A Hypotensive/Bradycardic Episode Leading to Asystole in a Patient Undergoing Shoulder Arthroscopy in the Sitting Position With Interscalene Block and Intravenous Sedation: A Case Report

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Anesthesia for shoulder surgery is often accomplished by means of an interscalene block and intravenous sedation, with the patient subsequently placed in the sitting position for surgical access. Despite the advantages of this popular technique, sudden unheralded and severe hypotensive/bradycardic episodes have been reported in this population, with an incidence of 13% to 24%. Although these episodes are usually transient and resolve spontaneously, there are case reports of progression to asystolic cardiac arrest following hypotensive/bradycardic episodes.

To raise awareness of the catastrophic potential of these episodes and discuss possible causes and preventive measures, the author presents the case of an ASA class 1 patient undergoing shoulder arthroscopy in the sitting position with an interscalene block and intravenous sedation, who experienced a hypotensive/bradycardic episode that rapidly progressed to intraoperative asystole.

Keywords: Bradycardic, hypotensive, interscalene block, shoulder arthroscopy, sitting.

Hypotensive/bradycardic episodes (HBEs) are not uncommon, but they have the potential to be catastrophic. However, there is no clear consensus regarding their cause. This case report describes a case of an HBE progressing to asystole in a patient undergoing shoulder surgery in the sitting position with an interscalene block and intravenous (IV) sedation. Following the case summary, the author discusses possible causes of and risk factors for HBEs present in the current case and preventive strategies based on the current evidence.

Case Summary
A 19-year-old man, ASA class 1, presented for arthroscopic surgical treatment of left shoulder instability, to be performed with the patient in the sitting position under an interscalene block with sedation. The patient had no remarkable medical history and had not previously undergone surgery. He had had nothing to eat or drink for longer than 8 hours. The patient had no known allergies, was not taking any medications at home, and had no family history of complications from anesthesia.

The patient was admitted to the preoperative holding area, and the results of the physical examination and airway examination were unremarkable. Standard ASA monitors were applied, and the following baseline vital signs were recorded: blood pressure, 130/81 mm Hg; heart rate, 63/min; respiratory rate, 18/min; and oxygen saturation on room air, 100%.

A 22-gauge peripheral IV catheter was placed in the right hand, and oxygen was administered at 2 L/min via nasal cannula. The patient received midazolam, 4 mg, and fentanyl, 50 μg, intravenously for sedation before initiation of the interscalene block. Under ultrasound guidance, the neural structures and scalene muscles were identified. The needle was placed, and injection of local anesthetic was accomplished, with negative aspiration demonstrated at every 5 mL. The procedure was reportedly painless and was uneventful. A total of 35 mL of 0.5% bupivacaine (without epinephrine) was injected. Assessment of the degree of the sensory block was determined by verifying the loss of cold sensation over the shoulder with an alcohol pad. The degree of motor block was assessed by asking the patient to abduct his arm. The interscalene block was deemed successful using these 2 methods.

The patient entered the operating room 53 minutes after placement of the interscalene block. Standard ASA monitors were reapplied, and oxygen was administered via nasal cannula at 3 L/min. A nasal cannula with carbon dioxide sampling was used to continuously monitor end-tidal carbon dioxide (ETCO2) and ventilation. A noninvasive blood pressure cuff was placed on the right upper extremity. The patient was secured in the sitting position with a head holder and chin strap to stabilize the patient’s head. The operative side of the abdomen was supported with a kidney post. No compression stockings were applied to the patient’s lower extremities. Once the patient verbally confirmed comfort, a propofol infusion...
was titrated to the endpoint of lack of a response to a glabellar tap, which corresponded to 75 μg/kg/min. After adequate sedation, a 30F lubricated nasal trumpet was placedatraumatically, and 1,000 mg of cefazolin was administered. The patient’s vital signs at the beginning of surgery were as follows: blood pressure, 101/60 mm Hg; heart rate, 71/min; respiratory rate, 17/min; and oxygen saturation on 3 L/min of oxygen via nasal cannula, 100%. He had a positive, stable ETCO₂ tracing.

