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External Chest Wall Stimulation to Suppress a Permanent Transvenous Pacemaker in a Patient during Endovascular Stent Graft Placement

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ENDOVASCULAR stent graft placement recently has become an alternative to open laparotomy in the treatment of abdominal aortic aneurysms. During deployment of the aortic stent graft, temporary asystole is induced to prevent distal device migration, which may result in ineffective aneurysm exclusion by the stent graft or possible aortic damage by device embolization. Currently, high-dose adenosine usually is used to induce temporary high-degree atrioventricular block and asystole during deployment of the device. ^{2,3}

Patients with permanent pacemakers present a unique problem. There must exist a temporary way of inhibiting the pacemaker during the action of adenosine. Although newer pacemaker generators can be inhibited temporarily by perioperative reprogramming, older pacemakers may have limited programmability, making this task impossible. In this case report, we describe the perioperative use of external chest wall stimulation for temporary inhibition of a pacemaker during endovascular repair of an abdominal aortic aneurysm in a patient with an older generator with limited programmability.

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Case Report

An 86-yr-old man presented for repair of an 8-cm infrarenal abdominal aortic aneurysm with bilateral iliac artery extension. His medical history was significant for atrial fibrillation with a slow ventricular response requiring insertion of a ventricular pacemaker (model number 505; Cardiac Pacemaker Incorporation, St. Paul, MN) 17 yr before admission. CXR showed a pacemaker generator and an intact ventricular lead. The electrocardiogram showed atrial fibrillation with a ventricular response of 55–60 beat/min with intermittent paced ventricular beats with 1:1 capture.

Preoperative evaluation of the pacemaker confirmed a normally functioning unipolar VVI pacemaker. Application of a magnet over the generator did not result in an end-of-life signal (a decrease in the ventricular pacing rate), indicating that the generator had an adequate battery charge, obviating the need for generator change. The pacemaker had limited programmability and could only be programmed to a ventricular rate of 30 beat/min. Because of this generator feature, external chest wall stimulation was planned to inhibit the pacemaker temporarily during intraoperative administration of adenosine.

After radial and pulmonary arterial catheterization, an epidural catheter was inserted for perioperative anesthesia. Two electrocardiogram monitoring electrodes were placed along the vector between the cathode (negative electrode) and anode (positive electrode) of the pacemaker generator to optimize the ability of the pacemaker generator to detect an externally generated signal (fig. 1). The electrocardiogram monitoring pads were connected to an external temporary transvenous single-chamber demand pulse generator (model 5375; Medtronic®, Minneapolis, MN) via a temporary pacemaker cable with alligator clip connections (model 5807; Medtronic). The temporary external generator was set to 80 beat/min, which was greater than the rate of the internal pacemaker. The generator output was slowly increased until the internal pacemaker was inhibited (3 milliamperes); at this output, the internal pacemaker sensed the external signals as intrinsic QRS complexes and was inhibited. No discomfort was reported by the patient during electrical stimulation. After confirmation of the ability to inhibit the pacemaker temporarily with external chest wall stimulation, the surgical procedure com-

After positioning of the aortic stent graft, temporary asystole was induced during deployment of the device. The patient was sedated with etomidate, and the temporary generator was activated (fig. 2). Adenosine (24 mg) was administered *via* the central venous catheter followed by a bolus dose of normal saline. (In our unpublished experience, 24 mg adenosine given *via* the central circulation provides an

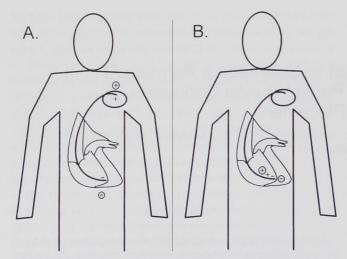


Fig. 1. Placement of electrocardiogram (ECG) pads for external chest wall stimulation for (A) unipolar and (B) bipolar lead configuration. With unipolar detection and pacing, the ECG leads are placed above and below the positive and negative polarities of the pacemaker. If bipolar detection and stimulation are used, ECG leads are placed in the vicinity of the distal end of the pacemaker lead over the right ventricular apex. Filled circles = chest wall ECG pads; + and - = pacemaker lead polarity.

adequate period of asystole [10-30 s] for deployment of the stent graft in most patients.)

Approximately 7 s after administration of adenosine, the patient became asystolic, the angioplasty balloon was inflated, and the stent graft was deployed. Proper positioning of the stent graft at the aneurysm neck was confirmed by fluoroscopy. Deployment of the device was completed after approximately 45 s. During this period, the patient was asystolic, with occasional atrioventricular nodal or ventricular escape beats. There were no ventricular beats produced by the patient's permanent pacemaker. After deployment of the stent graft, the external demand pulse generator was turned off, and the patient's internal pacemaker immediately began to function at its preset level (fig. 2B). The patient's heart rate returned to its preasystolic rate as the effects of adenosine dissipated. The rest of the procedure was uneventful, and the patient was taken to the postanesthesia care unit in stable condition. The patient had an uneventful recovery and was discharged home on postoperative day 2.

