Activation of the Bezold-Jarisch Reflex in the Sitting Position for Shoulder Arthroscopy Using Interscalene Block

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A retrospective analysis of 116 patients who underwent shoulder arthroscopy in the sitting position with interscalene block (ISB) revealed 20 patients who experienced potentially dangerous vasovagal events characterized by sudden severe hypotension and bradycardia (Group 1). The event occurred 61 ± 18 min after the block placement. Ninety-six patients (Group 2) did not experience a vasovagal event. Of the patients in Group 2, 18 received β-adrenergic blockers for increasing heart rate and/or arterial blood pressure (Group 2B) while 78 did not (Group 2A). The number receiving β-adrenergic blockers was significantly greater than in Group 1 (18/96 vs 0/20, P < 0.05). There were no significant demographic or baseline hemodynamic differences between groups, but the β-adrenergic blocker and vagal groups showed significantly greater intraoperative peak heart rates (P < 0.05). All patients received epinephrine in their local anesthetic for ISB, incision sites, and articular irrigating solution. Total and weight-corrected epinephrine doses differed significantly between groups (lowest in Group 2A, P < 0.01). Activation of the Bezold-Jarisch reflex, induced by increased circulating epinephrine levels and the sitting position, is the postulated mechanism.


Arthroscopy of the shoulder is becoming more common, particularly with patients in the sitting position (1,2), a position favored by surgeons for three reasons: 1) there is no need for unnatural traction on the shoulder which may compromise capsular repairs (2,3); 2) the procedure may be converted to open surgery without repositioning (2); and 3) the incidence of traction neuropathies is significantly decreased (2,4,5). Interscalene block (ISB) is used for arthroscopic shoulder surgery (6,7). We have noted a high incidence of vasovagal episodes associated with use of the ISB for shoulder arthroscopy in the sitting position. These episodes consist of sudden hypotension and/or bradycardia, frequently associated with symptoms of lightheadedness or nausea and, in one case, asystolic cardiac arrest requiring reuscitation. We undertook this chart review to investigate the frequency of these events and associated factors.

The purpose of this study is to: 1) alert anesthesiologists to the possibility of potentially dangerous vasovagal events when using ISB with local anesthetic and epinephrine for shoulder arthroscopy in the sitting position; 2) describe the timing and presentation of this series of events so that progression from prodromal symptoms to cardiovascular collapse may be avoided; 3) present our hypothesis as to the mechanism of this newly described problem; and 4) propose therapeutic interventions.

Methods

After approval by our institutional review board, patients who had undergone shoulder surgery from May 1990 through April 1993 were identified from operating room records. The subset of patients who had ISB for arthroscopic surgery in the sitting position was identified. The anesthesia records of this subset were reviewed for vasovagal episodes which we defined as hypotension and/or bradycardia requiring treatment by the administration of atropine, glycopyrrolate, or ephedrine. We defined hypotension as a rapid (less than 5 min) decline in systolic blood pressure to less than 100 mm Hg or a change greater than 30 mm Hg from the highest to the lowest recorded values in that same time period. Bradycardia was similarly defined, as a rapid (less than 5 min) decline in heart rate to less than 60 bpm or a change more than 30 bpm from the highest to lowest recorded values. These events were frequently associated with symptoms of nausea, sweating, restlessness, lightheadedness, or confusion, but the presence of these symptoms was not necessary.
in order to make the diagnosis. All patient charts were reviewed for ASA physical status, age, gender, height, weight, side of surgery, volume and dose of local anesthetic, dose of epinephrine in local anesthetic, and use of medications intraoperatively. Epinephrine dose was the sum of that administered in the ISB and in the incision sites with local anesthetic for hemostasis. Cardiovascular variables recorded for all patients included preanesthetic baseline, first intraoperative, and highest heart rate and systolic blood pressure. For patients who experienced vasovagal episodes, the lowest heart rate and systolic blood pressure immediately after the event were noted. For patients receiving β-adrenergic blockers, heart rate and blood pressure 10 min after the last dose of β blocker was chosen for comparison to the vagal group (lowest). Statistical analysis consisted of analysis of variance (ANOVA) for comparison of age, height, weight, total and weight-corrected dose of epinephrine, and all time intervals. χ² analysis was used to compare ASA physical status, gender, side of surgery, and intraoperative use of β blockers. Paired t-tests were performed to compare within-group changes in cardiovascular variables while ANOVA with post hoc Student-Newman-Keuls testing was used to compare the between-groups data. A P value of less than 0.05 was considered significant. All data are reported as mean ± SD.

