RESEARCH

Correlation Between Electroencephalography-Based Sedation Indices (BIS and qCON) and Mean Arterial Pressure (MAP) in Sedated Postoperative Patients in the Intensive Care Unit

ABSTRACT

Background: Sedation is a critical component in the management of postoperative patients in the intensive care unit (ICU). Electroencephalography (EEG)-based indices, such as the bispectral index (BIS) and quantum consciousness index (qCON), are increasingly used to objectively assess sedation depth. However, their relationship with hemodynamic parameters, particularly mean arterial pressure (MAP) as a reflection of sympathetic activity, remains unclear.

Objective: To evaluate the correlation between BIS and qCON with MAP in sedated postoperative patients upon ICU admission.

Methods: This analytical observational study with a cross-sectional design included 38 postoperative patients admitted to the ICU. All patients received a standardized sedative-analgesic regimen of midazolam and fentanyl. Sedation depth was assessed using BIS and qCON obtained from the CONOX® monitor sequentially, while MAP was measured 10 minutes after sedation was administered. Data were analyzed using Spearman's correlation test, with a p-value < 0.05 considered statistically significant.

Results: The median BIS and qCON values were 61 (59–71) and 60 (59–70), respectively, indicating moderate sedation. The mean MAP was 90.9 ± 10.2 mmHg. BIS showed a moderate positive correlation with MAP ($\rho = 0.481$, $p = 0.017$), with an R^2 of 0.231, while qCON also demonstrated a moderate positive correlation with MAP ($\rho = 0.433$, $p = 0.022$), with an R^2 of 0.187. These findings indicate that higher sedation index values, reflecting lighter sedation, were associated with higher MAP.

Conclusion: BIS and qCON are moderately correlated with MAP in sedated postoperative ICU patients, indicating an association between sedation depth and sympathetic activity. However, the modest strength of this relationship suggests that MAP is influenced by multiple factors beyond sedation depth.

Keywords: BIS; ICU; mean arterial pressure; postoperative; qCON; sedation

INTRODUCTION

Sedation and analgesia are essential components in the management of critically ill patients in the intensive care unit (ICU), particularly in postoperative patients requiring mechanical ventilation and adequate pain control. The primary goals of sedation are to reduce physiological stress, suppress excessive sympathetic responses, and improve patient comfort as well as ventilator synchrony.[1] Inappropriate sedation depth, whether in the form of over-sedation or under-sedation, has been associated with increased morbidity, hemodynamic instability, and poor clinical outcomes in ICU patients.[2]

Objective assessment of sedation depth has evolved toward electroencephalography (EEG)-based monitoring, particularly the bispectral index (BIS) and the quantium consciousness index (qCON), which reflect cortical activity and the level of central nervous system depression. While BIS has been extensively validated and is widely incorporated into ICU sedation protocols, qCON is a more recently developed multiparametric EEG-derived index that remains relatively uncommon in routine ICU practice; consequently, additional clinical evidence is still needed to support its applicability in critically ill populations.[3]

In ICU practice, analgesia is commonly achieved using a combination of sedatives and opioids, such as midazolam and fentanyl, which influence both neurological and hemodynamic responses through complex and often non-linear pharmacodynamic interactions.[4,5] Mean arterial pressure (MAP) is a key parameter reflecting organ perfusion and is influenced by autonomic nervous system balance, particularly sympathetic activity. Activation of the sympathetic nervous system, such as in conditions of pain or inadequate sedation, increases catecholamine release, leading to elevated heart rate, vasoconstriction, and increased MAP. Conversely, deeper levels of sedation suppress sympathetic outflow, resulting in vasodilation, reduced systemic vascular resistance, and decreased MAP. Therefore, MAP may be an indirect marker of sympathetic nervous system activity in sedated patients.[6]

The relationship between EEG-based sedation indices (BIS and qCON) and hemodynamic parameters such as MAP is not always linear or consistent in ICU patients. This inconsistency may be explained by multiple confounding factors, including underlying disease severity, vasoactive drug use, pharmacological interactions, and interindividual variability.[4,7,8]

