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Original Article

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Effect of Adding Dexmedetomidine to 0.5% Bupivacaine on Scalp Block on Intraoperative Hemodynamics During the First Hour of Surgery and Anesthetic Requirement in Intracranial Surgery

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ABSTRACT

OBJECTIVE Dexmedetomidine has been used as a perineural local anesthetic (LA) adjunct to improve the quality of block and decrease opioid consumption. This study aims to determine the efficacy of adding dexmedetomidine to 0.5% bupivacaine on scalp block on hemodynamic responses during the first hour of surgery, intraoperative propofol and fentanyl doses, and analgesic requirements in the first 24 hours post-craniotomy.

METHODS A prospective randomized controlled trial was conducted in forty-seven elective craniotomy patients receiving a scalp block with either 1 mcg/kg of dexmedetomidine (group D) or normal saline (group C) added to 0.5% bupivacaine (20 mL in total). Intraoperative blood pressure and heart rate at baseline and at 22 other time points during the first hour following the skin incision as well as the amount of intraoperative propofol and fentanyl and postoperative tramadol doses during the first 24 hours were collected and analyzed. The student t-test was used to compare means between groups, while repeated measure ANOVA with Bonferroni correction was used for comparing repeated means within each group. *P*-value less than 0.05 was considered statistically significant.

RESULTS During skull pin fixation (T4), the mean arterial pressure (MAP) and heart rate (HR) of both groups increased from baseline, but there were no statistically significant differences between groups. During the first hour of the operation (T7–T22), both groups had lower MAPs than their baselines, and group D had lower MAPs than group C at all time points. Intraoperative doses of fentanyl (mcg/kg) and propofol (mg/kg) in group D were significantly lower than those in group C, $p = 0.027$ and $p = 0.030$, respectively.

CONCLUSIONS The addition of 1 mcg/kg dexmedetomidine tends to enhance the efficacy of scalp block with 0.5% bupivacaine in attenuating intraoperative hemodynamic responses during the first hour of surgery and reducing intraoperative fentanyl and propofol requirements during intracranial surgery.

KEYWORDS scalp block, dexmedetomidine, bupivacaine, hemodynamic response

INTRODUCTION

Stabilizing hemodynamics in patients undergoing intracranial surgery is vital, particularly in patients at risk due to impaired cerebral autoregulation caused by brain pathology. Not only does this improve surgical outcomes, but it also helps prevent complications such as intracerebral hemorrhage, brain ischemia, and ruptured aneurysms caused by acute hemodynamic changes. Slightly higher blood pressure (BP) during neurosurgical procedures such as skull pin insertion, skin incision, or dural opening can result in increased cerebral blood flow and cerebral blood volume, followed by an increase in intracranial pressure (1, 2). For craniotomy, blocking the nerves that supply the relevant region of the scalp can attenuate the nociceptive response and thus control high BP and tachycardia. Additionally, it has shown evidence of effectively controlling post-operative pain in patients undergoing craniotomy for supratentorial brain lesions (3-5).

In neurosurgical anesthesia, an intravenous bolus or infusion of dexmedetomidine as an anesthetic adjuvant can reduce sympathetic response and hemodynamic variability associated with skull pin application and during an operation (6, 7) and can reduce intraoperative opioid usage (8). Nowadays, dexmedetomidine has been extensively added to local anesthetics as a perineural adjunct to prolong the duration of analgesia, sensory block, and motor block (9). However, the efficacy of dexmedetomidine in combination with local anesthetics in scalp blocks has a had only a limited number of clinical research studies. The primary purpose of this study was to determine whether there was any difference in hemodynamic response between scalp block with bupivacaine and scalp block with bupivacaine and dexmedetomidine, especially during intense pain stimuli such as skull pin insertion, skin incision, and dural opening during intracranial surgery. The secondary objective of the study was to determine if the addition of dexmedetomidine to bupivacaine had any effects on intraoperative opioid consumption, propofol use, and postoperative analgesic requirements.

