New Discharge Criteria Decrease Recovery Room Time after Subarachnoid Block

Christian M. Alexander, M.D.,* Lynn E. Teller, M.D.,* Jeffrey B. Gross, M.D.,† Denise Owen, B.S.N.,‡
Cornelia Cunningham, B.S.N.,‡ Flory Laurencio, R.N.‡

The authors completed a two-phase study to determine criteria that might predict hemodynamic stability during recovery from subarachnoid block (SAB). Patients' supine and sitting (2 min) blood pressures were determined at 30-min intervals in the recovery room (RR). In the first group of 26 patients, retrospective analysis revealed that the orthostatic decrease in mean arterial pressure (MAP) never exceeded 15% following two successive orthostatic decreases of 10% or less. This finding was validated prospectively in a second group of 26 patients. Following two successive orthostatic MAP decreases of 10% or less, none of 65 orthostatic challenges resulted in an MAP decrease of more than 15%; conversely, in the absence of two successive MAP decreases of less than 10%, 5 of 51 orthostatic challenges resulted in an MAP decrease of greater than 15% ($P < 0.02$). Had patients been discharged from the RR based on two successive MAP decreases of less than 10%, 35 of 52 patients could have been discharged from the RR 76 ± 6 min (mean ± SE) sooner than they would have under usual empirical discharge criteria of supine hemodynamic stability, regression of sensory level to T10, and return of toe movement. Following SAB, hemodynamic stability may return before sensory and motor function; for many patients, orthostatic testing following SAB may safely decrease the amount of time spent in the RR. (Key words: Anesthetic techniques: spinal, blood pressure changes. Recovery: discharge criteria; spinal anesthesia.)

SUBARACHNOID BLOCK (SAB) frequently causes hypotension as a result of sympathetic blockade and decreased cardiac output.[1] This autonomic instability may continue into the postoperative period,[2] and the hemodynamic shifts associated with transfer to the recovery room (RR) may cause significant hypotension in some patients. One of the primary reasons for observing patients in the RR after SAB is to ensure that they will be hemodynamically stable when they return to the less closely monitored setting of the surgical ward. The most conservative practice is to observe patients in the RR until the effects of the SAB have completely dissipated.[3] However, this may require patients to remain in the RR for many hours after surgery is complete.

To avoid inordinately long RR stays, our practice has been to use less stringent, empirical criteria for discharging patients from the RR after SAB: regression of sensory level to T10 and return of motor function to the lower extremity (toe movement). Our implicit assumption is that these indicators of recovery correlate with the return of autonomic function. Unfortunately, there are no data indicating whether this assumption is valid, or whether different criteria could decrease the length of RR stays without compromising patient safety. We designed the present study to quantitate the relationship between sensory and motor recovery and the return of hemodynamic stability. Our goal was to define criteria that would predict subsequent hemodynamic stability, thereby minimizing RR time following SAB.

Methods

Fifty-two consecutive patients receiving SAB for general, urologic, or orthopedic surgical procedures consented to participate in our Institutional Review Board-approved study. Patients with a history of cerebrovascular disease or evidence of myocardial ischemia were excluded. With patients sitting, plain 0.5% tetracaine was injected into the subarachnoid space (8–16 mg, at the discretion of the attending anesthesiologist) and we immediately returned patients to the supine position. An investigator recorded the time of drug injection and the highest level of sensory anesthesia that developed. Patients' operating room management did not differ from the care they would have received had they not been participating in this study. They received iv sedation as needed with combinations of midazolam and fentanyl; iv fluid replacement was based on clinical signs. Hypotension (systolic blood pressure < 100 mmHg) was treated with ephedrine (5 mg) or phenylephrine (100 μg) as well as by increasing the rate of iv fluid administration.

