Demand Pacemaker Malfunction Due to Abnormal Sensing

Report of Two Cases


SUMMARY

Two cases with external demand pacemakers are presented because of abnormal prolongation in the pacing interval. In both cases, pacemaker inhibition was caused by signals which were not recorded by the conventional surface electrocardiograms. In one case, inhibition was related to a partial lead fracture which generated a voltage transient in the region of the T wave. In the other case, inhibition was caused by current emitted from a faulty pacemaker unit. In both cases precise localization of the problem was possible by simple bedside recordings and measurements.

Additional Indexing Words:

Pacemaker failure Lead fracture Biphasic stimulation

Abnormal inhibition of demand pacemakers due to oversensing is a complex event which is rarely diagnosed thoroughly and convincingly and accordingly its potential implications may go unappreciated. This paper reports two cases of abnormal demand pacemaker inhibition due to oversensing. In the first case, pacemaker inhibition was caused by potentials generated from a broken lead, while in the second case, the inhibition was due to erratic potentials emitted by a faulty pacemaker. These cases illustrate the bedside maneuvers and measurements required to accurately isolate which part of the pacemaker-electrode complex is faulty.

Case Reports

Case 1

A 64-year-old woman was admitted to the Toronto General Hospital because of a history of syncopal attacks. At the time of her admission the electrocardiogram showed second degree atioventricular (A-V) block with a 2:1 conduction ratio. The conducted QRS complexes showed right bundle branch block and left posterior hemiblock. A 6F bipolar electrode catheter was inserted through the right antecubital vein and positioned in the apex of the right ventricle under fluoroscopic control. The catheter was connected directly to an external Medtronic pulse generator (Model 5880) and set to operate in a demand mode. The bipolar pacing threshold was 1.0 mA.

Intermittently it was noted that the actual rate at which the pacemaker discharged was slower than the

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rate at which the unit had been set (fig. 1). This occurred regardless of body position, respiratory cycle or set frequency of pacing. Furthermore, changing the pacemaker unit did not alter the abnormality. In this example the abnormal pacemaker discharge interval was prolonged by 300-440 msec above the set rate. The intermittent slowing was seen only when the pacemaker was functioning in the demand mode and when pacemaker discharges followed pacemaker stimulated beats. The pacemaker discharge rate was equal to the set rate whenever the pacemaker unit was set to operate in a fixed rate mode and whenever a pacemaker escape beat followed a sinus conducted beat (fig. 2). The prolongation in stimulus interval above the set rate suggested that the T wave was inhibiting the pacemaker.

To localize the abnormality, the patient was paced in a unipolar mode by connecting the cathode of the pulse generator to one of the catheter leads, and the anode to a needle under the skin (fig. 2). Unipolar intracavitary electrograms were obtained from the free catheter lead. Both leads of the electrode catheter were alternately used for demand pacing and recording. Pacing through the proximal electrode was normal (top tracing) with the discharge interval being precisely equal to the set rate. Simultaneous electrograms recorded from the distal pole disclosed voltage transients on the descending portions of many of the T waves 300-440 msec after the beginning of the antecedent QRS complex. Pacing through the distal electrode (bottom tracing) produced frequent prolongation in the pacemaker discharge interval (beats 2, 7, 8 and 9). The prolongation following beat 4 permitted a sinus capture beat to emerge (beat 5). The latter beat was followed by a demand paced beat (6) at the set interval. At no time was the pacemaker discharge interval prolonged when it followed a spontaneous beat. A simultaneous electrogram recorded from the proximal pole did not disclose voltage transients.

The abnormal prolongation in the pacing interval was not seen with every cycle. Figure 3 discloses why the abnormal pacemaker resetting occurred following some beats and not others. The heart in this figure was

**Figure 1**

Pacing throughout was set at a cycle interval of 780 msec. Beats 1-7, driven in fixed rate mode, show normal pacing intervals. Beats 8-22 are driven in the demand mode. Beats 8, 10, 12, 14, 16, 17, 18, 19 and 21 show abnormal prolongation by 300-440 msec above the preset pacing interval of 780 msec.

**Figure 2**

The top strip shows unipolar pacing in demand mode via the proximal pole while an electrogram is recorded from the distal pole. The pacing rate equals the set rate. Numerous voltage transients (see arrows) are recorded on the T waves. The bottom strip shows unipolar pacing in demand mode via the distal pole, while an electrogram is recorded from the proximal pole. No artefacts are recorded. Beats 2, 7, 8 and 9 show prolongation of the pacemaker discharge interval over the set rate. Beat 5 is a sinus conducted beat and it is followed by a normal pacemaker escape interval.