METHODS

This single-center, prospective randomized controlled trial was conducted at Maharaj Nakorn Chiang Mai Hospital from April 1, 2020 to August 31, 2022. The trial enrolled 48 patients who underwent an elective craniotomy to remove intracranial tumors. The study was approved by the Institutional Review Board of the Faculty of Medicine, Chiang Mai University (ANE-2562-06956), and registered on Thaiclinicaltrials.org (ID: TCTR20200106002). Written informed consent was obtained from patients who met the inclusion criteria one day before the scheduled surgery who were then assigned in a 1:1 ratio either to a study group receiving scalp block with dexmedetomidine and bupivacaine or to a group receiving bupivacaine only (Figure 1). The inclusion criteria were age 18 to 65 years, ASA physical status I-III, Glasgow Coma Scale scores 14-15, and scheduled for elective craniotomy. Exclusion criteria included known allergic reactions to bupivacaine and/or dexmedetomidine, coagulopathy, hypertension, clinically increased intracranial pressure, history of bradyarrhythmia, history of myocardial ischemia or history of traumatic brain injury.

Procedures

The random allocation for both arm groups was generated using a computer-generated randomization sequence. Each random number was then concealed in an opaque, sealed envelope prepared by personnel not connected with the study. A nurse anesthetist not participating in the anesthetic care of the patients opened each envelope and prepared the drugs immediately before the scalp block was performed. The patients, anesthetists, neurosurgeons, data collectors, and outcome assessors were all blinded to the randomization assignments.

Before induction of anesthesia, BP, pulse oxygen saturation, heart rate (HR), and ECG rhythm were monitored. After performing a radial arterial catheterization for direct BP measurement, the patient's baseline (T0) hemodynamic paameters were recorded. All patients received pre-oxygenation with 100% oxygen for three to five minutes, fentanyl 2 mcg/kg IV, and propofol IV starting at an effect-site concentration (Ce) of 3.0 mcg/mL by a target-controlled infusion (TCI) using a Schneider model syringe pump. Propofol was titrated to maintain bispectral index (BIS) values in the range of 40-60, followed by rocuronium at 0.8 mg/kg IV to enable endotracheal intubation. Scalp block was performed in both arm

