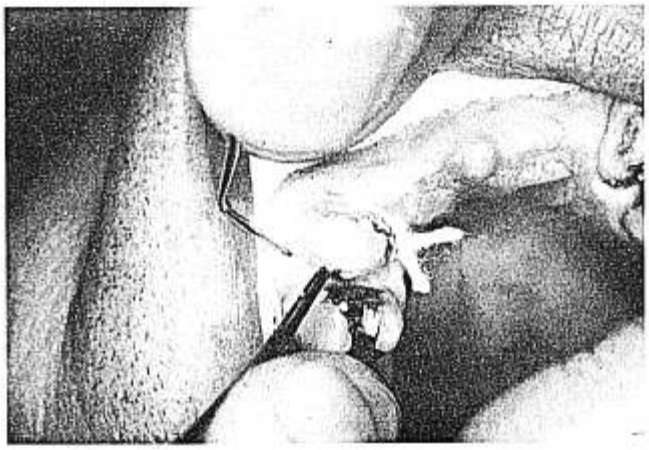

FIG. 1. Placement of gingival retraction cord impregnated with 8% racemic epinephrine for tissue retraction and hemostasis.

amount of l-epinephrine contained in the gingival retraction cord used in this case (*i.e.*, approximately 21 inches) is equivalent to a 210 $\mu g \cdot kg^{-1}$ dose, approximately 100 times the arrhythmogenic ED_{50} for submucosal epinephrine administered concomitantly with halothane![3]

Satisfactory gingival retraction and hemostasis for dental reconstruction is possible without the use of epinephrine-impregnated cord. Gingival retraction cord impregnated with either zinc chloride or aluminum sulfate are easily attainable for this purpose. Both of these agents are clinical astringents that have no systemic effects, and their use is advised when gingival retraction and hemostasis is required for multiple teeth.

REFERENCES

1. Raventos J: Action of fluothane—a new volatile anesthetic. Br J Pharmacol 11:394–409, 1956
2. Katz RL, Mateo RS, Papper EM: The injection of epinephrine during general anesthesia with halogenated hydrocarbons and cyclopropane in man: halothane. ANESTHESIOLOGY 23:597–600, 1962
3. Johnston RR, Eger EI, Wilson C: A comparative interaction of epinephrine with enflurane, isoflurane, and halothane in man. Anesth Analg 55:709–712, 1976
4. Malamed SR: Handbook of medical emergencies in the dental office. St. Louis, CV Mosby, 1978, pp 241–291
5. Brill N, Krasse B: The passage of tissue fluid into the clinically healthy gingival pocket. Acta Odontol Scand 16:233–245, 1958

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Pacemaker Syndrome during Anesthesia

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Many patients require anesthesia and surgery previously having had permanent pacemakers inserted for conduction abnormalities, particularly sick sinus syndrome and symptomatic bradycardia. Although many complications of artificial pacing such as power source failure, pulse generator malfunction, electrode fracture, and malpositioning of the electrode are well known, we present an unusual problem in a patient with a permanent ventricular demand pacemaker that has become known as "pacemaker syndrome."

REPORT OF A CASE

A 77-year-old man had, over several months, noted increasing "dizziness," especially when rising from bed in the morning. On auscultation, bilateral carotid bruits were noted. Carotid arteriograms revealed complete occlusion of the left internal carotid artery and an 80% stenosis of the right proximal internal carotid artery. He also had a history of sick sinus syndrome with profound bradycardia for which a permanent transvenous ventricular demand pacemaker had been inserted 4 years previously. Other significant medical problems were compensated congestive heart failure, chronic obstructive pulmonary disease, and diffuse peripheral vascular disease. His medications included digoxin, procainamide, meclizine, dipyridamole, and nitroglycerin. Preoperative evaluation included 12 lead EKGs, with and without a magnet, that revealed a functioning demand pacemaker set at 72 beats/min. The patient was scheduled for a right carotid endarterectomy under general anesthesia.

Premedication consisted of meperidine 25 mg, diphenhydramine 25 mg, and glycopyrrolate 0.2 mg im. A radial artery catheter was inserted percutaneously for continuous arterial pressure monitoring. Following pancuronium (1 mg iv), a rapid sequence induction for endotracheal intubation was accomplished following thiopental 150 mg and succinylcholine 80 mg iv. Anesthesia then was maintained with 60% nitrous oxide, fentanyl 0.35 mg, and pancuronium 5 mg iv.

