In the assessment of velocity of flow through a tube, the diameter of the tube is the primary determinant, but the length must also be considered.[8] Halving the length of a catheter halves the resistance to flow, so in the selection of a catheter to use for transtracheal ventilation, it is reasonable to pick the shortest one available.

Transtracheal appliances for emergency access to the airway are commercially available. Most of these are designed to assist placement of a tube through the cricothyroid membrane, which was unavailable in this case because of its involvement in the tumor. Although percutaneous dilational tracheostomy devices may be useful in experienced hands, they may have devastating complications. In general, problems are associated with injury to the tracheal walls and the use of positive-pressure ventilation, and the resulting widespread emphysema.[9] As anesthesiologists, we are most experienced with the use of intravenous catheters. If familiar equipment and techniques are used, the likelihood of traumatizing the airway in emergency situations is reduced.

In summary, this case demonstrates that a chronically progressively obstructing airway may be enlarged to a cross-sectional area that is adequate for spontaneous ventilation, at least for a short period of time, by placing one or more 12-G transtracheal intravenous catheters. This case also demonstrates that the decision threshold to use this therapeutic option, which has not been reported previously, should be reasonably low for anesthesiologists faced with the patient who needs just a slight enlargement of his or her natural airway to sustain life.

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Therapeutic Suppression of a Permanent Ventricular Pacemaker Using a Peripheral Nerve Stimulator

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Patients with permanent cardiac pacemakers present a potential problem for the anesthesiologist. The operating room represents an electrical environment that may interfere with normal pacemaker function.[1–3] Specifically, the ability of the pacemaker to sense electromagnetic potentials other than intrinsic myocardial potentials may lead to inhibition. Even inadvertent pacemaker reprogramming has been described.[4,5] This environment is especially dangerous when electrocautery is used, and numerous reports have delineated adverse effects, which include pacemaker inhibition as well as the precipitation of various dysrhythmias, including ventricular fibrillation and asystole.[6–8]

Optimal cardiac output may be difficult to maintain in patients with ventricular pacemakers. Patients with pacemakers also may have intrinsic cardiac disease and poor ventricular function. Furthermore, ventricular pacemakers eliminate the augmentation of ventricular filling by atrial contraction, and wall motion is asynchronous and dysfunctional during ventricular pacing.[9,10] An im-

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portant corollary to this problem is that a demand ventricular pacemaker, even when inserted for appropriate indications, may, if the pacemaker suppresses a slower sinus rhythm, prove detrimental to the patient whose cardiac function is inadequate. Pharmacologic interventions such as atropine or sympathomimetic agents often are useful in accelerating an intrinsic sinus rhythm and restoring cardiac performance.

We report a case in which our ability to interfere with normal pacemaker function under general anesthesia using a peripheral nerve stimulator significantly increased the cardiac output with a lower heart rate after the failure of chronotropic drugs to increase the intrinsic rate.

Case Report

A 63-yr-old woman with severe claudication was admitted for an aortofemoral bypass. She had a history of hypertensive heart disease with left ventricular hypertrophy and diastolic dysfunction, intermittent paroxysmal supraventricular tachycardia, and atrial fibrillation, and a history of congestive heart failure. A demand ventricular cardiac pacemaker had been inserted 1 month previously for episodes of profound bradycardia after spontaneous conversion from atrial dysrhythmia. The ventricular inhibited pacemaker rate was 70 beats per min. Her medications included verapamil 240 mg/day, ferrous sulfate, and acetaminophen with oxycodone as needed for low back pain.

She was admitted with a heart rate of 76 beats per min (sinus rhythm), a blood pressure of 150/70 mmHg, and unlabored respirations. Her lung fields were clear; other than a soft S4 and left ventricular enlargement, her cardiac examination was normal. Except for an hematocrit of 33%, her laboratory evaluation was normal. Her ECG revealed left ventricular hypertrophy with strain and left atrial enlargement. A chest roentgenogram confirmed left ventricular hypertrophy but demonstrated clear lung fields.

