siderable caution when using rectal methohexital. As demonstrated by our two patients, previous safe use is not always indicative of future responses.

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Transesophageal Indirect Atrial Pacing for Drug-resistant Sinus Bradycardia

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Hemodynamically deleterious bradycardia consequent to intrinsic or drug-related sinus node dysfunction is not uncommon in patients under general anesthesia. While bradycardia can be associated with hypotension that could impair vital organ perfusion in patients with cardiac disease, it can also predispose to life-threatening tachyarrhythmias in patients with the sick-sinus syndrome[1] or congenital or acquired QT-interval prolongation.[2] Further, bradycardia may not respond to chronotropic drug treatment, or the treatment may lead to worse arrhythmias. If either or both of these occur, it may not be possible to continue with anesthesia and surgery, or temporary cardiac pacing may need to be instituted. Available routes for emergency pacing include transvenous,[3] transcutaneous,[4] and transesophageal indirect atrial pacing (TAP).[5]

Transvenous atrial or ventricular pacing can be difficult to establish in patients undergoing surgery, particularly when access is limited by the surgical field or patient's position. Also, fluoroscopy may be required to correctly position electrodes (particularly with atrial or atrioventricular (AV) sequential leads). Though less invasive, transcutaneous indirect ventricular pacing may not provide reliable capture in some patients (*e.g.*, those with obesity or pulmonary emphysema) and does not preserve atrial transport function. The latter can contribute importantly to cardiac output in patients with intact AV conduction and who are not in atrial fibrillation, but who have myocardial functional impairment.[6,7] TAP, in contrast, preserves atrial transport function, is relatively noninvasive and easy to establish in patients under general anesthesia, and has been suggested as alternative management to drugs for hemodynamically disadvantageous bradycardia in anesthetized patients.[8] In addition, we have recently determined TAP thresholds in 100 anesthetized adult surgical patients, 38 of whom showed hemodynamic

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improvement with TAP for sinus bradycardia ≤ 60 beats per min or AV junctional rhythm.[9] We report here two patients receiving general anesthesia who required TAP as an emergency measure to restore and maintain a hemodynamically effective rhythm after chronotropic drugs had failed to correct bradycardia with hypotension.

Case Reports

Case 1

An 81-yr-old woman (47 kg) was admitted for surgical repair of a femoral neck fracture. Significant past medical history included hypothyroidism consequent to I^{131} therapy for Grave's disease, hypertension, organic brain syndrome with dementia and depression, and a syncopal episode 3 yr prior to admission that was diagnosed as a vasovagal event. Current oral medications included levothyroxine (0.1 mg once daily), hydrochlorothiazide (50 mg once daily), ergoloid mesylate (2 mg four times daily), haloperidol (5 mg twice daily), benztropine mesylate (2 mg once daily), and potassium supplementation (20 mEq three times daily). Physical findings and laboratory values were unremarkable except for serum ionized potassium (3.1 mEq/l) and glucose (145 mg/dl). The chest radiograph showed cardiomegaly, and the 12-lead ECG was read as normal sinus rhythm with voltage criteria for left ventricular hypertrophy and T-wave inversion (I, II, aVF, and V3-6) and flattening (III and aVL). Serial cardiac enzymes were normal and repeat ECGs unchanged. Potassium supplementation was continued prior to surgery, and an additional 40 mEq was administered intravenously overnight prior to surgery.

