

CASE REPORTS

5. del Nido PJ, Williams WG, Villamater J: Changes in pericardial surface pressure during pulmonary hypertensive crises after cardiac surgery. *Circulation* 76(suppl 3):93-96, 1987
6. Belik J, Light RB: Effect of increased afterload on right ventricular function in newborn pigs. *J Appl Physiol* 66:863-869, 1989
7. Vlahakes GH, Turley K, Hoffman JIE: The pathophysiology of failure in acute right ventricular hypertension: Hemodynamic and biochemical correlations. *Circulation* 63:87-95, 1981
8. Martin DE, Rosenberg H, Aukburg SJ, Bartkowski RR, Edwards MW, Greenhow DE, Klineberg PL: Low-dose fentanyl blunts circulatory responses to tracheal intubation. *Anesth Analg* 61:680-684, 1982
9. Hickey PR, Hansen DD, Wessel DL, Lang P, Jonas RA, Elixon EM: Blunting of stress responses in the pulmonary circulation of infants by fentanyl. *Anesth Analg* 64:1137-1142, 1985
10. Hickey PR, Hansen DD, Wessel DL, Wessel DL, Lang P, Jonas RE: Pulmonary and systemic responses to fentanyl in infants. *Anesth Analg* 64:483-486, 1985
11. Hickey PR, Wessel DL, Streitz SL, Fox ML, Kern FH, Bridges ND, Hansen DD: Transcatheter closure of atrial septal defects: Hemodynamic complications and anesthetic management. *Anesth Analg* 74:44-50, 1992
12. Faithfull NS, Haider R: Ketamine for cardiac catheterization. *Anaesthesia* 26:318-323, 1971
13. Coppel DL, Dunder JW: Ketamine anaesthesia for cardiac catheterization. *Anaesthesia* 27:25-31, 1972
14. Hickey PR, Hansen DD, Cramolini GM, Vincent RN, Lang P: Pulmonary and systemic hemodynamic responses to ketamine in infants with normal and elevated pulmonary vascular resistance. *ANESTHESIOLOGY* 62:287-293, 1985
15. Murray JP, Lynn AM, Stamm SJ, Herndon PS, Kawabori I, Stevenson JG: Hemodynamic effects of ketamine in children with congenital heart disease. *Anesth Analg* 63:895-896, 1984
16. Wolfe RR, Loehr JP, Schaffer MS, Wiggins JW: Hemodynamic effects of ketamine, hypoxia, and hyperoxia in children with surgically treated congenital heart disease residing >1,200 meters above sea level. *Am J Cardiol* 67:84-87, 1992
17. Hickey PR, Hansen DD: Fentanyl- and sufentanil-oxygen-pancuronium anesthesia for cardiac surgery in infants. *Anesth Analg* 63:117-124, 1984
18. Anand KJS, Hickey PR: Halothane-morphine compared with high-dose sufentanil for anesthesia and postoperative analgesia in neonatal cardiac surgery. *N Engl J Med* 326:1-9, 1992
19. D'Ambra MN, LaRaia PJ, Philbin DM, Watkins WD, Hilgenberg AD, Buckley MJ: Prostaglandin E₁: A new therapy for refractory right heart failure and pulmonary hypertension after mitral valve replacement. *J Thorac Cardiovasc Surg* 89:567-572, 1985
20. Frostell C, Fratacci M-D, Wain JC, Jones R, Zapol WM: Inhaled nitric oxide: A selective pulmonary vasodilator reversing hypoxic pulmonary vasoconstriction. *Circulation* 83:2038-2047, 1991
21. Roberts JD, Polaner DM, Todres ID: Inhaled nitric oxide (NO): A selective pulmonary vasodilator for the treatment of persistent pulmonary hypertension of the newborn (PPHN) (abstr). *Circulation* 84:321, 1991

Anesthesiology
78:376-379, 1993
© 1993 American Society of Anesthesiologists, Inc.
J. B. Lippincott Company, Philadelphia