The patient received oral ranitidine and diazepam, and her physical status on arrival to the operating room was unchanged from admission. She was monitored with continuous ECG (leads II and V5), pulse oximeter, esophageal stethoscope, intraarterial catheter, pulmonary artery catheter, and mass spectroscopy. Anesthesia was induced with sufentanil, thiopental, and vecuronium and was maintained with sufentanil, isoflurane, nitrous oxide, and vecuronium. After 1 h of surgery, the patient remained hemodynamically stable, with a heart rate of 78 mmHg, blood pressure of 126/66 mmHg, cardiac output of 4.6 l/min, and pulmonary capillary wedge pressure of 13 mmHg.

During the next 15 min, however, her heart rate slowly decreased, and the demand ventricular pacemaker began functioning at a rate of 70 beats per min. With a pacemaker-induced rhythm, the blood pressure decreased to 85/50 mmHg, cardiac output decreased to 2.5 l/min, and the pulmonary capillary wedge pressure was unchanged. Lactated Ringer's solution and albumin were infused to increase the pulmonary capillary wedge pressure to 18 mmHg, but the patient remained hypotensive, with a blood pressure of 87/55 mmHg and a cardiac output of 3.0 l/min.

A total of 2 mg atropine was given intravenously over the following 30 min in an attempt to increase her intrinsic heart rate to greater than 70 beats per min, but with only transient benefit. During periods of sinus capture, her blood pressure was immediately restored to >120/60 mmHg and her cardiac output to > 4.5 l/min. The sufentanil was discontinued, and anesthesia was maintained with isoflurane. Neuromuscular blockade was maintained with pancuronium. Because sinus rhythm was not restored, isoproterenol was infused at 1 μg/min, but it resulted in ventricular ectopy and hypotension and so was discontinued.

In an attempt to suppress the electronic pacemaker function and allow the patient's sinus rhythm to pace the heart, a peripheral nerve stimulator (Mini-Stim; Life-Tech, Inc., Houston, TX) was placed on the left shoulder, ipsilateral to the electronic pacemaker apparatus, and was discharged in the twitch mode at a frequency of 2 Hz. The pacemaker immediately was suppressed. The patient's sinus mechanism captured at 60 beats per min, and blood pressure and cardiac output were instantly restored to normal (fig. 1). When the peripheral nerve stimulator was withdrawn, the paced rhythm returned, and again the blood pressure decreased. Peripheral nerve stimulator suppression of the pacemaker was continued until the patient's intrinsic rate exceeded 70 beats per min.

In the intensive care unit, after surgery, the cardiology consultant reprogrammed the pacemaker to a demand rate of 50 beats per min, with no further episodes of hypotension. The patient recovered uneventfully.

Discussion

Electronic cardiac pacemakers are capable of inducing a stable heart rate and rhythm in patients whose intrinsic conduction system has become nonfunctional. However,

