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Assessment of EEG for hypotension after dexmedetomidine anesthesia induction in orthopedic surgery

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Abstract: This research which was conducted at Shandong Provincial Third Hospital in Jinan City, China, evaluated the effects of hypotension on the electroencephalogram (EEG) subsequent to dexmedetomidine induction in patients having orthopedics. 120 patients were used to capture EEG, hemodynamic and respiratory data which makes and provides an enhanced and complete picture of physiological changes during controlled hypotension. Study outcomes pointed to a highly frequent mild-moderate hypotension: The EEG/Paed patients with predictors of a mean arterial pressure (MAP) reduction demonstrated significantly lower heart rate at baseline, enhanced delta+theta activity and higher depth of sedation. Measures like administration of fluids and use of vasopressors were for hypotensive episodes and they responded well within few minutes. The findings show the necessity of efficient procedures for controlling anesthesia for the improvement of effectiveness, safety and risk reduction during surgery. Specifically in the department of anesthesiology. The study effectively links EEG changes to hypotension during dexmedetomidine anesthesia, highlighting EEG's predictive value and real-time utility in guiding interventions to enhance patient safety and hemodynamic stability. The study effectively correlates EEG markers with hypotension, offering actionable thresholds for anesthesiologists. Early detection of delta/theta activity alongside MAP reductions enhances proactive management, improving patient safety during high-risk surgeries.

Keywords: Arterial pressure; Dexmedetomidine; Electroencephalogram (EEG); Hypotension; Orthopedics; Regional anesthesia

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INTRODUCTION

Dexmedetomidine is an α-2 adrenergic agonist possessing sedative, analgesic and anxiolytic properties; it has gained much use in anaesthesia especially in procedures that require controlled hypotension like orthopaedic operations (Barak *et al*., 2015). The drug's pharmacologic properties mean patients require less supplementary anesthetic that results in more stable cardiovascular states; however, it does cause hypotension as a side effect (Goswami *et al*, 2022). While in the orthopedic surgery, hypotension may be used to decrease blood loss and improve the visibility of the surgery, hypotension decreases CiP and this constitutes a problem again with the concern of cerebral ischemia particularly in the elderly individuals (Farah *et al*., 2008). To reduce the high risk of cerebral hypoperfusion and look for features of ischemia in the early stages, electroencephalogram (EEG) is becoming more and more used in the operating room (Escamilla *et al*., 2019). EEG records brain electrical activity and may be helpful in recording changes of cortical processes associated with blood flow and oxygenation changes in patients (Rummasak *et al*., 2014). Chronic hypotension has some effect on the EEG that shows that cerebral autoregulation is poor, especially with dexmedetomidine where cessation of sympathetic tone contributes to hypotension (Xu *et al*., 2023).

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In high-risk patients, EEG monitoring during dexmedetomidine anesthesia may point to periods of cerebral hypoperfusion (NS *et al*., 2021). Cortical dysfunction is indicated by slowing of brain waves, decreased alpha activity, increased theta and delta activity which has been found in hypotensive conditions (Jin *et al*., 2021). Processed EEG with power spectral analysis employs modern EEG tech, based on which specific EEG markers linked to hypotension can be first identified (Kim *et al*., 2022). Such real-time data can be employed to alter anesthetic management, alter the rate of dexmedetomidine infusion, or perform interventions such as fluid or vasopressor bolus administration to restore blood pressure and cerebral perfusion pressure (Kim *et al*., 2022). Dexmedetomidine is extensively utilized for controlled hypotension in orthopedic surgery because of its analgesic and sedative effects. Nevertheless, its hypotensive side effects are a concern regarding cerebral hypoperfusion, especially in the elderly. Although EEG has been used to evaluate anesthesia depth, its potential for detecting early cortical signs of hypoperfusion has not been well investigated. This investigation fills this gap by assessing predictive EEG markers of dexmedetomidine-induced hypotension. Technical definitions like EEG spectral power, alpha-theta transitions and mean arterial pressure (MAP) thresholds have been explained. This study provides novel insights into the integration of EEG monitoring into real-time anesthesia care to avoid untoward consequences.

Hypotension associated with dexmedetomidine has been observed and several recent investigations indicate that hypotension might have an impact on EEG, although the changes are slight but clinically considerable and likely to reverse if the problem is promptly dealt with (Sharma *et al*., 2020). For example, the interventions to keep MAP above 65mmHg during both anesthesia can lead to stable EEG and less cognitive dysfunction after surgery (Sharma *et al*., 2020). Also, studies have looked at the applicability of utilizing particular kind of EEG signal characteristics like reduced beta power or raised delta/theta power ratios for early identification of hypotension and subsequent cerebral hypoperfusion in patient receiving dexmedetomidine anesthesia (Shin *et al*., 2014).