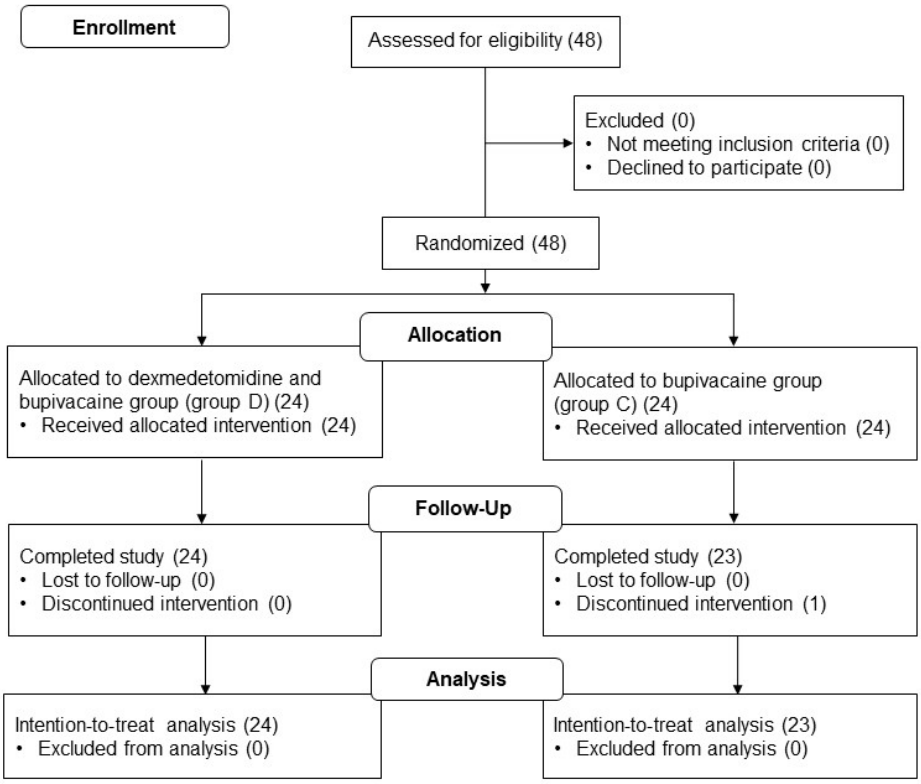

Figure 1. Consort flow diagram

groups who received a 20 mL solution containing either dexmedetomidine 1 mcg/kg (0.01 mL/kg) and 0.5% bupivacaine (group D) or 0.9% NaCl 0.01 mL/kg and 0.5% bupivacaine (group C). Six nerves supplying the scalp (supratrochlear, supraorbital, zygomaticotemporal, auriculotemporal, greater occipital, and lesser occipital nerves) were blocked bilaterally using the technique described by Pinosky et al. (10). Then 2% lidocaine 2 mL was injected at each skull pin site marked before skull pin application by a neurosurgeon and applied after five minutes of scalp block. Anesthesia was maintained with propofol TIVA-TCI, rocuronium, and fentanyl to achieve balanced anesthesia. If the BP and HR increased by more than 20% from baseline (T0), intraoperative fentanyl 0.5-1 mcg/kg was intravenously administered, with supplemental nicardipine or esmolol IV if the BP and HR still increased by more than 20% from baseline after being given fentanyl 1 mcg/kg. Bradycardia was treated by the administration of IV atro-pine 0.6 mg. Hypotension was treated by the administration of IV ephedrine in 3 mg boluses or IV norepinephrine in 10 mcg boluses. After the operation was finished, neuromuscular blockade was reversed with 0.05 mg/kg neostigmine and 0.02 mg/kg atropine. Removal of the endotracheal tube was done if the patient fulfilled the criteria for extubation before transfer to the neurosurgical intensive care unit. Intravenous tramadol was used postoperatively as rescue analgesia.

Systolic BP (Ps), diastolic BP (Pd), mean arterial pressure (MAP), and HR were recorded and analyzed at 23 timepoints: 1) T0 at baseline, 2) T1 before scalp block, 3) T2 one minute after scalp block, 4) T3 before a skull pin fixation, 5) T4 during a skull pin fixation, 6) T5 one minute after a skull pin fixation, 7) T6 five minutes after a skull pin fixation, 8) T7 during skin incision, 9) T8 one minute after skin incision, 10) T9 to T20 every five minutes for one hour after skin incision, 11) T21 during skull opening, and 12) T22 during the dural opening. Perioperative data, including total propofol infusion dose, total fentanyl dose given, adverse hemodynamic events, e.g., hypotension (MAP < 65 mmHg or decrease > 20% of baseline and treatment with a vasopressor drug), hypertension (treated with an anti-hypertensive drug), and bradycardia (HR < 60 /min), and postoperative analgesic requirements with tramadol IV in 24 hours were all collected.

Sample size calculation and statistical analysis

Based on a previous study by Kumar et al. (11), two repeated mean changes of baseline pre-block MAP and during skull-pin fixation MAP in two independent groups were used: 86.37±10.55 to 110.37±17.39 mmHg in the control group, and 83.22±11.13 mmHg to 89.46±16.56 mmHg in the dexmedetomidine with bupivacaine group, respectively. For a desired power of 0.80 and a type I error of 0.05, the total number of participants in both study arms was 48, including 20% in case some participants dropped out of the study.

Discrete categorical data are presented as frequency (percent) and are compared between groups using chi-square or Fisher's exact test as appropriate. Continuous data such as arterial pressure and HR are presented as mean ± SD and compared between groups using the independent t-test for normal distribution data or the Mann-Whitney U test for non-normal distribution data, while repeated measure ANOVA with Bonferroni correction was used for comparing repeated means within the group. A P-value less than 0.05 was considered statistically significant. All statistical analyses were performed using Stata statistical software version 16.1 (StataCorp LLC, College Station, TX, USA).