The patient was brought unpremedicated to the holding area where routine and central venous pressure monitoring was established. Noninvasive arterial pressure was 136/80 mmHg and heart rate 80 beats per min (sinus rhythm) prior to induction of general anesthesia. The latter was with thiopental (total 300 mg), metocurine (total 20 mg) and fentanyl (50 μg). Anesthetic induction produced hypotension (systolic pressure ~ 60 mmHg) and sinus bradycardia (45 beats per min). While atropine (total 3 mg) produced a transient increase in heart rate to 60 beats per min and systolic pressure to 80–90 mmHg, by 20 min blood pressure and heart rate had again decreased to former levels. Meanwhile, tracheal intubation was performed; mechanical ventilation begun; and a catheter was inserted into the radial artery for blood pressure monitoring. With the return of bradycardia (about 40 beats per min), due to alternating sinus- or AV-junctional-origin rhythms, and hypotension (systolic pressure 50–60 mmHg), consecutive bolus doses of epinephrine (100, 50, and 50 μg) were administered. These produced hypertension (to 180/90 mmHg) and sinus tachycardia (120 beats per min), as well as ventricular bi- and trigeminy and ST-segment depression (I, II, and V5 monitored leads). Lidocaine (50 mg) was administered for ventricular arrhythmias, and the ST-segment depression reversed with the return (within several minutes) of bradycardia and hypotension. Since the patient had intact AV conduction, it was decided to attempt TAP with an 18-Fr esophageal stethoscope (Electromedics, Inc., Englewood, CO) modified for TAP by insertion of a bipolar pacing electrode (Tapcath®, Arzco Medical Electronics, Inc., Vernon Hills, IL).** With the midpoint between bipolar electrodes (0.8-cm spacing) 30 cm from the infraalveolar ridge, the threshold for atrial capture at a pulse width of 10 ms was 5 mA (model 7 Transesophageal Cardiac Stimulator, Arzco Medical Electronics, Inc.). TAP at rates between 75 and 80 paced pulses per min (ppm) and three times the threshold mA produced a systolic pressure of 110–120 mmHg. Since TAP was effective in restoring blood pressure, it was decided to proceed with surgery, which was considered urgent. TAP was continued, without the need for additional vasopressors, until shortly after the beginning of surgery, after which the patient had a hemodynamically effective spontaneous rhythm. The TAP threshold was unchanged after the patient was turned to the left lateral decubitus position. Anesthesia during surgery was maintained with lorazepam (total 2 mg) and isoflurane (up to 0.5%) as tolerated.

Shortly after arrival in the recovery room (the patient's trachea remained intubated with the TAP stethoscope in place), bradycardia and hypotension (systolic pressure 50–60 mmHg) recurred. TAP was resumed with a satisfactory hemodynamic effect. Attempts to discontinue TAP, however, resulted in 9–12-s periods of asystole with hypotension. A cardiologist was consulted, and a transvenous ventricular pacing lead was inserted. Ventricular pacing at 80–100 ppm failed to increase systolic pressure much above 70 mmHg. TAP was therefore resumed and continued until, in the intensive care unit, temporary transvenous AV sequential pacing leads were positioned and were functioning. The TAP stethoscope was kept in place for 24 h, however, to provide backup pacing if necessary. It was removed prior to extubation of the trachea, and the transvenous electrodes were removed several days later, after the patient had had an adequate spontaneous rhythm for 48 h.

Case 2

A 52-yr-old woman (50 kg) was scheduled for revision of total hip surgery performed 5 months previously. Past medical history included rheumatoid arthritis, peptic ulcer disease, and psychiatric treatment for a hysterical personality disorder. Previous surgeries included a partial gastrectomy, lumbar laminectomy, and carpal tunnel release, all performed with general anesthesia without complications. The patient was receiving no prescription medications at the time of surgery, and physical examination (except for the above-noted personality disorder, lateral eyebrow loss, and coarse hair), laboratory values, chest radiograph, and 12-lead ECG were all unremarkable.

Venous access and direct arterial monitoring were established in the holding area. Midazolam (total 3 mg) and fentanyl (100 μg) were also given. Prior to induction of anesthesia, sinus rhythm (56 beats per min) was present, with a blood pressure of 102/64 mmHg. Anesthesia was induced with thiopental (total 400 mg), fentanyl (total 150 μg), and succinylcholine (100 mg). The trachea was easily intubated and mechanical ventilation begun. With informed consent, a TAP stethoscope, similar to the one used in the first patient, was also positioned for determination of pacing thresholds (see above**).[9] Isoflurane (up to 2% inspired) and metocurine (total 17 mg) were used to maintain anesthesia. Shortly after beginning surgery, the patient's heart rate decreased rather suddenly, to 38–40 beats per min. Sinus rhythm appeared to be present. Systolic arterial pressure, which had been 100–110 mmHg, decreased to about 65 mmHg. Two bolus doses of ephedrine (5 mg) failed to increase heart rate above 45 beats per min or systolic pressure above 70 mmHg. Since the TAP electrode was in place (the pacing threshold was 3 mA at an infraalveolar ridge to electrode distance of 29 cm and a 10-ms pulse width), it was decided to use TAP in preference to further drug therapy to increase heart rate. With TAP between 70 and 80 ppm, a hemodynamically effective rhythm was maintained for the duration of surgery. Attempts to discontinue TAP resulted in sinus or AV junctional bradycardia and hypotension (systolic pressure < 80 mmHg). After reversal of neuromuscular blockade (neostigmine 2.5 mg and glycopyrrolate 0.5 mg), the patient had an adequate spontaneous rate and rhythm, so that TAP was discontinued and the trachea extubated. The patient had no further bradycardia or hypotension in the recovery room, but she con-

