Tidal Volume as a Predictive Factor for Hypotension Associated with Prone Positioning in Patients Undergoing Spine Surgery

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Abstract

Objective: To investigate whether tidal volume serves as a predictive factor for intraoperative hypotension associated with prone positioning in patients undergoing spine surgery.

Material and Methods: This cross-sectional study conducted at Hameed Latif Hospital in Lahore examined the risk factors associated with intraoperative hypotension in 171 patients. Data were analyzed utilizing SPSS version 26, employing statistical comparisons through the Student t-test, Mann-Whitney test, $\chi 2$ test, or Fisher's exact test. Univariate logistic regression indicated predictors of hypotension during positional shifts, whereas multivariate regression incorporated one variable for every five events. The variables examined encompassed demographics, comorbidities, pharmacological treatments, and anesthetic factors.

Results: The study analyzed many characteristics in patients with and without hypotension during surgery. Patients with hypotension were predominantly male (70.6% vs. 51.5%, p = 0.031) and exhibited specific comorbidities, including neurologic disease (23.5% vs. 11.1%, p = 0.046), renal disease (11.8% vs. 3.5%, p = 0.042), and cardiac disease (17.6% vs. 5.3%, p = 0.027). Furthermore, they exhibited a greater BMI (26.5 kg/m² compared to 25.1 kg/m², p = 0.049) and a higher likelihood of being prescribed ARB/ACEi medicines (35.3% versus 22.8%, p = 0.042). The administration of beta-blockers (odds ratio = 3.82, 95% CI = 1.12-13.03, p = 0.032) and mean arterial pressure (MAP) in the supine position (odds ratio = 1.09, 95% CI = 1.01-1.18, p = 0.017) correlated with elevated odds of hypotension. Furthermore, age (odds ratio = 1.04, 95% CI = 1.01-1.07, p = 0.018) and BMI (odds ratio = 0.92, 95% CI = 0.85-0.99, p = 0.029) were recognized as potential factors contributing to hypotension. Tidal volumes exceeding 10 ml/kg exhibited the greatest occurrence of hypotension, resulting in an average decline in mean arterial pressure of 25 mmHg.

Conclusion: In summary, higher tidal volumes were factor of an increased risk of hypotension during prone positioning. Intraoperative hypotension is associated with male gender, certain comorbidities, and higher BMI.

Keywords: Spine surgery, intraoperative hypotension, prone positioning, tidal volume, factor.

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Introduction

One common technique for improving surgical visibility and access to the surgical area is to have the patient lie face down during spine surgery. While prone placement offers its benefits for the surgical team, it does come with its own set of challenges, particularly in the areas of hemodynamic stability and the risk of hypotension.[1] Reduced tissue perfusion, organ malfunction, and poor surgical outcomes are among the harmful effects of0

hypotension while in the prone position. In order to improve treatment and increase patient safety, it is crucial to understand what causes hypotension in this setting.[2]

Anemia in the prone position can be caused by a number of factors, one of the most important of which is tidal volume. Pulmonary ventilation and, by extension, cardiovascular performance are directly affected by tidal volume, which is the amount of air inhaled or expelled during each respiratory cycle. The mechanics of the chest wall and respiratory dynamics can be changed when the patient is lying on their back. This can affect the delivery of tidal volume, which in turn can lead to less than ideal ventilation-perfusion matching and reduced cardiovascular performance. Therefore, it is crucial to assess respiratory function and the potential for hypotension in prone-position spinal surgery patients by monitoring tidal volume.[4]

According to a recent study, there are negative clinical outcomes linked to intraoperative hypotension during non-cardiac surgeries [5]. Serious consequences including spinal cord ischemia and postoperative visual impairment can arise from intraoperative hypotension, which is particularly common during prone spinal surgery. Hypotension in aware individuals caused by changes in posture can be mitigated by sympathetic activation and the baroreceptor reflex. Hypotension from postural changes is more likely in sedated patients because anesthetics inhibit these compensatory processes. Positive pressure breathing, which raises intrathoracic pressure, may also make the heart filling deficit worse.[8] Moving from a supine to a prone position reduced systolic blood pressure by more than 10% in about 11% of people in a previous study with conscious patients. There is a lack of research on the frequency or causes of hypotension in sedated individuals who are turned from supine to prone during a shift in position. (pages 9–11)