Intraoperative Pacemaker-mediated Tachycardia: A Complication of Dual-chamber Cardiac Pacemakers

E. Samain, M.D., J. Marty, M.D.,† H. Dupont, M.D.,‡ E. Mouton, M.D.,§ J. M. Desmots, M.D.†*

PACEMAKER dysfunctions commonly reported during the perioperative period include pulse generator inhibition or reprogramming induced by electromagnetic

interference emitted by electrosurgical diathermy units.¹⁻³ However, more complex or specific complications, such as pacemaker-mediated tachycardia (PMT), may be observed with recently designed dual-chamber pacemakers.^{4,5} In this report, we describe an unusually sustained PMT that occurred intraoperatively in a patient with an atrioventricular sequential pacemaker.

Case Report

A 71-yr-old, 110-kg man was admitted for elective total knee replacement in October 1990. In January 1989, evaluation of several syncopal episodes had revealed intermittent complete heart block. Consequently, a Telectronics (model Aurora 6291, Sydney, Australia) atrioventricular sequential programmable pacemaker was inserted

* Assistant Professor of Anesthesiology, University of Paris VII.

† Professor of Anesthesiology, University of Paris VII.

‡ Resident in Anesthesiology, Department of Anesthesiology, Bichat Hospital.

§ Staff Cardiologist, Department of Cardiology, Bichat Hospital.

Received from Bichat Hospital and University of Paris VII, Paris France. Accepted for publication October 16, 1992.

Address reprint requests to Dr. Samain: Département d'Anesthésiologie, Hôpital Bichat, 46, rue Henri-Huchard, 75018 Paris, France.

Key words: Complications: tachycardia. Heart: pacemaker.

CASE REPORTS

just inferior to the left clavicle and attached to a Prothia-Ozypka endocardial atrial pacing lead (Type OZY KY61) and to a APC endocardial ventricular pacing lead (Type 3472/MAPC). The pacemaker was programmed to the DDD mode. The program parameters are listed in table 1. Since insertion of the pacemaker, regular clinical follow-up had been unremarkable, and the pacemaker was functioning normally 4 months before surgery. Medical history revealed hypertension, controlled by nicardipine and enalapril. Preoperative physical examination showed an obese elderly man. Electrocardiography (ECG) revealed normal atrial contractions at a rate of 68 beats/min and 100% synchronous ventricular pacing.

Oral lorazepam (1 mg), nicardipine (20 mg), and atropine (1 mg) were given 1 h before surgery. After insertion of a radial intraarterial catheter, anesthesia was induced with thiopental and fentanyl intravenously and tracheal intubation was facilitated with pancuronium. Anesthesia was maintained with nitrous oxide in oxygen (60–40%), isoflurane, and increments of fentanyl as required clinically. Fifteen minutes after induction of anesthesia, occasional premature atrial contractions were seen on the ECG monitor. They were sensed by the atrial lead of the pacemaker as they occurred after postventricular atrial refractory period (PVARP), and thus were followed by paced ventricular complexes. Anesthesia was deepened with fentanyl 150 µg and increased concentration of isoflurane (1% end tidal isoflurane).

Since the pacemaker was located far from the surgical field, a unipolar electrosurgery unit (Statome 820, type 322700, EMC, Montrouge, France) was used. The dispersive plate was placed on the opposite thigh, and the surgeons were asked to use the diathermy sparingly. Twenty minutes after induction, the tourniquet on the left thigh was inflated and the skin was incised. As soon as cutting current was used, a tachycardia at a fixed rate of 117 beats/min began. Simultaneously, systolic arterial pressure decreased from 110 to 75 mmHg. Interruption of electrosurgery did not restore normal rhythm. The ECG monitor showed ventricular pacing at a regular rate of 117 beats/min. The dysrhythmia was initially thought to be due to a reprogramming of the pacemaker induced by electrical interference from electrosurgery. To correct hypotension, presumed to be partly due to the tachycardia, a strong magnet was applied over the generator to convert the pacemaker to the VOO mode (asynchronous ventricular pacing) at a fixed-rate of 87 beats/min. Decreasing the ventricular rate induced only a slight increase in blood pressure (10 mmHg). Anesthesia was lightened and 6 mg ephedrine was administered intravenously. On three occasions, removal of the magnet was followed immediately by a return to ventricular pacing at a rate of 117 beats/min. It was thus decided to maintain magnet application until reprogramming became possible.