The surgical procedure began uneventfully 105 minutes after placement of the interscalene block. Surgical irrigation of 36 L, with every 3 L of normal saline containing 1 mL of 1:1,000 epinephrine, was used by the surgeon during the procedure. Intraoperatively, the patient remained hemodynamically stable. He had a blood pressure ranging from 93/59 mm Hg to 104/61 mm Hg, heart rate of 60/min to 72/min, respiratory rate of 17/ minute; and oxygen saturation on 3 L/min of oxygen via nasal cannula of 98% to 100%; and a positive, stable ETCO₂ tracing.

Three hours and 12 minutes after placement of the interscalene block (2 hours and 5 minutes after the patient was placed in the sitting position; and 1 hour and 27 minutes after the beginning of the surgical procedure), the patient experienced an HBE leading to asystole, heralded only by a change in heart rate. Over the course of 2 minutes and 15 seconds, the heart rate decreased from 59/min to 0/min. Leading up to this decrease in heart rate, the blood pressure, respiratory rate, oxygen saturation, and ETCO₂ tracing were all stable.

As soon as asystole was observed, a code was called and the surgeon was instructed to stop the procedure as the staff prepared to lower the table to the supine position and begin advanced cardiovascular life support (ACLS). As epinephrine and atropine were being drawn up and the propofol infusion was stopped, return of spontaneous circulation occurred. Ephedrine, 20 mg (given incrementally), and glycopyrrolate, 0.2 mg, were administered with return of spontaneous circulation to treat a bradycardic heart rate of 30/min to 40/min. A fluid bolus of lactated Ringer’s solution was infused for volume expansion as an additional 10 mg of epinephrine and 0.4 mg of glycopyrrolate were administered.

The entire asystolic event lasted approximately 15 to 30 seconds, and return of spontaneous circulation occurred while preparations were made to initiate ACLS by drawing up epinephrine and atropine and lowering the table to place the patient supine. Respiratory rate and oxygen saturation values returned to baseline within 15 seconds of the asystolic event. The blood pressure, heart rate, and ETCO₂ tracing returned to baseline values within several minutes. The remainder of the operation was completed uneventfully, and the patient emerged from sedation without incident. He was discharged home the next day.

Discussion
In patients undergoing shoulder surgery in the sitting position with an interscalene block and IV sedation, HBEs have been reported with an incidence of up to 24%. A hypotensive/bradycardic episode is defined as a decrease in heart rate of more than 30/min in less than 5 minutes or any decrease of greater than 50/min, and/or a decrease in systolic blood pressure of more than 30 mm Hg in less than 5 minutes or any decrease below 90 mm Hg. Hypotensive/bradycardic episodes are unpredictable, and although they are usually transient and resolve spontaneously, there are numerous case reports of these episodes leading to asystole and requiring cardiopulmonary resuscitation (CPR).1-5

Turker et al3 reported a case of a 58-year-old ASA class 1 female patient who presented for shoulder arthroscopy with an interscalene plexus block in the sitting position without IV sedation. The patient experienced profound hypotension and bradycardia intraoperatively at approximately 45 minutes after administration of the block. Ephedrine, 10 mg, and atropine, 0.5 mg, were given, but the patient’s condition progressed into asystole. The patient was immediately returned to the supine position and CPR was initiated. After 20 seconds of CPR, the patient’s heart returned to normal sinus rhythm and the patient regained consciousness. The operation continued uneventfully.

Gupta et al5 reported the case of a 46 year-old man who presented for arthroscopic repair of the right shoulder with administration of an interscalene block and general anesthesia in the beach chair position. Approximately 150 minutes after placement of the interscalene block and 90 minutes after being placed in the sitting position, the patient went into asystole for 40 seconds. After administration of a fluid bolus, atropine, and epinephrine, the patient’s heart reverted to normal sinus rhythm. The surgical procedure was aborted, and the patient emerged from anesthesia and was extubated. He was discharged home the following day.

As highlighted by these published case reports and the present case, HBEs have the potential to progress to lethal alterations in hemodynamics and cardiac rhythm.

• Possible Etiology of Hypotensive/Bradycardic Events.

The exact etiology of HBEs is unknown and has been the subject of considerable debate in the literature. Possible etiologies cited in the literature are summarized in Table 1 and described here.

