 5818 bipolar electrode. The threshold for bipolar pacing was 0.7 mA measured with an external 5840 Medtronic pulse generator. Over the next three days the ECG showed occasional prolongation of the spike to spike (SS) interval to less than 2 automatic intervals (fig. 1). We interpreted these pauses as either sensing of the T-wave or voltage afterpotential (or both) because application of the magnet over the pulse generator consistently restored regular pacing. Some of the prolonged SS intervals were less than the sum of the automatic interval and the paced (delivery) refractory period of the generator, suggesting partial recycling from a signal of marginal amplitude for sensing. Chest X-ray revealed good electrode position and no evidence of perforation. We considered the abnormality benign and indeed the patient became asymptomatic and returned to work.

For several years on regular follow-up visits, the ECG continued to show conducted sinus rhythm with right bundle branch block and left anterior hemiblock so that evaluation of pacemaker function necessitated application of the magnet. We electively replaced the pulse generator on 4/26/73. The chronic threshold for bipolar pacing was 3.5 mA measured with an external 5880A Medtronic pulse generator. The unipolar ventricular electrogram displayed normal intracavitary morphology and neither the unipolar (from tip and proximal electrodes) nor bipolar electrograms registered the artifacts commonly seen with an intermittent wire fracture; the amplitude of the P wave was less than 0.25 mV. A new Medtronic demand pulse generator (Model 5842) was implanted. Ventricular capture occurred only after application of the magnet.

A 24-hour Holter recording in January 1975 revealed normal pacing and sensing, except for a single pause considered to represent T wave sensing. We failed to detect further irregularity until 5/2/75 several months after the start of transtelephone monitoring. The patient now appeared to be pacemaker dependent and the SS interval occasionally lengthened to less than two automatic intervals. Application of the magnet again resulted in consistently regular SS intervals. The patient remained well and was followed at weekly intervals over the telephone. We finally rejected the diagnosis of T-wave sensing in January 1977 when the SS interval lengthened to more than twice the automatic interval. The patient returned for evaluation and a long ECG showed normal pacing during spontaneous respiration. The automatic interval was unchanged. There was no pacemaker sound or twitching of the diaphragm or intercostal muscles. Deep inspiration unmasked the abnormality and caused irregular and occasionally prolonged inhibition of the pacemaker. Transient suppression was consistently reproducible with deep respiration, straining, the Valsalva maneuver, coughing, sneezing, and laughing (figs. 2, 3). Inhibition occurred both in the supine and sitting positing. 

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From the Division of Cardiology, Department of Medicine, The Genesee Hospital and University of Rochester School of Medicine and Dentistry, Rochester, New York.

Address for reprints: Dr. S. Serge Barold, The Genesee Hospital, 224 Alexander Street, Rochester, New York 14607.

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tions. None of these maneuvers could inhibit the pacemaker when the magnet was applied. Very long rhythm strips with the magnet did not produce evidence of an intermittent wire fracture because the spike did not change in size (in leads usually unaffected by respiration) and the SS interval remained normal and never doubled suddenly. The magnet was then removed and the following tests were performed during spontaneous respiration; a) raising of the arms; b) bending and turning of the trunk; c) isometric exercise techniques to produce musculoskeletal potentials capable of suppressing unipolar demand pacemakers. The SS intervals remained normal during all these procedures.

The size and direction of the pacemaker spikes in the 12-lead paced ECG had not changed since 1979. The implanted pulse generator was removed with the tentative diagnosis of an intermittent wire fracture near the distal end of the pacing catheter. The permanent electrode was immediately connected to an external 5880A Medtronic pulse generator because the patient was pacemaker dependent. Neither the 5880A Medtronic pulse generator nor an old 5840 model (both on full demand sensitivity) could be inhibited by any of the maneuvers that previously suppressed the implanted pacemaker. These external pacemakers were tested both during bipolar and unipolar pacing (the latter with the tip and proximal electrodes in turn functioning as the cathode). We measured the pacing thresholds with a model 209A Cordis Pacer Systems Analyzer (PSA) delivering a 1 msec constant voltage pulse. The results did not suggest a wire fracture or insulation break (table 1). Bipolar and unipolar pacing with a 10 volt constant voltage output did not stimulate the left hemidiaphragm. With the Cordis PSA unit connected to the permanent electrode, we repeated all the maneuvers that had suppressed the implanted pacemaker. These consistently inhibited the PSA unit during bipolar and unipolar demand pacing, the latter with the tip as the cathode (figs. 4, 5). There was no inhibition when the proximal electrode became the cathode. During some of the long period of inhibition the special sensing light on the Cordis PSA flashed several times indicating that more than one signal was being detected by the unit. Unipolar and bipolar fixed rate pacing with the Cordis PSA unit showed no inhibition.

The ventricular electrograms of spontaneous beats could not be recorded as there was no underlying rhythm. We, therefore, recorded the voltage waveform from the two pacemaker terminals during demand pacing with the Cordis PSA unit. The following arrangements were evaluated: 1) bipolar pacing (fig. 4); 2) unipolar pacing with the tip as the cathode (fig. 5); 3) unipolar pacing with the proximal electrode as the cathode. Very long recordings were obtained on an Electronics for Medicine Recorder (filter settings: 1-500 Hz). Inhibition only occurred with 1) and 2) and the pacemaker waveform did not show the characteristic sudden changes in voltage seen with an intermittent wire fracture. Finally, the waveforms were recorded on a Hewlett-Packard storage oscilloscope and then all the inhibiting maneuvers were repeated during bipolar and unipolar demand pacing. It then became evident that the inhibiting maneuvers created electrical noise from musculoskeletal artifacts, some of which were of sufficient amplitude to be sensed by the demand pacemaker (figs. 4, 5).