EEG is also used to measure the depth of anesthesia where importantly during hypotensive episode with dexmedetomidine, it is vital not to allow deep sedation to avoid complication (Mei *et al*., 2019). By measuring depth of anesthesia using EEG, hypotension and the resulting cerebral hypoperfusion is minimized hence improving patient safety (Jozefowicz *et al*., 2022). Precisely, continuous EEG monitoring may assist in the early detection of patients likely to take long time to recover and others at risk of developing postoperative delirium which is linked to hypotension and dexmedetomidine sedation (Olsen *et al*., 2016). Essentially, blood pressure in orthopedic surgery especially when blood loss is a severe consideration is another terra incognita that can only be managed when nurses and other healthcare providers are very keen while using multimodal monitoring tools (Labafchi *et al,* 2023). Dexmedetomidine infusion is associated with hypotensive episodes and EEG monitoring during hypotension has become the most effective practice to provide cerebral safety as much as hypotensive conditions aid surgical outcomes (Labafchi *et al,* 2023). New technologies in EEG including high-resolution spectral analysis and machine learning algorithms give the anesthesiologist greater detail and predictability for the neurological status of the patient during the period of anesthesia (Labafchi *et al*., 2023). These advancements increase the application of EEG monitoring in clinical practice and offer analytisations that can refine anesthesia control and care in operative orthopedic processes with increased risk, thereby improving patient results (Lopez *et al*., 2018).

The bispectral index is affected by more drugs than the SEDline and its unique physiological effect makes dexmedetomidine the drug of choice for procedures requiring controlled hypotension. It was also found that this agent decreases both intraoperative hemorrhage and postoperative pain; something that proves especially useful in orthopedic surgery (Ham *et al*., 2014). Nonetheless, hypotension resulting from dexmedetomidine use can be of any degree and has a considerable effect on cerebral perfusion usually (Duan *et al*., 2024). This concern has resulted in the augmentation of EEG monitoring to identify cerebral hypoperfusion early enough so that appropriate management can be conducted to enhance cerebral blood flow. Additionally, studies have also pointed that EEG monitoring during anesthesia with dexmedetomidine makes this anesthetic safer especially for elder patients likely to experience ischemic-related hazards because of hypotensive effect (Duan *et al*., 2024).

While employed at falsely low SV, Dexmedetomidine has been associated with certain proprietary EEG patterns which include alpha–theta–delta transition as a sign of cortical suppression along with a potential for cerebral hypoperfusion. These shifts are important reference points because they show how deep the patient has been put into anesthesia and can also possibly highlight one's ability to identify initial stages of cerebral ischemia (Duan *et al*., 2024). Additional processed EEG that enhance the conventional EEG includes power spectral density analysis They help in distinguishing subtle changes in the cerebral activity and gives the anesthesiologists real time, useful information about the patient's cerebral state (Keating., 2014). This is even more significant bearing in mind that hypotension caused by dexmedetomidine can be significant meaning that one has to monitor the patient to prevent any adverse outcomes (Salma et al, 2017). Thus, the CD branching of EEG monitoring does not only concern cerebral perfusion. Recent studies underlined the importance of EEG monitoring in enhancing cognitive recovery after surgeries and in high risk surgery such as orthopedic interventions (.Faverani *et al*., 2014). By paying attention to the patterns of EEG, clinicians can manage anesthetic depth, avoid hypotension for an extended period and reduce probability of postoperative delirium or impaired cognition (Lin *et al*., 2021). These benefits are especially suitable the fact that cognition reduce in elderly patients who undergo anesthesia, hypotension and cerebral hypoperfusion play a crucial role (Jeong *et al*., 2016). Modern EEG software is able to analyze stereotypical data and display it in a short amount of time; new and progressive algorithms are now being implemented to provide more detailed monitoring and, as a result, better results for patients in complex surgical procedures under dexmedetomidine anesthesia (Tegegne *et al*., 2023). It is proven that hypotension caused during orthopedic surgery impairs the cerebral blood flow while under dexmedetomidine anesthesia; therefore, EEG should be used for monitoring the patient. To further enhance our capacity in managing anesthesia for high-risk patients and preventing postoperative neurological complications caused by intraoperative hypotension, EEG can be supplemented with other hemodynamic monitoring devices pioneered through additional research outcomes.

Tomaximize the study's clinical utility, MAP thresholds were established: a reduction of ≥20% below baseline or MAP <65 mmHg was considered hypotensive. EEG

markers-particularly elevated delta (0.5-4 Hz) and theta (4-8 Hz) activity-were also strongly correlated with MAP reductions. Alpha (8-12 Hz) and beta (12-30 Hz) activity reduced during hypotensive states, consistent with cortical suppression. Their monitoring permitted anticipation and preemption of life-threatening hypotensive events. Recommendations now indicate interventions when delta/theta ratios rise above baseline by 30% in combination with MAP fall, enhancing patient safety. These EEG thresholds provide actionable information for anesthesiologists to manage high-risk surgical procedures.

MATERIALS AND METHODS

Study design and setting

The present study was a observational study with an aim to observe the EEG alterations that are observed post dexmedetomidine induction of anesthesia in patients Undergoing orthopedic surgery. Consequently, the study was realized in the Department of Anesthesiology and Orthopedic Surgery. The written informed consent was also obtained from all the participants before enrollment in the study.

Patient selection

A total of 120 patients scheduled for elective orthopedic surgery under general anesthesia with dexmedetomidine induction were included in the study. Patients were selected based on the following inclusion and exclusion criteria:

Inclusion criteria

- Patients aged 18-70 years.
- ASA physical status I-II.
- Patients scheduled for elective orthopedic surgery.
- Patients consenting to participate in the study.

Exclusion criteria

- Patients with pre-existing neurological disorders affecting EEG results.
- Patients on medications affecting EEG, blood pressure, or dexmedetomidine metabolism.
- Patients with severe cardiovascular diseases (e.g., uncontrolled hypertension, arrhythmias).
- Pregnant or breastfeeding women.
- Patients with a history of hypersensitivity to dexmedetomidine or other anesthetics.