** This is an investigational device. Use for emergency pacing and investigation of TAP thresholds has been approved by our hospital and institutional human subject committees. Use for pacing is awaiting approval by the Food and Drug Administration.

tinued to be hypothermic (intraoperative esophageal temperature 34.5° C) for several hours despite the use of warm blankets and warming of all intravenous fluids in both the operating and recovery rooms.

Discussion

Two patients are described who developed hemodynamically deleterious bradycardia due to sinus node dysfunction or AV junctional rhythm during general anesthesia. The patients either did not respond well to or had complications with chronotropic drug treatment. TAP, as we have described elsewhere,[9] however, brought prompt restoration of a hemodynamically effective rhythm.

Based on our experience, bradycardia due to sinus node dysfunction or AV junctional rhythm is not uncommon in anesthetized patients, although the exact incidence of bradycardia in such patients has not to our knowledge been reported. Likely, intrinsic sinus node dysfunction contributed to bradycardia in the first patient, which is suggested by her previous syncopal episode. The depressant actions of intravenous and inhalation anesthetics on sinus node function also could have contributed to bradycardia in either of our two patients.[10,11] Except for ergoloid mesylate, which can be associated with bradycardia, drugs the first patient was receiving at the time of surgery are not known to produce bradycardia, although negative chronotropic interactions with anesthetic drugs cannot be dismissed. Hypokalemia may have contributed to bradycardia in the first patient, since serum potassium values of 2.6 and 2.9 mEq/l were recorded during the intra- and early postoperative periods. Nevertheless, based on review of available studies,[11] there does not appear to be a specific association between hypokalemia and intraoperative bradycardia.

Hypothyroidism almost certainly contributed to bradycardia in the first patient and possibly also in the second patient. Sinus bradycardia is said to be common in hypothyroid patients,[12] and thyroid function test results that became available after the first patient's surgery are consistent with the diagnosis of hypothyroidism: resin triiodothyronine uptake (T_3Ru) = 47 units (normal 25–35 units); thyroxine radioimmunoassay (T_4RIA) = 2.4 units (normal 5–12.5 units); free thyroxine index − free thyroxine factor (FTI − T_7) = 1.12 (normal 1.2–4.2); and thyroid-stimulating hormone radioimmunoassay (TSH RIA) = 10 units (normal 0.2–5 units). The first patient, like the second, tended to be hypothermic throughout her operative and immediate postoperative course (34.0–35.5 °C). Hypothermia in either patient likely contributed to bradycardia. With the second patient, the psychiatric history, lateral eyebrow loss, and coarse hair, as well as the hypothermia during and shortly after surgery, are consistent with hypothyroidism. Thyroid function tests subsequent to surgery in the second patient, however, were within the normal range except for low T_4RIA (4.2 units) and borderline low FTI − T_7 values (1.29).

Regardless of the cause for bradycardia in either of our two patients, it was associated with serious circulatory impairment and therefore required treatment. Treatment at first was attempted with chronotropic drugs and vasopressors, which proved ineffective or caused adverse effects. TAP, however, provided immediate and effective treatment for bradycardia without side effects. In addition, in the first patient, TAP was superior to ventricular pacing, illustrating the importance of preserved atrial transport function in this patient,[6,7] although we do not know her left ventricular filling pressures. Yet, in the absence of bradycardia, tachycardia, or hypotension, this patient's central venous pressure was below 15 mmHg.

Acute onset atrial fibrillation with bradycardia, however, might be reversed by cardioversion, and in that way TAP could be effective if bradycardia due to a mechanism other than AV block or atrial fibrillation persisted after cardioversion. We note that TAP has also been used to terminate atrial flutter and supraventricular tachyarrhythmias due to reentry (*i.e.*, paroxysmal supraventricular tachycardia).[5,13] Finally, the stethoscope modified for TAP[9] and used in our two patients cannot be relied upon to provide ventricular pacing,†† although Anderson and Pless have used a balloon catheter to provide ventricular[14] or AV sequential pacing from the esophagus.[15] Nevertheless, our two case reports clearly confirm the value of TAP as effective management for circulatory imbalance caused by sinus or AV junctional bradycardia.