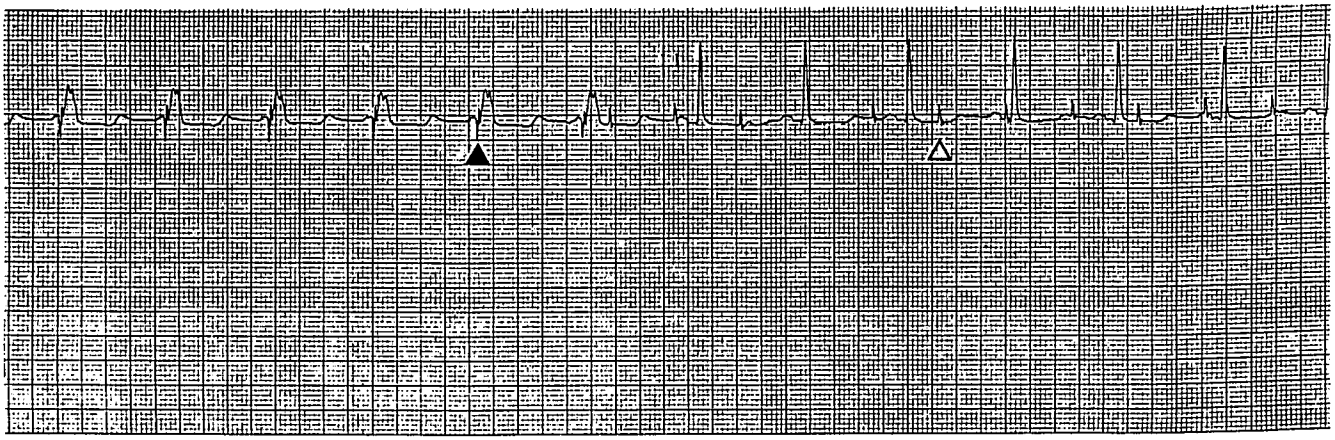

FIG. 1. A paced rhythm at 70 beats per min yields to a sinus rhythm at 68 beats per min. Note that pacemaker spikes (filled arrowhead) stop immediately as PNS spikes (open arrowhead) appear.

reestablishment of a stable heart rhythm does not necessarily imply recreation of normal cardiac function. For normal contractile function, sequential contraction of atria and ventricles is necessary, as is the simultaneous contraction of the left and right ventricles. Ventricular demand pacemakers do not allow sequential atrioventricular contraction. In the presence of a normal left ventricle, atrial contraction contributes minimally to left ventricular performance. However, in the presence of abnormal ventricular diastolic function (left ventricular hypertrophy or chronic congestive heart failure), atrial contraction enhances ventricular performance significantly. Benchimol *et al.*[9] demonstrated a 30–50% increase in cardiac index and a 33% improvement in tension–time index with atrial pacing when compared with ventricular pacing in patients with compensated congestive heart failure. Hartzler *et al.*[10] showed that after cardiac surgery, atrioventricular sequential pacing augmented cardiac index by 26–50% over ventricular pacing, and that ventricular pacing alone decreased cardiac output in the majority of patients for whom postoperative pacing was indicated.

Atrioventricular sequential pacemakers improve cardiac output, more than do ventricular pacemakers, by preserving the atrial contribution to ventricular filling. But even with these atrioventricular sequential devices, the left ventricle does not eject blood normally. Because the pattern of conduction system depolarization is abnormal, the pattern of myocardial contraction also is abnormal. Badke *et al.*[11] demonstrated a 27% decrement in left ventricular systolic performance and the appearance of dyskinetic segments during ventricular pacing, due to abnormal patterns of depolarization.

That this patient would have abnormal diastolic heart function could have been predicted from her history of chronic hypertension. This was proven by the diminution in cardiac performance when her ventricular demand pacemaker began capturing at a rate of 70 beats per min. Given this information, an atrioventricular sequential pacemaker should have been placed originally. If a ventricular demand unit was chosen, a demand rate of 50 beats per min would have provided better overall cardiac function.

When faced with the dilemma presented here, the goal of the anesthesiologist is to restore atrioventricular sequential contraction. One approach is to use chronotropic drugs to increase the sinus rate, as was done here, but without success. Not only did the drugs have no effect on sinus rate, but also isoproterenol led to ventricular dysrhythmias and hypotension. Remaining options included atrial pacing using transvenous pacing wires or external suppression of the existing pacemaker. Transesophageal atrial pacing also is an acceptable atrial pacing modality[12] and has been used intraoperatively to treat anesthesia-induced bradyarrhythmia.[13,14]