Results

One hundred sixteen patients who had undergone ISB for arthroscopic shoulder surgery in the sitting position were identified. Group 1 (n = 20) consisted of patients who experienced a vasovagal reaction as defined above. Group 2 (n = 96) consisted of patients who did not experience such an event. Group 2 patients received β-adrenergic blockers intraoperatively (for treatment of tachycardia and/or hypertension) with significantly greater frequency than those in Group 1 (18/96 vs 0/20, P < 0.05, χ²). This asymptomatic group was then, in a post-hoc fashion, divided according to whether a patient received intraoperative β-adrenergic blockers (Group 2A, no β blockers, n = 78; Group 2B, β blocker, n = 18). Demographics for the two groups did not differ with respect to age, ASA physical status, gender, height, weight, or side of surgical procedure (Table 1). The dose (42 ± 8 mL) and choice of local anesthetic and vasoconstrictor varied and included lidocaine, mepivacaine, and bupivacaine.

Baseline heart rates for the groups did not differ (Group 1, 76 ± 12 bpm; Group 2A, 79 ± 15 bpm; Group 2B, 84 ± 10 bpm; P = 0.21) and all demonstrated an increase over time (Figure 1). Group 1 (patients who experienced vasovagal symptoms) accelerated from 76 ± 12 to 81 ± 20 to 96 ± 19 bpm (baseline, first intraoperative, highest heart rate). The highest heart rate was significantly greater than baseline (P < 0.01). The vasovagal event resulted in a precipitous drop in heart rate to 55 ± 19 (P < 0.01, highest to lowest). Group 2B, which received β-adrenergic blockers, manifested a similar, but more pronounced, increase in heart rate than Group 1 (84 ± 10 to 98 ± 13 to 110 ± 18; baseline to first intraoperative and highest, P < 0.01). Ten minutes after β blocker administration, the heart rate declined to 85 ± 13 bpm (P < 0.01, highest to lowest) and thereafter remained stable.

Table 1. Demographic Data

<table>
<thead>
<tr>
<th>Group</th>
<th>Vasovagal reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1, yes (n = 20)</td>
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<tr>
<td>Age (yr)</td>
<td>34 ± 16</td>
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<tr>
<td>ASA status</td>
<td>17 I/II</td>
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<tr>
<td>Gender (M:F)</td>
<td>3 III</td>
</tr>
<tr>
<td>Side (R:L)</td>
<td>12:8</td>
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<tr>
<td>Height (cm)</td>
<td>177 ± 11</td>
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<tr>
<td>Weight (kg)</td>
<td>74.2 ± 11</td>
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<tr>
<td>Epinephrine, total dose (µg)</td>
<td>222 ± 77</td>
</tr>
<tr>
<td>Epinephrine, weight corrected dose (µg/kg)</td>
<td>3.0 ± 1.2</td>
</tr>
<tr>
<td>β adrenergic blocker (%)</td>
<td>0</td>
</tr>
</tbody>
</table>

Data reported as mean ± sd.

*P < 0.05, χ².

Figure 1. Within and between groups comparison of heart rate at baseline, first intraoperative, highest and lowest recorded values. a) Within groups comparison: † significantly different than baseline P < 0.01; ‡ significantly different than highest P < 0.01. b) Between groups comparison: *significantly different than Group 1 or Group 2A, P < 0.01; ‡ significantly different than Group 2A P < 0.01; ƒ significantly different than Group 1 P < 0.01. c) Lowest value is that obtained immediately after onset of symptoms for Group 1 and 10 min after administration of last dose of β blockers for Group 2B.
Group 2A, those patients who experienced no symptoms and received no β blockers, demonstrated a slight but significant increase in heart rate from 80 ± 15 to 82 ± 12 to 86 ± 14 (P < 0.01 baseline to highest). Between groups comparison revealed that the first intraoperative heart rate for Group 2B (95 ± 13 bpm) was significantly greater than that of the other two groups (Group 1, 81 ± 20; Group 2A, 82 ± 12; P < 0.01). The highest recorded heart rate was significantly greater in Group 2B (110 ± 18 bpm) than Group 1 (96 ± 19 bpm; P < 0.01) which was significantly greater that of Group 2A (86 ± 14 bpm; P < 0.01). Time to development of the peak heart rate from time of block was 61 ± 18 min in Group 1 and 57 ± 18 min in Group 2B (P = 0.44).