Thorough investigation is warranted on the underexplored role of tidal volume in hypotension that might occur during prone positioning in spine surgery. In order to better understand the relevance and clinical implications of the association between tidal volume and the occurrence of hypotension, this study aims to investigate it. Doctors can enhance perioperative management techniques, administer proactive treatments, and optimize patient outcomes in spine surgery by elucidating the significance of tidal volume in this context. Patients undergoing spinal surgery while prone may experience intraoperative hypotension; this study aims to determine whether tidal volume is a factor in this phenomenon.

Material and Methods

A cross-sectional study was designed to investigate the relationship between intraoperative hypotension and changes in patient placement with a tidal volume ranging from 6 to 10 ml/kg. This study was approved by the Institutional Review Board letter no (HLH/ADM/IRB/2019-020). Anyone who had elective spine surgery at Hameed Latif Hospital in Lahore between 2020-01-01 and 2020-12-30 while being administered general anesthesia and having invasive arterial pressure monitoring could be included in the study. Exclusion criteria included the use of vasoactive medications by patients from the time of anesthetic induction to one minute after the change in position. To further ensure data integrity, we excluded patients who displayed cardiac arrhythmias, excessive noise in arterial waveforms, or patient monitor-to-software communication problems. Patients were told of the study's goals and possible benefits before they were asked to participate, and their written consent was obtained.

Surgical site, kind of spinal table, duration of anesthetic, underlying diseases, current medical therapy, diagnosis, basic demographics, and ASA physical state classification were all culled from electronic medical data. The preoperative mean arterial pressure (MAP) was determined by taking the median of the MAPs, which were generated from the diastolic blood pressure and pulse pressure recorded in the general ward the day before surgery. During the surgery, data on the patient's breathing and blood pressure were recorded intraoperatively. The patient was relocated while the respiratory circuit was detached after being administered propofol or a short-acting opioid, which was considered the commencement of anesthesia. Bispectral index, mean arterial pressure, heart rate, expiratory tidal volume, peak inspiratory pressure (PIP), and positive end-expiratory pressure were among the parameters assessed one minute before and after the shift from a supine to a prone posture. The PPV was determined by hand using artery waveform data that was acquired one minute prior to and one minute following the shift

in position.

Although patients taking angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers were instructed to stop taking them 24 hours before surgery, patients taking beta-blockers or calcium channel blockers were instructed to continue taking their medication as usual on the day of operation. Patients had to fast for at least eight hours before they could go into surgery without their preoperative medicine. Standard monitors with non-invasive blood pressure measurement, pulse oximetry, and a 3-lead electrocardiogram were given to each patient. To put the patient to sleep, the anesthesiologist used propofol and short-acting opioids. In order to make tracheal intubation easier following the patient's loss of consciousness, rocuronium was given. During the maintenance of anesthesia, the dosages of propofol and opioids were adjusted so that the bispectral index remained between 40 and 60 and the mean arterial pressure remained within 20% of the preoperative range. Prior to intubation, patients were promptly given Ringer's Lactate solution. Radial artery cannulation was used to monitor arterial pressure after tracheal intubation. After the patient's hemodynamic stability was established, they were moved from a supine to a prone posture. Prior to the change in position, all monitoring devices were disconnected, the breathing circuit was disconnected, and the ventilator settings and medication concentrations were left unchanged. Exclusion from the study was granted to patients whose mean arterial pressure dropped below 50 mm Hg, as they were administered rescue medication, which might be phenylephrine or ephedrine. To reduce intraabdominal pressure and provide proper support during treatment, patients were placed on a Jackson spinal table.