Unfortunately, transesophageal pacing devices are not available to us in our operating room. The ability of extrinsic electrical stimuli to inhibit pacemaker output, especially in the operating room environment, is well known.[1–8] Unipolar electrocautery is notorious for producing pacemaker dysfunction or failure. We chose to use to our advantage the ability of external stimuli to interfere with pacemaker function. The Life-Tech Mini-Stim peripheral nerve stimulator provided 300-V (current = 30 mA) stimuli of 0.25-ms duration at a rate of 120 pulses/min. Electrical stimuli applied over the left shoulder were sufficient to inhibit ventricular pacing. The pacemaker was programmed to sense myopotentials of 2.5 mV or greater. Because of the distance of the peripheral nerve stimulator from the heart, the intrinsic cardiac rhythm was unaffected by the stimulator. The improvement in cardiac output was dramatic, and pacemaker suppression was necessary for much of the remainder of the case. Had suppression of the demand pacemaker unmasked a profound bradycardia that worsened this patient's hemodynamic status, the nerve stimulator simply would have been withdrawn. Severe bradycardia certainly can occur with sick sinus syndrome, but the gradual diminution in the sinus rate after induction of anesthesia made this unlikely.

Alternatively, the pacemaker could have been reprogrammed. However, a cardiologist was not available to reprogram the pacemaker during the operation. Once in the surgical intensive care unit, the patient's cardiologist reprogrammed the pacemaker for a rate of 50 beats per min, and the patient was hemodynamically stable for the remainder of her postoperative course.

In conclusion, it cannot be assumed that the presence of a ventricular inhibited pacemaker in a patient with intrinsic heart disease provides a "safety net" during the administration of anesthesia. However, as this case demonstrates, proper function could be detrimental to the patient. Indications for the pacemaker as well as its function should be addressed in view of the underlying disease. Finally, the use of a repetitive external stimulus supplied by a peripheral nerve stimulator in this case provided a temporary alternative to reprogramming by restoring sinus rhythm and acceptable blood pressure and cardiac output.

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Anesthetic Considerations in Patients Receiving Colony-stimulating Factors (G-CSF and GM-CSF)

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In an effort to achieve the maximum therapeutic benefit, chemotherapeutic agents are administered to the point of severe hematopoietic toxicity. This aggressive therapy leads to marrow aplasia with thrombocytopenia, anemia, and neutropenia. In an effort to decrease the period of neutropenia and thereby lessen the risk of infection, recent investigation has centered on the administration of granulocyte–macrophage and granulocyte colony-stimulating factors (GM-CSF and G-CSF, respectively) to increase granulocyte production.[1,2] Although these new interventions effectively increase granulocyte production, adverse cardiovascular and pulmonary effects have occurred.[2,3]

Currently, use of these agents is limited; however, early success suggests that their administration to patients with neutropenia of various causes will continue to increase. In addition, these agents have recently been released for general medical use and are no longer restricted to specialized centers and protocols. Patients with neutropenia frequently require intraoperative and intensive care management for various diagnostic and therapeutic modalities.

Currently there is no information about these factors in the anesthesia literature. This report presents the anesthetic treatment of a patient receiving GM-CSF, reviews the current information available concerning colony-stimulating factors, discusses their adverse physiologic effects, and comments on the anesthetic implications of patients receiving these agents.

CASE REPORT

A girl aged 4 yr and 3 months and weighing 15.4 kg presented for treatment of juvenile chronic myelogenous leukemia. After preconditioning with cytosine arabinoside and total body irradiation, a matched unrelated bone marrow transplant was performed. Posttransplant graft function showed persistent neutropenia, anemia, and thrombocytopenia. Seventy days posttransplant, the absolute neutrophil count was 400–500 neutrophils per mm^3. At this point, therapy with GM-CSF ($20\ \mu g \cdot kg^{-1} \cdot day^{-1}$) was started.

Because of persistent fever, further testing was performed and included computed tomography of the head, which showed opacification of the maxillary sinuses compatible with sinusitis. Follow-up chest x-rays revealed a diffuse interstitial pattern compatible with pulmonary edema, a small right-sided pleural effusion, and a left lower-lobe infiltrate. Based on these findings, the patient was scheduled for bron-

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