Despite the increasing use of EEG-based monitoring in ICU sedation, current evidence remains heterogeneous and insufficient to fully explain its relationship with clinical outcomes and physiological responses. Thus, there remains a significant knowledge gap regarding the relationship between cortical activity suppression, as measured by BIS and qCON, and sympathetic nervous system responses reflected by MAP in critically ill patients.[9] Evaluating both indices simultaneously in this clinical context may also contribute to the limited body of evidence on qCON in ICU sedation monitoring, an area where data remain considerably less robust than for BIS. Therefore, this study aims to evaluate the correlation between MAP and EEG-based sedation depth parameters (BIS

and qCON) in sedated postoperative patients in the ICU.

METHODS

This analytical observational study employed a cross-sectional design involving postoperative patients admitted to the ICU at Adam Malik Hospital, Medan, and Haji General Hospital, Medan (North Sumatera Provincial Hospital). Ethical approval was obtained from the Health Research Ethics Committee of Universitas Sumatera Utara, along with research permits from the respective hospitals. Written informed consent was obtained from all patients and/or their legal representatives prior to participation in the study.

This study included adult postoperative patients aged 18–60 years who underwent non-neurosurgical surgery under general anesthesia and were admitted to the ICU postoperatively without metabolic and neurological dysfunction. Exclusion criteria comprised patients with a preoperative Glasgow coma scale (GCS) score < 15, a history of central nervous system disorders, hearing impairment, and those undergoing combined general and regional anesthesia techniques. During the observation period, all patients received a standardized sedative-analgesic regimen consisting of midazolam at 0.05 mg/kg/h and fentanyl at 0.5 mcg/kg/h.

During the observation period, patients receiving low-dose vasopressor support for transient postoperative hypotension were not excluded, as this reflects routine clinical practice in postoperative ICU care. However, the type and dose of vasoactive agents, intraoperative fluid balance, and postoperative pain scores were not systematically recorded as part of this study and therefore could not be incorporated into the analysis. This decision was made to preserve the real-world applicability of the findings while acknowledging the inherent variability in postoperative ICU populations.

The sample size for this study was calculated using a standard formula for correlation analysis of numerical variables, incorporating the expected correlation coefficient, type I error ($\alpha = 0.05$), and type II error ($\beta = 0.20$) to ensure adequate statistical power. Based on these parameters and an anticipated correlation coefficient of 0.5, the minimum required sample size was determined to be 38 subjects.[8]

Baseline demographic data, including age, sex, body mass index, ASA physical status, type of surgery, and duration of surgery, were recorded upon ICU admission. At the same time, sedation depth was assessed using the BIS and the qCON index obtained from the CONOX® monitor (Fresenius Kabi, Germany), while hemodynamic parameters, including systolic and diastolic blood pressure, MAP, and heart rate, were simultaneously measured and recorded.

All study variables were measured upon ICU admission using standardized monitoring methods. Sedation depth was assessed using EEG-based parameters, including the BIS and the qCON, obtained from the CONOX® monitor (Fresenius Kabi, Germany). Both indices range from 0 to 100, with lower values indicating deeper levels of sedation.[10]

Mean arterial pressure (MAP), as the primary hemodynamic parameter, was calculated using the standard formula:

MAP = (Systolic Blood Pressure + (2 × Diastolic Blood Pressure)) / 3

These parameters were measured using standard bedside monitoring and reflect the balance of systemic vascular resistance and cardiac output. Hemodynamic parameters measurements, including MAP, were recorded 10 minutes after analgosedative regimens were administered; EEG-based parameters were recorded sequentially within 30 minutes upon postoperative ICU admission to ensure temporal consistency and reduce measurement bias.

Data were analyzed using SPSS version 29. Categorical variables were presented as frequencies and percentages (n [%]), while continuous numerical data were presented as median values (minimum–maximum). Normality of numerical data was assessed using the Shapiro–Wilk test. The correlations between BIS and qCON with MAP values were analyzed using Spearman's correlation test due to non-normal data distribution. A p-value < 0.05 was considered statistically significant.