**Figure 3**

Demand pacing in unipolar mode through the distal pole. Lead 2 and a distal pole electrogram are recorded. Voltage artefacts follow every beat. Three artefacts, A₁, A₂ and A₃, are of sufficient amplitude to reset the pacemaker thereby prolonging the cycle for the beats labelled X.
paced in the demand mode through the distal pole, while an electrogram was recorded from the same pole. A 10,000 ohm series resistor was used to attenuate the signal recorded from this pole in order to prevent electrocardiographic preamplifier saturation. Every beat was followed by an artefact within the previously noted interval. However, only three of these artefacts (A1, A3 and A5) were of sufficient amplitude to reset the pacemaker and thereby delay the beats labelled X.

These findings suggested that the artefacts recorded were most likely due to a partial fracture of the distal lead. To further clarify this, we applied a constant DC current of 0.25 mA through the distal pole while an electrogram was recorded from the same lead (fig. 4). This was performed while the heart was paced in a unipolar mode through the proximal electrode. Artefacts of varying amplitudes were seen falling in the previously noted interval after each beat. No artefacts were recorded from the proximal pole when the DC current was applied through it.

The observations to date localized the abnormal pacemaker inhibition to a voltage artefact generated by a partial fracture of the distal lead.

During spontaneous beating, no artefacts were recorded from either lead (fig. 5). This coincided with the fact that demand beats always occurred at the set rate when they followed sinus propagated beats (fig. 2). To clarify the essential role of paced beats in the production of these artefacts, the output of the pulse generator was gradually reduced until it reached a sub-threshold level, at which time the ventricles were driven from the sinus node with a 2:1 A-V conduction ratio (fig. 6). The pacemaker was allowed to continue to inject sub-threshold pulses through the distal pole.

![Figure 4](image)

**Figure 4**
Demand mode pacing through the proximal pole. A DC current of 0.25 mA was injected through the distal pole and an electrogram recorded from this lead. Voltage transients of varying amplitude and duration follow every beat.

![Figure 5](image)

**Figure 5**
Electrograms are recorded from the distal and proximal leads during sinus rhythm with 2:1 A-V conduction. No artefacts are recorded.

A simultaneous electrogram was recorded from this electrode. Again, an attenuating 10,000 ohm series resistor was used to prevent electrocardiographic preamplifier saturation. Each QRS produced a tiny deflection labelled by dots in the distal pole electrogram.

Voltage transients were recorded following some spontaneous beats provided these beats were followed by a properly timed sub-threshold pacemaker stimulus. Beat number 2 is followed by an artefact (A) on its T wave. These observations indicate that spontaneous beats provided the necessary electrode separation and hence increased the lead resistance, while the sub-threshold stimulus provided the required current to generate the artefact.

The patient was paced unipolar in the demand mode through the proximal electrode and experienced no additional difficulties. Two days later a permanent transvenous electrode and a demand pacemaker were installed and this resulted in normal pacing. Using an ohmmeter, the resistance across each lead was measured when the temporary pacing catheter was
removed. When the distal electrode was not deformed there was no measurable resistance across it. When the distal three inches of this lead was twisted, wide changes in resistance were seen up to a value of over 2,500 ohms. Resistance across the proximal electrode was zero and constant despite twisting of the catheter. This confirmed the presence of a partial fracture confined to the distal portion of the distal electrode.

Comments

Biphasic current flow is normally used in biological pacemakers such as the one used here, to prevent electrolysis with subsequent damage to the tissues by sodium hydroxide. To achieve this, the areas under the positive and negative voltage waveforms must equal each other (fig. 7). A capacitor is therefore included in the pacing circuit to provide biphasic current flow. Accordingly, after every discharge the pacemaker is charged by a low amplitude current. This current decreases quickly and by the time the pacemaker refractory period ends the current is too small to produce inhibition of it. Sudden changes in the electrode catheter resistance as manifestations of a broken wire can, in the presence of this current, produce a potential difference at the electrode terminals sufficient to inhibit a demand pacemaker. To explain a similar case of inhibition caused by a wire fracture, Lasseter et al. showed in a physical model that a resistance change of 1,000 ohms can inhibit a pacemaker by generating a potential difference of about 4 mV at the pacemaker terminals. In contrast to our case, they did not record the inhibiting potentials in vivo as we did.

Since the current emitted by the pacemaker was constant, the amplitude of the artefacts must have depended on the degree of wire separation. Thus, a greater separation produced a greater resistance change which in turn created a large voltage artefact across the broken lead. Variations in artefact amplitude determined whether or not the pacemaker would be inhibited.

The artefact occurred only when the heart was being paced. During spontaneous beating there was no current source during systole to act in concert with the wire separation. The normal pacing threshold suggests that the wire was intact at the onset of pacing, while the timing of the artefacts indicates that the wire separation occurred towards the end of ventricular systole. These observations stress the notion that pacing threshold alone is not a sensitive test of wire integrity. Therefore, oversensing resulting in pacemaker inhibition should be a strong warning to the possible existence of a wire break. Moreover it must be emphasized that the type of abnormality referred to here is very similar in timing to cases reported by Barold, Kosowsky and others as T wave sensing. One cannot help but wonder whether many of the so-called T wave sensing problems are really partial wire breaks producing a T wave artefact. This is especially suggestive since Barold notes that "T wave sensing of paced beats is in our experience far more common than sensing of spontaneous T wave voltage which occurs rarely with implantable pacemakers."

