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Atrioventricular Sequential Pacemaker Inhibition by Transurethral Electrosurgery

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Inhibition and/or modulation of cardiac pacemaker function caused by electromagnetic interference (EMI) emitted by the electrosurgical unit (ESU) used during transurethral resection of prostate (TURP) have been described previously.[1–6] These reports detail the particular problems involved with demand-type pacemakers. In this presentation, we describe a unique case of atrioventricular (A-V) sequential pacemaker interference by cystoscopic electrosurgery.

REPORT OF A CASE

A 72-yr-old, 47-kg man, who, 4 yr prior to admission, had a Cordis Model 233F‡ A-V sequential programmable pacemaker placed for complete heart block, presented to undergo TURP. The pacemaker mode was DDD, indicating dual chamber pacing, dual chamber sensing, and dual response (capable of triggering or inhibiting the pacing circuit in response to sensing parameters) at 80 paces per minute.

Channel 1 (atrial lead) sensitivity was 0.5 mv with a pulse duration/output current of 2/12 msec/mA; channel 2 (ventricular lead) was set at 1.3 mv sensitivity with an output of 0.6/6. Prior cardiac catheterization had documented the absence of coronary artery occlusion and the presence of mild aortic stenosis/insufficiency without gradient. Medical therapy included procainamide 500 mg q6h, theophylline 100 mg bid, digoxin 0.125 mg qd, captopril 6.25 mg q6h, isosorbide 20 mg qid, and isoetharine inhaler prn.

Physical examination revealed a cachectic, elderly male in mild respiratory distress without evidence of congestive heart failure. Supine arterial blood pressure ranged from 90–110 mmHg systolic and 60–70 mmHg diastolic. ECG showed 100% A-V sequentially paced rhythm at 80 bpm. Serum sodium was 132 mEq/l, potassium 4.3 mEq/l, blood urea nitrogen 28 mg/dl, creatinine 1.3 mg/dl, hematocrit 35.7%, digoxin level 1.1 μg/ml, and procainamide level 10.8 μg/ml.

Spinal anesthesia to a T6 sensory level was successfully induced without significant hemodynamic change, using 10 mg tetracaine and 0.2 ml epinephrine 1:1000. During the application of cutting current utilizing a Bovie™ 400-SR ESU with the dispersing electrode placed on the right thigh, continued atrial pacing spikes were observed without corresponding ventricular impulse or capture (fig. 1). Simultaneous radial arterial pressure tracing showed absence of the corresponding waveforms, and the Nellcor™ N-100C pulse oximeter also indicated an absence of pulsatile flow. The urologist was informed of the problem; interruption of ESU use resulted in immediate return of pacemaker function to full A-V sequential mode with myocardial capture and restoration of pulsatile arterial flow.

The magnet mode in this case was DOO at a rate of 70 bpm, with a back-up mode of VOO at 52.5 bpm. Application of a magnet over the pacemaker generator for several seconds between ESU use, however, had no effect. During ESU use, the pacemaker not only failed to pace sequentially, but also did not change to the fixed (asynchronous) mode. The surgical procedure continued with the surgeon applying short bursts of cutting current, each time inhibiting pacemaker function. On every occasion, cessation of ESU use promptly led to return of proper sequential pacing. Following surgery, the patient recovered from his anesthetic and was transferred to the floor in stable condition.

DISCUSSION

Despite improvements in shielding and filtering from electrical interference, electrosurgery can cause significant malfunction of demand pacemaker impulse generation. Experimentally produced electromagnetic interference has variably produced complete pacemaker cut-off; dysrhythmias, including both tachycardia and bradycardia; and reversion to fixed-rate impulse generation.[7] Although coagulation current used during TURP procedures has no effect, cutting current at high frequencies (up to 2500 kc/sec) can suppress the output of a bipolar demand ventricular pacemaker.[1] EMI resulting in ventricular asystole was similarly described with a unipolar demand pacemaker during prolonged cutting current use, while coagulation current or short bursts of cutting current yielded no malfunction.[3]

Smith *et al.*[2] have reported demand pacemaker malfunction attributable to "grounding failure" caused by a fouled contact on the dispersing electrode. In that case, interference caused a decrease in heart rate from 72 to 40 bpm with both cutting and coagulation use of the ESU. Even with an apparently normally functioning dispersing electrode, radiofrequency suppression of a VVI-type (ventricular paced/sensed/inhibited mode)

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‡ Cordis Corporation Manual, August 1983.

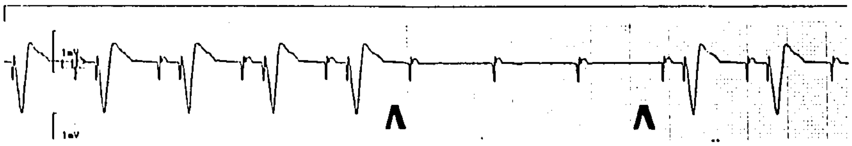

FIG. 1. Computer-generated electrocardiographic tracing, representative of the electromagnetic interference of pacemaker function observed in this case.

demand pacemaker can occur.[6] Altering the placement of the dispersing electrode would not be expected to modify the probability of interference, inasmuch as "stray current" from the ESU is much greater than the area defined by its electrodes.[8]

Additional problems have arisen with the change from previous analog pacing systems to the more recent digital/programmable impulse generators. In such devices, automatic reversion back to fixed-rate pacing may not occur with exposure to intermittent or rapidly changing electromagnetic fields, as is often seen during ESU use.[9] Complete inhibition of function may occur, instead. Furthermore, A-V sequential pacemakers, with more sensitive pulse generators than the earlier fixed-rate, R wave-inhibited or R wave-stimulated standby ventricular pacemakers, have exhibited temporary rate changes when active electromagnetic fields were externally applied.[10] Thus, the application of an external magnet to convert the pacemaker to an asynchronous mode may activate the rate-changing command, allowing EMI to change randomly the firing rate of the impulse generator.[9]