The course of anesthesia initially was uneventful, with a sinus rhythm at a rate of 75–80 beats/min and a systolic arterial blood pressure varying between 140 and 160 mmHg. Because systolic blood pressure increased to 180 mmHg during initial vessel dissection, a sodium nitroprusside infusion, 33 $\mu g \cdot min^{-1}$, was employed for less than 15 min. One per cent lidocaine then was infiltrated into the tissues surrounding

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the carotid sinus, and a carotid Sundt shunt (Heyer–Schulte) was inserted. Approximately 10 min after the nitroprusside infusion was discontinued, the sinus rate slowed from 75 beats/min and ventricular pacing began at a rate of 72 beats/min. Concomitant with the onset of ventricular pacing, the arterial blood pressure decreased from 150/70 to 100/60 mmHg. After approximately 10 paced beats, sinus rhythm returned and arterial blood pressure increased within two heart beats to 150/70 mmHg. These changes in blood pressure were quite abrupt and were thought to be due to carotid sinus stimulation producing reflex vasodilation and bradycardia, the latter being masked by artificial ventricular pacing. Shortly thereafter, reversion to paced rhythm was associated with another simultaneous decrease in blood pressure (systolic blood pressure fell from 140 to 80 mmHg). Atropine 0.3 mg iv restored sinus rhythm and the blood pressure immediately increased to 140/70 mmHg. Atropine was administered three times in the ensuing 90 min for similar episodes of hypotension. Multiple similar episodes of hypotension too brief for the administration of atropine also occurred. Because of the acuteness and the intermittent nature of these events, metabolic derangements seemed unlikely to be a factor. Therefore, these periods of intermittent hypotension associated with artificial pacing still were attributed to surgical manipulation of the carotid sinus, causing reflex bradycardia and vasodilation, although the surgical team denied any stimulation of the carotid sinus.

The residual neuromuscular blockade was reversed with pyridostigmine 10 mg and glycopyrrolate 0.4 mg iv, and the trachea was extubated without difficulty. He then was transported to the recovery room, where, while lying quietly and without any stimulation, he experienced a period of paced rhythm associated with a decrease in blood pressure from 140/80 mmHg to 80/60 mmHg. Clearly, surgical stimulation could not have precipitated this episode of hypotension, which led to the conclusion that ventricular pacing was directly responsible for this phenomenon. Additional iv atropine was administered, and calcium (2 $mg \cdot min^{-1}$) and isoproterenol (1.0 $\mu g \cdot min^{-1}$) infusions were instituted with restoration of the blood pressure upon the return of the sinus rhythm.

Following discharge from the recovery room, several pacemaker-induced hypotensive episodes occurred. Postoperative electrolytes and blood counts were within normal limits. During spontaneous ventilation with a venturi mask at a $F_{I_{O_2}}$ of 0.40, blood gas analysis revealed pH_a 7.38, Pa_{CO_2} 42 mmHg, Pa_{O_2} 101 mmHg. The patient was transferred to the medical intensive care unit for definitive therapy. While the systolic blood pressure was less than 100 mmHg, a pacing thermodilution flow-directed catheter (Swan–Ganz®) was inserted. Pulmonary artery diastolic was 13 mmHg and pulmonary capillary wedge 11 mmHg. With the institution of temporary A-V sequential pacing at 100 beats/min, systolic pressure increased to 160 mmHg. At that time, cardiac index was 2.92 l/M^2 and systemic vascular resistance (SVR) was 1,299 $dynes \cdot s \cdot cm^{-5}$. No hypotensive episodes had occurred for several hours when the temporary pacing catheter failed to capture. The flow-directed catheter was withdrawn and replaced with a hexapolar pacing wire accompanied by the reinstitution of A-V sequential pacing. Permanent A-V sequential pacing was recommended, however, the patient refused any further operative therapy. The hexapolar pacing wire was removed. All monitoring was discontinued and the patient transferred to the neurosurgical floor, where records indicate systolic blood pressures fluctuated between 100 and 160 mmHg.

Discussion

Pacemaker syndrome is an established entity in the cardiology literature.[1–4] Several mechanisms for this syndrome have been proposed. Hypotension has been attributed to the decreased cardiac output (CO) found with pacing and decreased SVR secondary to reflex peripheral vasodilation.[1–3]

When compared with a sinus rhythm, ventricular pacing causes a decrease in CO, for which two mechanisms have been proposed. The first mechanism is that the aberrant conduction pathways utilized during ventricular pacing alter the sequence of myocardial depolarization, resulting in a decreased stroke volume. After comparing the hemodynamic changes during atrial, ventricular, and atrioventricular pacing, Samet *et al.*[5] concluded that the utilization of aberrant conduction pathways had no effect on CO, as output was nearly identical in patients being atrially paced and in those being sequentially paced, both being approximately 20% higher than in patients being paced ventricularly.

The second mechanism for decreased CO during ventricular pacing is the loss of the ventricular filling supplied by atrial contraction, known as the "atrial kick." The atrial kick increases the CO by about 20%, which closely corresponds with the observed decrease seen during ventricular pacing. For this reason, the loss of the atrial kick with the associated decrease in CO has been proposed by several authors as the mechanism responsible for the hypotension of pacemaker syndrome.[2,4]

Because CO and SVR are directly proportional to BP (BP = CO · SVR), changes in CO of this magnitude (20%) could not result in the 50% reduction in BP seen in our patient. These factors may contribute to the observed hypotension but cannot be the sole cause. Similarly, heart rate does not seem to play a role as A-V sequential pacing corrects the hypotension at the same rate as ventricular pacing causes hypotension. Our patient experienced marked hypotension when HR decreased only slightly from 75 to 72 beats/min.

