Perioperative Pacemaker-Mediated Tachycardia in the Patient with a Dual Chamber Implantable Cardioverter-Defibrillator

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Patients with cardiac implantable electronic devices are at additional risk for arrhythmias while undergoing surgical procedures. In this case report, we present a patient with a dual chamber implantable cardioverter-defibrillator who developed intraoperative pacemaker-mediated tachycardia causing significant hemodynamic instability. Management of this arrhythmia can be particularly challenging, because standard application of a magnet does not affect the pacing functions of an implantable cardioverter-defibrillator. Awareness by the anesthesiologist and timely coordination with the cardiac electrophysiology team helped to optimize care for this patient. (Anesth Analg 2013;116:307–10)

Pacemaker-mediated tachycardia (PMT) refers to a rapid heart rate facilitated by the presence of a cardiac dual chamber pacing device set to an atrial tracking mode.[1] We present the clinical case and management of this arrhythmia in a patient with a dual chamber implantable cardioverter-defibrillator (ICD) that occurred during and immediately after hip surgery. The role of the anesthesiologist-specific interventions and preventative steps during and before surgery are discussed.

CASE DESCRIPTION

This case is reported in accordance with our IRB guidelines. An 89-year-old man with a history of atrial fibrillation, coronary artery disease, and congestive heart failure presented for open reduction and fixation of the right hip. The patient underwent coronary artery bypass graft surgery 20 years before admission. Presently, the patient was diagnosed to have ischemic cardiomyopathy with a reduced left ventricular ejection fraction of 30%. Twenty months before hospitalization he received a Boston Scientific dual chamber ICD (model Teligen DR E-110; Natick, MA). The cardiac electrophysiology service interrogated the ICD preoperatively and determined that it was functioning normally. No prior cardiac arrhythmic events were recorded. The antitachyarrhythmia functions were turned off, and defibrillator pads were applied to the anterior and posterior aspects of the patient's chest. The ICD pacing mode was kept in the previously programmed DDD mode (Fig. 1), because the patient

was pacing nondependent and because the surgery was below the umbilicus.[2]

Anesthesia was induced with etomidate, fentanyl, and vecuronium and maintained with desflurane, fentanyl, and vecuronium. The patient was stable with a heart rate between 60 and 70 beats per minute with occasional premature ventricular contractions (PVCs), and a systolic blood pressure of 120 mm Hg. One hour into the procedure, the patient experienced a rapid ventricular paced rhythm at 105 beats per minute causing a decrease in systolic blood pressure to 80 mm Hg necessitating phenylephrine injection. The tachycardia spontaneously terminated 10 minutes later. A second ventricular paced tachycardia episode occurred 45 minutes later and also terminated spontaneously. At the end of the procedure, the patient remained in normal sinus rhythm with stable vital signs. He was successfully tracheally extubated and transferred to the postanesthesia care unit where the cardiac electrophysiology service was consulted.

During ICD interrogation in the postanesthesia care unit, the patient developed a third episode of the rapid ventricular paced tachycardia (Fig. 2). The ICD interrogation revealed this arrhythmia to be a PMT. The programmed postventricular atrial refractory period (PVARP) was determined to be shorter (280 milliseconds) than the actual retrograde conduction time (295 milliseconds). This allowed for retrograde conduction of a PVC to the atrium facilitating the development of PMT. The ICD was reprogrammed to increase the PVARP to 320 milliseconds, which prevented further recurrences of this arrhythmia.

DISCUSSION

Patients with cardiac implantable electronic devices are at additional risk for arrhythmias while undergoing surgical procedures as reflected in practice recommendations on perioperative management published recently by the American Society of Anesthesiologists and by the Heart Rhythm Society.[2,3] There are, however, less common types of arrhythmias that are not reflected in the current practice guidelines[2,3] that may cause significant hemodynamic instability. Intraoperative occurrences of PMT in patients with cardiac implantable electronic devices have been described,[4–6] but there is little information on the intraoperative management of PMT in patients with an ICD.

Figure 1. Baseline programmed bradytherapy settings for the reported patient with a pacing mode of DDDR, which allows for potential pacemaker-mediated tachycardia (PMT). Note that the maximum tracking rate is set to 120 beats per minute, which would be the rate required for 16 consecutive beats to trigger the PMT intervention algorithm. Also note the dynamic postventricular atrial refractory period (PVARP) set range between 240 milliseconds (during faster heart rates) up to 280 milliseconds (during slower heart rates). AV= atrioventricular; VRP = ventricular refractory period; PVC = premature ventricular contraction.

