Haemodynamic changes during prone positioning in anaesthetised chronic cervical myelopathy patients

INTRODUCTION

Patients with chronic degenerative changes at the cervical spine region present with symptoms of myelopathy which include pain, neurological deficits and autonomic dysfunction. These patients require surgery either in supine or in prone position to relieve the neck pain and prevent further progression of spinal cord compressive symptoms. Cervical myelopathy patients who require decompressive laminectomy are positioned prone to facilitate the surgical procedure. Cardiovascular physiological changes following prone positioning under anaesthesia include reduced venous return due to increased intra-abdominal pressure resulting in reduced stroke volume, increased sympathetic response, increased heart rate, increased...
systemic and pulmonary vascular resistance and reduced left ventricular compliance due to increased intra-thoracic pressure. All these changes can result in reduced cardiac output and systemic hypotension.

When cervical myelopathy patients are positioned prone under anaesthesia, they carry a higher risk of developing haemodynamic changes due to combined effects of prone positioning, anaesthesia, cervical cord compression and autonomic nervous system dysfunction. These haemodynamic changes can compromise spinal cord perfusion and result in spinal cord ischaemia. Studies describing the haemodynamic changes during prone positioning in cervical compressive myelopathy patients are lacking. Hence, this study was designed to assess the haemodynamic changes following prone positioning in cervical myelopathy patients.

**METHODS**

The study was registered with the ClinicalTrials.gov (ID: NCT03027817). After obtaining Institute ethics committee approval and informed consent from patients, 30 adult ASA I-III patients, aged 18–65 years, symptomatic for more than six months and belonging to Nurick’s grading system ≥2 were recruited [Table 1]. All patients underwent decompressive laminectomy in prone position under general anaesthesia. Patients with atlanto axial dislocation, spinal tumour pathology, diabetes mellitus, on anti-hypertensive medications affecting the autonomic system and who required awake prone positioning were excluded from this study. Demographic data collected include age, gender, co-morbid conditions, Nurick’s grading, height and weight (if patients could not stand, approximate weight was noted).

Before the start of surgery, patients were connected to standard monitoring which includes an electrocardiogram, blood pressure, pulse oximetry and capnography. Four NICOM® sensors (Cheetah Medical, Inc., Massachusetts, USA) were attached at the back of the patient symmetrically in the four quadrants of the back (one each on the upper aspect of the scapula and one each on posterior costal margins) [Figure 1]. These sensors were connected to the NICOM monitor through a cable, and the monitor continuously displayed the haemodynamic variables. Baseline haemodynamic measurements were made before the administration of anaesthetic medications. Anaesthesia was induced intravenously with fentanyl 2 µg/kg and thiopentone 3–5 mg/kg. Muscle relaxation was achieved with intravenous vecuronium 0.1 mg/kg, and the patient was ventilated with 100% oxygen. Two minutes later, xylocaine 1 mg/kg was given intravenously. After a minute, the trachea was intubated, and anaesthesia was maintained with O2 + air (50:50) and 1 MAC sevoflurane with hourly supplementation of 1 µg/kg fentanyl. The patient was positioned prone over pillows under chest and abdomen and the head was fixed in Mayfield’s skull pins. The sensors were secured with an adhesive tape. All patients received a constant rate (10 ml/kg/hr) of fluid administration during the study period.

The haemodynamic data were collected before induction, 2 minutes after induction and after intubation, after prone positioning (before skull pins fixation) and thereafter every 5 minutes until 20 minutes or until surgical incision [Figure 2]. Prior to skull pins insertion, each site was infiltrated with one ml of Lidocaine 2%. The haemodynamic parameters that were recorded using the NICOM® monitor include heart rate (HR), non-invasive mean arterial pressure (MAP), cardiac output (CO), cardiac index (CI), stroke volume (SV), stroke volume index (SVI), stroke

| Table 1: Nurick’s grading system |
|---|
| **Grade** | **Findings** |
| 0 | Signs or symptoms of root involvement but without evidence of spinal cord disease |
| 1 | Signs of spinal cord disease but no difficulty in walking |
| 2 | Slight difficulty in walking that does not prevent full-time employment |
| 3 | Difficulty in walking that prevents full-time employment or the ability to do all housework |
| 4 | Able to walk only with someone else’s help or with the aid of a frame |
| 5 | Chairbound or bedridden |

**Figure 1: Picture depicting position of sensor placement**
volume variation (SVV), total peripheral resistance (TPR) and total peripheral resistance index (TPRI).

All haemodynamic alterations (< or > 20% of baseline) were treated and interventions were noted. Hypotension was treated with intravenous boluses of mephentermine 6 mg and hypertension was treated with intravenous bolus of fentanyl 1 µg/kg.