In a small but controlled study, Alicandri *et al.* compared CO, plasma volumes, and autonomic function as measured by cardiovascular responses to the Valsalva maneuver and found that in pacemaker syndrome patients these indices met or exceeded those found in control patients.[1] Therefore, pacemaker syndrome appears to result from a decreased or at least an inappropriately low SVR. In fact, several authors have found that as opposed to the normal compensatory increase in systemic vascular resistance (+22%) in response to a decrease in CO, patients with pacemaker syndrome exhibit either no change or a slight decrease (−1.5%) in systemic vascular resistance. These findings as well as the finding of right atrial cannon waves in patients with pacemaker syndrome led Alicandri *et al.* to propose an atrial vasodilatory reflex as the mechanism responsible for the production of pacemaker syndrome. According to them, either retrograde A-V conduction or inappropriately timed spontaneous atrial contraction leads to atrial contraction against closed valves, thereby creating acutely elevated atrial pressures.

These elevated pressures then stimulate atrial *A* vagal receptors, which are known to cause peripheral vasodilation.[6] Although this proposal appears sound, no direct evidence for it exists and not all authors agree with this proposed mechanism.

As noted in our case report, the definitive therapy is A-V sequential pacing, which eliminates inappropriate timing of atrial contraction. In situations in which sequential pacing is not available, therapy aimed at restoring a sinus rhythm is indicated. Because the pathophysiologic mechanism of pacemaker syndrome is an inappropriately low systemic vascular resistance, the use of vasopressors and volume expansion in the acute situation would have a strong theoretical basis, provided a sinus rhythm could not be restored.

We have presented a patient with pacemaker syndrome. These patients develop hypotension with the onset of ventricular pacing and may have symptoms of hypotension, including syncope. Although cardiac output falls, it does not fall any more than in control patients. These patients have normal intravascular volumes and normal cardiovascular responses to the Valsalva maneuver. Reflex vasodilation arising from the stimulation of atrial stretch receptors therefore appears to be responsible for the hypotension. A-V sequential demand pacing is the definitive therapy for this syndrome.

References

1. Alicandri C, Fouad FM, Tarazi RC, Castle L, Morant V: Three cases of hypotension and syncope with ventricular pacing: Possible role of atrial reflexes. Am J Cardiol 42:137–142, 1978
2. Erbel R: Pacemaker syndrome. Am J Cardiol 44:771, 1979
3. Lewis ME, Sung RJ, Alter BR, Myerburg RJ: Pacemaker-induced hypotension. Chest 79:354–355, 1981
4. Miller M, Fox Stewart, Jenkins R, Schwartz J, Toonder FG: Pacemaker Syndrome: A non-invasive means to its diagnosis and treatment. Pace 4:503–506, 1981
5. Samet P, Castillo C, Bernstein WH: Hemodynamic sequelae of atrial, ventricular, and sequential atrioventricular pacing in cardiac patients. Am Heart J 72:725–729, 1968
6. Guyton AC: Textbook of Medical Physiology. Philadelphia, WB Saunders, 1976, pp 271, 474

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Precautions in the Anesthetic Management of a Patient with Creutzfield–Jacob Disease

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Creutzfield–Jacob Disease (CJD) (subacute spongiform encephalopathy) is a rare, noninflammatory disease of the central nervous system.[1] We describe our anesthetic experience in a patient with CJD and discuss precautions in managing patients with this diagnosis and precautions in handling the equipment used to anesthetize them.

Report of a Case

A 56-year-old man was referred to our hospital for neurologic evaluation. He had been healthy until 8 months ago, at which time he began to experience mild vertigo. In the next 6 months, he subsequently began to have bouts of mild dysphagia and horizontal diplopia. He progressively became more confused and occasionally displayed unusual outbursts of emotion, delusional thinking, and frank hallucinations. At the time of admission he was suffering from dementia, ataxia, and diffuse myoclonic jerks.

Laboratory values for his blood, urine, and cerebrospinal fluid (CSF) were normal. A computerized axial tomographic scan of the brain revealed only minimal atrophy of the cerebral cortex. An electroencephalogram displayed frequent generalized irregular slowing. He was scheduled for a stereotaxic brain biopsy.

No preoperative medication was given. In addition to disposable masks and hats, both operating room and anesthesia personnel wore gowns and gloves. A 16-gauge catheter was inserted iv in the right arm, and anesthesia was induced with thiopental iv. The trachea was intubated during paralysis induced by pancuronium and anesthesia maintained with 50% oxygen and nitrous oxide. The operation lasted approximately 1 h. Vital signs were stable without autonomic dysfunction. Anesthesia and recovery were uneventful.

All disposable equipment was incinerated and discarded. The laryngoscope then was exposed for 1 h to a 5% solution of sodium hypochlorite. Surgical instruments were autoclaved for 1 h at 121° C (15 PSI). The operating room table, floor, anesthesia machine, and other large surfaces were wiped with the sodium hypochlorite solution.

The brain biopsy revealed neuronal degeneration, gliosis, and vacuolation characteristics of CJD. At the time of this report, the patient has continued to deteriorate.

Discussion

We presented the above case for two reasons. First, there is no documentation of anesthetic experience in a

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