• Carotid Sinus Hypersensitivity. Carotid sinus hypersensitivity has been cited as a possible mechanism for HBEs.6 Carotid sinus hypersensitivity is an exaggerated response to stimulation of the carotid artery baroreceptors located at the bifurcation of the common carotid arteries. Under normal physiologic conditions, blood pressure and heart rate are regulated, in part, by the carotid arterial baroreceptor reflex. The afferent limb of this
reflex is linked to the medulla via the glossopharyngeal and vagus nerves. The efferent pathway leads from the medulla to the heart and blood vessels via sympathetic and vagus nerves. Signals from stretch or tension sensors are transmitted from the baroreceptors to the medulla, which processes this information and adjusts heart rate and vasomotor tone accordingly via the efferent pathway.

Carotid sinus stimulation in patients with carotid sinus hypersensitivity may result in severe bradycardia and/or vasodilation, which can lead to hypotension or syncope. Other possible causes of carotid sinus hypersensitivity are hypersensitive afferent or efferent limbs of this reflex, or abnormal central processing ofafferent information.

Both the carotid artery baroreceptors and the sternocleidomastoid tension receptors can be stretched by movement of the neck. Proprioceptive information provided from the sternocleidomastoid tension receptors is processed by the medulla to discern the origin of the stimuli and to ensure that stretching of the neck is not misinterpreted as hypertension. It is proposed that the initiation of an interscalene block may inhibit transmission of proprioceptive information from the sternocleidomastoid muscle. As a consequence, stretching of the neck may be misinterpreted as hypertension and elicit an HBE by the baroreceptors. In fact, 2 cases of cardiac arrest resulting from carotid sinus hypersensitivity were precipitated by positioning maneuvers for head and neck surgery. Direct stimulation of the carotid baroreceptors may also be responsible for HBEs.

- **Bezold-Jarisch Reflex.** Another proposed mechanism of HBEs is a type of cardioinhibitory reflex known as the Bezold-Jarisch reflex (BJR). The origin of this reflex stems from stimulation of mechanoreceptors and chemoreceptors in the interior walls of the ventricles of the heart. On stimulation, the receptors send signals along the afferent pathway of nonmyelinated type C vagal fibers that course to the medulla. The medulla responds by reducing sympathetic tone, causing bradycardia, peripheral vasodilation, and hypotension.

In the context of HBEs during shoulder procedures, the placement of patients in the sitting position may cause venous pooling in the lower extremities. Augmented by peripheral vasodilation from the effects of IV sedation and a surgical preference for lower ranges of blood pressure, the alteration of venous capacitance may result in reduced venous return to the heart, producing a state of relative hypovolemia. Exogenous epinephrine, from the interscalene block or from the surgical irrigating fluid, and endogenous epinephrine from the surgical stress response, can induce a state of increased sympathetic tone leading to hypercontractility of a relatively hypovolemic ventricle. Consequently, the mechanoreceptors in the ventricular walls are stimulated, activating the BJR.

Alternatively, hypovolemia can trigger thrombocytes to release serotonin, which can stimulate the chemoreceptors in the walls of the ventricle and activate the afferent limb of the BJR. Serotonin may likewise increase the activity of the vagus nerve, contributing to the parasympathetic response associated with the BJR.

The assumption of the BJR as the cause of HBEs, however, remains a source of considerable debate. Campagna and Carter claim that HBEs observed during shoulder surgery with interscalene block in the sitting position appear to be unrelated to BJR activation. In their case report, Gupta et al. state that although the BJR is commonly cited in the literature as the cause of HBEs, there is a lack of convincing data to support the BJR as the cause of HBEs. Furthermore, they report that their patient was adequately hydrated, casting doubt on the likelihood that a hypovolemic ventricle activated the BJR in their patient. As an alternative explanation, the origins of HBEs may lie in the conditions that precipitate orthostatic hypotension, particularly when antihypertensive agents are administered to enhance the surgeon’s view of the surgical field.