Baseline systolic blood pressure between groups was not different (Group 1, 122 ± 13; Group 2A, 130 ± 17; Group 2B 122 ± 11 mm Hg: P > 0.05) (Figure 2). Within groups analysis revealed that Group 1 patients experienced an increase in systolic blood pressure from 122 ± 13 to 138 ± 18 to 147 ± 20 mm Hg (baseline, first intraoperative, highest). The first intraoperative and highest values recorded were both significantly greater than baseline (P < 0.01). Lowest systolic pressure during the vasovagal episode was 86 ± 27 mm Hg which was significantly less than the highest (P < 0.01). Group 2B patients once again manifest a similar but more pronounced pattern than Group 1. Systolic pressure increased from 122 ± 11 to 142 ± 18 to 160 ± 16 mm Hg (P < 0.01 baseline to first intraoperative, highest). Ten minutes after administration of β blockers, systolic blood pressure declined to 134 ± 13 mm Hg (P < 0.01, highest to lowest). Patients in Group 2A experienced a significant increase only when comparing the baseline and highest values (130 ± 17 to 135 ± 18 to 141 ± 18 mm Hg, P < 0.01). There was no difference between groups for the first intraoperative systolic pressure (P = 0.94) although Group 2B proceeded then to develop a significantly higher systolic pressure (160 ± 16 mm Hg) than either of the other two groups (Group 1, 147 ± 20 mm Hg, P < 0.05, Group 2, 140 ± 18 mm Hg, P < 0.01) at the highest value.

Total administered dose of epinephrine included that from ISB and skin incision sites injected by the surgeon into the surgical portal sites. In addition, all patients received a continuous, pressurized intravenous infusion of 0.9% saline containing 0.3 μg/mL epinephrine at approximately 100 mL/min, for which no estimate of absorption is made. Epinephrine dose proved to be significantly larger in Groups 1 (222 ± 77 μg; 3.0 ± 1.2 μg/kg) and 2B (228 ± 72 μg; 3.1 ± 1.3 μg/kg) than Group 2A (173 ± 79 μg; 2.5 ± 1.1 μg/kg) P < 0.005. The most commonly administered drug in Group 2B was labetalol (13 patients average 18 mg), followed by metoprolol (4 patients, average 3 mg), and 1 patient received 30 mg esmolol.

Time of onset of symptoms from ISB placement was 60 ± 18 min (range 45–110 min), from attainment of the beach-chair position to symptoms 33 ± 15 min (range 20–90 min) and from start of surgery to symptoms 17 ± 15 min (10–75 min). Prodromal symptoms consisted of nausea (n = 8), sweating (n = 7), lightheadedness (n = 3), anxiety (n = 3), or no symptom (n = 4); several patients experienced more than one symptom. Therapy consisted of atropine (n = 11), glycopyrrolate (n = 5), and/or ephedrine (n = 9). Several patients received multiple therapeutic interventions.

Discussion

Complications of both the sitting position and ISB are well described (8–10). The combination of the sitting position, regional anesthetic technique, an awake patient, and surgical procedure may result in another side effect, vasovagal episodes, which if anticipated, are of minor significance, but which in the extreme may lead to cardiac arrest. There are many possible causes of the observed symptomatology. Intravenous uptake of local anesthetic can be considered, but time from block placement to onset of symptoms was 61 ± 18 min. This is significantly longer than that which
would be expected for peak local anesthetic uptake from an ISB which is approximately 30 min for bupivacaine (11,12) and less than 20 min for lidocaine without epinephrine (13). Rach et al. (14) using mepivacaine 10 mg/kg and Tetzlaff et al. (15) using the same dose of alkalinized mepivacaine for ISB reported no episodes of toxicity. The central nervous system symptoms of lightheadedness and nausea were unlike those of local anesthetic toxicity and promptly resolved after the administration of atropine, glycopyrrolate, or ephedrine. Extensive spread of local anesthetic to the epidural or subarachnoid space may cause cardiovascular compromise but is rare. None of the patients experienced bilateral Horner’s syndrome, contralateral arm weakness, or apnea (16).