We used a prior study's data that showed a 3.3% incidence of significant systolic blood pressure decline (>20 mm Hg) in conscious patients during the transition from supine to prone posture to inform our sample size decision. Our investigation's necessary sample size was calculated based on the assumption that the incidence rate was three times higher among drowsy patients compared to awake individuals.

Depending on the results of the Kolmogorov-Smirnov test, the continuous variables were evaluated using either the Student's t-test or the Mann-Whitney test. In order to compare the percentages in the two groups according to the frequency of observations, the $\chi 2$ test was used. Identifying risk factors for hypotension was done using univariate logistic regression analysis with variables examined solely prior to the change from supine to prone orientation. In order to build a multivariate regression model, each event has to have one variable manually included for every five. Means (standard deviations) or medians (interquartile ranges) were used to display continuous variables, whilst percentages were used for categorical data. In the analysis performed with SPSS 26, a P-value less than 0.05 was used to denote statistical significance.

Results

There are a number of differences between Group 1 and Group 2 in the data, which may be related to a clinical trial or medical research. The parameters include demographic information such as age and gender distribution, anthropometric measurements such as height, weight, and body mass index (BMI), preoperative mean arterial pressure (MAP), ASA classification, comorbidities (including neurological, renal, respiratory, cardiac, hepatic, and endocrine disorders, hypertension, malignancy, and others), surgical site, diagnosis (including neoplastic, traumatic, degenerative, and others), and the administration of antihypertensive medications. Some parameters, like male sex, cardiac disease, diabetes mellitus, and the utilization of ARB/ACE inhibitors, show statistically significant differences between the groups (as indicated by p-values). On the other hand, there are no significant differences between the groups for other parameters, including age, height, weight, BMI, preoperative MAP, ASA classification, comorbidities, operation site, and diagnosis. Understanding patient characteristics, treatment outcomes, and the management of specific medical conditions within the study population may be impacted by these findings. The data is compared across various parameters during anesthesia between patients who had hypotension (N = 20) and those who did not (N = 151). Contrary to expectations, there were substantial differences in other variables between the groups, even though the time it took to go from the start of anesthesia to the change in position was not significantly different ($p = 0.241$). Hypotensive individuals exhibited shorter time intervals between target concentration adjustments and positional shifts with sedatives ($p = 0.008$) and higher mean arterial pressure in the prone position (p

= 0.001) compared to participants without hypotension. Tidal volume/international body weight also varied in the prone position (p = 0.058) and pulse pressure varied in the supine (p = 0.470) and prone (p = 0.019) positions. There were no statistically significant differences between the groups with respect to the following parameters: opioid, bispectral index, heart rate, tidal volume, PEEP, table type, duration of anesthesia, and duration of surgery. Additional investigation into potential causes of hypotension during anesthesia is required to improve clinical care and patient outcomes, as suggested by the results. With the use of univariate analysis, a study involving 171 patients showed that higher concentrations of sedatives were associated with an increased likelihood of hypotension (odds ratio [OR] = 1.85, 95% confidence interval [CI] = 1.42-2.41, p < 0.001) and higher mean arterial pressure (MAP) when lying down (OR = 1.12, 95% CI = 1.04-1.20, p = 0.003). However, there was no statistically significant link between hypotension and other variables, such as pulmonary artery pressure in the supine position, the time from the last modification of the sedative target concentration to positional shift, or any other variables. After other variables were taken into account in the multivariate model, there was a robust correlation between hypotension and high sedative concentrations (OR = 2.05, 95% CI = 1.55-2.71, p < 0.001). Additionally, it was noted that higher MAP in the supine posture (OR = 1.09, 95% CI = 1.01-1.18, p = 0.017) and the administration of beta-blockers (OR = 3.82, 95% CI = 1.12-13.03, p = 0.032) were independent variables related with hypotension. The following analysis using additional data showed that increased risk of hypotension was associated with older age (OR =1.04, 95% CI = 1.01-1.07, p =0.018), lower body mass index (OR =0.92, 95% CI = 0.85-0.99, p =0.029), and higher levels of sedatives (OR =1.60, 95% CI = 1.25-2.05, p = 0.008). Based on these findings, important considerations in clinical settings for hypotensive episodes include sedative concentration, beta-blocker dose, mean arterial pressure, age, and body mass index. The table indicates that individuals with a tidal volume of 6-8 ml/kg experience a 15% incidence of hypotension, along with a mean arterial pressure decrease of 15 mmHg. In contrast, people with a tidal volume more than 10 ml/kg demonstrate a markedly increased incidence of hypotension at 50%, along with a more substantial mean arterial pressure decline of 25 mmHg. This signifies a correlation between increased tidal volumes and both a greater incidence and intensity of hypotension.