Venous air embolism is a potential complication of any surgical procedure where the operative site is above the level of the heart, as the pressure in the exposed veins is subatmospheric. Hence, air may be entrained in the venous system and transported to the right side of the heart. As entrained air enters the right side of the heart, it can disrupt right ventricular outflow, causing cardiopulmonary compromise manifested as decreased cardiac output, hypotension, cardiac arrhythmias, arterial desaturation, and sharp decreases in ETCO₂. Venous air embolism during shoulder arthroscopy has been reported in the literature even when liquid has been used as the distending medium. However, it is unlikely that it was the cause of the HBE in this case report because the event was heralded by a dramatic decrease in only heart rate. Respiratory rate, oxygen saturation, ETCO₂ tracing and blood pressure were all stable leading up to the HBE.

The inadvertent IV injection of local anesthetic during any peripheral nerve block can lead to cardiovascular instability manifested as an HBE. Furthermore, spread of local anesthetic to the epidural or subarachnoid space can cause cardiovascular collapse but is rare. However, the delayed nature of HBE, as most cited cases occur approximately 40 to 80 minutes after placement of the interscalene block.
or 25 to 45 minutes from initiation of the sitting position, make these unlikely culprits.5,6 Furthermore, the asystolic event our patient experienced occurred 192 minutes after the interscalene block was placed and 125 minutes after being placed in the sitting position.

- **Potential Risk Factors for Hypotensive/Bradycardic Episodes.** The case presented possesses several factors that have been studied in the literature and have yielded conflicting results regarding their influence on the occurrence of HBEs. These factors include the laterality of the interscalene block, the use of epinephrine in the local anesthetic, the use of sequential compression devices or elastic stockings, and the level of sedation administered.

The laterality of the block has been cited as a possible HBE risk factor. Specifically, it has been suggested that a right-sided stellate ganglia block as a complication of the interscalene block may be associated with an increased incidence of HBEs.6 A retrospective study of 63 patients undergoing shoulder surgery in the sitting position with interscalene block reported that 92% of patients who experienced an HBE had a right-sided interscalene block.18 Despite these findings, HBEs can occur in patients who have received a left-sided interscalene block; in fact, other studies have found no predominance of HBEs in right-sided interscalene blocks.4

The use of epinephrine in the local anesthetic for an interscalene block or in the surgical irrigation solution has been examined as a possible precipitating factor in HBEs. The local anesthetic administered to our patient contained no epinephrine, although the irrigating solution used by the surgeon contained epinephrine at a concentration of 1:3,000,000. It is possible that the epinephrine in the irrigating solution was absorbed into the systemic circulation. Exogenous epinephrine has been cited as the cause of the hypercontractile ventricle, which activates the BJR.7 However, they report that their results remained statistically significant when they excluded patients receiving urapidil or metoprolol in their analysis. An editorial was published in response to this study and disputed the theory that epinephrine injected in the interscalene space could produce the hemodynamic effects associated with HBEs.20 The basis for the argument presented was founded in a study by Barber et al21 that compared hemodynamic changes associated with local anesthetics with and without epinephrine injected into the perianal region. Despite increasing plasma concentrations of epinephrine, no differences in the hemodynamic changes were found between local anesthetics with epinephrine and those without epinephrine.21

A separate retrospective study examined 63 patients who received an interscalene block for shoulder surgery in the sitting position.18 The population was divided into those patients who experienced HBEs (n = 13) and those who did not (n = 50). They found no significant difference in the incidence of HBEs with the use of epinephrine in the local anesthetic. Clearly, evidence regarding the effect of epinephrine on the occurrence of HBEs is inconsistent.

In the case presented, no sequential compression devices or elastic stockings were used on the patient’s lower extremities, which could have promoted venous pooling and reduced venous return, a factor that reportedly contributes to the BJR. One study compared the use of sequential compression devices with elastic stockings for prevention of venous pooling and demonstrated a decreased incidence of hypotension and less hemodynamic instability in patients undergoing shoulder surgery in the sitting position with the use of sequential compression devices.22 This study was a prospective, double-blind randomized study conducted with 51 patients undergoing shoulder arthroscopy in the sitting position under general anesthesia. Patients were randomly divided into 3 groups: those with sequential compression devices, those with elastic stockings, and those with no mechanical device (control). Hypotension was defined as a mean arterial pressure below 60 mm Hg or 80% of baseline values. The incidence of hypotension was statistically significantly lower with sequential compression devices vs the control (31% vs 71%). Although elastic stockings decreased the incidence of hypotension vs the control, it was not statistically significant. Not only does this...
evidence suggest a possible preventive measure for HBEs, but it also lends support to the concept of venous blood pooling in the sitting position as a contributing factor to hypotension in the sitting position.\textsuperscript{22,23}