RESULTS

Forty-eight patients were enrolled in the study by randomization into group D (24 patients) and group C (24 patients). However, 1 patient from group C was excluded from the study due to a technical error in data collection. No significant differences were found between groups in demographic characteristics (Table 1). The surgical sites were not different between the two groups and were mostly performed in the frontal and frontotemporal regions. The time from scalp block to skull pin insertion was 13.00±6.26 minutes in group D and 13.17±5.73 minutes in group C, p = 0.921. The operating-room extubation rate was

Table 1. Demographics data

Variable	Group D n=24 (%)	Group C n=23 (%)	*p*-value
Gender (male/female) (%)	6/18 (25.0/75.0)	9/14 (39.1/60.9)	0.359
Age (years)	48.29±10.74	47.00±13.32	0.716
BMI (kg/m^2)	23.10±2.43	23.88±4.45	0.457
GCS score	15	15	
ASA PS (1/2/3) (%)	6/16/2 (25.0/66.7/8.3)	5/17/1 (21.7/74.0/4.3)	0.963
Surgical diagnosis (%)			0.008
Meningioma	12 (50.0)	3 (13.0)	
Glioma	4 (16.7)	2 (8.7)	
Astrocytic tumor	3 (12.5)	6 (26.1)	
Pituitary	2 (8.3)	0 (0.0)	
Brain metastasis	1 (4.2)	3 (13.0)	
Others	2 (8.3)	9 (39.2)	
Surgical site (%)			0.250
Frontal	6 (25.0)	6 (26.2)	
Parietal	0 (0.0)	2 (8.7)	
Temporal	2 (8.3)	3 (13.1)	
Occipital	0 (0.0)	1 (4.3)	
Fronto-temporal	12 (50.0)	8 (34.8)	
Fronto-parietal	3 (12.5)	0 (0.0)	
Temporo-occipital	0 (0.0)	1 (4.3)	
Fronto-temporo-parietal	0 (0.0)	1 (4.3)	
Parieto-occipital	0 (0.0)	1 (4.3)	
Temporo-parietal	1 (4.2)	0 (0.0)	
Duration of surgery (min)	226.33±71.15	296.70±122.51	0.020
Duration of anesthesia (min)	298.58±69.75	384.61±132.03	0.007
Time from scalp block to skull pin insertion (min)	13.00±6.26	13.17±5.73	0.921
Time from scalp block to skin incision (min)	45.04±2.04	48.70±2.11	0.220
Operating room extubation (%)	16 (61.5)	10 (38.5)	0.147

BMI, body mass index; GCS, Glasgow coma score; ASA PS, American Society of Anesthesiologists physical status

greater in group D (61.5% vs. 38.5%, $p = 0.147$).

The hemodynamic profiles (Ps, Pd, MAP, and HR) at 23 different timepoints are shown in Figures. 2-5. The baseline (T0) Ps, Pd, and MAP were comparable between group D and group C. HR in group D was greater than group C ($p = 0.038$), but it was comparable prior to scalp block (T1). In group D, the Ps and MAP increased immediately one minute after scalp block (T2) and persisted higher than baseline for about 13 minutes (T3). In contrast, the HR of group D dropped at the first minute after scalp block (T2), which was 73.38± 13.29, compared to 79.48±14.68 minutes of group C, $p = 0.142$, and group D remained lower consistently for one hour after skin incision. During skull pin fixation (T4), the BP values of each group were not different from their baselines and were comparable between groups, but the MAP of group C increased significantly from T1 (before scalp block), with a mean difference of 13.35 ± 3.18 mmHg, 95%CI [0.5, 26.2], $p = 0.031$, while the MAP of group D did not increase significantly. At one minute after skin incision (T7), hemodynamic responses (Ps, Pd, and MAP) were attenuated and decreased from baseline values in both groups. In group D, the mean difference of MAP between T7 and T1 was -23.67 mmHg, 95%CI [-34.9, -12.5], $p < 0.001$, while it was -21.52 mmHg, 95%CI [-33.0, -10.1], $p < 0.001$ in group C. Additionally, patients in group D had lower Ps, Pd, MAP, and HR than those in group C persistently for one hour after skin incision (T7-T21) and during the dural opening