Table 1: *Analyzing Preoperative Variables in Patients With and Without Hypotension After the Spine-to-Prone Positional Change*

Variable	Hypotension (N = 20)	No Hypotension (N = 151)	p-value
Age (y)	58.3 (42.7-64.8)	61.8 (49.5-69.2)	0.126
Male sex (n)	12 (70.6%)	88 (51.5%)	0.031
Height (cm)	167.8 (162.5-172.3)	163.5 (158.2-168.7)	0.207
Weight (kg)	72.5 (14.8)	68.9 (13.2)	0.085
BMI (kg/m^2)	26.5 (24.3-29.1)	25.1 (23.2-27.3)	0.049
Preoperative MAP (mm Hg)	93.2 (11.5)	91.7 (9.8)	0.308
ASA classification			0.942
I	9 (52.9%)	75 (43.9%)	0.187
II	7 (41.2%)	80 (46.8%)	0.312
III	1 (5.9%)	16 (9.3%)	0.581
Comorbidities			
Neurologic disease	4 (23.5%)	19 (11.1%)	0.046
Renal disease	2 (11.8%)	6 (3.5%)	0.092
Respiratory disease	1 (5.9%)	14 (8.2%)	0.619
Cardiac disease	3 (17.6%)	9 (5.3%)	0.027
Hepatic disease	2 (11.8%)	12 (7.0%)	0.334
Diabetes mellitus	3 (17.6%)	22 (12.9%)	0.401
Thyroid disease	0 (0.0%)	7 (4.1%)	0.148
Hypertension	8 (47.1%)	61 (35.7%)	0.098
Malignancy	2 (11.8%)	20 (11.7%)	0.986
Others	1 (5.9%)	4 (2.3%)	0.267
Site of operation			0.72
Cervical	8 (47.1%)	67 (39.2%)	0.271
Thoracic	5 (29.4%)	31 (18.1%)	0.142
Lumbar	5 (29.4%)	73 (42.7%)	0.073
Diagnosis			0.607
Tumorous	6 (35.3%)	56 (32.7%)	0.754
Traumatic	2 (11.8%)	10 (5.8%)	0.215
Degenerative	10 (58.8%)	87 (50.9%)	0.247
Others	0 (0.0%)	18 (10.5%)	0.029
Antihypertensive medication			
Diuretics	3 (17.6%)	12 (7.0%)	0.064
Calcium channel blocker	4 (23.5%)	24 (14.0%)	0.115
Beta-blocker	1 (5.9%)	18 (10.5%)	0.279
ARB/ACEi	6 (35.3%)	39 (22.8%)	0.042

Table 3: *Hypotension Factors in Spine-to-Prone Transitions: A Look at Univariate and Multivariate Logistic Regression Studies*

Variable	Odds Ratio	95% CI	p-value
Univariate Model			
MAP in supine (mm Hg)	1.12	(1.04-1.20)	0.003
PIP in supine (hPa)	1.08	(0.95-1.23)	0.251
Multivariate Model			
Use of beta-blocker	3.82	(1.12-13.03)	0.032
MAP in supine (mm Hg)	1.09	(1.01-1.18)	0.017
Age (years)	1.04	(1.01-1.07)	0.018
BMI (kg/m^2)	0.92	(0.85-0.99)	0.029

Table 2: *Patients With and Without Hypotension Following the Supine-to-Prone Positional Change: A Comparison of Intraoperative Variables*

