Pacemaker-mediated Tachycardia Induced by Intraoperative Somatosensory Evoked Potential Stimuli

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Permanent cardiac pacing is a reliable and effective form of therapy for a variety of slow and fast dysrhythmias. In addition to being relatively long-lived and trouble free, newer generations of these increasingly sophisticated devices have been designed to provide greater programmable and telemetric capabilities. Such advances have supported the increased use of the more complex dual chamber and rate responsive pacemakers, which not only ensure a minimum heart rate but attempt to optimize the patient’s hemodynamic status by varying the pacing rate in accordance with metabolic needs. Performing appropriately they offer considerable benefits. Should aberrations in function occur, however, adverse results may be profound unless there is early recognition and prompt corrective action. The operating room and other anesthetizing areas present several unique opportunities for pacemaker dysfunction, (e.g., cautery, muscle tremor, inadvertent damage to leads or generator) of which the anesthesiologist must be aware. Whereas in many instances the pacemaker problems encountered in this setting are readily identified by a transient or prolonged decrease in heart rate, we report a case in which an acceleration of the ventricular rate occurred in association with profound hypotension.

CASE REPORT

The patient was a 78-year-old woman with a history of Parkinson’s disease treated with levodopa (L) and carbidopa (C). She was admitted following an accidental fall for stabilization of the first two cervical vertebrae. The etiology of the fall was considered to be toxicity from excessive doses of L and C. Neurologic examination prior to surgery revealed findings compatible with Parkinson’s disease but no significant sensory or major motor deficits. Her past medical history was positive for symptomatic sinus node dysfunction for which a dual chamber, unipolar pacemaker (Pacemaker AFP, model 283, Sylmar, California) was implanted 1.5 years previously. The mode of pacing was DDD (DD pacing with automatic extension of the atrial refractory period following premature ventricular contraction (PVC) detection) (table 1) and the programmable parameters are listed in table 2. The functional status of the pacing system had undergone routine analysis two months prior to the present admission. At that time noninvasive capture and sensitivity thresholds were normal except for a low sensed p-wave amplitude and consequent intermittent failure to sense intrinsic atrial activity.

At the time of surgery intraarterial pressure monitoring was established. After fiberoptic nasotracheal intubation general anesthesia was administered. Once the patient was stable somatosensory evoked potential (SEP) testing (TECA, Model TE4, Pleasantville, New York) was initiated. The SEP procedure involved repetitive transcutaneous electrical stimulation using gradually increasing energy levels (output 0- 500 V, impedance <3,000 ohm) delivered at intervals of 300 ms. Because stimulation of the left posterior tibial nerve at the ankle elicited no cortical response with maximal stimulation (approximately 100 mA), SEP stimulation was initiated at the left median nerve at the wrist. A cortical response was elicited, but the stimulation was associated with the sudden onset of tachycardia and hypotension, which persisted until cessation of current delivery. The dysrhythmia was initially thought to be ventricular tachycardia occurring either as a primary event or as a consequence of current leak from the stimulating device. Analysis of the monitored electrocardiogram (fig. 1), however, revealed the tachycardia to be due to ventricular pacing at the rate of 100 beats/ min. Rechallenging the patient with upper extremity SEP stimuli had the same effect and the SEP testing was abandoned. The effects of an external magnet were not tested. It was decided to proceed with surgery without SEP monitoring. Both anesthesia and surgery were without further event, and the patient awoke without cardiac or neurologic deficit. (The TECA 4 was retested after this event and a malfunction of stimulation frequency was noted, i.e., 500 beats/min rather than the intended 200/min. Even with a stimulus rate of 200 beats/min the pacemaker described would presumably have tracked the SEP, but at a different ventricular response rate.)

DISCUSSION

Retaining specifics of pacemaker terminology may be difficult for persons not engaged frequently in their use. The most widely used system at this time is that developed by the Inter-Society Commission on Heart Disease (ICHD). A three-position ICHD Code was originally established in 1974 and subsequently expanded to five positions to cover both programmable and antitacharyrhythmia functions (table 1). The three-position code remains the most widely used. The first letter represents the chamber paced, the second the chamber sensed, and the third the expected response to a sensed event. Much more complex specific and pictorial codes are used primarily

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by pacing electrophysiologists. Unipolar pacing systems have a negative stimulating electrode in the cardiac chamber and a positive electrode at some distance from the heart, e.g., at the pulse generator. Bipolar systems have both the positive and negative electrodes in the chamber being paced. If functioning in an asynchronous mode, a pacemaker delivers electrical impulses without regard for any intrinsic cardiac electrical activity. For function in a synchronous capacity sensing circuits must be present and able to respond (e.g., triggered or inhibited) to intrinsic depolarization.

