

Total Spinal Anesthetic after Continuous Posterior Lumbar Plexus Block

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THE lumbar plexus is derived from rami from the first through the fourth lumbar nerves and is primarily responsible for innervation of the extensor and adductor compartments of the thigh. Blockade of the lumbar plexus using a posterior approach, *via* either a single injection or continuous technique using an indwelling catheter, is an attractive alternative for postoperative pain management in patients undergoing surgery of the hip and above the knee. This technique may enable blockade of all branches of the lumbar plexus, most notably the lateral femoral cutaneous, femoral, and obturator nerves. The genitofemoral and ilioinguinal nerves, also formed from contributions of ventral rami of the first and second lumbar nerves, are generally not considered incorporated in the block of the plexus. Also referred to as the continuous psoas compartment block, it is considered reliably safe with few complications.^{1,2}

We report the case of a total spinal anesthetic after a posterior lumbar plexus blockade performed during general anesthesia.

Case Report

A 45-yr-old woman (weight, 68 kg), American Society of Anesthesiology (ASA) class III, presented for left total hip arthroplasty for traumatic arthritis following a past motor vehicle collision. General endotracheal anesthesia with postoperative continuous lumbar plexus blockade to facilitate postoperative pain control was discussed and planned with the patient preoperatively. Appropriate ASA monitors were used with the addition of a right radial arterial pressure monitor.

Intravenous induction was performed with propofol and vecuronium. The patient was positioned right decubitus. Maintenance was accomplished with isoflurane, oxygen, nitrous oxide, and supplemented with fentanyl. The operative procedure was uneventful. No residual neuromuscular blockade was confirmed with a peripheral nerve stimulator and the patient was spontaneously breathing at the end of surgery.

With the patient remaining in the right decubitus position, she was prepped for insertion of a continuous lumbar plexus catheter using a posterior approach by the regional anesthesia attending as previously described.³ Landmarks included the superior border of the iliac crest of the operative hip, the third through fifth lumbar spinous processes to

identify the midline, and a 3- to 5-cm perpendicular line from the midline at approximately the level of the posterior superior iliac spine. The intersection of these two lines represents the point of needle insertion. A 4-in insulated Contiplex Touhy needle (B.Braun Medical, Bethlehem, PA) was inserted perpendicular to all planes using a peripheral nerve stimulator. The needle was advanced until contact with the transverse process of the fifth lumbar vertebrae was met then angled in a caudal direction. A myotonic response of the quadriceps muscles was observed, consistent with femoral nerve distribution. This was elicited at 1.5 mA and decreased to 0.5 mA at a depth of approximately 9 cm. A 40-ml mixture of 1.5% mepivacaine and 0.75% ropivacaine was injected after a negative aspiration for blood, air, and cerebral spinal fluid. A catheter was then inserted to 14 cm at the skin and secured with tape and benzoin.

The patient was then positioned supine and isoflurane was discontinued, at which time it was noted she was hypotensive with systolic blood pressure of 80 mmHg, confirmed by both arterial trace and cuff pressure. This was immediately treated with fluids and vasoactive agents consisting of intermittent boluses of ephedrine and phenylephrine, which improved blood pressure. It was also noted the patient had become apneic. Her pupils were dilated at 6 mm bilaterally and sluggish to light reaction. A suspicion of intrathecal injection with total spinal anesthesia was considered. All extremities of the patient were flaccid and did not react to painful stimuli. The patient was transferred to the postanesthesia recovery unit for postoperative ventilation, intubated, and placed on a ventilator. She was given propofol and midazolam to maintain her sedation and hypnosis.

After approximately 140 min, the patient was awake and opening her eyes upon command. The patient recovered spontaneous respirations after 180 min. After 260 min, she was moving all extremities with head lift, at which time she was extubated uneventfully and transferred to an intermediate monitoring unit for postoperative care.

The lumbar plexus catheter was left *in situ*, but not infused. The catheter was assessed postoperative day 1 by aspiration and clear fluid was withdrawn. Laboratory analysis showed a glucose level of 84 g/dl. Intrathecal placement was confirmed, and the catheter was removed. The patient recovered without further sequelae. There were no motor or sensory deficits appreciated prior to discharge. When the patient was questioned regarding recall of the event, she stated that she "remembers being numb to her ears."