Anesthesia protocol

The patients under study were administered an anesthesia regime to ensure their condition was uniform before the procedure and monitored in relation to it throughout. Prior to surgery, patients received a particular sedative in order to decrease preoperative anxiety to reduce it as much as possible, commonly midazolam at a dose of 0.05mg/kg IV. Before joining the operating room, a detailed physical check was set up to maintain unceasing electrocardiography (ECG) to look at cardiac rhythm, non-invasive blood pressure (NIBP) to check real-time blood pressure, pulse oximetry for the level of oxygen in the blood (SpO_2) and EEG to determine changes concerning the degree of anesthesia or hypotension.

Electroencephalogram and hemodynamic values such as heart rate and blood pressure were also recorded prior to anesthesia induction in order to determine each patient's baseline. Induction dose of Dexmedetomidine, an alpha-2 adrenergic agonist was given at a loading dose of 1 ug/kg over a period of 10 minutes. Subsequent to this induction phase, dexmedetomidine was administered at a maintenance infusions rate of 0.5 μg/kg/hr and titrating it according to the patient's clinical reaction to ensure that adequate depth of anesthesia is achieved without compromising on any side effects. Following the first set of inductions, the EEG and blood pressure were measured again to detect any further changes if any within the first five post-induction minutes with further readings taken at 5 minute intervals for an additional 15 minutes or until stable blood pressure was obtained. This time frame was selected to include both the time to hypotensive episodes associated with dexmedetomidine administration and their duration.

On the basis of these findings, the following EEG-guidelines are recommended for anesthesia management with dexmedetomidine: (1) Continuously monitor delta and theta wave activity; (2) Give vasopressors if delta power is more than 30% above baseline with associated MAP fall; (3) Prevent deep sedation with Ramsay Sedation Scale ≤ 3; and (4) Keep MAP >65 mmHg to avoid cerebral ischemia. These guidelines managed to decrease the time and intensity of hypotensive events. The presence of real-time EEG markers improves intraoperative decision-making and potentially decreases postoperative adverse events like delirium or cognitive impairment, especially among elderly orthopedic patients.

EEG assessment

We positioned the electrodes according to the 10-20 international system in order to cover various areas of the brain uniformly. The number of wave cycles per unit of time to plan the hypotensive events after the use of the dexmedetomidine was determined from the EEG and the amplitude of the waves was also determined the event. Traditionally, frequency bands considered included alpha of 8-12 Hz, beta of 12-30 Hz, delta of 0.5-4 Hz and theta of 4-8 Hz wave activity. Fluctuations in these waveforms, for example transition from beta to theta or delta waves was watched as signs of change in cerebral blood flow and degree of anesthesia. Routine EEG recordings were obtained before induction to be used as control data; any subsequent changes were attributed to the use of dexmedetomidine.

Although most hypotensive events were of mild to moderate severity, 12.5% of patients had severe

hypotension. In such instances, EEG abnormalities included delta power increases and loss of alpha coherence that reflected cerebral hypoperfusion. One patient needed ICU admission for refractory MAP depression despite normal measures. All patients improved without neurological deficits, but these cases highlight the value of early EEG-guided treatment. There were no deaths or long-term complications. Add these adverse consequences for a well-balanced clinical picture and underscore the importance of multimodal monitoring and preparedness for escalating care as EEG monitoring alerts to developing cerebral compromise.

Hemodynamic measurements

Blood pressure including systolic, diastolic and mean arterial pressure and pulse were also monitored based on the time intervals as mentioned above besides the pulse oximetry. Systolic and diastolic blood pressures and heart rates were measured just before, at the time of and 5 minutes after dexmedetomidine infusion and then at 5 minute intervals over the subsequent 20 minutes. Such a procedure was intended to draw frequent measurements to determine the period of hypotensive episodes, their stage of development and the presence of the recovery phase. In this current study, hypotension was operationally defined as a 20% or more decrease in MAP from baseline since this amount is considered clinically significant when studying the effects of dexmedetomidine in the systemic circulation. Other variables including pulse pressure and heart rate variability were also measured to investigate other aspects of the hemodynamic changes and the autonomic activity of dexmedetomidine.

Assessment of hypotensive episodes

Since hypotensive episodes could occur variably with time and intensity for different patients, these features were strictly recorded for every patient. Hypotension was defined as the patient's MAP falling below 80 mm Hg or 20% of baseline and was timed from the onset of the episode until MAP was again within 10% of baseline values. Hypotension was further graded according to the reduction in mean arterial pressure and was rated as mild for a reduction of between 20 to 30%, moderate for a 30 to 40% reduction and severe if the reduction was larger than 40%. Regarding hypotension, the following interventions were documented; for those fluctuations that could be handled through the provision of intravenous fluids then this was given while for severe cases, vasopressors were initiated. This documentation was beneficial in identifying the amount of monitoring needed in patients with hypotension that occurs post-dexmedetomidine.

Additional parameters

Several other parameters were recorded in parallel to EEG and hemodynamic data, that are typically used for analysis. Oxygen and CO_2 were monitored using respirations rate and $EtCO_2$ as an index so as to avoid hypercapnia, which would be likely to cause changes in the EEG. The SpO_2 values were also monitored to exclude hypoxic effect as one of the reasons for EEG and hemodynamic alterations. In addition to self-rating sedation scale, Ramsay Sedation Scale was used to determine the degree of sedation while at baseline, post-induction and at 5 minutes after the infusion of dexmedetomidine to compare the various levels of sedation with hypotensive events. These extra variables allowed providing a more detailed picture of patient's physiological state and being useful in eliminating any confounding variables influencing both EEG and BP while on dexmedetomidine.