†† Unpublished personal observations.

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Postoperative Independent Lung Ventilation in a Single-lung Transplant Recipient

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Transplantation of the human lung was first performed in 1963.[1] Prior to 1978, more than 40 transplants were performed, but the longest survivor lived only 10 months.[2] The major causes of mortality were bronchial dehiscence or respiratory failure due to infection or rejection. Lung transplants are now more successful and more common.[3] Perioperative care of these patients is interesting and challenging for anesthesiologists due to the dramatic hemodynamic and respiratory changes that occur, but there has been little in the anesthesia literature regarding the perioperative care and course of these patients.[4] We report a case of unique perioperative physiology successfully treated with independent lung ventilation in a single-lung transplant recipient.

CASE REPORT

A 50-yr-old woman with advanced pulmonary emphysema due to α_1-antitrypsin deficiency was referred for consideration for single-lung transplantation. The diagnosis of α_1-antitrypsin deficiency had been made at age 32 yr, when she noted exertional dyspnea. At the time of evaluation she was short of breath at rest, could walk only 10 feet without stopping, and used continuous nasal oxygen. She had lost 3.6 kg over the previous 6 months, and now weighed 43 kg; her height was 162 cm.

Preoperative evaluation revealed pulmonary artery pressures of 40 mmHg systolic and 26 mmHg diastolic. Pulmonary capillary wedge pressure was 4 mmHg. Cardiac output was 4.04 $l \cdot min^{-1}$. Pulmonary function tests revealed forced vital capacity 0.96 l (30% of predicted) and FEV_1 0.22 l (9% of predicted). Medications included metaproterenol and ipratropium inhalers. A lung ventilation/perfusion (V/Q) scan revealed multiple bilateral matched ventilation and perfusion defects, with 65% of ventilation and perfusion to the right lung. Arterial blood gas measurements are reported in table 1. The preoperative chest x-ray demonstrated hyperinflation with bullous formation. Other laboratory values were normal.

The patient was brought to the operating room for left lung transplantation. Anesthesia was induced with thiopental 250 mg and fentanyl 250 μg (in divided doses over 5 min), and intubation was facilitated with succinylcholine 100 mg after a defasciculating dose of pancuronium 2 mg. An 8-Fr bronchial blocker catheter with a 20-ml balloon (Fogarty Occlusion Catheter, Baxter model 62-080-8/14F) was placed alongside a 7-mm single-lumen endotracheal tube and was advanced into the left mainstem bronchus with the aid of fiberoptic bronchoscopy to allow selective ventilation of the right lung during the transplantation. A pulmonary artery catheter was inserted after induction and revealed values similar to those measured during the preoperative evaluation. Arterial oxygenation was easily maintained (arterial blood gas [ABG] measurement 3, table 1).

Intermittent doses of ephedrine (5 mg each) and phenylephrine (40–80 μg) were required in order to maintain systolic blood pressure in the 80–100-mmHg range over the 1st h of the surgery, while the surgeons performed the abdominal part of the operation in order to pass a portion of omentum into the thorax with which to "wrap" the eventual bronchial anastomosis.[5] During passage of the omentum, arterial blood pressure decreased to below 50 systolic, necessitating treatment with 300 μg epinephrine and 30–45 s of external cardiac massage. The arterial blood pressure returned to 80–100 mmHg systolic, and dopamine was started at a dose of 7 $\mu g \cdot kg^{-1} \cdot min^{-1}$. A pneumothorax was suspected but could not be demonstrated by the surgeons, and no air was obtained *via* a chest tube. It was noted that if positive pressure ventilation was suspended for short periods (15–45 s), the systolic arterial pressure increased from the 80–90 mmHg range (with dopamine) to 100–110 mmHg. Attempts were made to improve hemodynamics by changing the rate or depth of ventilation at normocapnia, but were not successful.

The anesthesia team made the diagnosis of air-trapping in severely emphysematous lungs causing a decrease in venous return due to a rise in intrathoracic pressure, and deliberate hypoventilation was instituted in an attempt to stabilize the patient's hemodynamics. In an attempt to reduce any bronchospastic contribution to air-trapping, several doses of a metaproterenol inhaler were administered *via* the

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