Bhatia and Goldschlager cited "oversensing" as the most common cause of abnormal pauses in paced rhythms.[11] Extracardiac myopotentials from the diaphragm or pectoralis muscles, for example, account for over 50% of oversensing, which particularly inhibits pacemaker output of unipolar systems. Although EMI may elicit a series of pacemaker malfunctions, as detailed earlier, the most likely cause of the pacemaker malfunction in the case described above appears to have been inhibition of the ventricular pacing circuit by the atrial pacing spike. This type of "crosstalk," or the sensing by one electrode of the other electrode's output stimulus in the DVI or DDD pacing system, is a known cause of malfunction,[11] although previously described only through experimental alteration of R wave sensitivity.[12] The possibility that the pacemaker generator had sensed the ESU as R waves, inhibiting ventricular output and thereby changing the pacing mode to AOO, also exists. If that were the case, however, the atrial channel output should also have been inhibited, since the atrial channel sensitivity (0.5 mv) was set to less than that of the ventricular channel (1.3 mv).

With the advent of more sensitive and versatile programmable pacemakers, the incidence of interference from ESU use may be expected to increase. Consequently, when ESU use is anticipated during anesthesia for A-V sequential pacemaker-maintained patients, the following guidelines are suggested.

1. Thorough preoperative evaluation including knowledge of: a) reason for pacemaker insertion; b) pacemaker, magnet, and back-up mode programming; and c) proper function of the pacemaker (consultation with the cardiologist/internist).
2. Careful monitoring, including continuous ECG with hardcopy capability for comparison and quantitative measurement.
3. Placement of a properly functioning ESU dispersing electrode distant from the pacemaker impulse generator (even though the current spread is minimally affected).
4. Limitation of surgical cutting current to short bursts with frequent pauses.
5. Possibly reprogramming the pacemaker to the asynchronous (fixed-rate) mode, if tolerated hemodynamically by the patient. This maneuver should *not* be performed with a magnet during ESU use, since random reprogramming may result in uncontrolled pacemaker function.[9]
6. Having a programming device readily available during surgery, should hemodynamic instability require pacemaker reprogramming from its back-up mode.

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Spinal Anesthesia with Isobaric Bupivacaine in Infants

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Previous studies have reported the efficacy of spinal anesthesia with hyperbaric tetracaine in infants.[1,2] In adults, isobaric 0.5% bupivacaine is an appropriate local anesthetic for spinal anesthesia.[3] The addition of epinephrine to intrathecally administered bupivacaine does not significantly increase the duration of analgesia.[4] However, there is no information on the use of intrathecal bupivacaine alone or with epinephrine in infants.[5] The aim of this study was to evaluate in infants under 6 months of age the efficiency and the hemodynamic effects of spinal anesthesia with isobaric 0.5% bupivacaine alone and combined with epinephrine (1:200,000).

MATERIALS AND METHODS

Twenty-eight infants undergoing inguinal hernia repair were studied after approval by the Institutional Investigation Committee and parental consent. Sixteen infants 2.7 ± 1.3 months of age (mean ± SD) (range 1.5–5 months), weighing 4.8 ± 1.0 kg (range 3.5–6.6 kg) received isobaric 0.5% bupivacaine (group I), and 12 infants 2.3 ± 1.2 months of age (range 1–5 months), weighing 4.5 ± 1.3 kg (range 1.6–6.8 kg), received isobaric 0.5% bupivacaine with epinephrine 1:200,000 (group II). They were not premedicated, and had fasted for 4 h before anesthesia. Heart rate was continuously recorded from the ECG and arterial pressure measured with an automated blood pressure cuff every 5 min. After insertion of a venous cannula for infusion of 5% dextrose in 0.18% NaCl (4 $ml \cdot kg^{-1} \cdot h^{-1}$), lumbar puncture was performed at L4–L5 interspace using a pediatric lumbar puncture needle (22-gauge, 4 cm) while the infant was awake and in lateral or sitting position with the chin held upward.[6] When free flow of cerebrospinal fluid was obtained, bupivacaine was injected using a 1-ml syringe over a 20-s period. The dose of 0.5% bupivacaine administered depended on the infant's weight: 1.25 mg (0.25 ml) under 2 kg, 3.75 mg (0.75 ml) between 2 and 5 kg, and 5 mg (1 ml) above 5 kg. Mean dose of bupivacaine was 0.80 ± 0.2 $mg \cdot kg^{-1}$ in group I and 0.86 ± 0.1 $mg \cdot kg^{-1}$ in group II. After injection, the infant was placed in the supine position.

Measurements recorded during this study were: 1) onset and duration of motor blockade by observation of the movements in the lower limbs, 2) cutaneous analgesia assessed every 5 min by dermatome level of subjective change to pinprick, 3) heart rate and blood pressure recorded before insertion of the venous cannula, 5, 10, 15 min after intrathecal bupivacaine administration and 30 s after surgical incision (22 ± 6 min), and 4) respiratory function monitored during the first four postoperative hours by Respitrace[7] in four of the 28 infants who were premature and were 37, 46, 48, and 50 weeks conceptual age at the time of surgery. Apnea was defined as the cessation of breathing for longer than 20 s or a shorter period associated with bradycardia, cyanosis or pallor.[8] Respitrace showed the abdominal, the rib cage, and the sum movements. Central apnea is the absence of chest wall movements on trend event recordings, whereas obstructive apnea is the absence of sum movements, despite respiratory efforts indicated by paradoxic movements of rib cage and abdominal compartments.

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