When patients arrived in the RR and every 30 min thereafter, the following variables were measured or noted: 1) level of sensory anesthesia to pinprick, 2) presence of observable toe movement, and 3) heart rate and systolic and diastolic blood pressure (Dinamap). After helping patients to sit with their legs over the side of the stretcher for 2 min, we remeasured their blood pressures and heart rates. If patients complained of dizziness or chest pain while sitting, they were immediately returned

TABLE 1. Combined Data for Retrospective and Prospective Patient Groups (mean ± SE)

	Patients with One or More Episodes of MAP Decreases of >10% in RR	Patients Having No Episodes of MAP Decreases of >10% in RR	All Patients
N	19	33	52
Age (yr)	66 ± 2	65 ± 2	65 ± 1
Dose (mg)	10.3 ± 0.3	11.3 ± 0.4	10.9 ± 0.3
Peak sensory level in OR (thoracic dermatome)	5.0 ± 0.4	5.1 ± 0.3	5.0 ± 0.2
Time from injection of anesthetic until arrival in RR (min)	114 ± 10	97 ± 6	103 ± 5
Time from injection of anesthetic until toe movement (min)	224 ± 16	220 ± 9	221 ± 9
Time from arrival in RR until toe movement (min)	116 ± 13	123 ± 9	120 ± 8
Sensory level on arrival in RR (thoracic dermatome)	5.4 ± 0.4	6.1 ± 0.4	5.8 ± 0.3
Sensory level at time of toe movement (thoracic dermatome)	11.3 ± 0.1	11.3 ± 0.1	11.3 ± 0.1
Intraoperative fluids (ml)	742 ± 57*	939 ± 58	867 ± 44

* $P < 0.05$ compared with patients not developing orthastatic hypotension.

to the supine position. These assessments continued at 30-min intervals until patients were able to move their toes, at which time they were discharged from the RR. For each pair of blood pressure determinations, MAP was calculated (MAP = ⅓ × systolic + ⅔ × diastolic) and was used to determine the percentage change in MAP that occurred when patients went from the supine to sitting position.

After studying our first group of 26 patients, retrospective analysis of the data determined the degree of hemodynamic stability associated with our usual, empirical discharge criteria, i.e., toe movement and regression of sensory level to T10 and criteria that could predict in advance which patients would subsequently be hemodynamically stable and therefore could be discharged safely from the RR before complete regression of the block had occurred. It appeared that patients whose MAP was stable during two successive orthostatic challenges (<10% decrease in MAP, v.i.) never manifested an MAP decrease of >15% in subsequent tests. Because this was a retrospective generalization, we sought, therefore, to independently validate it by prospectively studying a second group of 26 patients.

Patients in the second group were not discharged immediately after they demonstrated hemodynamic stability with two successive negative orthostatic challenges. Rather, they remained in the RR until their sensory levels regressed to T10 and toe movement returned. During this time orthostatic testing continued at 30-min intervals, allowing us to determine the incidence of orthostatic instability developing between the time when patients first proved to be hemodynamically stable and the time when they would routinely be discharged.

Continuous variables were evaluated using analysis of variance. For contingency tables, we used Fisher's exact test or chi-square analysis as appropriate. $P < 0.05$ indicated significance.

Results

Data from our first group of 26 patients showed that at the time of discharge from the RR based on our usual criteria (hemodynamic stability in the supine position with toe movement present), two patients had orthostatic MAP decreases of 10–15%. Patients meeting these routine criteria have not developed postdischarge complications in our experience. Therefore, it appeared that patients could be safely discharged from the RR if one could predict that subsequent orthostatic blood pressure changes would not exceed 15%. Based on a retrospective analysis of our data, we observed that once hemodynamic stability had returned, as demonstrated by less than 10% decrease in MAP on two successive orthostatic challenges, subsequent stability was ensured: patients never developed blood pressure decreases of >15%. Interestingly, there was no correlation between the level of sensory block and the degree of concurrent orthostatic blood pressure change.