RESULTS

The study included a total of 38 postoperative patients who met the inclusion criteria. The mean age of the participants was 46.6 ± 11.52 years, ranging from young adults to the elderly. The gender distribution showed a predominance of male patients (63.2%) compared to females (36.8%). In terms of physical status, the majority were classified as ASA III (63.2%), indicating patients with severe systemic disease, while the remaining 36.8% were ASA II. The types of surgery varied, with digestive surgery being the most common procedure (44.7%), followed by oncologic surgeries (18.4%). The detailed demographic and clinical characteristics are summarized in Table 1.

Table 2 demonstrates that patients were hemodynamically stable upon ICU admission, with a mean heart rate of 81.18 ± 12.94 beats/min and a mean MAP of 90.9 ± 10.2 mmHg. Blood pressure values were within acceptable clinical ranges, with a median systolic blood pressure of 120 mmHg (95–171) and a mean diastolic blood pressure of 76.18 ± 8.77 mmHg. Oxygenation was well preserved, as reflected by a median SpO_2 of 99% (97–100).

Table 3 shows that the median BIS and qCON values were 61 (59–71) and 60 (59–70), respectively, indicating a relatively homogeneous depth of sedation within the moderate range. The close agreement between BIS and qCON suggests consistency between EEG-based indices in reflecting cortical suppression at the time of ICU admission.

Tables 4 and Table 5 demonstrates statistically significant moderate positive correlations between EEG-based sedation indices (BIS and qCON) and MAP upon ICU admission. BIS showed a moderate correlation with MAP ($\rho = 0.481$, $p = 0.017$), with an R^2 value of 0.231, indicating that approximately 23.1% of MAP variability could be explained by BIS. Similarly, qCON demonstrated a moderate positive correlation with MAP ($\rho = 0.433$, $p = 0.022$), with an R^2 value of 0.187, suggesting that 18.7% of MAP variability was attributable to qCON.

Notably, BIS exhibited a slightly stronger correlation and higher explanatory power compared to qCON, suggesting a marginally greater

sensitivity in reflecting hemodynamic changes. Overall, these findings indicate that lighter levels of sedation, as reflected by higher EEG-derived indices, are associated with increased MAP, supporting the physiological link between cortical activity and sympathetic nervous system activation. However, the moderate strength of these correlations and relatively low R^2 values also highlight the multifactorial nature of hemodynamic regulation in critically ill patients.

Table 1. Characteristics of the study sample

Variable	Total (n = 38)
Sex, n (%)	
Male	24 (63.2)
Female	14 (36.8)
Age, years (Mean ± SD)	46.6 ± 11.52
Body weight, kg (Mean ± SD)	67.79 ± 7.98
Height, cm (Mean ± SD)	165.32 ± 4.75
Body mass index (BMI), kg/m² (Mean ± SD)	24.78 ± 2.61
ASA Physical Status, n (%)	
ASA II	14 (36.8)
ASA III	24 (63.2)
Duration of surgery, minutes (Mean ± SD)	172.37 ± 80.77
Type of surgery, n (%)	
Digestive	17 (44.7)
Genitourinary	2 (5.3)
Gynecologic	4 (10.5)
Skin and soft tissue	2 (5.3)
Orthopedic	2 (5.3)
Thoracic and cardiovascular	2 (5.3)
Plastic surgery	1 (2.6)
Oncologic	7 (18.4)
Spinal neurosurgery	1 (2.6)

Table 2. Hemodynamic parameters upon ICU admission

Variable	Total (n = 38)
Heart rate, beats/min	81.18 ± 12.94
Systolic blood pressure (SBP), mmHg	120 (95 – 171)
Diastolic blood pressure (DBP), mmHg	76.18 ± 8.77
Mean Arterial Pressure (MAP), mmHg	90.9 ± 10.2
Oxygen saturation (SpO_2), %	99 (97 – 100)