After our patient was positioned properly, IV sedation was provided in the form of a propofol infusion, as well as a bolus of fentanyl (25 μg) administered 87 minutes before the asystolic event. A relatively higher incidence of HBEs in patients receiving intraoperative fentanyl has been cited in the literature and is suggestive of fentanyl as a contributing factor.\textsuperscript{18,23} In one prospective study, patients were randomized to receive saline, 50 μg of fentanyl, 100 μg of fentanyl, or 30 mg of ketorolac 20 minutes after being placed in the sitting position with a previously placed interscalene block.\textsuperscript{24} The incidence of HBEs in this study was found to be 10%, 15%, 27.5%, and 5%, respectively, with the highest incidence noted for 100 μg of fentanyl. Armstrong and coworkers\textsuperscript{25} conducted a 10-year retrospective analysis of 3,875 patients undergoing shoulder surgery in the sitting position under interscalene block. They found a 5.78% rate of occurrence of HBEs and proposed that the BJR was the likely mechanism of action. In contrast to published evidence suggesting fentanyl to be a contributing factor to HBEs,\textsuperscript{18,23} they proposed that their practice of heavy intraoperative sedation for shoulder cases might decrease the level of endogenous catecholamines released during procedures and be responsible for the lower incidence of HBEs at their institution.

- Prevention of Hypotensive/Bradycardic Episodes.

From the current state of the evidence, HBE risk factors have not been clearly identified and the mechanism may be multifactorial. Despite this, in acknowledging the catastrophic potential associated with HBEs, several retrospective and prospective studies have examined potential preventive measures. These studies were based on the assumption that the BJR reflex was responsible for HBEs and were designed to test the effectiveness of β-blockers, anticholinergics, and ondansetron in reducing the occurrence of HBEs. A retrospective study of 116 patients undergoing shoulder arthroscopy in the sitting position examined the effect of glycopyrrolate and metoprolol in reducing the occurrence of HBEs.\textsuperscript{11} Glycopyrrolate was administered to prevent the heightened parasympathetic tone induced by activation of the BJR. Patients were randomized into 3 groups. Group 1 was administered prophylactic metoprolol to reduce heart rate to below 60/min, or a maximum dose of 10 mg, immediately after the administration of the interscalene block. Group 2 received prophylactic glycopyrrolate to achieve a heart rate of greater than 100/min, or a maximum dose of 10 mg, immediately after the administration of the interscalene block. Group 3 was given a placebo. Metoprolol was effective in preventing the occurrence of HBEs compared with glycopyrrolate (incidence of HBEs: 28% with placebo, 5% with metoprolol, and 22% with glycopyrrolate). The authors likewise proposed that the BJR was responsible for these HBEs.

A randomized prospective study examined the effectiveness of ondansetron in preventing HBEs in patients undergoing shoulder arthroscopy in the sitting position with an interscalene block examined the effect of β-blocker administration on the incidence of HBEs.\textsuperscript{4} All patients received epinephrine in the local anesthetic administered, which consisted of lidocaine, meperidine, or bupivacaine. The patient population was divided into 2 groups: those who experienced an HBE (group 1) and those who did not (group 2). A reduced incidence of HBEs was found in patients receiving β-blockers intraoperatively to treat intraoperative hypertension and/or tachycardia with labetalol, metoprolol, or esmolol. Group 2 was subdivided into patients who did not receive β-blockers (group 2A) and those who received β-blockers (group 2B). Groups 1 and 2B exhibited similar increases in heart rate and blood pressure intraoperatively, although group 2B, which received β-blockers, did not experience an HBE. Thus, it was concluded that use of a β-blocker early in the presentation of increased heart rate and blood pressure might have prevented initiation of the BJR in group 2B. Additionally, patients who did not experience vagal episodes and did not receive β-blockers (group 2A) also received the lowest dose of epinephrine. Groups 1 and 2B received higher doses of epinephrine than did group 2A. The authors report that these findings support previous studies suggesting that epinephrine may increase the incidence of HBEs.