We conducted a pilot study on ten patients to calculate the sample size. Calculated effect size $f = 0.88$ and the minimum correlation between time points was 0.009 for a change of mean arterial pressure over different time points. Thus, sample size estimation came to 20 patients for 8 time points with an $\alpha$ of 0.05 and $\beta$ of 0.8. Quantitative data were expressed as mean ± standard deviation (SD) and qualitative data as percentages. Normality of the data was tested using Shapiro-Wilk test. Repeated measures analysis of variance (ANOVA) was used for analysing the haemodynamic parameters across different time points with Bonferroni post hoc corrections. Mixed models ANOVA was used for analysing the difference between groups [Nurick’s high (4,5) vs. low grade (2,3) and mephentermine utilisation vs non-utilisation]. Bivariate Spearman’s correlation was done to assess associations between MAP and age, number of levels of compression, Nurick’s grade, CO, HR, TPR and SV. The significantly correlated variables were entred into a linear regression model to assess the independent predictability of MAP. SPSS software (SPSS Statistics for Windows, Version 17.0. Chicago: SPSS Inc.) was used to perform statistical analysis. A $P$ value of < 0.05 was considered statistically significant.

**RESULTS**

Data were available for all the 30 patients. Patients’ demographic profile is given in Table 2. Majority of the study population were males (87%) and belonged to high Nurick’s grading system (74%). Haemodynamic changes during the study period are given in Table 3. Cardiac output and cardiac index remained stable during the entire study period and the changes which occurred during post induction (decrease) and post-intubation (increase) time points were not significant ($P = 0.186$). Heart rate fluctuated significantly during the study period ($P < 0.001$). Heart rate increased following intubation and then decreased at 10, 15 and 20 minutes after prone positioning. SV reduced after induction and then increased after prone positioning at 10 min, 15 min and 20 min. These changes were significant ($P = 0.002$). Mean blood pressure decreased significantly at the following time points – post induction, post prone positioning, and 15 and 20 minutes after prone positioning ($P = 0.001$). However, there was a significant increase in MAP following intubation. Sixty percent of the patients experienced at least one episode of hypotension and required mephentermine boluses. Cardiac output remained stable during episodes of hypotension and mephentermine boluses. Changes in TPR and TPRI mirrored the changes in MAP and these changes were significant ($P < 0.001$). Stroke volume variation progressively decreased from baseline (16.8%) to end of the study period (15.1%).

Both low and high Nurick’s grade patients experienced a similar type of haemodynamic changes during the study period. Heart rate, cardiac output, stroke volume, total peripheral resistance and number of levels of spinal cord compression were associated with MAP changes during the study period and not age and Nurick’s grading [Table 4]. Number of levels of spinal cord compression had a negative linear correlation with MAP ($\beta$ co-efficient: -1.40, $P$ value = 0.006), which means that the patients with more levels of spinal cord compression had more chances of developing hypotension.

**DISCUSSION**

This study found 60% incidence of hypotension in cervical myelopathy patients during prone positioning under anaesthesia with stable cardiac output during...
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In the study period, there was a reduction in total peripheral resistance associated with increased stroke volume in the prone position. These findings were not affected by the patients' Nurick's grading and age.

Hypotension (≥20% decrease from baseline) occurred more frequently following prone positioning than in the immediate prone positioning phase due to the skull fixation during laminar surgical field. Following this, there was an increase in the vasoconstrictor response of the patients undergoing surgery in prone position. In patients undergoing surgery in prone position, blood pressure increased and there was no significant increase in mean arterial pressure, which prevented compensatory vasodilation in peripheral resistance. On the contrary, we found a decrease in stroke volume and an increase in peripheral resistance.