Discussion

The lumbar plexus blockade, using a posterior approach, is a relatively safe alternative for postoperative pain management in patients undergoing surgery above the knee. Combined with a sciatic nerve block, anesthesia to the leg can be accomplished. Continuous techniques using indwelling catheters for peripheral nerve blocks have been used widely in Europe and are becoming more popular in the United States. Despite infrequent occurrence of complications with these procedures, subcapsular renal hematoma,⁴ psoas hematoma and plexopathy,⁵ and even inadvertent coagulation of the catheter tip at the surgical site impeding catheter removal⁶ have been reported.

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We report a case of inadvertent total spinal anesthesia after lumbar plexus block. Although this complication has been reported in the French literature,⁷ to our knowledge, it has not been reported in the United States. Our case presents critical points both of our technique and of the procedure as a whole.

The lumbar plexus originates from the ventral rami of the first four lumbar nerves and lies within the psoas muscle and anterior to the transverse processes of the corresponding lumbar vertebrae. Care must be taken to maintain perpendicular needle insertion, and the direction should be in a cranial or caudal fashion with regard to the transverse processes. A more medial direction may result in intrathecal puncture or dural sleeve contact similar to a paramedian approach to the epidural space.

The exact mechanism behind this complication in our case is unknown. Perhaps a more medial approach was not appreciated or use of improper landmarks was used because our patient was obese.

Hypotension after lumbar plexus block can occur due to epidural spread of local anesthetic, especially with volumes greater than 20 mL,^{8,9} and it should be suspected as a side effect rather than a complication of the procedure. We considered this as well as an etiology for the hypotension encountered. However, apnea and dilated bilateral pupils are more consistent with intrathecal injection and total spinal anesthesia. We used a large volume (40 mL), which we have amended to 20 mL in our practice, not to avoid inadvertent intrathecal injection but to decrease the likelihood of epidural spread. Also, we do not routinely perform peripheral nerve blocks in anesthetized patients, although time constraints created difficulty with preoperative placement. Having the patient anesthetized removes the patient as a monitor of this complication because the patient is unable to communicate any unusual symptoms, such as complete motor block or dyspnea. Further, a test dose was not used,

because it would not have been detected in our patient who was anesthetized. However, in our opinion, a test dose should be performed in the awake patient and thorough aspiration for cerebral spinal fluid should be routine for all patients. Intrathecal placement of either the needle and/or catheter is difficult to determine. A loss of resistance technique, along with the use of a nerve stimulator, would help determine this since cerebral spinal fluid should be apparent from the hub of the needle. Our needle was connected to injection tubing. Spinal fluid may have been overlooked when we aspirated because it was mixed with the local anesthetic in the small caliber tubing.

Use of peripheral nerve blockade either for regional anesthesia or postoperative pain management is gaining popularity in the United States. Although this type of blockade is considered relatively safe, especially in experienced hands, the operator must maintain constant vigilance for potential complications.

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A Complication of Left Heart Bypass: A Transesophageal Echocardiographic Finding

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LEFT heart bypass (LHB) is a commonly used technique in descending thoracic aortic aneurysm surgery. It is used primarily to help reduce the incidence of postoperative paraplegia and paraparesis. Bypass is achieved by cannulation of a pulmonary vein, left atrium, or left ventricle and delivery of the blood *via* a centrifugal pump to an aortic site distal to the aneurysm. We describe the intraoperative course and transesophageal findings of inadvertent cannulation of the left pulmonary artery instead of the pulmonary vein in LHB for descending aortic aneurysm surgery.

A 52-yr-old woman was scheduled for descending thoracic aortic aneurysm resection. The patient had been in good health before the aneurysm was discovered on a routine chest radiograph. Cardiac catheterization revealed a large calcified saccular aneurysm distal to the left subclavian artery extending to the proximal descending thoracic aneurysm. There was an anomalous origin of the left internal carotid artery from the right innominate artery. The left vertebral artery was also noted to have a moderate sized ostial lesion. The coronary arteries were normal. Left ventricular systolic function was normal with an ejection fraction of 62%. The left ventricular end-diastolic pressure was 12 mmHg. The aortic and mitral valves were normal.