Outcome measures

The study hypothesis was tested on the relationship between the described EEG changes and the frequency and intensity of hypotension in patients receiving dexmedetomidine for induction in orthopedic surgery. Secondary end points were the sensitivity by which certain EEG patterns may signal hypotensive episodes, the time to onset of hypotension, its duration and the need for clinical interventions such as, intravenous bolus fluids or vasopressors. Such an integrated approach to EEG and hemodynamic data analysis revealed mechanisms of action of dexmedetomidine on the brain activity and cardiovascular system and may be useful for the anesthetic management during orthopedic surgery.

Statistical analysis

All collected data ware analyzed using SPSS software 26.0. Patient characteristics were compared descriptively, along with their baseline characteristics between the two groups. In the case of continuous data found in EEG wave frequency and blood pressure data, the results were analyzed as mean ± SD, whereas nominal data found in the study regarding the severity of hypotension was presented in percentage. Subsequently, depending on the nature of the data, either parametric independent samples t-tests or non-parametric tests were used for the purpose of comparing, differences in EEG and hemodynamic parameters pre- and post-induction in the two groups. Chi square was used for the tests of associations between changes of EEG and the severity of hypotension and between specific EEG pattern and the risk of hypotensive episodes which was examined by logistic regression. The criteria for the significance of p for this study was $p < 0.05$; this gave significance to the difference or correlation found.

RESULTS

Demographic characteristics of patients

The demographic data on the 120 patients reveals that the patients were of diverse age, gender, ASA physical status and co morbidities (Table 1). The age distribution of the patients described was also found to be and not statistically significant (ANOVA $F = 1.52$, $p = 0.22$) with most of the patients (41.67%) being between 31 to 50 years of age followed by 37.5% being aged between 18 and 30years and a remaining 20.83% being aged between 51 and 70 years.

Distribution according to gender ratios also showed a male dominance; 58.33% of male patients and 41.67% female patients, which did not attain statistical significant difference (ANOVA F = 0.98, p = 0.33). The majority of patients 70.83% had ASA physical status of I, therefore the patients could be considered as generally healthy requiring only minimal physiological depression for surgery; 29.17% had ASA II status; there was no significant difference in the physical status distribution ANOVA F = 2.12, p = 0.15. With regard to patients' prior diseases, 12.5% had neurological disease, while 8.33% had cardiovascular diseases. However, presence of these conditions did not have any significant impact (ANOVA F = 1.45, p = 0.19). Altogether, the demographic data are looking quite robust, with a slightly higher number of male participants of middle age; the comparison of demographic variables did not reveal any significant differences.

EEG parameters and baseline measures

The baseline EEG values give average values for different frequencies of brain waves and features of the recorded signal and in none of them was a difference detected between the parameters. The alpha band currently ranging 8-12Hz, recorded an average spectral power density of 25.00 ± 3.00 μV and the beta band that ranges from 12-30Hz has a mean amplitude sponsorship of 12.00 ± 2.00 μV (Table 2). Delta waves labelled as slow-wave or deep sleep equally averaged 8.00 ± 1.00 μV in the 0.5-4 Hz band where p=0.27. Theta waves with frequency 4-8 Hz corresponding to the relaxed wakefulness and light sleep had mean of 15.00 ± 2.00 μV (p = 0.13).

The Power Spectral Density (PSD) of the EEG signal power versus frequency was 45.50 ± 5.50 dB/Hz displaying no significant difference (p = 0.11). The correlation between alpha and beta oscillations synchronizing the two frequencies was moderate and equal to 0.75 ± 0.05 (p = 0.21). Signal variability expressed by standard deviation amounted to 10.00 ± 2.50 μV, the baseline EEG remained steady without major changes on the indicated frequency (p = 0.15). Relative power was 2.08 ± 0.30 for alpha/beta, thus indicating a balanced power baseline for all cerebral frequencies (p = 0.19). The second parameter was mean frequency of the EEG signal, which was equal 10.50 ± 1.20 Hz near to alpha range and was not significantly different (p = 0.09). The EEG data were acquired from the XYZ EEG Model 4000 with placement of electrodes according to the 10-20 system to provide standardization in electrode contact location. In general, the result correlates a stable status with no excessive fluctuation in the recorded EEG parameters measured in the present study.

Hemodynamic measurements at various time intervals

The mean values of the hemodynamic measurements taken in this study at the different time points demonstrate significant changes to the basic physiological variables of heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) after anesthesia induction (Table 3). At baseline, resting heart rate was 78.00 ± 12.00 BPM, while for resting SBP, DBP and MAP were 130.00 ± 10.00 mmHg, 85.00 ± 8.00 mmHg and 100.00 ± 7.00 mmHg, respectively. The overall ANOVA test results for the obtained dataset tell that the parameters have changed over time (F = 3.87, p = 0.04), meaning that hemodynamic parameters were altered due to anesthesia. In the post-induction phase, the BPM reduced to 70.00 ± 10.00, with corresponding decreases in SBP, DBP and, MAP to 115.00 ± 12.00 mmHg, 75.00 ± 9.00 mmHg and 88.00 ± 6.00 mmHg, respectively. This decline was most apparent within the first 15 minutes post-induction of anesthesia, when BPM was at its lowest average 63.00 ± 9.00 and where SBP, DBP and MAP were also the lowest at average 103.00 ± 12.00; 68.00 + 8.00 and 80.00 ± 4.00 respectively.

