Original Article

DOI: 10.7860/JCDR/2025/79290.21163

Anaesthesia Section

Comparison of Dexmedetomidine-Bupivacaine Combination versus Bupivacaine alone for Scalp Block in Attenuating the Haemodynamic Response to Skull Pin Placement during Neurosurgical Procedures: A Randomised Controlled Study

EMIN JIMMY[1], ARPANA KEDLAYA[2], NAYANTHARA JOACHIM[3]

ABSTRACT

Introduction: Skull pin placement in neurosurgical procedures can cause acute nociceptive stimuli that may result in an acute haemodynamic response and rise in intracranial pressures. Pain management is crucial to treat this detrimental stimulus.

Aim: To compare the effects of dexmedetomidine as a local anaesthetic adjuvant to bupivacaine versus bupivacaine alone in scalp block for attenuation of haemodynamic response to skull pin placement in neurosurgical procedures.

Materials and Methods: A double-blinded randomised controlled study was done at St. John's Medical College Hospital, Bengaluru, Karnataka, India where 70 patients were randomly placed into one of two groups (35 each). Patients in Group D received scalp block with 1 µg/kg dexmedetomidine and 25 mL of 0.25% bupivacaine. Group C received 25 mL of 0.25% bupivacaine alone. Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and Mean Arterial Pressure (MAP) were recorded at baseline and at 1, 3, 5, 10 and 15 minutes post scalp block. Haemodynamic changes were analysed using the unpaired Student's t-test in both groups.

Results: Demographic variables including age, gender, weight and American Society of Anaesthesiologists (ASA) status were comparable between the groups. A significant increase in HR from baseline to one and three minutes after scalp block in both groups (-6.89 beats/min, p-value=0.001 and -4.89 beats/min, p-value=0.049 in Group D; -8.4 beats/min, p-value <0.001 and -7.34 beats/min, p-value <0.001 in Group C, respectively). There was also significant decrease in HR from baseline to 15 minutes after scalp block (4.8 beats/min, p-value=0.006) in Group D. In both groups, there was a clinical increase in BP from baseline to 1 and 3 minutes after scalp block (Group D baseline MAP 86.5±7.1 mmHg, at 1 minute 90.5±7.5 mmHg, at 3 minutes 89.2±6.5 mmHg; Group C baseline MAP 89.5±8.7 mmHg, at one minute 92.7±9.0 mmHg, and at three minutes 92.0±7.9 mmHg). After 10 and 15 minutes of scalp block, the mean BP of the patients in Group D (84.9±5.8 mmHg and 83.9±5.7 mmHg) was significantly lesser as compared to that of patients in Group C (88.9±8.8 mmHg and 87.9±8.8 mmHg).

Conclusion: Scalp nerve block is extensively used for intraoperative haemodynamic stability and opioid-sparing effects. It has proven to provide better postoperative analgesia in patients undergoing craniotomies. In present study, the addition of 1 µg/kg of dexmedetomidine with 25 mL of 0.25% bupivacaine for a scalp block minimises the haemodynamic response to skull pin placement when compared to 0.25% bupivacaine given alone in patients undergoing craniotomies for Central Nervous System (CNS) pathologies.

Keywords: Adjuvants, Craniotomy, Haemodynamics, Nerve blocks, Regional anaesthesia

INTRODUCTION

Anaesthesia for intracranial surgeries in patients with elevated Intracranial Pressure (ICP) poses unique challenges for anaesthesiologists. Procedures such as laryngoscopy, skull pin placement, surgical incision and tissue dissections involving the periosteum and dura can trigger painful stimuli. These stimuli may lead to sudden spikes in BP and HR, which can exacerbate ICP, increase the risk of complications and elevate the likelihood of aneurysm rupture [1]. Brain hyperaemia and raised ICP, resulting from increased oxygen demand and catecholamine release due to postoperative pain, may put patients at risk for intracranial haematoma. Effective pain management is essential in preventing these systemic alterations to enhance long-term results and promote better recovery. In addition, early postoperative pain management helps to not only reduce chronic pain states brought on by surgical tissue damage but also the emergence of central sensitisation [2,3].