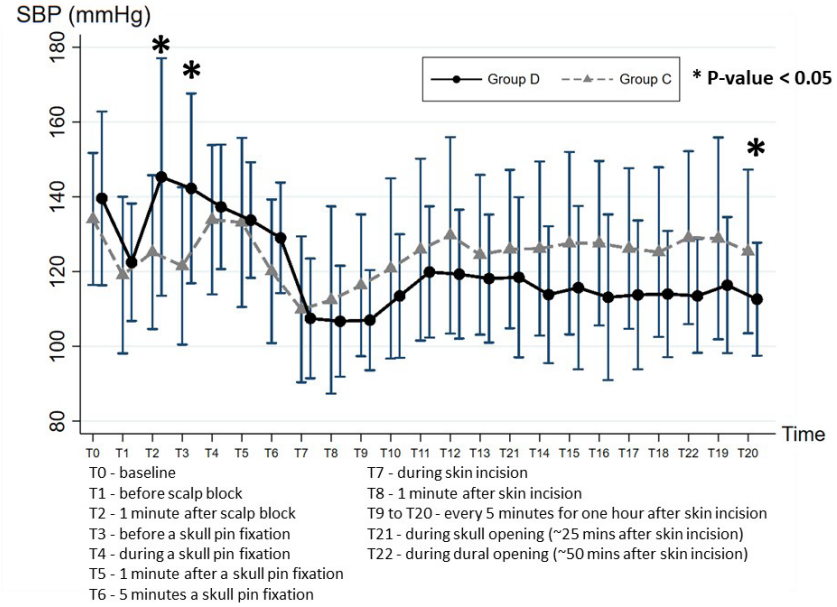

Figure 2. Systolic blood pressure at different time points

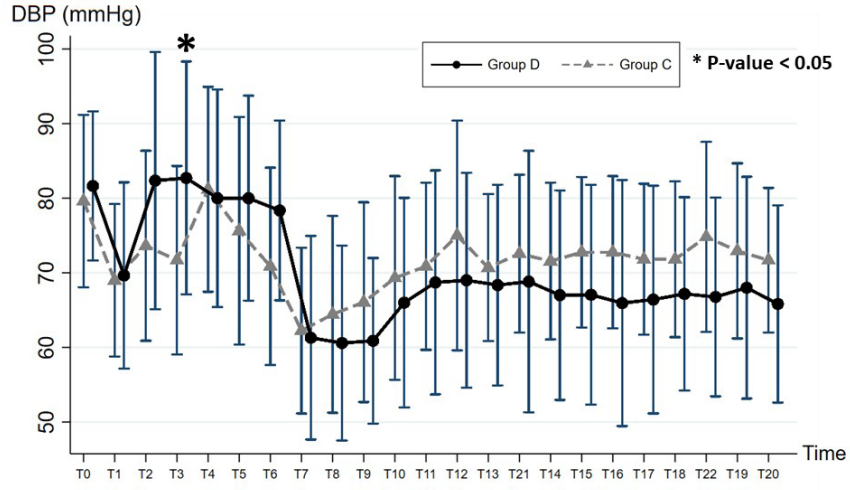

Figure 3. Diastolic blood pressure at different time points

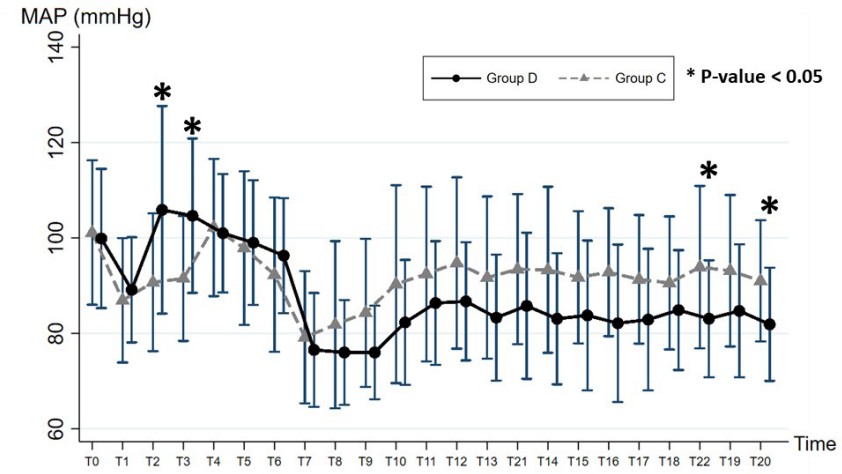

Figure 4. Mean arterial pressure at different time points

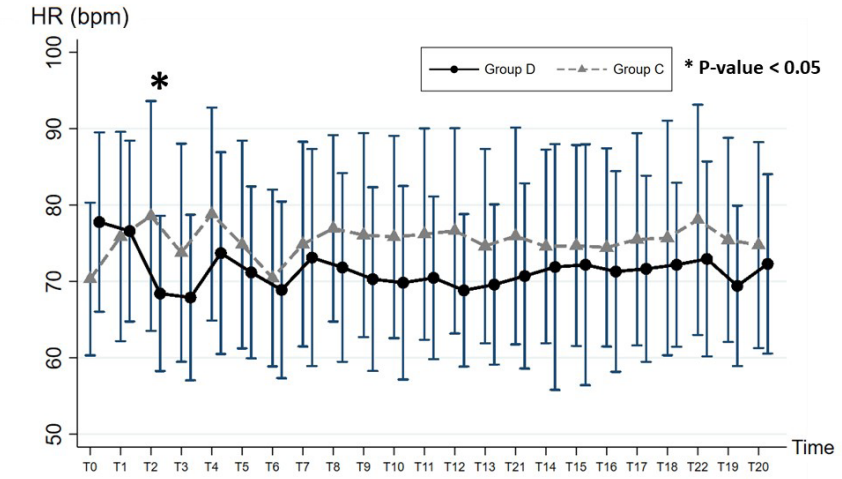

Figure 5. Heart rate at different time points

Table 2. Intraoperative anesthetic requirements

Variable	Group D (n=24)	Group C (n=23)	*p*-value	95%CI
Intraoperative fentanyl use first hour (mcg/kg)	2.63±0.89	3.04±0.96	0.134	(-0.1,1.0)
Intraoperative fentanyl use (mcg/kg)	3.66±1.35	4.61±1.51	0.027	(0.1,1.8)
Intraoperative fentanyl use (mcg/kg/hr)	0.74±0.19	0.75±0.24	0.771	(-0.1,0.1)
Intraoperative propofol use (mg/kg)	29.69±8.32	35.80±10.23	0.030	(0.6,11.5)
Intraoperative propofol use (mg/kg/hr)	5.97±0.82	5.78±1.24	0.548	(-0.8,0.4)

which occurred about 50 minutes after skin incision (T22), but the MAP values of group D from T15 to T22 (which was about 35 minutes after skin incision) were significantly lower than those of group C. (Figure 4)

As to secondary outcomes, intraoperative doses of fentanyl (mcg/kg) and propofol (mg/kg) in group D were significantly lower than those of group C (Table 2). The postoperative tramadol dose, as a rescue drug in the first 24 hours was 1.88 ± 0.25 mg/kg in group D and 2.31 ± 0.20 mg/kg in group C, $p = 0.193$. The incidences of intraoperative hemodynamic adverse events