Figure 2. Ongoing pacemaker-mediated tachycardia (PMT) episode recorded during dual chamber implantable cardioverter-defibrillator (ICD) interrogation postoperatively. The intracardiac atrial and ventricular electrograms and the lead III surface electrocardiographic (ECG) tracings are recorded. Measured retrograde ventricular-atrial time is 295 milliseconds, which exceeds the programmed dynamic postventricular atrial refractory period (PVARP) (240–280 milliseconds). The ICD senses retrogradely conducted atrial events as intrinsic atrial events (AS) and tracks them by pacing the ventricle (VP) following the programmed atrioventricular (AV) delay (260 milliseconds). This results in a tracking rate of 108 beats per minute (295 + 260 = 555 millisecond period), which fails to trigger a device intervention to terminate PMT. In Boston Scientific devices, PMT termination algorithm is only triggered at the maximum tracking rate, which is preset here at 120 beats per minute. With transient faster ventricular-atrial conduction, possibly as a result of a spontaneous change in autonomic tone, the first shorter ventricular-atrial interval is still sensed beyond the PVARP but the subsequent retrograde atrial event (asterisk) is within the dynamic PVARP interval as evidenced by the "(AS)" with parenthesis notation indicating it is refractory. As a result, the atrial event was never sensed by the device and the AV delay window was never triggered and consequently no ventricular pacing occurred. Instead, the device paced the atrium at the lower rate limit, timed from the preceding sensed atrial event. This atrial paced event is documented on the marker channel as "AP" and occurs at exactly 1000 milliseconds (60 beats per minute—the programmed lower rate limit) from the last sensed atrial event, with subsequent resumption of AV sequential pacing. In the figure inset, note the different morphology of the retrogradely conducted atrial event (A) compared with the paced atrial event (B).

PMT can occur in any ventricular pacing configuration set to track atrial activity (i.e., DDD, VDD). PMT is induced by retrograde conduction of an impulse from the ventricle to the atrium (Fig. 3). If the retrograde impulse is sensed in the atrium at a time that is beyond the programmed PVARP, the impulse is interpreted by the cardiac rhythm device as an intrinsic atrial event, which then prompts pacing the ventricle after the programmed atrioventricular (AV) delay. The result is a paced ventricular impulse that also conducts in a retrograde fashion and the process repeats itself resulting in a "positive-feedback" reentrant tachycardia. The heart rate during PMT is determined by the sum of the ventricular-atrial conduction time and the set AV delay (Fig. 3), while its upper rate is limited by the programmed maximum tracking rate of the pacing device. The most common trigger for PMT is a PVC. Other forms of AV dyssynchrony may also initiate this event. Those include atrial undersensing with antegrade AV block, a loss of atrial capture, or ventricular pacing in the absence of atrial activity (atrial oversensing).[1]

More than 50% of patients receiving dual chamber pacemakers have been identified as having ventricular-atrial conduction, thus making them susceptible to PMT.[7,8] Another
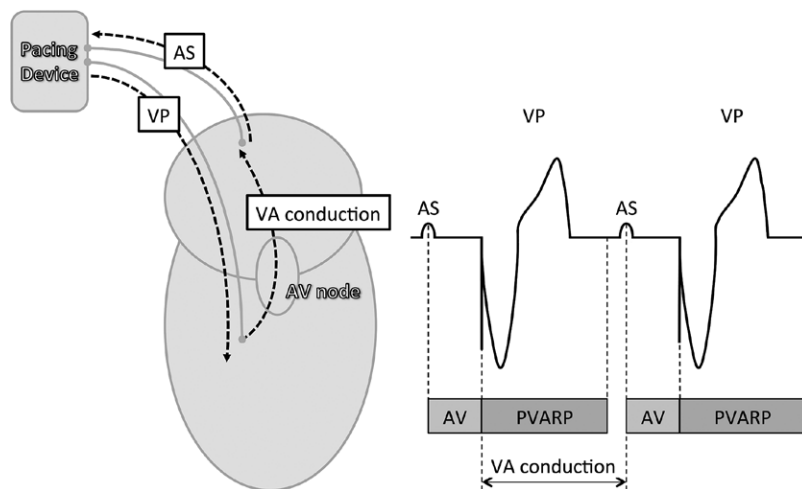
Figure 3. The schematics of pacemaker-mediated tachycardia (PMT) as a "positive-feedback" endless loop reentrant tachycardia. The left side of the diagram represents a series of events consisting of ventricular-atrial (VA) conduction and atrial sensing (AS) of a retrograde p-wave "tracked" by ventricular pacing (VP) after the programmed atrioventricular (AV) delay causing the next cycle of PMT. The right side of the figure represents a schematic electrocardiogram during PMT accompanied by the pacemaker timing windows. The heart rate during PMT is composed of the sum of the VA conduction time and the preset AV delay. PVARP = postventricular atrial refractory period.