The patient fasted for 8 h prior to surgery. The patient was admitted to the hospital on the day of surgery. Bilateral intravenous catheters were placed along with a 20-gauge radial arterial catheter in the right arm. An intrathecal catheter was placed in the L3-L4 interspace for cerebrospinal fluid (CSF) drainage and pressure monitoring. Standard American Society of Anesthesiology (ASA) monitors were also applied. Induction was accomplished with fentanyl, thiopental, and pancuronium. A left 35-French double-lumen endobronchial tube was inserted. Inhalational anesthesia was maintained with isoflurane. Central venous pressure monitoring was obtained with a right internal jugular 12-French catheter. A pulmonary artery catheter was not placed due to previously reported latex sensitivity in the patient. A right

femoral 20-gauge arterial line was also placed and monitored. A transesophageal echocardiography (TEE) probe was inserted. TEE confirmed that the patient had normal left and right ventricular systolic function and no valvular dysfunction. An intracardiac shunt was ruled out by two-dimensional echo and color Doppler. Intrathecal pressure was measured throughout the procedure with occasional aspiration of CSF fluid from the intrathecal catheter to maintain a pressure less than 10 mmHg.

The patient was positioned in the right lateral position for a left thoracotomy incision, and one-lung ventilation was started after incision. After dissection of the aneurysm and preparation of the left femoral artery and left superior pulmonary vein for cannulation, the patient was anticoagulated. Cannulae were placed in the left femoral artery and the left superior pulmonary vein. Left heart bypass commenced. One minute later, the aorta was cross-clamped and aortotomy was performed. Several minutes later, the blood return to the left atrial cannula was noted to be dark. The perfusionist commented that the left heart bypass flows were exceedingly high. At this point, the patient was tolerating LHB well and was stable hemodynamically, and oxygen saturation measured by pulse oximetry (SpO_2) was 100%. Concerns about the adequacy of LHB prompted arterial blood gases to be drawn and the heart to be reexamined with TEE. A right radial arterial blood gas test revealed a pH of 7.42 and partial pressure of oxygen (pO_2) of 268, and a simultaneous right femoral arterial blood gas was pH 7.35 and pO_2 22. TEE reconfirmed that there were no new findings, especially intracardiac shunt. However, the mid-esophageal pulmonary artery split view at 0° demonstrated a new echodense signal in the left pulmonary artery (fig. 1, 2).

Because the surgical repair was already underway, the decision was made to complete the surgery instead of attempting to rectify the problem. Repair was accomplished using a tube graft with closure of the aneurysm sac over the tube graft. Total aortic cross-clamp time was 25 min. After completion of the surgery, the double lumen endobronchial tube was changed to a single lumen endotracheal tube. The patient remained intubated and was transferred to the cardiothoracic intensive care unit in stable condition.

The patient was extubated on the same day of surgery in the intensive care unit. The intrathecal catheter was kept in place and monitored for 3 days. Neurologic exams were normal throughout the hospital course. The

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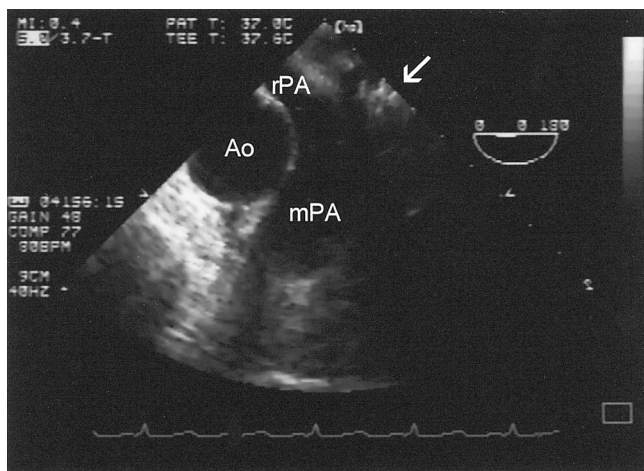


Fig. 1. The transesophageal echocardiography (TEE) image of mid-esophageal pulmonary artery split view at 0°. Ao = aorta in cross-section; mPA = main pulmonary artery; rPA = right pulmonary artery. Arrow indicates echodensity in left pulmonary artery.

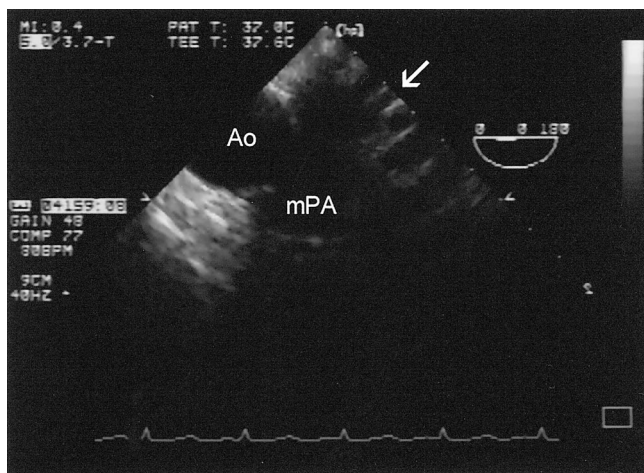


Fig. 2. The transesophageal echocardiography (TEE) image of mid-esophageal pulmonary artery split view at 0°. Ao = aorta in cross-section; mPA = main pulmonary artery. Arrow indicates echodensity in left pulmonary artery.

patient was discharged to go home on postoperative day 6 without any neurologic sequelae.