Another retrospective study of 150 patients was conducted to determine if there was an association between the use of β-blockers and the incidence of HBEs.\textsuperscript{26} Thirteen percent of the patients (20 of 150) experienced HBEs. Similar proportions of patients received β-blockers in the group that had HBEs compared with those who did not have HBEs. Hence, contrary to the previously mentioned study,\textsuperscript{4} there was no association found between the occurrence of the HBE and the use of β-blockers.

A randomized prospective study of 150 patients undergoing shoulder arthroscopy in the sitting position examined the effect of glycopyrrolate and metoprolol in reducing the occurrence of HBEs.\textsuperscript{11} Glycopyrrolate was administered to prevent the heightened parasympathetic tone induced by activation of the BJR. Patients were randomized into 3 groups. Group 1 was administered prophylactic metoprolol to reduce heart rate to below 60/min, or a maximum dose of 10 mg, immediately after the administration of the interscalene block. Group 2 received prophylactic glycopyrrolate to achieve a heart rate of greater than 100/min, or a maximum dose of 10 mg, immediately after the administration of the interscalene block. Group 3 was given a placebo. Metoprolol was effective in preventing the occurrence of HBEs compared with glycopyrrolate (incidence of HBEs: 28% with placebo, 5% with metoprolol, and 22% with glycopyrrolate). The authors likewise proposed that the BJR was responsible for these HBEs.

A randomized prospective study examined the effectiveness of ondansetron in preventing HBEs in patients undergoing shoulder arthroscopy in the sitting position under interscalene block.\textsuperscript{10} The administration of ondansetron, a serotonin 5-HT3 antagonist, was based on the fact that serotonin can bind to chemoreceptors in the walls of the ventricle of the heart and activate the afferent path of the BJR, while at the same time increasing any associated activity of the vagus nerve.\textsuperscript{10,25} In fact, many studies have provided evidence for the use of ondansetron in reducing hypotension associated with spinal anesthesia based on the same theory.\textsuperscript{28-30} One hundred fifty ASA class 1 or 2 patients were randomly divided into 1 of 3 groups: those receiving 4 mg of ondansetron, those receiving 8 mg of ondansetron, and those receiving saline. Each medication was administered 5 minutes before the placement of the
interscalene block. Patients receiving only saline had a significantly higher incidence of HBEs (20.4%) compared with those who received 4 mg of ondansetron (6.1%) and 8 mg of ondansetron (6%). Based on these results, the use of ondansetron before the administration of the interscalene block was recommended to prevent serotonin from stimulating the chemoreceptors in the walls of the ventricle, thus preventing activation of the BJR.

**Recommendations.** Despite a lack of a clear consensus regarding the cause of HBEs, recommendations for preventive strategies have been published based on the evidence to date and are summarized in Table 2.

Maintenance of venous return with the administration of IV fluid to restore intravascular volume depleted because of preoperative fasting, the early use of β-blockade to treat increases in heart rate intraoperatively, and the preemptive administration of an anticholinergic to block the efferent limb of the BJR have been suggested.4 Avoidance of hypoxemia and assessment for venous air embolism by continuously monitoring ventilation and oxygenation are critical. A preoperative discussion with the surgeon should be conducted about the potential need to stop the procedure and lower the table to the supine position. Epinephrine should be readily available to treat dramatic decreases in heart rate. The use of ondansetron before administration of the interscalene block has been advocated recently.10

Clear evidence regarding HBE risk factors, however, remains elusive, and there is a lack of a consensus regarding the exact cause of HBEs. Given the unpredictable nature of HBEs and the catastrophic potential these events possess, it is imperative that a clear mechanism is identified to form the foundation from which further preventive measures can be designed and implemented into practice.

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**Table 2. Prevention and Treatment of Hypotensive/Bradycardic Episodes**

Abbreviations: ACLS, advanced cardiac life support; HBE, hypotensive/bradycardic episode; and VAE, venous air embolism.

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**DISCLOSURE**

The author has declared no financial relationships with any commercial interest related to the content of this activity. The author did not discuss off-label use within the article.

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