Different anaesthetic and pharmacological approaches have been used to reduce the haemodynamic response during skull pin placement. These methods include the administration of narcotics, antihypertensives and enhancing the depth of anaesthesia with both intravenous (i.v.) and inhalational agents. While opioids are commonly used as primary analgesics, their side-effects such as sedation and respiratory depression-can complicate neurological evaluations and potentially increase ICP during and after surgery. Consequently, incorporating non opioid analgesics into a multimodal pain management approach may be beneficial for postcraniotomy pain. Additionally, performing nerve blocks for the scalp can

help manage hypertension and tachycardia, reduce the need for vasodilators and minimise the requirement for deeper planes of anaesthesia early in the procedure, all of which can help prevent an increase in cerebral blood flow and ICP [4].

Scalp nerve blocks using local anaesthetics effectively reduce sympathetic responses. Bupivacaine, a long-acting local anaesthetic, mainly targets sensory nerve fibres while minimising cardiac and neurotoxic effects [5]. To enhance or extend the action of local anaesthetics, adjuvants are often added. Among these, alpha-2 agonists are commonly used in conjunction with local anaesthetics. Dexmedetomidine is commonly chosen as an adjuvant due to its established efficacy in neuraxial anaesthesia techniques. It inhibits aδ and C fibres and acts on the α2 receptors in peripheral vascular smooth muscle cells to constrict the peripheral blood vessels, reduce the absorption of local anaesthetics and prolong the block time [6]. It also produces central analgesia and has anti-inflammatory effects, further delaying absorption of local anaesthetic drug. Dexmedetomidine is known to be eight times more selective than clonidine (1620:1) in binding to all three α2 receptor subtypes (A, B, C), hence appearing to be a better choice than clonidine [7,8].

Similarly, incorporating dexmedetomidine with bupivacaine in scalp nerve blocks can prolong pain relief and delay the need for additional analgesics. While dexmedetomidine has been studied extensively as an adjuvant, there appears to be lacunae in the use of the drug as a regional block adjuvant to local anaesthetics [9]. Scalp blocks particularly have commonly been given with plain local anaesthetics while supplemented with i.v. and inhalational agents. Very few studies have used dexmedetomidine as an adjuvant to local anaesthetics in the scalp nerve block itself [10,11]. Hence, present study intended to note the efficacy of dexmedetomidine as an adjuvant to bupivacaine and to evaluate the haemodynamic stability, including HR and BP in patients undergoing elective craniotomy procedures.

MATERIALS AND METHODS

This double-blinded randomised controlled study was done over a period of two years, from August 2022 to August 2024, at St. John's Medical College Hospital, Bengaluru, Karnataka, India. The study was reviewed and approved by the Institutional Ethics Committee (IEC Study ref no: ST-138/2022).

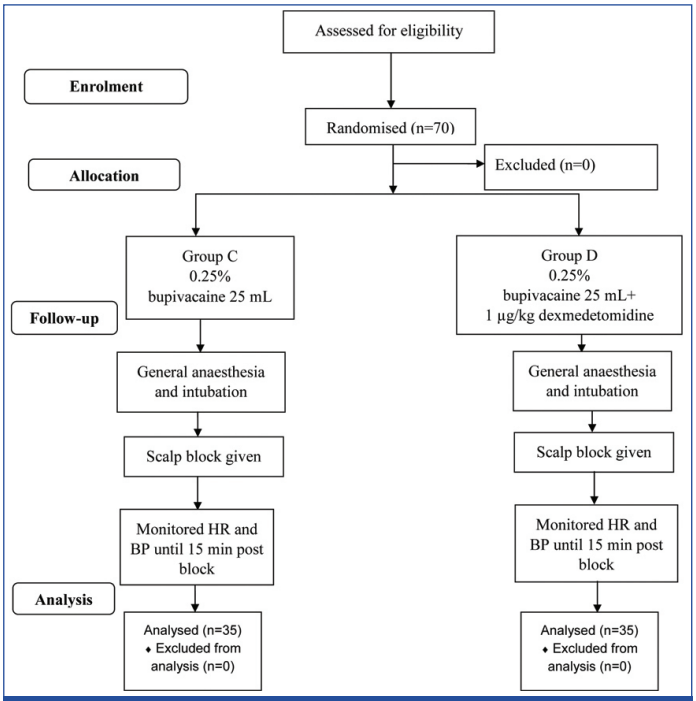
Inclusion criteria: All consenting patients aged between 18 and 59 years, classified as American Society of ASA grade I and II, who were posted for elective supratentorial craniotomy surgeries and requiring placement of Mayfield skull pins, were included in the study.