Analysis of data from our second group of patients, who were studied prospectively, revealed that orthostatic MAP decreases of >15% did not occur once patients fulfilled our hypothesized criterion: less than 10% decrease in MAP during two successive orthostatic challenges. Of the 65 orthostatic challenges performed in patients who fulfilled this criterion, none resulted in an MAP decrease of >15%. In contrast, five of 51 orthostatic trials in patients who had not fulfilled this requirement resulted in

MAP decreases of >15% ($P < 0.02$). Three of the second 26 patients never had two successive orthostatic blood pressure decreases of <10%. Their orthostatic MAP decreases were 11%, 13%, and 23% at discharge, when their sensory levels had regressed to T10 and toe movement had returned.

Of the 52 patients we studied, 35 completed two successive orthostatic challenges with <10% decrease in MAP 76 ± 6 min (mean ± SE, $P < 0.001$) before toe movement. Had these patients been discharged from the RR upon fulfilling the orthostatic criterion, this would have resulted in a significant saving of RR time. When all patients, including those in whom orthostatic testing would not have decreased RR time, were included in the calculation, the average saving of RR time was 51 ± 7 min ($P < 0.001$).

Continuous data from our two groups of patients are presented in table 1. Because these did not differ between groups, the following represent pooled data. Patients were between 35 and 90 yr of age (65 ± 1), received 10.9 ± 0.3 mg of tetracaine, and developed peak sensory levels of T5.0 ± 0.2; time from of injection of spinal anesthetic until toe movement was 221 ± 9 min. There was no correlation between orthostatic decrease in MAP (ΔP) and the concurrent level of sensory anesthesia. One patient complained of light-headedness during an orthostatic test; his sensory level was T4 and the MAP decreased from 78 to 46 mmHg (a 43% decrease). This resolved when he returned to the supine position and did not recur during subsequent tests.

In all, 19 of 52 individuals had one or more orthostatic MAP decreases of >10%; there were 30 such episodes, with a mean decrease of 16.0 ± 1.2%. These occurred at 167 ± 11 min after injection of spinal anesthetic, when pinprick sensory levels were T8.5 ± 0.5. There was no difference in age, tetracaine dose, sensory level upon arrival in the RR, time to return of toe movement, or RR time between the individuals who developed orthostatic decreases of >10% on one or more determinations in the RR and those who did not. Although the time from induction of anesthesia until the end of surgery was the same (OR time 103 ± 5 min), patients who demonstrated orthostatic MAP decreases of 10% or more in RR received less intraoperative iv fluid volume (742 ± 6 ml) than those whose blood pressures were stable during orthostatic testing (939 ± 6 ml, $P < 0.05$). Eighteen of 52 patients received ephedrine or phenylephrine in the operating room; there was no relationship between the need for intraoperative vasopressors and development of orthostatic blood pressure decreases in the RR.

Discussion

The goal of this investigation was to define RR discharge criteria that would safely allow patients to return to the surgical ward as soon as possible following SAB. There were no published data to indicate whether empirical discharge criteria requiring regression of sensory level to T10 and return of motor function to the lower extremities (toe movement) adequately predict hemodynamic stability or prolong RR time unnecessarily. Because 4% of our patients (two of 52) who fulfilled these criteria had subsequent orthostatic blood pressure decreases of 15% or greater, it appears that the empirical criteria do not predict absolute hemodynamic stability. Fortunately, in our experience, patients who have been discharged based on these clinical signs of regression of SAB do not develop postdischarge hemodynamic complications. This may be related to the fact that orthostatic testing represents a greater autonomic stress than those to which these patients are exposed during and after transfer to the surgical ward.

In patients recovering from SAB, MAP did not subsequently decrease by more than 15% provided there had been no more than a 10% orthostatic decrease in two successive determinations 30 min apart. This implies that it is safe, from a hemodynamic standpoint, to discharge patients from the RR after they meet this orthostatic criterion even if the level of sensory anesthesia is above T10 and motor function has not yet returned to the toes. Orthostatic testing appears to be at least as sensitive to hemodynamic instability as other, empirical discharge criteria (v.s.). In addition, it more closely approximates stresses such as those occurring during transfer by stretcher or transport in the elevator that patients routinely encounter en route to their rooms.