5% to 10% of patients will demonstrate ventricular-atrial conduction after the implantation of the device[7,8] probably due to previously unrecognized intermittent ventricular-atrial conduction. Moreover, ventricular-atrial conduction time varies among individuals as well as in the same individual as a result of variations in the autonomic nervous system tone.[9] It is important to appreciate that a multitude of factors in the perioperative period can transiently affect the autonomic nervous system and/or directly affect the AV node, thus altering its antegrade and retrograde conduction.[9–12] A decrease in retrograde conductivity may cause the ventricular-atrial time to be longer than the programmed PVARP, thus setting the conditions for PMT. Factors that might decrease the conductivity of the AV node include electrolyte imbalances (hyperkalemia, hypermagnesemia), changes in metabolic variables (hypoglycemia, hypoxia), alterations in acid-base status (acidosis/alkalosis), hypothermia, decreased sympathetic tone, increased vagotonia, myocardial ischemia, various drugs (muscarinic agonists, acetylcholinesterase inhibitors, β-blockers, calcium channel blockers, and antiarrhythmics), and some anesthetics (halothane, dexmedetomidine, and fentanyl).[9–12] During PMT, pharmacologic arterial blood pressure support may become necessary as a result of changes in left ventricular stroke volume due to right ventricular pacing and decreased ventricular filling time associated with a higher heart rate. An increase in the ventricular-atrial conduction time by some drugs including AV nodal blocking drugs[13] may also interfere with PMT termination algorithms.[13]

The PVARP is the primary setting of a cardiac electronic device in preventing PMT. This preset allows the pacemaker to ignore any atrial impulses that occur within a specified time period after ventricular activation. In addition, each device manufacturer has a different proprietary algorithm for detecting and terminating PMT. In Boston Scientific pacing devices, PMT is determined by an algorithm that detects 16 consecutive paced beats at the programmed maximum tracking rate with a ventricular-atrial time that

does not vary beyond 32 milliseconds. This prompts an automatic lengthening of the PVARP to 500 milliseconds for 1 cycle thereby interrupting PMT. In our patient, however, the algorithm failed to recognize PMT. This was attributed to a prolonged ventricular-atrial conduction time that resulted in a slower heart rate during PMT than the programmed maximum tracking rate. Other device manufacturers use different algorithms that may recognize PMT at rates lower than the maximum tracking rate, require fewer consecutive beats, or use other mechanisms to break PMT such as withdrawal of the next paced ventricular beat.[1]

One approach to the operating room management of suspected PMT in a patient with a pacemaker is the application of a magnet. Assuming that the magnet activates asynchronous AV pacing, atrial sensing will stop and the PMT will be terminated. This approach cannot be used with ICD, however, because magnet application does not affect the pacing function of the ICD. In such situations, short-lived treatment modalities such as an adenosine bolus[14] or carotid sinus massage[15] may be feasible and could be considered as a means to terminate this arrhythmia due to interruption of the retrograde AV node-dependent limb of the circuit. However, this tachycardia could easily recur with a single reentrant atrial event secondary to a PVC. Definitive management of PMT in this setting would require intraoperative reprogramming of the cardiac implantable electronic device.

Finally, preoperative interrogation of the ICD is important to confirm that the PMT intervention mode is activated. PVARP may be checked against the actual retrograde ventricular-atrial conduction time and reset to a longer period if needed. A programmed increase in the PVARP interval would result in an obligatory decrease in the maximal heart rate provided by pacing. Whereas this limit might be considered a disadvantage for the active ambulating patient, its clinical significance in the patient within the operating room or ICU is minimal. Alternatively, PMT can be abolished by switching to a pacing mode without atrial sensing

or tracking such as VOO, DOO, or VVI pacing modes. The disadvantage of this approach is that it may lead to the loss of AV synchrony. The DDI pacing mode would also avoid the occurrence of PMT because this mode allows atrial sensing but does not allow atrial tracking. This mode should only be used in patients with normal AV conduction and sinus rate higher than the programmed lower rate limit, because it may also promote significant AV dyssynchrony. Incorporating these steps into the preoperative consult for the anesthesiologist will help to prevent and manage this potentially hemodynamically significant and challenging arrhythmia intraoperatively. ■

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