Exclusion criteria: Patients with a history of uncontrolled hypertension, ischaemic heart disease, cardiac arrhythmia, bradycardia (HR ≤60 bpm), severe hepatic and renal disease, previous craniotomy, low Glassgow Coma Scale (GCS), use of alpha or beta blockers, allergy to study drugs and pregnant and lactating patients, were excluded from the study.

Sample size estimation: In a study done by Nagappa S et al., the mean maximum HR in scalp block with clonidine and ropivacaine was 96.88±12.83 bpm and 84.41±15.68 bpm, respectively [12]. Assuming a similar effect with bupivacaine and dexmedetomidine, and using a standard deviation of 12.83 and 15.68 for HR in both groups, with 1% level of significance and 90% power of the study, the required sample size was 35 patients in each group.

Study Procedure

Seventy patients were randomly assigned to two groups using computer-generated randomisation table [Table/Fig-1]. Allocation sequences were concealed in sealed, coded envelopes. To maintain blinding, a resident trainee who was not involved in the study, prepared two sets of 25 mL syringes on the day of surgery. Both the patient and the anaesthesiologist performing the block were blinded to group assignments. The control group (Group C) received 25 mL of 0.25% bupivacaine, while the dexmedetomidine group (Group D) received 25 mL of 0.25% bupivacaine added with dexmedetomidine (1 µg/kg) for the scalp block [13].



[Table/Fig-1]: Consort diagram.

The night before surgery, all patients received oral alprazolam 0.25 mg and pantoprazole 40 mg. Upon arrival in the operating room, an i.v. line was placed. As per ASA monitoring protocols, all patients' vitals including oxygen saturation (SpO_2), end-tidal carbon dioxide ($EtCO_2$), and electrocardiogram (ECG) trace, were recorded at baseline and intraoperatively. BPs were monitored through an invasive arterial line catheter throughout the procedure.

Anaesthesia was induced with midazolam 0.03 mg/kg, fentanyl 2 µg/kg, propofol 1 mg/kg, and atracurium 0.5 mg/kg. Maintenance included 50% oxygen in air and isoflurane, with an end-tidal concentration of 0.8-1 (Minimum Alveolar Concentration). A bilateral scalp block was administered using the assigned study medication by an investigator who was uninvolved in data collection or analysis. A 24-gauge needle was used to block the supraorbital and supratrochlear nerves near the supraorbital groove, the zygomaticotemporal nerve 1 cm from the outer canthus, the auriculotemporal nerve near the tragus and the lesser and greater occipital nerves along the line from the mastoid process to the occipital protuberance. A 15-minute interval was observed between intubation and skull pin fixation to minimise the impact of haemodynamic response.

HR and BP were recorded at baseline and at 1, 3, 5, 10-, and 15-minutes post scalp block. Bradycardia, defined as a 20% decrease from baseline HR, was treated with atropine 0.6 mg i.v. Hypertension, defined as a >20% increase from baseline mean BP, was managed with incremental i.v. propofol (10 mg, up to a maximum of 1 mg/kg) and esmolol (100 µg/kg) if needed. Hypotension, indicated by a >20% decrease in mean BP, was treated by reducing isoflurane concentration to 0.5%, with i.v. ephedrine 3 mg boluses administered if necessary.

Dexamethasone (0.08 mg/kg) was used as an antiemetic and antiepileptics were administered as required. Following the surgical procedure, patients were extubated once adequate respiratory effort and response to verbal commands were confirmed.