Routine discharge of patients from the RR based on the results of orthostatic blood pressure testing increases RR efficiency by redistributing utilization toward those patients who need intensive monitoring. Thirty-three of our 52 patients could have been safely discharged before toe movement occurred, representing a significant saving of RR time. Conversely, orthostatic testing identified two patients whose MAP decreased by >15% when discharge based on regression of sensory level and toe movement would otherwise have occurred. To increase patient safety, patients who continue to show orthostatic changes may need to remain in the RR until the level of SAB regresses further or until another underlying problem such as a fluid deficit has been corrected.

Any new discharge criterion must be applied judiciously. Patients who require observation for surgical or medical indications such as continued bleeding or ischemic ECG changes should not be discharged until those problems are resolved. RR personnel should verify that the level of sensory block is stable or receding. Also, to ensure that the sensory level of anesthesia is receding normally (and quickly diagnose the rare occurrence of an epidural hematoma), a member of the anesthesia care team should

confirm that patients have full return of motor and sensory function within a few hours after discharge from the RR. As in any patient recovering from spinal anesthesia, patients discharged on the basis of these orthostatic criteria must be cautioned not to attempt unassisted ambulation until full return of lower extremity sensory and motor function has been documented by the ward nurse.

The intraoperative courses of our 52 patients did not predict which patients would demonstrate a postoperative orthostatic blood pressure change of >10%. The occurrence of intraoperative hypotension requiring treatment with vasopressors or fluids did not predict orthostatic hypotension in the RR. The OR times for patients who did or did not manifest orthostatic changes were not significantly different, ruling out more recent injection of tetracaine as a cause of orthostatic change. However, patients who developed orthostatic hypotension received somewhat smaller volumes of intraoperative fluids than patients who were hemodynamically stable. Although the difference was small (<200 ml), differences in fluid replacement may have affected orthostatic blood pressure change.

Our observation that the magnitude of orthostatic BP change was independent of the level of sensory anesthesia may seem surprising. However, Roe and Cohn[4] and Kim et al.[5] demonstrated that autonomic function returns before motor or sensory function after SAB. Furthermore, Skagen et al.[6] found that sympathetic blockade by epidural anesthesia did not affect the reflex vasoconstriction of subcutaneous tissue and skeletal muscle in the lower extremities induced by a 45° head-up tilt. Their data[6] suggest that local mechanisms are responsible for vasoconstriction during head-up tilt in the presence of central sympathetic blockade. Therefore, the presence of orthostatic hemodynamic stability in the presence of relatively high levels of sensory and motor blockade could have resulted either from early return of sympathetic function or from local vasomotor factors.

In summary, we have demonstrated that patients may be discharged safely from the RR after SAB if they demonstrate <10% decrease in MAP in response to two orthostatic tests 30 min apart. Our previous empirical discharge criteria (regression of the level of sensory block to T10 and toe movement) do not guarantee hemodynamic stability and were associated with unnecessarily long RR stays in 35 of the 52 subjects. Routine use of our orthostatic discharge criteria for patients not requiring intensive monitoring for continuing medical or surgical indications would result in a significant saving of RR time.

References

1. Ward RJ, Bonica JJ, Freund FG, Akamatsu T, Danziger F, Englesson S: Epidural and spinal anesthesia: Cardiovascular and respiratory effects. JAMA 191:275–278, 1965
2. Greene NM: Physiology of Spinal Anesthesia, 3rd edition. Baltimore, Williams & Wilkins, 1981, p 125
3. Lund PC: Principles and Practice of Spinal Anesthesia. Springfield, Charles C Thomas, 1971, p 617
4. Roe CF, Cohn FL: Sympathetic blockade during spinal anesthesia. Surg Gynecol Obstet 136:265–268, 1973
5. Kim JM, LaSalle AD, Parmley RT: Sympathetic recovery following lumbar epidural and spinal anesthesia. Anesth Analg 56:352–355, 1977
6. Skagen K, Haxholdt O, Henriksen O, Dyrberg V: Effect of spinal sympathetic blockade upon postural changes of blood flow in human peripheral tissues. Acta Physiol Scand 114:165–170, 1982