Discussion

Endovascular stent graft placement recently has become an alternative surgical technique to repair abdominal aortic aneurysms. To minimize movement of the device during deployment, temporary asystole is induced using high-dose adenosine.

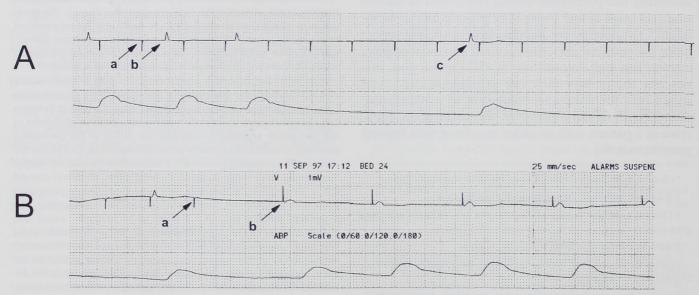


Fig. 2. Induction of asystole with adenosine and external chest wall stimulation. (*A*) The electrocardiogram and arterial pressure tracing immediately before induction of adenosine-induced asystole are demonstrated. Arrow a illustrates the temporary external pulse generator spikes, and arrow b is the patient's intrinsic QRS complex, which is not currently electronically paced. After the third QRS complex, the patient became asystolic secondary to the action of adenosine. The external pulse generator continues to inhibit the patient's permanent pacemaker and is thus temporarily asystolic. Although there are no intrinsic paced ventricular beats, the patient continues to have a few escape atrioventricular nodal or ventricular complexes (*arrow c*). (*B*) The electrocardiogram and arterial pressure tracing immediately before termination of adenosine-induced asystole. Arrow a indicates the last external pulse generator spike before cessation of external chest wall stimulation. Because the patient's own permanent transvenous pacemaker is no longer being inhibited by the external pulse generator, the permanent pacemaker begins to function to treat the patient's residual adenosine-induced bradycardia (*arrow b*).

In patients with pacemakers who undergo repair of endovascular aortic stent graft, administration of highdose adenosine is ineffective in producing the required period of asystole; during the action of adenosine, the pacemaker detects an interruption of ventricular systole and stimulates the ventricle. Preoperatively, patients with pacemakers should be seen by a cardiologist or cardiothoracic surgeon to assess the function of the pacemaker. If the patient is not dependent on the pacemaker and the programming capability of the pacemaker is not limited, the pacemaker may be turned off during the perioperative period. If the patient is dependent on the pacemaker or in the rare case of limited programmability of the pacemaker (e.g., minimal rate of ventricular stimulation by the pacemaker is 30 beat/min), other options include (1) temporary perioperative inhibition of the pacemaker during administration of adenosine (which is not a function of all pacemakers); (2) temporary reprogramming of the pacemaker to a subthreshold output to prevent ventricular capture; (3) external chest wall stimulation by an external single-chamber demand pulse generator; or (4) induction of induced hypotension to avoid the need for reprogramming of the pacemaker.

External chest wall stimulation offers a convenient way to temporarily inhibit a VVI pacemaker, obviating the need for pacemaker programming. 4,5 To maximize sensing of the external chest wall stimulation, the pacemaker should be programmed to a unipolar VVI mode, and the sensitivity should be maximized. If a unipolar mode is used, both cutaneous electrodes should be positioned over the generator and lead parallel to the cathode/anode vector to optimize the ability of the generator to sense the external signal (fig. 1).^{6,7} If a bipolar lead configuration is used, the sensitivity of external stimulation is decreased. External chest wall stimulation may be possible; however, the external cutaneous electrodes should be positioned over the right ventricular apex. In either case, the position of the cutaneous electrodes may need to be adjusted to optimize sensing of the external signal by the pacemaker generator.

Previously, this method of external chest wall inhibition has been used to detect underlying rhythms.^{4,5,7} This method has been replaced by direct inhibition of the pacemaker *via* reprogramming in newer generators. Other pacemakers (*i.e.*, dual-chamber pacemakers) may sense external stimuli as atrial or ventricular activity, depending on the orientation of the external electrodes, thus resulting in rapid ventricular pacing. Although most dual-chamber pacemakers have program-

mable capabilities, only some can be programmed off or set to subthreshold values to eliminate capture. Other dual-chamber pacemakers may require programming to a unipolar VVI mode with subsequent intraoperative external chest wall stimulation to inhibit the pacemaker.

Induction of temporary ventricular fibrillation with a transvenous ventricular electrode attached to an alternating current generator is an alternative to adenosine for producing a period of nonpulsatile blood flow in the aorta. We believed that this alternative was too dangerous *vis à vis* reprogramming the pacemaker, damaging the pacing leads, and possibly inducing tissue damage secondary to burns at the patient's generator site. Alternatively, the mean arterial pressure could have been decreased temporarily using short-acting vasodilator agents during deployment of the stent to minimize the chance of stent graft embolization.

We presented a case of a patient undergoing repair of an endovascular aortic aneurysm with an implanted permanent pacemaker that could not be reprogrammed to a mode that would result in temporary inhibition of the pacemaker. Using equipment commonly found in a cardiac or vascular operating room, we described a straightforward technique of temporary inhibition of a pacemaker without the need for reprogramming of the pacemaker.

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