A maxilith bipolar pulse generator (Cardiac Pacemakers, Inc.) with a lesser sensitivity (narrow band) was then connected to the permanent electrode. Before closing the pacemaker pocket, we repeated all the inhibiting maneuvers, and the SS interval now remained absolutely constant. The patient has continued to be completely asymptomatic. Several Holter recordings taken over the next few weeks with instructions to take deep breaths, cough, laugh, sneeze intermittently, revealed regular pacing and sensing of ventricular extrasystoles without a single period of inhibition.

We tested the technical characteristics of the explanted pulse generator with two commercially available pacemaker analyzers which revealed normal function according

**TABLE 1.** Threshold Measurements with Cordis Threshold Analyzer (1 msec constant voltage pulse)

<table>
<thead>
<tr>
<th>Pacing mode</th>
<th>Voltage (volts)</th>
<th>Current (mA)*</th>
<th>Impedance (ohms)</th>
<th>Inhibition of Cordis PSA during deep respiration, etc.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bipolar</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 5 volts</td>
<td>5</td>
<td>11.2</td>
<td>446</td>
<td>Yes</td>
</tr>
<tr>
<td>Threshold</td>
<td>2.2</td>
<td>4.8</td>
<td>458</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Unipolar: Tip As Cathode</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 5 volts</td>
<td>5</td>
<td>11.6</td>
<td>431</td>
<td>Yes</td>
</tr>
<tr>
<td>Threshold</td>
<td>2.8</td>
<td>6.2</td>
<td>451</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Unipolar: Proximal Electrode as Cathode</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 5 volts</td>
<td>5</td>
<td>13.9</td>
<td>360</td>
<td>No</td>
</tr>
<tr>
<td>Threshold</td>
<td>1.7</td>
<td>4.4</td>
<td>386</td>
<td>No</td>
</tr>
</tbody>
</table>

*90 msec after onset of pulse.
†Cordis Pacing System Analyzer. Sensitivity 1 mV (square wave 45 msec in duration).
Discussion

Prolongation of the pacemaker spike to spike (SS) interval during deep respiration and the various maneuvers described in this report should always raise the suspicion of an intermittent electrode problem (wire fracture or insulation break) near the tip of the catheter inside the heart.16, 18, 21 The preoperative diagnosis of an intermittent electrode problem was unlikely in our case because application of the magnet did not produce ineffectual or attenuated pacemaker spikes and sudden doubling of the SS interval,18 though the absence of these findings cannot positively rule out the diagnosis. Indeed, an intermittent wire fracture may occasionally present as a pure sensing problem if its timing to specifications. A 209A Cordis Pacing System Analyzer (PSA) revealed a sensitivity of 1.6 mV (using a rectangular voltage pulse having a 45 msec duration and a rise time of less than 2 msec). A Medtronic 5300 PSA revealed normal sensing function (i.e., sensed R wave test signals: standard 4mV, 45 msec square wave signals).

FIGURE 2. A) Prolonged inhibition of bipolar demand pacemaker during a maximal inspiratory effort. B) Temporary inhibition during coughing (intervals in msec). Leads I, II and III were recorded simultaneously.

FIGURE 3. A) Apparent T wave sensing during a moderately increased inspiratory effort. B) Slightly increased inspiratory effort causes partial recycling of the pulse generator suggesting sensing of a signal close to the sensitivity of the demand circuit. The delivery (paced) refractory period of this particular pulse generator is about 250 msec. Compare with figure 1 (intervals in msec). Leads I, II, III were recorded simultaneously.
always allows the fractured ends to be in contact whenever the pacemaker delivers its impulse.\textsuperscript{16,18} In this situation, momentary changes in resistance cause voltage changes across the pacemaker terminals (false signals) with temporary inhibition\textsuperscript{22} and regular fixed rate pacing would therefore occur with the magnet.

Measurements of electrode impedance may also be normal if there is electrical continuity whenever the pulse generator fires. On this basis, we cannot agree with the conclusions of Peter et al.\textsuperscript{15} who felt that mere restoration of regular pacing with the magnet ruled out an intermittent wire fracture in their case of temporary inhibition of a unipolar demand pacemaker by myopotentials associated with inspiration. Moreover, these workers did not confirm their diagnosis by intracardiac recordings. We excluded an intermittent electrode problem in our case by recording the magnified pacemaker waveform directly from the two pacing terminals simultaneously with the surface electrocardiogram. With an intermittent fracture, prolongation of the SS interval would have coincided with sudden and large disruptions of the waveform characteristic of any transient electrode problem.\textsuperscript{21}

Mymin et al.\textsuperscript{3} described the apparent suppression of a single bipolar pulse generator by muscle potentials presumably originating near the implantation site. The geometry of the sensing electrode in a bipolar system makes this occurrence difficult to understand. A wire fracture or loose connection may also present with intermittent cessation of

\textbf{Figure 4.} Bipolar demand pacing. Storage oscilloscope recordings of the pacemaker waveform directly from the two pacing terminals. Deep inspiration causes continuous noise with intermittent peaks as high as 2 mV causing inhibition seen on the surface ECG.

\textbf{Figure 5.} Unipolar demand pacing (tip as cathode). Storage oscilloscope recordings of the pacemaker waveform directly from the two pacing terminals. In bottom panel, during deep inspiration, there is continuous noise from diaphragmatic myopotentials with peaks of about 2 mV causing pacemaker inhibition seen on the surface ECG.
pacemakers by skeletal muscle potentials. JAMA 223: 527, 1973
Inhibition of bipolar demand pacemaker by diaphragmatic myopotentials.
S S Barold, L S Ong, M D Falkoff and R A Heinle

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