Table 3. BIS and qCON values upon ICU admission

Variable	Total (n = 38)
BIS, (Median [Min – Max])	61 (59–71)
qCON, (Median [Min – Max])	60 (59–70)

Table 4. The correlation between BIS and MAP upon ICU admission

BIS	MAP	ρ	R^2	p-*value*
61 (59–71)	90.9 ± 10.2	0.481	0.231	0.017

Spearman's Correlation Test

Table 5. The correlation between qCON and MAP upon ICU admission

qCON	MAP	ρ	R^2	p-*value*
60 (59–70)	90.9 ± 10.2	0.433	0.187	0.022

Spearman's Correlation Test

DISCUSSION

The present study demonstrated a statistically significant moderate positive correlation between EEG-based sedation indices (BIS and qCON) and MAP upon ICU admission, indicating that lighter levels of sedation were associated with higher MAP. This finding is consistent with the fundamental physiological concept that inadequate sedation or nociceptive stimulation leads to activation of the sympathetic nervous system, resulting in increased catecholamine release, vasoconstriction, and elevation of arterial blood pressure.[10] Such responses have been widely described in critically ill patients, where stress and pain contribute significantly to hemodynamic fluctuations.[12]

The observed correlation between BIS and MAP ($\rho = 0.481$) and between qCON and MAP ($\rho = 0.433$) supports the notion that EEG-derived indices reflect, at least partially, the physiological consequences of sedation depth on autonomic function. Previous studies have demonstrated that BIS monitoring provides a continuous and objective assessment of cortical activity, allowing clinicians to titrate sedative agents more accurately and potentially reduce physiological stress responses.[13]

Although the Spearman's ρ values indicated moderate correlations, the low R^2 values ($R^2 = 0.231$ and 0.187) suggest that only a limited proportion of the variability in the EEG parameters could be explained by mean arterial pressure, implying a substantial influence of confounding factors. These may include, among others, anesthetic depth, anesthetic agents, cerebral autoregulation, arterial carbon dioxide tension, body temperature, and other physiological variables that affect EEG activity. Because these potential confounders were not evaluated or adjusted for in the present study, this represents a major limitation and warrants caution when interpreting the observed associations.

This finding aligns with previous literature indicating that the relationship between EEG-based sedation indices and systemic physiological responses is often inconsistent. A systematic review and meta-analysis reported that the correlation between BIS and clinical or physiological parameters in critically ill patients is heterogeneous and frequently limited by interindividual variability and complex ICU conditions.[14] Furthermore, studies conducted in ICU populations have shown that EEG-derived indices do not always correlate strongly with clinical or physiological outcomes. For instance, research in patients with severe traumatic brain injury demonstrated poor correlation between BIS values and clinical sedation parameters, as well as

no significant relationship with other physiological variables, highlighting the variability of EEG responses in critically ill states.[15]

The moderate correlation observed in this study may be explained by the multifactorial nature of MAP regulation in critically ill patients. Hemodynamic status in the ICU is determined by a complex interplay between cardiac output, systemic vascular resistance, intravascular volume status, and the use of vasoactive agents, with large-scale observational data demonstrating that MAP targets and achieved values vary widely across patient populations and clinical conditions.[16] Sedation itself has also been shown to influence MAP, often necessitating additional hemodynamic support, particularly in patients with higher illness severity or pre-existing cardiovascular compromise.[17]

Furthermore, considerable interindividual variability in hemodynamic responses, influenced by disease severity, age, baseline hemodynamic status, and pharmacological interactions, contributes to the non-linear relationship between sedation depth and MAP. Together, these findings suggest that while EEG-based indices such as BIS and qCON reliably reflect cortical suppression, they may not fully capture downstream autonomic and cardiovascular responses, since systemic hemodynamic regulation extends beyond central nervous system activity alone. Interestingly, BIS demonstrated a slightly stronger correlation with MAP compared to qCON, suggesting a marginally higher sensitivity in detecting changes related to sympathetic activity. BIS has been extensively validated and remains one of the most widely used EEG-derived indices for monitoring sedation depth, with several studies demonstrating its responsiveness to changes in cortical activity and anesthetic depth in both operative and critical care settings.[18,19] This broader validation and clinical familiarity may contribute to its slightly stronger association with physiological parameters such as MAP, particularly in dynamic ICU environments where rapid fluctuations in sedation depth and autonomic responses occur.