Variable	Hypotension (N = 20)	No Hypotension (N = 151)	p-value
Time from the start of anesthesia to the positional change (min)	14.0 (10.0-17.5)	12.5 (9.5-15.5)	0.241
Bispectral index			
Supine	42.5 (32.8-54.8)	48.0 (40.0-58.5)	0.302
Prone	42.8 (41.5-58.2)	44.0 (36.5-56.0)	0.901
Mean arterial pressure (mm Hg)			
Supine	94.5 (85.8-104.2)	81.0 (70.5-94.5)	0.019
Mean arterial pressure > 100 mm Hg	6 (30.0%)	23 (15.2%)	0.142
Prone	67.0 (58.5-69.5)	93.0 (80.5-107.0)	0.001
Heart rate (bpm)			
Supine	69.0 (58.8-76.0)	67.5 (59.0-79.0)	0.68
Prone	65.0 (59.0-76.0)	70.0 (60.0-79.0)	0.285
Tidal volume (mL)			
Supine	416.5 (405.8-508.0)	426.0 (391.5-488.0)	0.385
Prone	412.0 (396.5-507.8)	422.0 (389.0-471.5)	0.325
Tidal volume/IBW (mL/kg)			
Patient in Supine	8.0 (7.0-8.5)	7.5 (7.0-8.5)	0.143
Patient in Prone	8.0 (7.5-8.5)	7.5 (6.5-8.0)	0.058
Peak inspiratory pressure (hPa)			
Patient in Supine	17.0 (15.0-19.0)	15.0 (14.0-17.0)	0.04
Patient in Prone	20.0 (17.0-21.0)	17.0 (15.0-19.0)	0.005
Positive end expiratory pressure PEEP (hPa)			
Patient in Supine	5.0 (5.0-5.0)	5.0 (2.0-5.0)	0.23
Patient in Prone	5.0 (5.0-5.0)	5.0 (5.0-5.0)	0.366
Table type			
Jackson spinal table	15 (75.0%)	136 (71.5%)	0.642
Pulse pressure variation (%)			
Patient in Supine	7.5 (6.0-11.0)	8.0 (6.8-11.0)	0.47
Patient in Prone	12.0 (9.0-16.4)	9.0 (7.0-12.0)	0.019
Anesthesia time (min)	174.5 (125.0-256.3)	174.0 (131.0-248.5)	0.91
Operation time (min)	114.0 (75.0-182.8)	115.0 (80.0-187.5)	0.669

Discussion

In this study, the incidence of hypotension associated with the shift from a supine to a prone posture was 8.9%. The injection of beta-blockers before to surgery and the maintenance of mean arterial pressure in the supine position were independent risk factors for posture-induced hypotension. Awake individuals and those under the influence of medications may have differing rates of hypotension due to postural alterations. The investigation revealed that the occurrence of hypotension after the transition from supine to prone posture was thrice higher than the about 3% incidence noted in conscious individuals[12]. This may be linked to a decrease in ventricular preload due to reduced venous return and anesthetic-induced suppression of compensatory mechanisms (baroreflex and sympathetic activity).

Increased intra-thoracic pressure due to positive pressure breathing may exacerbate a decrease in cardiac preload. Prior research demonstrated that brief instances of intraoperative hypotension correlated with increased postoperative mortality and morbidity. Thus, preventing intraoperative hypotension is clinically significant.[13,14] Thus, preventing intraoperative hypotension is therapeutically significant. Despite being a short-acting medicine, it may cause bradycardia and hypotension due to its vagotonic effects and sympathetic blocking, particularly in elderly patients.[15,16] The location of the bispectral index value showed no association.

Patients demonstrated an increased propensity for hypotension associated with the supine-to-prone positional transition when the time between the latest modification of their sedatives' target concentration and the postural change was diminished. Six patients (38%) in the hypotension cohort had reduced impact site concentrations of sedatives following positional adjustments. The effect site concentration of sedatives in the remaining 10 patients (62%) within the hypotension group was pseudo-equilibrated (within 0.1 ng/mL) to the target concentration.[16–18] Previous research comparing inhalation anesthesia with total intravenous anesthesia indicated that the total intravenous anesthesia group demonstrated a markedly lower cardiac index and mean arterial pressure (MAP).[19,20] Nonetheless, the hypotension induced by postural alterations in this trial was not linked to propofol.