Discussion

TEE has been used to assist in the placement of left atrial cannula for LHB using the transeptal puncture method.¹ With the increased use of intraoperative TEE in cardiovascular surgery, considerations should be given for its use in confirming the correct location of the atrial or pulmonary venous cannula prior to commencement of the bypass. Left atrial cannulation could be assessed easily using the mid-esophageal four-chamber or two-chamber views. The left superior pulmonary vein is readily visible on TEE by first obtaining the mid-esophageal

four-chamber view and then focusing on the left atrium. Slight withdrawal and turning of the TEE probe to the left should bring out the superior pulmonary vein located superiorly to the left atrial appendage. The left lower pulmonary vein can often be visualized in the same position or with rotation of the imaging plane to 90°.

Aortic cross-clamping results in two major insults to the patient: distal organ ischemia and proximal hypertension. During aortic occlusion, the spinal cord must rely on collateral circulation because the clamp usually compromises normal circulation. The area of the spinal cord at greatest risk is that supplied by the anterior spinal artery. The thoracolumbar region of the anterior spinal artery is supplied primarily by the highly variable *arteria radicularis magna*. This vessel, also known as the *artery of Adamkiewicz*, has a highly variable aortic origin from between T5 to L2.

Proximal hypertension is dependent on the location of the clamp with more proximal placement resulting in a more significant increase in systemic blood pressure. This can result in increased myocardial wall stress and possibly subsequent myocardial ischemia. CSF pressure may also increase with proximal hypertension, which could reduce spinal cord perfusion pressures.²

Spinal cord injury following aortic cross-clamping can occur as early as 20–25 min.³ Controversy still exists as to the best approach to preserve spinal cord function in descending aortic aneurysm surgery. Some centers prefer to employ simple aortic cross-clamping with expeditious surgical repair, while others advocate the use of distal perfusion methods. The efficacy of LHB in reducing cord injury has been demonstrated when used as the sole neuroprotective intervention⁴ or when used in conjunction with CSF drainage.^{5,6} Supporters of left heart bypass suggest that other potential benefits are gained, such as reduction of both renal and cerebral complications.⁷

Complications of cannulation for LHB are relatively infrequent. Assurance of proper bypass and adequate perfusion and oxygenation should be sought prior to aortotomy. In our case, suspicion of a potential problem was raised only after subjective observation of the dark blood in the venous cannula. The determination of P_{aO_2} peripherally was helpful in detecting problems in LHB. The use of distal pulse oximetry is probably not useful due to the nonpulsatile nature of bypass in the distal circulation.

Left pulmonary artery cannulation for LHB has been described⁸ as an alternative to atrial or pulmonary vein cannulation. The proposed benefits to pulmonary artery cannulation are that it is readily accessible and is a sturdy structure, which allows for stable cannula position to ensure steady flows. Flow rates greater than 3 l/min can typically be achieved. This likely explains the exceedingly good bypass flow rates demonstrated by the patient.

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Complete Vasomotor Collapse: An Unusual Manifestation of the Carotid Sinus Reflex

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THE carotid sinus is a major part of the arterial baroreceptor reflex system.^{1,2} When the arterial vessel wall of the sinus is deformed during rapid pressure changes in the systemic arteries, the stretch receptors in the right and left carotid sinuses and aortic arch are activated, which elicits the threefold response of bradycardia, hypotension, and apnea.² Usually bradycardia is the most common response associated with the reflex.³ This case report describes sudden intraoperative hypotension without bradycardia, which we think was an unusual manifestation of the carotid sinus reflex.

Case Report

A 45-yr-old white man presented with recurrence of a right carotid body tumor. One year earlier, a carotid body tumor was removed, at which time the right internal jugular vein and right vagus nerve were excised due to tumor involvement. The tumor recurrence involved the right carotid artery, necessitating excision and replacement with an autologous vein graft. The preoperative assessment revealed normal electrocardiographic results and no history of dysrhythmias or syncope.