However, qCON has emerged as a promising alternative EEG-based parameter for evaluating sedation depth, utilizing multiparametric signal processing to provide a continuous and objective assessment of consciousness levels. Several studies have demonstrated that qCON performs comparably to BIS in assessing sedation depth, with good agreement across different sedation ranges, supporting its validity as a reliable monitoring tool.[20]

Although both BIS and qCON are derived from EEG signals, differences in their underlying algorithms and signal processing techniques may account for the observed variation. BIS is calculated using bispectral analysis incorporating phase relationships and frequency components of EEG signals, whereas qCON is derived from multiparametric spectral analysis designed to optimize responsiveness across different anesthetic agents. Despite these methodological differences, previous comparative studies have shown a good level of agreement between BIS and qCON across various levels of sedation, supporting their concurrent validity in assessing depth of sedation.[21]
Nevertheless, the relatively close values between BIS and qCON observed in this study indicate that both parameters provide comparable clinical information

regarding sedation depth. This finding is consistent with emerging evidence suggesting that newer EEG-based indices, including qCON, can serve as reliable alternatives to BIS in clinical practice, particularly when integrated into multimodal monitoring strategies.[20]

However, despite these promising results, data regarding the performance and clinical applicability of qCON in critically ill ICU patients remain limited. By demonstrating a strong agreement between qCON and BIS in mechanically ventilated critically ill patients, the present study helps address this knowledge gap and provides additional evidence supporting the use of qCON in the intensive care setting. Therefore, both BIS and qCON may be considered complementary tools in ICU sedation monitoring, allowing clinicians to better assess cortical suppression while acknowledging the inherent limitations of EEG-based metrics in reflecting systemic physiological responses.

This study has several limitations. The cross-sectional nature of this study represents an important limitation. Because sedation depth and MAP were assessed simultaneously at ICU admission, the findings reflect only a single-point snapshot of their relationship and may not capture temporal variations occurring during the ICU stay. Moreover, the study design precludes any inference of causality, and the observed associations should therefore be interpreted as correlational rather than causal. Future longitudinal studies incorporating repeated measurements are warranted to evaluate how the relationship between sedation depth and hemodynamic parameters evolves over time. The relatively small sample size and restricted population of postoperative patients without neurological impairment may limit generalizability to broader ICU populations. Importantly, MAP is regulated by multiple physiological and pharmacological factors beyond sedation depth, including the use of vasoactive medications, intravenous fluid administration, postoperative pain, surgical stress, and underlying comorbidities. These variables were not systematically standardized, recorded, and analyzed as part of the study protocol, which represents an important limitation. Consequently, the observed correlations between EEG-based sedation indices and MAP should be interpreted as reflecting broader hemodynamic associations rather than direct, isolated effects of sedation depth on blood pressure. Although a standardized midazolam–fentanyl regimen was applied, interindividual variability in pharmacodynamic response may have affected both EEG indices and hemodynamic parameters. Furthermore, BIS and qCON primarily reflect cortical activity and may not fully represent autonomic responses, which could explain the moderate and non-linear relationship observed with MAP.

CONCLUSION

This study demonstrates that both BIS and qCON show statistically significant moderate positive correlations with MAP in sedated postoperative patients upon ICU admission. These findings suggest that lighter levels of sedation, as reflected by higher EEG-derived indices, are associated with increased MAP, supporting the physiological link between cortical activity and sympathetic nervous system responses. However, the moderate strength of the correlations indicates that MAP is influenced by multiple factors beyond sedation depth alone. Both BIS and qCON provide comparable clinical

information in assessing sedation depth, highlighting their potential utility as complementary tools in ICU sedation monitoring.

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