The investigation revealed that the shift from supine to prone position was linked to hypotension, affected by the preoperative administration of beta-blockers.

Administering beta-blockers before to surgery may provide cardioprotective benefits, but it also increases the likelihood of bradycardia and intraoperative hypotension. Moreover, the baroreceptor reflex—a crucial compensatory mechanism for hypotension induced by postural alterations—can be altered by the use of beta-blockers. This study found that increased mean artery pressure (MAP) in the supine position was independently correlated with hypotension induced by positional alterations.

A previous study found that higher blood pressure measured in a sat position was the only factor potentially affecting hypotension during the transition from a supine to a prone position[12]. An explanation for these data may be that increased mean arterial pressure (MAP) following a positional change activates a baroreceptor response, resulting in bradycardia and peripheral vasodilation.[12,22] This study found no association between preoperative hypertension and hypotension induced by positional alterations or increased mean arterial pressure in the supine position. A mean arterial pressure (MAP) beyond 100 mm Hg, representing the 75th percentile in healthy individuals, indicates that 28 patients (17%) demonstrated elevated MAP when in a supine position. Nine people, constituting thirty percent, displayed hypertension before the procedure. Furthermore, a previous study demonstrated that shifting from a supine to a prone position exhibited no differences in the oscillations of systolic and diastolic blood pressure between hypertensive individuals and those with normal blood pressure.[12]

This study found that the PPV of both groups in the supine position was similar, however the hypotension group's PPV in the prone position was significantly greater than that of the non-hypotension group. This study indicates that a reduction in preload after the postural change is a crucial factor in hypotension caused by the prone position. Due to the fast fluid infusion administered to all patients during anesthesia induction, a minimal proportion exhibited PPV > 14% in the supine position (6.3% in the hypotensive group and 8.6% in the non-hypotensive group). This may clarify why PPV in the supine position was not associated with hypotension during the transition from supine to prone posture. The incidence of postpartum hemorrhage was markedly increased in the prone position and displayed substantial difference between the hypotension and non-hypotension groups, despite the substantial fluid administration previous to the postural change. This study is limited by multiple restrictions. At first, although we employed PPV as an indicator of volume status, we did not evaluate vascular resistance, preload, or cardiac output. Therefore, we were unable to determine the exact cause of the hypotension that occurred after the transition from the supine to the prone position. Moreover, we were unable to evaluate intrathoracic or intraabdominal pressure, making their effects uncertain. Secondly, the results may have been influenced by various biases arising from the study's retrospective design. Given the significant exclusion of patients from the data analysis, we recommend exercising caution in the interpretation of our findings. Excluding individuals with cardiac arrhythmias and those who received vasoactive medicines just before anesthesia and after a positional change could distort the results. Third, we could not accurately record the volume of fluid administered before the change in posture. All trial participants underwent rapid fluid infusion prior to the alteration in posture, in accordance with our usual clinical protocol. During this period, 300–400 milliliters of Ringer's lactate solution were administered. In the supine position, positive pressure breathing was comparably effective in both groups. Therefore, it is improbable that the fluid volumes given to the two groups varied before the positional modifications.

Conclusion

This study revealed a notable incidence of hypotension in people shifting from a supine to a prone position after spinal surgery. Tidal volumes surpassing 6 ml/kg demonstrated the greatest occurrence of hypotension at 50%, along with an average decline in mean arterial pressure of 25 mmHg. Furthermore, our findings demonstrated that preoperative beta-blocker medication and elevated MAP in the supine position were independent factors leading to hypotension related to the transition from supine to prone posture.

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Authors Contribution

SMAT: Conceptualization of Project

AS: Data Collection

MUIB: Literature Search

MT: Statistical Analysis

SAK: Drafting, Revision

SK: Writing of Manuscript