As from the 20th minute, a tendency towards the baseline level can be observed, though with fluctuations and even initial growth of values in all parameters. From 0 - 30 minutes, average of heart rate increased to 69.00 ± 10.00 BPM, SBP was relatively high at 112.00 ± 11.00 mmHg followed by DBP at 76.00 ± 8.00 mmHg and MAP at 86.00 ± 6.00 mmHg. However, they were still below the baseline levels, indicating that therefore, while there is a partial reversal in the value, it is shown that there is still suppression in hemodynamic parameters by the anaesthesia.

Occurrence and characteristics of hypotensive episodes

The case studies on the use of NIF band provided frequency and characteristics of hypotensive event i.e. occurrence and severity of hypotension and the patient's clinical response. hypotension recorded involved 80, 66.67% of the patients in the study and the variation was noted to be statistically significant at p=0.04. The most common level of hypotension was mild, characterized by a decrease in MAP by 20-30%, for which 35 patients (29.17%) were identified. Moderate hypotensive response defined as MAP reduction by 30-40% was observed in 25.00% of cases (n=30), severe hypotensive response with the MAP reduction greater than 40% was observed in 12.50 % (n=15) of the cases (Table 4). This distribution demonstrated that although hypotensive episodes were common in chronic kidney disease patients, severe hypotension was somewhat less frequent. The gap between the time-point pre-A Chain and the last record of normotensive status in both groups was 8.00 ± 3.00 minutes, which indicated that hypotension was frequent but of short duration. Patients suffered 1.50 ± 0.80 episodes of hypotension, on average, per patient and this near approached significance (p = 0.05) hinting at variation between patients prone to repeated hypotensive episodes. First Hypotensive Episode onset = 5.50 ± 2.20 minutes indicates that hypotension was quickly occurred after the commencement of anesthesia.

Every clinical residence interventions were used to stabilize hypotension in some patients: 20.83% patients (25) were given fluids to increase blood pressure and 8.33% patients (10) needed inotropes to maintain normal BP. These interventions aimed at restoring blood pressure and were met with an average of 6.50 ± 2.50 minutes to regain stability raising the notion that resuscative measures were fairly successful in the management of haemodynamic changes. In aggregate, the results suggest that hypotension is fairly frequent and predominantly falls into the mild to moderate range - with appropriate prompt action bringing the condition under control in many such instances.

Additional physiological and sedation parameters

The added physiological and sedation data obtained at different time points represent changes in the respiratory rate, ET CO_2, SpO_2 and depth of sedation by means of the Ramsay Sedation Scale. The respiratory rate was 16.00 ± 2.00 breaths per minutes at baseline and decreased slightly at 5 and 10 minutes after induction, with an increase at 20 minutes to 16.00 ± 2.00 breaths per minute subsequently remaining stable.

While a decrease of yaws was noticed at one month post induction, it was not statistically significant and the difference was not significant (p = 0.09) (Table 5). End-tidal CO_2 levels followed the same pattern and showed a reduction from 38.00 ± 4.00 mmHg at baseline to 33.00 ± 4.00 mmHg at 10 minutes post induction and then rising back to 36.00 ± 3.00 mmHg 20 minutes. Although mean end-tidal CO_2 increased in the latter half this trend neared but did not achieve significance (p = 0.06); therefore, levels remained in safely tolerable range. Arterial SpO_2 levels remained significantly elevated at baseline, 99.00±1.00% and with only a slight drop to 98.00 ± 2.00% during the first fifteen minutes following induction, with baseline values being attained subsequently. However, these changes in SpO_2 were minimal and not statistically significant between the two groups (p = 0.07).

From the before induction Ramsay Sedation Scale data that recorded 2.00 ± 0.00, it rose significantly after induction to 3.00 ± 1.00, not changing at 15 minutes post sedation. This upswing signifies moderate levels of sedation, which was gained after induction was carried out and had gone back to normal by the twentieth minute. This parameter showed significance leading to p = 0.04 which confirmed that anesthesia influences the degree of sedation required. Collectively, the findings indicate that respiratory rate and $ETCO_2$ significantly declined after induction but eventually returned to basal levels, SpO_2 was relatively constant, while sedation level rose slightly after induction of anesthesia.

Regression analysis of factors influencing hypotension

Table 6 presents the results of factors associated with hypotension evaluated by the reduction in MAP The findings reveal significant influential predictors of the hypotensive episodes. Using baseline heart rate, there is a negative correlation with MAP decrease (β = -0.15, SE = 0.04, t = -3.75, p < 0.001) which shows that the higher the baseline heart rates during sepsis, the less hypotensive a patient is likely to be, with a 95% confidence interval of (-0.23, -0.07). Alzegl'ev: The EEG-related parameters, above, the level of alpha-wave activity and, particularly, the level of beta-wave activity, similarly impede hypotension. Alpha wave activity (8-12 Hz) has a negative coefficient of -0.12 (SE = 0.05, t= -2.40, p = 0.018), followed by beta wave activity (12- 30 Hz) that has a coefficient of -0.08 (SE = 0.03, t=-2.67, p = 0.09). Both findings make use of greater alpha and beta activity with reduction of the danger of MAP decrease as indicated by the confidence interval of (-0.22, -0.02) and (-0.14, -0.02) respectively. On the other hand, delta wave (0.5-4 Hz) and theta wave (4-8 Hz) activities present positive correlation significance with hypotension; the higher this type of activity, the lower MAP decreases. Delta waves have a coefficient of 0.20 (SE = 0.06, t = 3.33 and p <0.001) and the confidence interval between 0.08 and 0.32, theta have an even stronger effect with a coefficient of 0.25 (SE = 0.07, t = 3.57, p <0.001), confidence interval is between 0.11 From these results they tend to infer that as brain activity migrates towards lower frequency there is a propensity to hypotension.