that required medical treatment, including bradycardia, hypotension, and hypertension, were not different between groups, as were the incidences of postoperative adverse hemodynamic events requiring rescue treatment. (Tables 3 and 4)

Table 3. Intraoperative hemodynamic adverse events that required medical treatment

Variable	Group D n=24 (%)	Group C n=23 (%)	*p*-value
Bradycardia	1 (4.17)	1 (4.35)	1.000
Hypotension	6 (25.00)	8 (34.78)	0.534
Hypertension	8 (33.33)	6 (26.09)	0.752

Table 4. Postoperative hemodynamic adverse events that required medical treatment

Variable	Group D n=24 (%)	Group C n=23 (%)	*p*-value
Bradycardia	0 (0.00)	1 (4.35)	0.489
Hypotension	0 (0.00)	0 (0.00)	
Hypertension	1 (4.17)	2 (8.70)	0.609

DISCUSSION

In addition to providing perioperative analgesia for craniotomy, a scalp block has been proven to attenuate the hemodynamic response to skull pinning and skin incision (3, 12, 13) more effectively than routine anesthesia with only intravenous or volatile anesthetic (3, 12). Similarly, the results of our study showed that scalp block with 0.5% bupivacaine in 20 mL attenuated the hypertensive response to noxious stimuli during the first hour of intracranial procedures, including skull pin fixation, craniotomy incision, as well as skull and dural opening. Although the neuroendocrine response has also been previously reported following skull-pin holder fixation (3), stress hormone levels were not investigated in this study.