It is also essential to point out that a similar type of artefact occurring with an R synchronous demand pacemaker could result in a very different abnormality in demand pacing. If the artefacts generated by the lead fracture occurred after the refractory period of the pacemaker unit was completed, this could result in premature pacemaker discharge following every paced beat. Thus, a pacemaker-driven ventricular tachycardia would result. We are not aware of such a case at this time.

Figure 7

This shows the biphasic nature of the pulse emitted by conventional pacing units of the type used here. The area under the negative portion equals the area under the positive portion.

Figure 8

During fixed rate pacing, the pacemaker discharge rate equals the set rate. In the demand mode, there are frequent periods of pacemaker inhibition lasting up to 2.5 sec.
Superficially, these pauses appeared to be related to P wave sensing. However, careful measurements eliminated that possibility.

In order to identify whether the inhibition was related to one of the electrode leads, the heart was paced in a unipolar mode. Irrespective of which pole was used for pacing, abnormal inhibition still occurred. During unipolar pacing the surface electrocardiogram (fig. 9) recorded voltage transients (see dots) which reset the pacemaker and produced the abnormal manifest discharge rate.

In figure 10, the first two strips show unipolar pacing via the proximal and distal electrodes, respectively. These electrodes were connected to the cathode while the skin was connected to the anode. In both cases, the voltage transients were negative. When the hook-up to the pulse generator was reversed, the transients were positive (bottom strip). These observations localized the source of the transients to the pacemaker unit. However, they failed to answer why these transients were recorded from the surface electrocardiogram only when unipolar pacing was performed but not during bipolar pacing. In order to examine what role the resistance imparted by the skin and body played in displaying the transients, we performed the following maneuver. The heart was driven in the bipolar mode and the output from the pacing box was monitored across a 1,000 ohm series resistor, as shown in figure 11. As one can see, numerous transients were recorded and these are seen to inhibit the pacemaker.

The patient’s faulty pulse generator was replaced and the abnormal inhibition disappeared. Figure 12 shows the output of the faulty unit on an oscilloscope. The top signal is a one second timing pulse. The sec-

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**Figure 9**
Unipolar pacing through the proximal electrode discloses numerous artefacts labelled with dots. These reset the pacemaker and are responsible for the prolongation in pacemaker discharge rate.

**Figure 10**
Unipolar stimulation of the heart with the cathode via the proximal (top strip) or distal pole (middle strip) produces negative artefacts labelled with dots. The polarity of the artefacts is reversed when the heart is driven by the distal pole through the anode (bottom strip).

**Figure 11**
Bipolar stimulation. The voltage drop across a 1,000 ohm resistor in series with the pacemaker is recorded. Numerous artefacts (see dots) are recorded which inhibit the pacemaker.
Demand Pacemaker Malfunction

Abnormal demand pacemaker discharge rate was traceable to a broken electrode in one patient, and a faulty pulse generator in a second. These cases illustrate that an abnormal signal may readily be sensed, yet not be apparent on the surface electrocardiogram. When an artefact is dependent on either the electrical or mechanical activity of the heart for its generation, it will usually occur at a fixed point in the cardiac cycle. In this situation, if the artefact itself is not recorded, the problem may be falsely attributed to “T” or “P” wave sensing. These cases are presented in order to describe in detail the bedside techniques required to identify the source of the abnormality.

The requirement for an accurate assessment is self evident since in both individuals serious faults existed which could have led to total pacemaker failure. We suspect that similar problems are not rare events in demand pacing systems, but their actual causes may easily be overlooked. Moreover, if these types of abnormality develop in permanently implanted systems, their complete diagnosis may be further constrained. However, the potential seriousness of demand pacemaker inhibition makes precise identification of the problem mandatory.

Comments

Although the faulty component(s) within the pulse generator were not identified, the pacing problem could be correctly localized to the pacemaker unit. The most interesting feature relates to the fact that the abnormal signals emitted by the pulse generator and hence responsible for inhibiting the unit, were recorded only when the patient was paced in a unipolar mode. During bipolar pacing the low level signals passed through the electrode system but did not register a measurable voltage on the surface electrocardiogram. During unipolar pacing the current responsible for the artefacts had to traverse the impedance posed by the patient’s skin and subcutaneous tissues. The value of this tissue is variously reported to be at least 500-1,000 ohms and of course produces an increased voltage drop as the current traverses its path. This higher voltage was seen on the surface electrocardiogram as positive or negative transients.

The role of the skin resistance could quite easily be simulated by interposing a 1,000 ohm series resistor during bipolar pacing and recording the voltage drop across this resistor. This, of course, resulted in recordable voltage transients (fig. 11).

It is of additional interest to note that the artefacts were emitted only when the pacemaker function switch was in the demand mode. Furthermore, the pacemaker could emit repetitive artefacts and hence produce prolonged pacemaker inhibition.

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