Table 1. Pacemaker Parameters

	Atrial	Ventricular
Resettable refractory period (ms)	240	280
Pulse width (ms)	0.5	0.5
Pulse amplitude (V)	5	5
Sensitivity (mV)	1	2.8

Telectronics, model Aurora 6291: mode DDD, dual-chamber; ventricular minimum rate, 55 ppm; magnet mode, VOO, fixed rate at 87 ppm; postventricular atrial refractory period (PVARP), 280 ms; atrioventricular delay, 180 ms; ventricular maximum rate, 120 ppm.

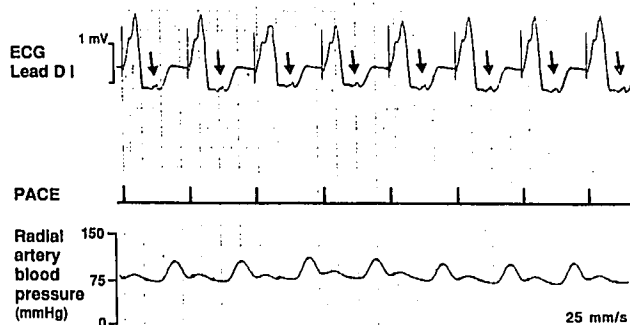


Fig. 1. Electrocardiograph and radial artery blood pressure recording during application of the magnet over the pacemaker generator, in VOO mode. Pacing rate is 87 beats/min. Each paced ventricular complex is followed by a retrograde atrial activation, seen 330 ms after ventricular depolarization (arrows).

The supervising engineer for this model of Telectronics pacemaker was called for reprogramming. Before his arrival, lead DI of the ECG and radial artery blood pressure were recorded (fig. 1). This record shows the cardiac pacemaker functioning appropriately in the magnet-mode VOO, with ventricular pacing at a rate of 87 beats/min. However, retrograde P waves, not visualized on the ECG monitor, were seen 330 ms after the ventricular spike, suggesting another explanation for the tachycardia. The recording obtained after removal of the magnet allows a diagnosis of pacemaker-mediated tachycardia to be made (fig. 2). In an attempt to terminate this phenomenon, successive applications and removal of the magnet were performed without success. It was decided to continue the surgical procedure with the magnet applied over the generator. On arrival in the recovery room, involuntary displacement of the magnet showed a return to the normal DDD mode with effective sensing of normal atrial contractions and 100% sequential ventricular pacing at a rate of 85 beats/min. Thirty minutes later, the pacemaker program was retested by the engineer. No malfunction was noted. To avoid recurrence of PMT, the postventricular atrial refractory period was increased to 360 ms. Recovery from anesthesia and the early postoperative period were uneventful.

Discussion

Advanced atrioventricular pacemakers have been developed to adjust the ventricular pacing rate according to physiologic changes of atrial rate and to maintain the contribution of atrial contraction to ventricular filling. These pacemakers are beneficial in patients suffering from cardiac conduction defects who have normal sinus node function.⁶ The usual mode of such pacemakers is the DDD mode, characterized by sense and trigger functions in the right atrium and the right ventricle, respectively.