The most likely cause of the observed events in those having ISB for shoulder arthroscopy in the sitting position is a form of vasovagal syncope mediated by the Bezold-Jarisch reflex. Neurologists and cardiologists use the tilt table diagnostically, with low-dose isoproterenol infusions to incite syncopal episodes in susceptible individuals. These patients experience hypotension, preceding profound bradycardia, and syncope due to changes in heart rate (17). The mechanism of the Bezold-Jarisch reflex is thought to be venous blood pooling (induced by the sitting position) and a heightened cardiac contractile state (induced by the β-adrenergic effects of epinephrine or isoproterenol) which result in reflex arterial vasodilation (mediated by activation of the parasympathetic nervous system) and a subsequent vagally mediated bradycardia (17,18). Patients with a positive result have an average 31 ± 15 mm Hg decline in mean arterial blood pressure and a decrease in heart rate of approximately 24 bpm from baseline (17). The onset of hemodynamic changes is usually 12–24 min but may be as long as 1 h after being tilted to 60° upright (19,20).

Our patients experienced a similar sequence of events to those undergoing tilt-table testing. The sitting position, which places the torso nearly vertical with the legs dependent, was attained 33 ± 15 min before the onset of symptoms. During the vasovagal event, they experienced a mean systolic blood pressure decrease of 36 mm Hg and a heart rate decrease of 21 bpm from baseline. Epinephrine at a low dose has primarily β-adrenergic agonist activity, similar to isoproterenol (21,22). Blood levels of epinephrine from the ISB, combined with the local infiltration at the portals sites by the surgeon, may have yielded sufficient catecholamine levels to incite the Bezold-Jarisch reflex when associated with placement of the patient in the sitting position. Kennedy et al (23) studied cardiovascular effects of increasing doses of epinephrine associated with supraclavicular block anesthesia. They noted a highly significant trend toward increasing cardiac rate, output, and stroke volume, while mean arterial pressure and total peripheral resistance decreased. These effects were linearly correlated with the dose of epinephrine from 25 to 400 µg. Duration of hemodynamic changes were 90 min for cardiac and 120 min for the peripheral effects. Endogenous catecholamines may have also played a critical role in our awake patient population.

Group 2 patients (no vasovagal event) were significantly more likely to have received a β-adrenergic blocker during the early phases of tachycardia and increasing systolic blood pressure (18/96 vs 0/20, P < 0.05, χ²). Group 2B patients, the subset who received β blocker, manifest cardiovascular alterations similar in timing and degree to Group 1 yet did not progress to vasovagal symptoms. Administration of β-adrenergic blockers in this group, as suggested by multiple authors (17,18,24), may have interrupted the early stages of the reflex.

Our observations are similar to those of Roch and Sharrock (25) and confirm the presence of this complication. They noted a 24% incidence of sudden hypotension and bradycardia in shoulder arthroscopy patients in the sitting position using ISB anesthesia (25). Time of onset of symptoms was 69 ± 28 min after the block compared with our 61 ± 18 min. They noted a systolic blood pressure and heart rate decline of 51 mm Hg and 16 bpm, respectively, during the event and prodromal symptoms were similar. Administration of preprevent β blockers was not addressed, nor did they speculate as to the cause of their findings. Additionally, Masuda and Fujiki (26) have induced sinus arrest and syncope after right stellate ganglion block and table tilt to 60°. They also attribute this to a vasovagal cardioinhibitory response. Rogers et al. (27) demonstrated a significantly slowed heart rate after right stellate ganglion block (86 ± 5 to 72 ± 4 bpm, P < 0.01) but not so after left-sided block. We, however, did not find a predominance of right-side blocks in the vasovagal group (P = 0.422, χ²).

Our recommendations for prophylaxis of vasovagal events in the sitting position are: 1) administration of adequate intravenous fluids to reestablish circulating blood volume after fasting; 2) administration of a β-adrenergic blocker early in the phase of increasing heart rate prior to initiation of the Bezold-Jarisch reflex (17); or 3) prophylactic administration of an anticholinergic to block the vagally mediated efferent limb of the reflex (18).

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References