Regarding locoregional analgesia, dexmedetomidine has been added to several routes of both central and peripheral neural blockade, e.g., epidural, caudal, and spinal, to enhance the duration of both sensory and motor blockade by local anesthetics (14). One prior study demonstrated that adding dexmedetomidine (1 mcg/kg) to 0.25% bupivacaine 20 mL for scalp block was very efficient in obtunding the hemodynamic response to skull pin placement (10). On the contrary, the author could not demonstrate an add-on effect of dexmedetomidine on scalp block with 0.5% bupivacaine when the skull pin was attached. This result is in concordance with a recent study by Sahana et al. (15) which mentions that the addition of dexmedetomidine (1 mcg/kg) to 25 mL of 0.5% ropivacaine for scalp block provided no extra advantage over 25 mL of 0.5% ropivacaine alone in attenuating the hemodynamic response to skull pin placement. In the present study, however, adding dexmedetomidine resulted in much lower BP and HR than that in patients who received scalp block with only 0.5% ropivacaine, at least during the first hour.

The mechanism of action of dexmedetomidine as an alpha2-adrenoceptor agonist in peripheral nerve blocks is not fully understood. Proposed mechanisms include central analgesia, vasoconstriction, and anti-inflammatory effects (16). However, none of these mechanisms can clearly explain the synergistic effect of alpha2-adrenoceptor agonists of dexmedetomidine when added to a local anesthetic in peripheral nerve blocks (14). In the present study, the plasma level of dexmedetomidine was not investigated, so we postulated that the initial hypertensive phase was due to the systemic effect of dexmedetomidine on the alpha2B adrenoceptor mediated vascular response from rapid plasma absorption. However, the subsequent hypotension was not confirmed as being an effect of dexmedetomidine mediated by alpha2A receptor on cardiovascular response and/or locoregional analgesia by enhancing the onset and efficacy of peripheral neural blockade.

Dexmedetomidine prolongs the duration of analgesia via a hyperpolarization-activated cation current that acts more selectively on C-fibers than on A-alpha fibers (17). C fibers are densely innervated in the scalp, which may explain the mechanism. A prior study concluded that the addition of dexmedetomidine (1 mcg/kg) to bupivacaine prolonged the pain-free period, and that a scalp block is a superior technique compared to scalp infiltration with 0.25% bupivacaine and dexmedetomidine (1 mcg/kg) in a 20 mL solution (18). Another study, which added dexmedetomidine 1.5 mcg/kg to levobupivacaine 36 mL for scalp block, reported a prolonged analgesic effect and better hemodynamic control post-craniotomy, despite no data on intraoperative hemodynamics and anesthetic requirements being revealed (19). A recent study by Lekprasert et al. (20) concluded that compared to 0.25% levobupivacaine with adrenaline (1:200,000), preoperative scalp block with added dexmedetomidine 1 mcg/kg increased time to the first analgesic and decreased intraoperative fentanyl use, while postoperative tramadol consumption was not statistically significantly

different between groups, which correlates with our study in which adding dexmedetomidine reduced the amount of fentanyl.

Limitations

There are several limitations in this study. The first is that the plasma level of dexmedetomidine was not investigated, so we postulated that the initial hypertensive phase was due to the systemic effect of dexmedetomidine on the alpha2B adrenergic receptor-mediated vascular response from rapid plasma absorption. Additionally, the subsequent hypotension was not confirmed to be an effect of dexmedetomidine mediated by the alpha2A receptor in the cardiovascular response and/or locoregional analgesia by enhancing the onset and efficacy of peripheral neural blockade. Secondly, authors could not demonstrate whether the scalp block succeeded or failed, and as there was no nociceptive index monitor to guide analgesic drug titration, fentanyl was given subjectively to diminish the hyperdynamic response. Lastly, our intraoperative study period was only one hour after the scalp block, which was insufficient to reveal how long dexmedetomidine affects perioperative hemodynamics in a craniotomy.

CONCLUSIONS

This study demonstrated that hemodynamic responses during the first hour of intracranial surgery are more attenuated with the addition of dexmedetomidine 1 mcg/kg to a scalp block with 0.5% bupivacaine. In addition, adding dexmedetomidine to a scalp block decreases intraoperative fentanyl and propofol requirements during intracranial surgery.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to report.

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