Pacemaker-mediated tachycardia is an inappropriate acceleration of heart rate sustained by the pacemaker.⁴

CASE REPORTS

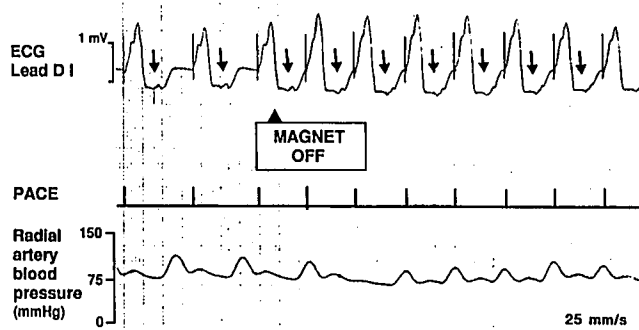


Fig. 2. Removal of the magnet from the pacemaker generator is followed immediately by a pacemaker-mediated tachycardia at a rate of 117 beats/min. Arrows show retrograde atrial activation.

A triggering event, most frequently a premature ventricular complex, establishes a condition favorable for retrograde electrical activation of the atria. This atrial activation is sensed by the pacemaker as a physiologic atrial event. Consequently, it triggers a paced ventricular complex and again a subsequent retrograde atrial activation. A pacemaker-mediated reentrant loop is therefore initiated. The retrograde limb of the reentrant loop is set up by retrograde ventriculoatrial conduction. The dual-chamber pacemaker serves as the anterograde limb. The rate of the PMT is determined by the sum of the atrioventricular interval and the retrograde ventriculoatrial conduction time.

In the present case, tachycardia was first attributed to a reprogramming of the pacemaker at submaximal ventricular tracking rate. This diagnosis was suggested because: (1) the tachycardia occurred during use of electrocautery; (2) 100% of ventricular complexes were paced at a regular rate of 117 beats/min, which is just below the programmed ventricular maximal rate of this device (table 1); and (3) no atrial activity was visualized on the ECG monitor. The correct diagnosis of PMT could be made only from the ECG recording, which showed a retrograde atrial P wave in the ST segment, occurring 330 ms after the ventricular spike and 180 ms between retrograde P waves and ventricular pacing, corresponding to the programmed atrioventricular delay. Hence, the summed atrioventricular (180 ms) and ventriculoatrial delay (330 ms) of 510 ms corresponds to the tachycardia rate of 117 beats/min.

The initiating event of the PMT cannot be identified precisely in this case, since the ECG display was altered by the use of electrocautery. Pacemaker-mediated tachycardia is initiated most frequently by a premature

ventricular complex and less commonly by a premature atrial complex.⁷ Other events, such as myopotentials or electromagnetic interference, have been reported occasionally to initiate PMT.⁸ Transient PMT has been recorded in an anesthetized patient during intraoperative transcutaneous somatosensory electrical potential stimulation, using short bursts of current delivered at interval of 300 ms.⁵ Cutting currents needed for electrosurgery have been reported to inhibit atrioventricular sequential pacemakers, but their ability to initiate PMT is unknown.^{2,9,10} It can be speculated that application of cutting current just after the PVARP may be detected by the pacemaker as atrial activity. This triggers a paced ventricular complex and initiates a PMT, whenever ventricular depolarization is conducted to the atrium.

It is of interest in this case that the PMT recurred each time the magnet was removed from the generator. The magnet mode of most dual-chamber pacemakers is the DOO mode, characterized by asynchronous sequential atrioventricular pacing.⁶ When the magnet is applied, the reentrant loop is interrupted. As the pacemaker imposes atrial contraction, retrograde activation of the atria becomes unlikely. The probability of recurrence of PMT when the magnet is removed is therefore very low with these pacemakers.¹¹

Although still in use today, the Aurora pacemaker belongs to a generation of pacemakers in which the magnet mode is VOO. When the magnet is applied over the generator, the reentrant loop is interrupted and 100% ventricular pacing is observed, but retrograde activation of the atria remains unchanged. Therefore, when the magnet is removed, retrograde activation of the atria can be sensed by the atrial lead of the pacemaker and PMT can be reinitiated. Termination of PMT associated with the use of these pacemakers is therefore more difficult. In most cases, it happens spontaneously when a ventricular depolarization is not driven to the atrium.

