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Case Report

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Successful treatment with transesophageal pacemaker for medically refractory bradycardia during endovascular thoracoabdominal aortic aneurysm surgery: 2 case reports

Abstract

We present two cases of external pacing using esophageal atrial pacing for the treatment of medically refractory bradycardia during endovascular treatment of thoracoabdominal aortic aneurysms (TAAA) under general anesthesia. TAAA are life-threatening medical emergencies. Prompt management is required to prevent rupture or further dissection. Management focuses on impulse and blood pressure control followed by endovascular repair. Beta-blockers and antihypertensives are considered first line agents. General anesthetics can potentiate the effect of these medications intraoperatively. In our patients, favorable outcome was achieved using transesophageal pacing. Currently, there is limited literature supporting the use of transesophageal pacing in the perioperative setting.

Keywords: thoracoabdominal aortic aneurysm (TAAA), transesophageal pacemaker, refractory bradycardia

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Elizabeth R Larsen MD,[1] K H Kevin Luk MD MS[2]

[1]Resident Physician, Department of Anesthesiology & Pain Medicine, University of Washington School of Medicine, USA
[2]Senior Associate Consultant / Assistant Professor, Department of Anesthesiology & Perioperative Medicine, Department of Critical Care Medicine, Mayo Clinic Arizona / Alix School of Medicine, USA

Correspondence: K. H. Kevin Luk, M.D., M.S, Department of Anesthesiology & Perioperative Medicine, Mayo Clinic Arizona, 5777 E Mayo Blvd, Phoenix, AZ 85054, USA, Email Luk.KeHangKevin@mayo.edu

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Abbreviations: TAAA, thoracoabdominal aortic aneurysm and dissection; TEVAR, thoracic endovascular aortic eepair

Introduction

Descending (Stanford Type B) thoracoabdominal aortic aneurysm (TAAA) and dissection are considered life-threatening medical emergencies that require prompt treatment of hypertension and tachycardia to prevent aneurysm rupture or further dissection. The pathogenesis of TAAA is related to an initial intimal injury with subsequent tear. Acute treatment focuses on rapid impulse control followed by blood pressure control. It should be noted that afterload should not be reduced until adequate impulse control has been achieved to prevent further stress on the aneurysm/dissection. Impulse and blood pressure control are achieved initially using intravenous agents such as esmolol, nicardipine, and nitroprusside infusions. Once the patient is stabilized, the intravenous regimen is transitioned to an oral regimen. Given the long-standing history of hypertension in some of these patients, it is not uncommon for multiple antihypertensives including beta-blockers, calcium channel blockers, central alpha-2 agonists, and nitrates to be used in various combinations for blood pressure management. With the advances in endovascular technology, it is now possible to repair some of these aneurysms/dissection in a minimally invasive fashion. Intraoperative blood pressure and heart rate homeostasis during endovascular management of the aneurysm or dissection (TEVAR) presents a unique challenge for anesthesiologists. The deployment of the endovascular stent requires neuromuscular blockade and controlled ventilation. This necessitates general endotracheal anesthesia, which can further exacerbate the pre-existing sympathetic blockade. This can result in hypotension and bradycardia, and in extreme cases can become refractory to vasopressor and inotrope administration. Excessive bradycardia can be treated with temporary pacemaker. The use of transesophageal electrodes was first reported in the 1950s in an effort to pace the heart and study the conduction system. However, there is limited recent report regarding the clinical application of transesophageal pacing as a rescue therapy for medically refractory bradycardia. We report two cases of severe, medically refractory hypotension and bradycardia, in the setting of endovascular TAAA repair under general anesthesia, and the first reported successful use of transesophageal pacemaker to correct the hemodynamics. The patient(s) or patient's family have provided "written HIPAA authorization" to publish this case report.

Case 1

66-year-old man with history of hypertension presented with a penetrating descending thoracic aortic ulcer with intramural hematoma. Patient was admitted to the intensive care unit and was placed on a labetalol infusion for impulse control. On hospital day 5, he was noted to be bradycardic and the infusion was switched to nicardipine. Later that morning, he presented for endovascular repair of his thoracic aorta. In the preoperative area, patient was noted to be normotensive in sinus bradycardia. A lumbar subarachnoid drain was placed by the anesthesiologist prior to induction of general anesthesia. After uneventful induction, an arterial catheter and large bore peripheral intravenous (IV) lines were placed. Patient became hypotensive and bradycardic. He was given escalating doses of phenylephrine, ephedrine, glycopyrrolate, and vasopressin. However, there was inadequate clinical response. When the patient failed to respond to the aforementioned agents, a norepinephrine infusion was started with additional norepinephrine, epinephrine, and vasopressin boluses. Blood pressure remained marginal with mean arterial pressure >60mmHg, but heart rate steadily trended down to the 30s. Atropine 0.5 and 1mg were administered without any effect on the heart rate. Beta-blocker toxicity was suspected, and glucagon was administered, which resulted in transient improvement of the heart rate. However, patient remained severely bradycardic and required 0.4mcg/kg/min of norepinephrine to maintain his blood pressure. An esophageal pacemaker was placed and the myocardium was captured

at 10mA. The patient was then paced at a rate of 80 beats per minute, which resulted in marked improvement of his hemodynamics. The rest of the procedure was uncomplicated and the patient remained intubated overnight with the esophageal pacemaker left in as backup. His native heart rate began to improve around midnight. He was extubated the next day and was free of neurological sequela from the procedure and anesthetic. His recovery was uneventful and was eventually discharged home with his family.