Table 3: Haemodynamic changes during the study period

|            | Baseline | Post Induction | Post Intubation | Pre prone | 5 min Post prone | 10 min Post prone | 15 min Post prone | 20 min Post prone | P      |
|------------|----------|----------------|-----------------|-----------|-----------------|-----------------|-----------------|-----------------|--------|
| CO (L/min) | 3.77±1.09 | 3.47±0.84      | 3.9±1.01        | 3.74±0.95 | 3.62±0.96       | 3.61±1          | 3.54±1.03       | 3.49±1.04       | 0.186  |
| CI (L/min/m²)| 2.25±0.59 | 2.06±0.46      | 2.32±0.54       | 2.23±0.51 | 2.17±0.56       | 2.17±0.59       | 2.12±0.63       | 2.1±0.64        | 0.224  |
| HR (beats/min)| 81±14    | 81±13          | 93±12           | 85±15    | 76±13           | 75±11           | 74±11           | 74±11           | <0.001 |
| SV (ml)    | 46.4±10.8 | 43.9±12.6*     | 42.6±11.1       | 44.0±12.1| 42.9±11.8       | 48.7±13.8       | 48.3±14.1       | 48.8±16.6*      | 0.002  |
| SV (ml/m²) | 27.8±5.8  | 26.2±6.9*      | 25.7±6.1        | 26.3±6.8 | 25.9±6.9        | 29.3±8.3*       | 29.1±8.4*       | 29.3±10.1*      | 0.005  |
| SV (%)     | 16.8±3.6  | 16.4±2.9       | 16.7±2.7        | 16.3±2.8 | 16.4±2.3        | 15.9±3.0        | 15.6±3.1*       | 15.1±3.4        | 0.008  |
| MAP (mm Hg)| 113.23±12.26| 96.03±17.51*   | 125.8±23.55*    | 98.7±19.52*| 91.9±19.93*    | 86.8±21.6*      | 83.7±15.15*    | 78.63±13.87*    | <0.001 |
| TPR (dyne/sec/cm²)| 2637.27±709.24| 2337.47±700.03| 2805.7±770.50 | 2297.83±660.55 | 2152.47±643.8  | 2006.1±710.64* | 2003.07±599.28*| 1923.9±579.83* | <0.001 |
| TPRI (dyne/sec/cm²/m²)| 4374.5±1049.67| 3863.73±1053.77| 4633.5±1151.42 | 3812.57±1045.41| 3574.2±1050.63 | 3283.77±1132.86*| 3333.5±1007.76*| 3221±1068.71* | <0.001 |

CO – Cardiac output, CI – Cardiac index, HR – Heart rate, SV – Stroke volume, SVI – Stroke volume index, SVV – Stroke volume variation, MAP – Mean arterial pressure, TPR – Total peripheral vascular resistance, TPRI – Total peripheral vascular resistance index. All values are in mean±SD. *P<0.05 compared to baseline; †P<0.01 compared to baseline; ‡P<0.05 compared to pre-prone.
Previous Studies have shown an association between prone positioning and a decrease in SV and CI.[15-18] The proposed mechanism of hypotension in these studies is that a reduction in venous return due to increased intra-abdominal pressure decreases the cardiac output thereby resulting in hypotension. As a compensatory mechanism, the body increases the total peripheral resistance and the heart rate to restore the blood pressure. In our study done on cervical myelopathy patients, we did not find a decrease in stroke volume, cardiac output and cardiac index after prone positioning. Instead, we found SV increase after prone positioning from baseline and prepositioning values associated with a significant decrease in total peripheral resistance and heart rates. One possibility could be due to the presence of autonomic dysfunction in these patients. As a result of this, these patients do not develop compensatory vasoconstriction which caused a fall in TPR thereby maintaining the CO. At the same time, due to chronic spinal cord compression, spinal cord pressure autoregulation might get compromised and in this situation, maintaining blood pressure is important to prevent ischemia. However, in clinical practice, unlike cerebral pressure autoregulation, autoregulation in the spinal cord is difficult to study and the data from the cerebral autoregulatory studies are extrapolated to spinal cord function. If impaired, then adequate pressure gradient is required to maintain perfusion to the spinal cord and maintaining global CO alone might not be sufficient. Though studies are available to prove the presence of autonomic dysfunction in chronic cervical myelopathy, we could not measure the autonomic function in our patients. Hence, we can only speculate this to be the most probable reason for the haemodynamic changes seen in our patients.

There was no significant difference in haemodynamic changes between patients with high and low Nurick’s grade. Rather, hypotension was associated with increasing number of levels of spinal cord compression. Sympathetic neurons arise from the thoracolumbar level, and hence, compression above its origin greatly affects the function of the sympathetic system. This could explain the correlation between blood pressure changes and increased number of levels of spinal cord compression. On the other hand, Nurick’s grading mainly deals with the functional activity (motor system) and hence did not correlate with the blood pressure changes.

Previous studies have shown that haemodynamic changes are dependent on the type of frame used to position the patients in prone. Jackson’s spine table, bolsters and pillows induce the least amount of haemodynamic changes.[19] In our study, all patients were placed on pillows and could be one of the reasons for the lack of CO changes.

There are however certain limitations to our study. We did not measure autonomic function in these patients which could have helped us to understand the reason for haemodynamic changes better. We did not include a control group (e.g., lumbar spine surgery) to document the contrasting haemodynamic changes.

**CONCLUSION**

Hypotension is common in cervical myelopathy patients when positioned prone. This hypotension is related to the number of levels of spinal cord compression. However, this hypotension was not associated with decreased cardiac output.

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**Conflicts of interest**

There are no conflicts of interest.

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