The potential for PMT depends upon the existence of ventriculoatrial conduction.¹² In patients with antegrade atrioventricular block, ventriculoatrial conduction is present in 25–32% of cases.¹³ Several techniques have been proposed to prevent PMT in DDD pacing.^{8,12} The most common is to provide a programmable atrial refractory period after ventricular depolarization, namely a PVARP. When the PVARP is adjusted to exceed ventriculoatrial conduction time, retrograde atrial depolarization cannot be sensed by the pacemaker. In our case, ventriculoatrial conduction time

CASE REPORTS

was 330 ms and PVARP was set at 280 ms. Increasing PVARP to 360 ms was sufficient to avoid recurrence of PMT. However, in some cases, ventriculoatrial time is much longer, up to 500 ms.⁷ Increasing PVARP above ventriculoatrial conduction time is therefore not possible, as it limits the ventricular maximum rate and thus decreases the physiologic adaptiveness. New solutions have been proposed in more recently designed pacemakers, such as automatic prolongation of PVARP after a premature ventricular beat or differentiation by the pacemaker between normal P waves and retrograde P waves.^{5,8}

In summary, identifying the cause of tachycardia from an electrocardioscope display in patients with dual-chamber pacemaker is difficult and may lead to a false interpretation. In the perioperative period, initiation of PMT in DDD pacing is favored by electromagnetic interference and the high incidence of premature ventricular complex. This case illustrates the importance to the anesthesiologist of knowing the characteristics and functions of a pacemaker, as this knowledge may be necessary for successful management of perioperative potentially life-threatening dysrhythmias.

References

1. Batra YK, Bali IM: Effect of coagulating and cutting current on a demand pacemaker during transurethral resection of the prostate: A case report. *Can J Anaesth* 25:65-66, 1978
2. Domino KB, Smith TC: Electrocautery-induced reprogramming of a pacemaker using a precordial magnet. *Anesth Analg* 62:609-612, 1983
3. Zaidan JR: Pacemakers. *ANESTHESIOLOGY* 60:319-334, 1984
4. Greenberg RM, Greenspon AJ, Bridenbaugh A, Brest AN: Pacemaker-mediated tachycardia: A complication of atrioventricular universal (DDD) pacemakers. *Arch Intern Med* 144:1061-1063, 1984
5. Merritt WT, Brinker JA, Beattie C: Pacemaker-mediated tachycardia induced by intraoperative somatosensory evoked potential stimuli. *ANESTHESIOLOGY* 69:766-768, 1988
6. Zipes DP, Duffin EG: Cardiac pacemakers, Heart Disease. Edited by Braunwald E. Philadelphia, WB Saunders, 1988, pp 717-741
7. Littleford PO, Curry RC Jr, Schwartz KM, Pepine CJ: Pacemaker-mediated tachycardias: A rapid bedside technique for induction and observation. *Am J Cardiol* 52:287-291, 1983
8. Jordaens L, Clement DL: The prevention of pacemaker mediated tachycardias in double-chamber pacing. *Acta Cardiol* 39:449-462, 1984
9. Dresner DL, Lebowitz PW: Atrioventricular sequential pacemaker inhibition by transurethral electrosurgery. *ANESTHESIOLOGY* 68:599-601, 1988
10. Shapiro WA, Roizen MF, Singleton MA, Morady F, Bainton CR, Gaynor RL: Intraoperative pacemakers complications. *ANESTHESIOLOGY* 63:319-322, 1985
11. Luceri RM, Castellanos A, Zaman L, Myerburg RJ: The arrhythmias of dual-chamber cardiac pacemakers and their management. *Ann Intern Med* 99:354-359, 1983
12. Ausubel K, Gabry MD, Klementowicz PT, Furman S: Pacemaker-mediated endless loop tachycardia at rates below the upper rate limit. *Am J Cardiol* 61:465-467, 1988
13. Goldreyer BN, Bigger JT: Ventriculo-atrial conduction in man. *Circulation* 41:935-946, 1970