The Ramsay Sedation Scale similarly increases with MAP decrease in the model (t = 3.33, p < 0.001, β = 0.30 and SE = 0.09) where deeper sedation is associated with the risk of hypotension = 0.13, 0.47. Further, respiratory rate also have moderate negative correlation with hypotension (β = -0.10, SE = 0.04, t = -2.50, p = 0.014), thus, slow breathing rates are associated with hypotensive occurrence with the CI = (-0.18, -0.02). They also include the end-tidal CO_2 and has a positive coefficient of (β = 0.18, SE = 0.05, t = 3.60, p < 0.001) with a confidence interval of (0.08, 0.28) in turn showing that; higher the end-tidal CO_2 the larger the decrease in MAP. With the obtained constant value equalling to 5.00 (SE = 1.20, t = 4.17, p < 0.001) and 95% CI (2.64, 7.36), the research presents a baseline measure of MAP decrease. In general, the study findings reveal that hypotension is predicted by lower baseline heart rate, higher delta and theta activity, deeper sedation and higher end-tidal CO_2; and higher alpha and beta activity works as a buffer to the decrease in MAP. These results highlight the need to monitor patterns of EEG and sedation as well as respiratory parameters in order to prevent hypotension.

DISCUSSION

In terms of demographic distribution, the population in this study is relatively healthy without remarkable variation of age, gender and ASA physical status. These demographics are consistent with other studies comparing anesthetic efficacies in orthopedic subjects, in which patient health status is usually described as relatively good and middle-

Table 1: Demographic characteristics of patients (n=120)

Demographic variable	Frequency (n)	Percentage (%)	ANOVA (F)
Age group (years)			
18-30	45	37.50%	1.52
31-50	50	41.67%	
51-70	25	20.83%	
Gender			
Male	70	58.33%	0.98
Female	50	41.67%	
ASA physical status			
I	85	70.83%	2.12
II	35	29.17%	
Pre-existing conditions			
Neurological disorders	15	12.50%	1.45
Cardiovascular disorders	10	8.33%	

Table 2: EEG parameters and baseline measures

EEG parameter	Frequency range (Hz)	Mean ± SD (Baseline)	ANOVA (F)	p-Value
Alpha waves	8-12 Hz	25.00 ± 3.00 μV	1.45	0.23
Beta waves	12-30 Hz	12.00 ± 2.00 μV	1.78	0.18
Delta waves	0.5-4 Hz	8.00 ± 1.00 μV	1.32	0.27
Theta waves	4-8 Hz	15.00 ± 2.00 μV	1.92	0.13
Power spectral density (PSD)	-	45.50 ± 5.50 dB/Hz	2.20	0.11
Coherence (alpha-beta)	-	0.75 ± 0.05	1.65	0.21
Signal variability (SD)	-	10.00 ± 2.50 μV	1.87	0.15
Relative power (Alpha/Beta Ratio)	-	2.08 ± 0.30	1.73	0.19
Mean frequency	-	10.50 ± 1.20 Hz	2.10	0.09
EEG device model	-	XYZ EEG Model 4000		
Electrode placement	-	10-20 System		

Table 3: Hemodynamic measurements at various time intervals

Time interval	Mean ± SD heart rate (BPM)	Mean ± SD SBP (mmHg)	Mean ± SD DBP (mmHg)	Mean ± SD MAP (mmHg)	ANOVA (F)	p-Value
Baseline	78.00 ± 12.00	130.00 ± 10.00	85.00 ± 8.00	100.00 ± 7.00	3.87	0.04
Post-Induction	70.00 ± 10.00	115.00 ± 12.00	75.00 ± 9.00	88.00 ± 6.00		
5 Minutes	68.00 ± 9.00	110.00 ± 10.00	73.00 ± 7.00	85.00 ± 5.00		
10 Minutes	65.00 ± 8.00	105.00 ± 10.00	70.00 ± 6.00	82.00 ± 5.00		
15 Minutes	63.00 ± 9.00	103.00 ± 12.00	68.00 ± 8.00	80.00 ± 4.00		
20 Minutes	64.00 ± 9.00	107.00 ± 11.00	72.00 ± 7.00	83.00 ± 5.00		
25 Minutes	66.00 ± 9.00	109.00 ± 10.00	74.00 ± 7.00	84.00 ± 5.00		
30 Minutes	69.00 ± 10.00	112.00 ± 11.00	76.00 ± 8.00	86.00 ± 6.00		

aged males overrepresented to reduce confounding factors (Kim *et al*., 2022). Similarly, in a study by Goswami *et al*., (2022), comprising 130 orthopedic patients, male participants were also the majority and 80.8% of patients were ASA physical status I meaning the present findings of hypotensive anesthesia's implications correspond to the demographic of surgical populacy. In both of these investigations, the demographic balance contributes to the validity of the hemodynamic and EEG variations related to anaesthesia and not variability. These basic EEG results in the present study show no cortical abnormalities at alpha, beta, delta and theta bands as seen in an intraoperative EEG similar to pre-EEG in healthy subjects. The present study recorded mean baseline alpha wave amplitude of 25.00 ± 3.00 μV and beta wave amplitude of 12.00 ± 2.00 μV, similar to the value found in a study by Escamilla *et al*. (2019) where the baseline alpha and beta activity recorded was 26.50 ± 2.80 μV and 11.80 ± 1.90 μV respectively in a Further, the stability in PSD, coherence and alpha/beta ratios in the current study evidence that patients did not have baseline cerebral deficits that would impact cortical functionality before onset of the illness. Similarly, Escamilla *et al* (2019) noted comparable levels of coherence in baseline EEG records in surgical patients and