In addition to the standard monitors, an intraarterial catheter and electroencephalogram leads were placed. Anesthesia was induced using 250 µg fentanyl, 150 mg propofol, and 14 mg cisatracurium. Tracheal intubation was accomplished without difficulty and without significant variations in heart rate and blood pressure. Isoflurane and oxygen were administered for maintenance anesthesia. When the right carotid artery was cross-clamped below the tumor at the common carotid artery bifurcation, the EEG indicated adequate collateral flow

and the blood pressure and pulse were stable. During resection of the isolated portion of the carotid artery, the blood pressure suddenly decreased to 0 mmHg. The absence of any systemic pressure was confirmed by absent heart sounds from the precordial stethoscope, loss of waveform on the pulse oximeter, and absence of any palpable femoral or left carotid pulsation. The EEG that had been normal for the 15 min after cross-clamping displayed evidence of global ischemia. However, the electrocardiogram was unchanged, showing normal sinus rhythm at 76 beats/min. Cardiopulmonary resuscitation was instituted and 0.5 ml epinephrine (1:10,000) was administered intravenously. During chest compressions, the arterial catheter showed systolic pressures of 30-40 mmHg, then rapidly increased to 260/140 mmHg with a tachycardia of 150 beats/min at 45 s after epinephrine administration. Soon the blood pressure, heart rate, and EEG returned to baseline values. Arterial blood gas and chemistry measurements were normal as was follow-up electrocardiogram and cardiac enzyme studies. The subsequent surgical and postanesthetic course was uneventful.

Discussion

We believe that, during surgical dissection and excision of the recurrent tumor, the surgical instruments perturbed the right nerve of Hering and produced supramaximal stimulation of the afferent limb of the baroreceptor reflex. The noteworthy features of this event were the absence of bradycardia and the extent of the vasomotor collapse.

The afferent limb of the baroreceptor reflex is composed of two separate nerves. One nerve, the nerve of Hering, carries impulses from both the carotid sinus and the carotid body.² It is a branch of the glossopharyngeal (IX) cranial nerve. The other nerve is the aortic depressor nerve, a branch of the vagus, which carries impulses from stretch receptors in the aortic arch. The receptors in the vessel wall of the carotid sinus and the aortic arch are composed of mechanosensitive ion channels,⁴ which respond to deformation of the vessel wall with changes in blood pressure. The generated impulses are carried in the afferent fibers to synapse with secondary neurons in the nucleus tractus solitarius of the dorsal medulla. The

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efferent limb of the reflex is also twofold: one part is the vagus nerve with predominant innervation of the sinoatrial node in the right atrium⁵; the other part is the sympathetic innervation to the blood vessels.

When the baroreceptor reflex is activated, usually by increases in blood pressure, impulses in the vagal efferent component of the reflex are increased, releasing acetylcholine at the muscarinic receptors in the sinoatrial node, slowing the heart rate. However, impulses in the sympathetic efferent component are decreased and the reduced release of norepinephrine causes arteriolar and venous dilation with resultant hypotension. During surgical procedures in the neck (e.g., carotid endarterectomy), mechanical stimulation of the carotid sinus commonly elicits the baroreceptor reflex with bradycardia and occasionally hypotension. Administration of a muscarinic blocker such as atropine or field application of a local anesthetic typically eliminates further reflex activity.

The mechanosensitive ion channels are normally responsive to vessel wall stretching in the pressure range of 60–200 mmHg,^{1,2} and they are most affected by the rate of pressure change (i.e., the rate of increase of left ventricular pressure, dP/dt), and the diastolic blood pressure.⁶ In our patient, the blood pressure initially was stable, fluctuating in a small range with normal values. Probably, the mechanical stimulation of the carotid sinus or the nerve of Hering itself provided the afferent input necessary for a maximal response from the sympathetic component of the reflex, namely complete cessation of sympathetic activity to the vasculature. Experiments in animals have demonstrated cessation of postganglionic sympathetic nerve activity in response to electrical stimulation of the carotid sinus nerve.⁷ Human experiments using pharmacologic manipulation of systemic blood pressure to elicit the baroreceptor reflex have also demonstrated sympathetic ablation in response to acute blood pressure elevation.^{8,9} Shoukas *et al.*¹⁰ contend that the reduction of sympathetic activity is equally manifested in both arterial and venous components of the vascular tree. In our patient, the sudden decrease in pressure from normal levels to 0 suggests both reduction in afterload by arteriolar dilatation and reduction in preload by an increase in venous capacitance.