Case 2

84-year-old man with history of diabetes mellitus type 2, severe chronic obstructive pulmonary disease, chronic kidney disease stage 4, cerebrovascular disease, poorly controlled hypertension and a large thoracic aortic aneurysm causing hoarseness who underwent a TEVAR and a right carotid to left carotid bypass seven days prior now presenting with a large type 1 endoleak. He is scheduled for trans-arterial coil embolization of the left subclavian artery. The patient's preoperative blood pressure regimen consists of metoprolol extended release 50mg every 12 hours and clonidine 0.2mg every 12 hours. Prior to induction of general anesthesia, the patient was noted to be normotensive and in sinus bradycardia of 40-45 beats per minute. After induction, the patient became hypotensive and his heart rate remained at 40 beats per minute. Boluses of ephedrine, glycopyrrolate, and atropine in escalating doses were given without adequate hear rate response. An esophageal pacemaker was inserted and the myocardium was initially captured at 15mA. The current was gradually reduced to 8mA and the pacing rate was set at 80 beats per minute, which resulted in improvement of hemodynamics. The case proceeded uneventfully and the patient was extubated at the end of the procedure after removal of the transesophageal pacemaker. Patient's heart rate returned to the mid-40's and his blood pressure was noted to be normal in the immediate postoperative period. The patient made adequate recovery and was discharge to a skilled nursing facility.

Discussion

The medical management of descending TAAA focuses on impulse and blood pressure control. Beta-blockers, especially esmolol, with anti-chronotropic and anti-inotropic effects are considered first line agent in the management of TAAA. This decreases the shearing force across the aortic arch and prevents propagation of the dissection. Afterload reduction agents, such as angiotensin converting enzyme inhibitors, angiotensin-receptor blocker, calcium channel blockers, nitrates, and hydralazine can be added after adequate impulse control has been achieved to further control the blood pressure.

Labetalol is a non-specific beta-blocker with an IV beta to alpha blockade ratio of 6.9:1. The volume of distribution is about 200-800L with a significant extravascular depot. Metabolism is mostly hepatic with a half-life of 6 hours. Labetalol infusion is indicated for management of hypertensive urgency or emergency with a starting dose of 2-4mg/min for a total of up to 300mg only based on manufacturer recommendation. In the first case, the labetalol infusion was administered for 4 days, resulted in accumulation of the drug in the patient's extravascular depot. Beta-blocker toxicity is the likely culprit of the refractory bradycardia, which is further exacerbated by the sympathectomy by the potent inhaled anesthetic. In the second case, the patient was on both a beta-blocker and a central alpha-1 agonist, which resulted in significant suppression of the patient's endogenous adrenergic system. With the addition of potent inhaled anesthetics, the result is a similar clinical scenario to case one. Treatment of beta-blocker toxicity begins with optimization of preload, followed by administration of atropine, vasoactive agents, calcium, insulin along with dextrose, and in severe cases glucagon and intralipid.

In medically refractory cases, external pacing is indicated. External pacing options include transcutaneous, transvenous, and trans-esophageal. Transcutaneous pacing is the simplest way to pace the heart, but it requires a higher current to capture the myocardium. This can result in significant discomfort to the patient and requires sedation. In addition, depending on the patient's positioning and procedure performed, the chest might not be accessible to the anesthesiologist to place the electrodes, and patient movement with external pacing may interfere with the surgical procedure. Transvenous pacemaker is the most stable way to capture the myocardium with leads directly placed in the myocardium. However, it requires central venous access and fluoroscopy for accurate placement of the electrodes. Esophageal electrode placement is a relatively noninvasive technique, which was first described in 1982.[1] When compared to other external pacing methods, esophageal pacemaker offers ease of placement in the operating room since most cases the anesthesiologist would be at the head of the bed and there is no need for fluoroscopy or invasive lines. Gallagher and colleagues[1] demonstrated that esophageal pacemakers could accomplish atrial pacing with minimal discomfort to the patient, at lower pacing thresholds than other external pacing devices. At our institution, we use a commercially available esophageal pacemaker (TAPSYSTEM™, Cardio Command, Tampa, FL) that also serves as a temperature probe.[2] Anatomically, the esophagus is in close proximity to the posterior wall of the atrium. This allows for atrial pacing in patients with an intact atrioventricular node. The cases presented involves the use of general anesthesia for the endovascular management of TAAA and its related complications. We hypothesized that the interaction between the general anesthetic and multiple antihypertensives results in significant synergistic effect of hypotension, bradycardia, and anti-inotropy in our two patients. Esophageal pacing was initiated once conventional medical therapies failed to correct the hemodynamics adequately. Both of our patients had excellent neurological and cardiovascular outcomes owing to the prompt correction of hemodynamics.

In summary, esophageal pacemaker offers a convenient and effective intraoperative tool for anesthesiologists to help correct hemodynamics in patients who are suffering from medically refractory bradycardia during endovascular TAAA repair under general anesthesia.

Financial disclosures

None.

Conflicts of interest

None.

Author contributions

Elizabeth R. Larsen: This author helped prepare the entire manuscript.

K. H. Kevin Luk: This author collected the clinical data and prepared the entire manuscript.

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