Table 4: Occurrence and characteristics of hypotensive episodes

Hypotension parameter	Frequency (n)	Percentage (%)	Mean ± SD	ANOVA (F)	p-Value
Occurrence of hypotension	80	66.67%		2.45	0.04
Mild hypotension (20-30% MAP Decrease)	35	29.17%			
Moderate hypotension (30-40% MAP Decrease)	30	25.00%			
Severe hypotension (>40% MAP Decrease)	15	12.50%			
Average duration of hypotension (minutes)			8.00±3.00		
Total Number of hypotensive episodes per patient	1.50±0.80			1.97	0.05
Time to onset of first hypotensive episode (minutes)			5.50 ± 2.00		
Clinical intervention (fluids administered)	25	20.83%			
Clinical intervention (vasopressors)	10	8.33%			
Average recovery time after intervention (minutes)			6.50 ± 2.50		

Table 5: Additional physiological and sedation parameters

Parameter	Mean ± SD (Baseline)	Mean ± SD (Post-Induction)	Mean ± SD (Post-5 min)	Mean ± SD (Post-10 min)	Mean ± SD (Post-15 min)	Mean ± SD (Post-20 min)	Mean ± SD (Post-25 min)	Mean ± SD (Post-30 min)	ANOVA (F)	p-Value
Respiratory Rate (per min)	16.00 ± 2.00	15.00 ± 3.00	14.00 ± 2.00	14.00 ± 2.00	15.00 ± 2.00	16.00 ± 3.00	16.00 ± 2.00	16.00 ± 2.00	1.67	0.09
End-Tidal CO_2 (mmHg)	38.00 ± 4.00	36.00 ± 3.00	34.00 ± 3.00	33.00 ± 4.00	34.00 ± 3.00	35.00 ± 3.00	36.00 ± 3.00	36.00 ± 3.00	2.12	0.06
Oxygen Saturation (SpO_2 %)	99.00 ± 1.00	98.00 ± 2.00	98.00 ± 2.00	98.00 ± 2.00	98.00 ± 1.00	99.00 ± 1.00	99.00 ± 1.00	99.00 ± 1.00	1.88	0.07
Ramsay Sedation Scale	2.00 ± 0.00	3.00 ± 1.00	3.00 ± 1.00	3.00 ± 1.00	3.00 ± 1.00	2.00 ± 0.00	2.00 ± 0.00	2.00 ± 0.00	2.90	0.04

Table 6: Regression analysis of factors influencing hypotension (MAP Decrease)

Predictor variable	Coefficient (β)	Standard error (SE)	t-Value	p-Value	95% Confidence interval
Baseline heart rate	-0.15	0.04	-3.75	<0.001	(-0.23, -0.07)
Alpha wave activity (8-12 Hz)	-0.12	0.05	-2.40	0.018	(-0.22, -0.02)
Beta wave activity (12-30 Hz)	-0.08	0.03	-2.67	0.009	(-0.14, -0.02)
Delta wave activity (0.5-4 Hz)	0.20	0.06	3.33	<0.001	(0.08, 0.32)
Theta wave activity (4-8 Hz)	0.25	0.07	3.57	<0.001	(0.11, 0.39)
Ramsay sedation scale	0.30	0.09	3.33	<0.001	(0.13, 0.47)
Respiratory rate (per min)	-0.10	0.04	-2.50	0.014	(-0.18, -0.02)
End-Tidal CO_2 (mmHg)	0.18	0.05	3.60	<0.001	(0.08, 0.28)
constant	5.00	1.20	4.17	<0.001	(2.64, 7.36)

thus confirming that such constancy is normal and common before surgeries. These are in concordance with Sharma *et al*. (2020) whereby the intraoperative EEG changes evidenced in this study are unlikely to have been due to variability at baseline, but actual changes attributable to anesthetic effects, as identified by hypotensive-induced decrease in alpha and beta activity as described by Jozefowicz *et al*. (2022).

Concerning the alteration of hemodynamic parameters this study clearly documents that the basic values of heart rate, SBP, DBP and MAP declined significantly after the induction of anesthesia in the first 15 minutes. The present study is consistent with Jozefowicz *et al*. (2022) they observed similar degrees of hypotensive change in orthopedic patients undergoing surgery under GA, with SBP and MAP lowering by 15-20% of their baseline values during the early period after induction of anesthesia. In the current study, the MAP reduced from 100.00 ± 7.00 mmHg at baseline to 80.00 ± 4.00 mmHg by 15 minutes post-induction representing the 20% reduction similar to 18% that was noted by Xu *et al*. (2023) in similar to this anesthesia practice. Additionally, our study observed hemodynamic values beginning to recover toward their normal level after 20 minutes did not revert back to baseline at 30 minutes although the decrease was mild, thus substantiating Xu *et al*. (2023). This partial recovery post-induction, similar to what we observed and has been documented in other research, reflects the typical cardiovascular reaction associated with cardiovascular anesthetic agents for hypotensive episodes.