Pacemaker-mediated tachycardia (PMT) is an inappropriate acceleration of heart rate that is initiated and sustained by the pacemaker. This terminology is generally applied to a condition occurring in patients having dual chamber, atrial tracking units in which an initiating event (often a PVC) establishes a condition favorable for retrograde electrical activation of the atria, which in turn is sensed by the pacemaker as an intrinsic atrial event, triggering a ventricular pacing stimulus and again subsequent retrograde atrial activation. This cycle is repeated until something occurs to interfere with reentry loop. During this type of PMT the paced ventricular rate is typically equal to the programmed upper rate limit of the device, and the A-V interval is automatically prolonged to meet this requirement. Although a very common problem in early generations of DDD pacemakers, PMT has been curtailed by the capability of prolonging the postventricular atrial refractory period (PVARP) as a programmable option. This prevents sensing of the retrograde atrial depolarization and thus interrupts the pacemaker limb of the reentry tachycardia.

The mechanism of the tachycardia in our patient is different in that it did not depend upon a reentry loop. Rather it resides with the mistaken interpretation by the pacemaker of the SEP stimulus as its own intrinsic atrial event. The pacemaker begins to track the SEP stimulus once the energy delivered by the latter exceeds the sensitivity threshold of the atrial channel. Figure 1 demonstrates SEP stimulus artifacts in the electrocardiogram. Although the transcutaneous stimuli occur at the rate of 300/min, the rate of ventricular pacing is only 100/min.

The reason for this is demonstrated in the lower panel of figure 1. The fourth SEP stimulus on this tracing is presumably the first of voltage great enough to be sensed by the atrial channel and results in a ventricular paced event after the programmed A-V interval (165 ms). The PVARP commences with the ventricular event and extends 325 ms thereafter. During this period of time the atrial channel is blinded to electrical activity and therefore cannot respond to the fifth and sixth SEP stimuli. The seventh stimulus, however, occurs during a receptive period of atrial sensitivity and results once again in a paced ventricular response. This sequence is repeated until cessation of the SEP and results in the pacemaker tracking every third SEP, thus producing a ventricular rate of 100/min. With neither intrinsic nor pacemaker delivered atrial pacing, the atrial component of ventricular filling was lost, and profound hypotension ensued. Interference with normal pacemaker activity by endogenous or exogenous sources of electric or magnetic energy is a well-recognized problem associated with pacing. Although considerable protection has been designed into these devices, it is not always adequate. Unipolar pacemakers are particularly prone to these sources of interference because of the relatively large sensing "antenna" established between the

<table>
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<th>Table 2. Programmed Parameters (Pacemaker AFP, MODEL 283)</th>
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<td><strong>Atrial</strong></td>
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<td>Refractory period</td>
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<td>Pulse amplitude</td>
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<td>Sensitivity</td>
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Mode DDx
Rate 60 ppm
A-V interval 165 ms
Maximum tracking rate 110 ppm
Magnet on
Blanking period 13 ms
pacemaker generator (acting as the positive electrode, with metallic case serving as ground) and the negative electrode tip, compared to the distance between the two electrodes of a bipolar lead. In most instances electrical interference is interpreted as a ventricular electrical event and the pacemaker is inhibited. In our patient the SEP was of enough voltage to be sensed by the atrial channel (threshold 0.5 mV) but below the threshold of the ventricular channel (threshold 2.0 mV). Had the ventricular threshold been lower or the SEP voltage higher the pacemaker might have been inhibited completely. Temporarily programming the pacemaker to DVI (atrial and ventricular pacing but only ventricular sensing) for SEP testing would have prevented the PMT because there is no sensing or tracking by the atrial channel in this mode. This pacing mode i.e., DVI, would have maintained A-V sequential pacing and the hemodynamics associated with it. Reprogramming temporarily to the DOO mode would likewise have preserved sequential pacing but eliminated sensing by the ventricular channel as well, thereby converting the pacemaker to an asynchronous mode, as would the use of an external magnet. Both of these latter modes would have wasted pacemaker energy and posed a risk of arrhythmia due to competition with the patient’s intrinsic electrical activity.

It is not clearly understood why our patient had no cortical response to posterior tibial nerve stimulation. There were no significant preoperative or postoperative sensory deficits. The stimulation was begun just after intubation. Muscle paralysis may have made nerve location with a twitch response impossible. Bolus medication, including the sodium thiopental used for induction of anesthesia, may have a measurably negative effect on the SEP for 5–10 min. In addition, in the elderly, cortical evoked potentials from the lower extremity may be rather small and difficult to discern. This is compounded by the anatomic location of the electrical response deep in the interhemispheric fissure and by high skin impedance at the stimulation site.14

In summary, this case report demonstrates the importance to the anesthesiologist of understanding pacemaker function and dysfunction, especially so in view of the increasing complexity of these devices. While absence of effective cardiac pacing is rather easily recognized, the role of the pacemaker in mediating tachyarrhythmias may be less obvious. Knowledge of the particular type of pacemaker being utilized and its programmed settings, coupled with careful review of the electrocardiogram, should reveal the source of rhythm problems and suggest their solutions. On occasion the pacemaker’s programming device can be used to obtain telemetry data (helpful in troubleshooting) or to change a pacemaker function. If doubts exist concerning the patient’s rhythm or the pacemaker’s function, cardiology consultation should be obtained.

REFERENCES