The absence of bradycardia in association with the hypotension made the diagnosis difficult. Anaphylaxis and venous air embolism were other considerations; however, no recent medications or blood products had been given and, though the end tidal CO_2 decreased to near 0, no mill-wheel murmur was detected when listening for heart sounds. Because the sinoatrial node is innervated primarily by the right vagus nerve with little involvement from the left vagus nerve,⁵ the predominant source of cholinergic-induced bradycardia associated

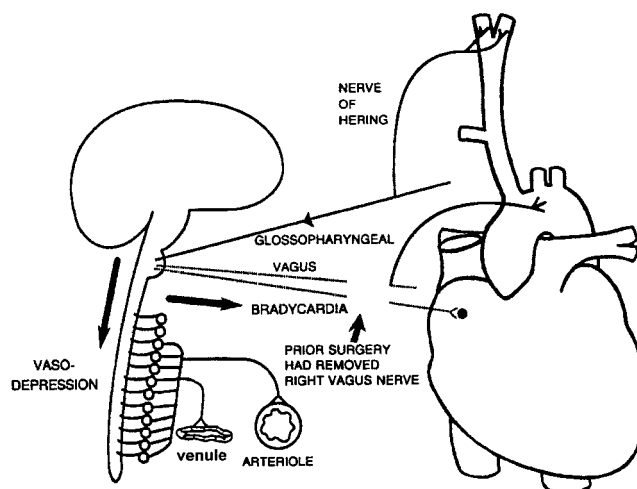


Fig. 1. Stimulation of the carotid sinus and the aortic arch produces afferent impulses in the glossopharyngeal and vagal depressor nerves. The efferent limbs are the vagus to the sinoatrial node and the decrease in sympathetic activity to the vasculature. In our case the vagal component was absent due to previous surgical excision.

with the baroreceptor reflex was missing because the right vagus nerve was excised during the previous surgery (fig. 1).

Exactly how the nerve of Hering was stimulated to elicit such a profound hypotensive response is unclear. In experimental animals, the stimuli used in baroreflex studies include electrical stimulation of the carotid sinus and aortic depressor nerves, with supramaximal intensity for 0.1 ms at 1–200 Hz.⁷ Perhaps the use of electrocautery was the precipitating stimulus in our case. It is also possible that simple mechanical stimulation, often seen in carotid surgery, was the cause.

Anesthetic agents can have a profound effect on the baroreceptor reflex. Propofol at infusion rates of $200 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, but not $100 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, will block sympathetic nervous system-mediated responses when baroreceptors are stimulated experimentally, but it has no effect on the vagal component.¹¹ The propofol we used on induction could cause initial hypotension by a baroreceptor reflex-related sympathetic block, but it would be an unlikely etiologic factor an hour after induction. The isoflurane we used for maintenance of anesthesia could have contributed to the hypotension. McCallum *et al.*¹² showed that 1.5% isoflurane abolished all sympathetic reflex responses to bilateral carotid artery occlusion. Prior to the vascular collapse, our patient was stable with a 1.1% end-tidal concentration of isoflurane, but it can be surmised that this inhalational agent exacerbated the hypotensive response to carotid sinus stimulation.

Early animal experiments by Glick and Braunwald¹³ indicated that the heart rate response to baroreceptor stimulation was mediated both vagally (increased efferent activity) and sympathetically (decreased efferent ac-

tivity). If such were the case in humans, our patient would have manifested at least partial reduction of heart rate by the cessation in sympathetic activity at the sinoatrial node. However Leon *et al.*¹⁴ observed that humans, adrenergically blocked with 10 mg propranolol intravenously, responded with both bradycardia and tachycardia to pharmacologically induced hypertension and hypotension, respectively. These investigator concluded that although β -adrenergic activity may influence basal heart rate, it exerts little if any influence on baroreflex heart rate control. This conclusion is consistent with our case because the hypothesized supramaximal carotid sinus nerve stimulation produced profound vascular collapse, presumably through reflex sympathectomy, but the heart rate was unchanged due to vagal denervation of the sinoatrial node.

In summary, we present a case of acute vasomotor collapse, which resulted from intraoperative dissection of recurrent carotid body tumor. Prior excision of the right vagus nerve prevented any heart rate response, namely bradycardia.

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