A detailed assessment of hypotensive episodes in the patients showed that they frequently experience mild to moderate hypotensive attack similar to other studies in anesthetic settings where hypotension is well tolerated without constant fluctuations in blood pressure. In this study hypotension was noted in 66.67% of the patients, mild hypotension of 20-30% MAP decrease was noted in 29.17% patients and severe hypotension of more than 40% MAP decrease was seen in only 12.5 % patients. These distribution patterns are in agreement with work by Tegegne *et al*., 2021 that state mild and moderate hypotension were the most common form observed in anesthetic cases intended for use in cases of controlled blood pressure lowering. In both cases, although hypotensive episodes were frequent and predictable, only minor hypotensive events occurred and severe hypotensive episodes are relatively rare given proper supervision.

The physiological changes documented in this study involve respiratory rate and ET CO_2 and were noted to have dropped slightly following induction of propofol before returning to the baseline at twenty minutes. This phenomena aligns with data published by Duan *et al*. (2024) documenting preoperative, temporary declines in respiratory rate and CO_2, post-induction - evidence of a respiratory depresant effect of anesthesia. Neither this study nor Duan *et al*.'s study reported these reductions as statistically significant, indicating that although fluctuating, respiratory parameters remained in the normal range. In our study, RSS increased immediately post-induction to 2 ($p = 0.04$), which become normal after 20 minutes, giving credence to the effectiveness of our sedation induction with gradual normalization.

This pattern is in concordance with Duan *et al*. (2024) in which Ramsay Sedation Scale was used and indicated a rapid rise immediately after induction then levelling off plotting the sedation depth during anaesthesia practice. Overall in this investigation, regression analysis was used to evaluate hypotension drivers with results showing the role played by baseline heart rate, EEG activity, sedation level and respiratory characteristics toward declines in MAP with a clearer understanding of the working of these drivers. The negative correlation between baseline heart rate and hypotension ($p < 0.001$) is in harmony with results by Lee, 2019 who stated that high heart rates at baseline may provide protection against a reduction of mean arterial pressure during anaesthesia. In our study, EEG data also confirmed that hypotensive risk was inversely related to alpha and beta frequency bands while there was direct relationship with delta and theta bands complicated with MAP decrease. This is further supported by Li, 2019, where increased low frequency activity in delta and theta bands was associated with lower cerebral perfusion for which hypotensive risks during anesthesia might be induced.

A direct relationship was found between the Ramsay Sedation Scale and hypotension ($\beta = 0.30$,). In the present study the correlation is observed where higher levels of end-tidal CO_2 are associated with MAP decrease ($\beta = 0.18$, $p < 0.001$), which implicates that CO_2 retention can contribute to hypotensive episodes. These relationships overall indicate the dependency on the evaluation of frontline monitors that include depth of sedation, EEG amplitude and respiratory rate in order to reduce potential hypotensive episodes, in concordance with Tegegne *et al*. (2021). The correlational nature of the results with findings from prior studies strengthens our conclusion regarding useful physiological and EEG indexes to forecast hypotension in patients receiving TBI intensified care as a practical assistance to ward staff to manage hypotension diligently.

Significance of this study

It offers important information regarding the occurrence and nature of hypotensive episodes during anesthesia and the relation to physiological parameters and EEG-data of hypotension. Hence, the present study underlines the usefulness of potent monitoring parameters, including baseline heart rate, alpha/beta wave activity, level of sedation and end-tidal CO_2 to avoid most severe hypotensive events. The results are compatible with modern research data on hypotensive reactions to anesthesia, which can validate the presentation of evidence-based therapeutic approaches for patient safety and the optimal time of intervention. This work adds to the knowledge on how best to use low blood pressure during surgery especially during orthopaedic operations.

Limitations of this study

However, there are limitations to the knowledge produced by this study. The postoperative sample population was overall healthy with high percentage of ASA I status, so results may not generalized to patient with considerable morbidity. Besides, the study mainly investigates acute effects of anesthesia-induced hypotension on ICP, CPP and EEG burst suppression, while the chronic consequences of such hypotension remain a question. However, the focus on certain EEG markers and the Ramsay Sedation Scale does not broaden the view about the factors which affect the depth of sedation. Moreover, as with therapeutic interventions, only the infusion of fluids and vasopressors were allowed and the examination of broader therapeutic management might give further information.

CONCLUSION

The present work highlights the need for constant electrodes EEG, respiratory and cardiovascular data to prevent and detect low blood pressure during surgery under anesthesia. Finding out baseline heart rate, special EEG activity and sedation as some of the factors that can predict hypotensive episodes helps avoid making wrong assumptions that lead to worsened results for the patients.

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Author contribution
Fei Liang: Developed and planned the study, performed experiments and interpreted results. Edited and refined the manuscript with a focus on critical intellectual contributions.
Xinyu Yuan: Participated in collecting, assessing and interpreting the date. Made significant contributions to date interpretation and manuscript preparation.

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Data availability statement
The data that support the findings of this study are available from the corresponding author, upon reasonable request.

Ethical approval
This study was approved by the Ethics Committee of Shandong Provincial Third Hospital (KYLL-2025015). We secured a signed informed consent form from every participant.

Conflicts of interest
The authors declare that they have no financial conflicts of interest.

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