STATISTICAL ANALYSIS

Data analysis was conducted using MS Excel and Statistical Package for the Social Sciences (SPSS) version 27.0. Categorical data were presented as frequencies and percentages, while continuous data

were summarised as means with standard deviations or medians with interquartile ranges. Haemodynamic changes (HR, SBP, DBP) were analysed using linear mixed models. Statistical significance was set at p-values ≤0.05 in each group. Haemodynamic changes over time in both groups were analysed using the unpaired Student's t-test.

RESULTS

Demographic observations (age, gender, weight and ASA status) were comparable between both the groups, as noted in [Table/Fig-2]. There was significant increase in HR from baseline to one and three minutes after scalp block (p-value <0.05) in both groups, as noted in [Table/Fig-3] (-6.89 beats/min, p-value=0.001 and -4.89 beats/min, p-value=0.049 in Group D; -8.4 beats/min, p-value <0.001 and -7.34 beats/min, p-value <0.001 in Group C, respectively). In Group C, this rise was also seen at 5 mins after scalp block (-3.94 beats/min, p-value=0.002). There was a significant decrease in HR from baseline to 15 minutes after scalp block in Group D (4.8 beats/min, p-value=0.006). At all other time points there was no significant difference noted. A significant difference in HR between both groups at 5, 10 and 15 minutes as noted in [Table/Fig-4].

Demographics		Group D (mean±SD)	Group C (mean±SD)
Mean age (years)		37.12±11.391	42.12±13.17
Gender (Male:Female)		24:11	20:15
Mean weight (kg)		58.4±10.9	55.28±7.4
ASA-PS	I	19	21
	II	16	14

[Table/Fig-2]: Demographic distribution in the study.

Change in HR from baseline to different time points	Group D (35)		Group C (35)	
	Mean difference	p-value	Mean difference	p-value
After 1 min	-6.89	0.001*	-8.4	<0.001*
After 3 mins	-4.89	0.049*	-7.34	<0.001*
After 5 mins	-0.74	1.000	-3.94	0.002*
After 10 mins	1.54	1.000	-1.66	1.000
After 15 mins	4.80	0.006*	2.57	0.529
	F (3.098, 105.328)=27.88 with p-value <0.001*		F (3.15, 107.19)=48.76 with p-value <0.001*	

[Table/Fig-3]: Variations of HR within the groups.
*Significant difference at p≤0.05, p-value for significance calculated using unpaired t-tests

Heart Rate (HR) (Beats/min)	Group D (35) Mean±SD	Group C (35) Mean±SD	p-value
Baseline	82.4±11.2	83.8±11.3	0.591
After 1 min	89.3±10.1	92.2±8.6	0.191
After 3 mins	87.3±8.8	91.2±8.5	0.062
After 5 mins	83.1±7.9	87.8±9.7	0.03*
After 10 mins	80.8±7.1	85.5±8.5	0.015*
After 15 mins	77.6±6.9	81.3±7.8	0.04*

[Table/Fig-4]: Variation of HR between two groups.
*Significant difference at p≤0.05, p-value for significance calculated using unpaired t-test

SBP, DBP and MAP differed significantly between baseline to different time points in both the groups (D and C), as seen in [Table/Fig-5].

As noted in [Table/Fig-6], there was an increase in BP from baseline to 1st and 3rd minute after scalp block in both groups of patients, but this was not statistically significant. In Group D, there was a significant decrease in mean BP from baseline to 15 minutes after scalp block. A significant difference in BPs between the groups was observed at 10 and 15 minutes after scalp block; however, no such difference was noted at other time points. After 10 and 15 minutes of scalp block, the mean BP of the patients in Group D (84.9±5.8 mmHg and 83.9±5.7 mmHg) was significantly lesser as compared to that of patients in Group C (88.9±8.8 mmHg and 87.9±8.8 mmHg).

DISCUSSION

Acute arterial hypertension may lead to cerebral aneurysm rupture, pulmonary oedema and disruption of autoregulation in neurosurgical patients. Preventing an acute rise in ICP, particularly during skull pin placement, is crucial since the scalp is densely innervated with type C fibres. Although scalp infiltration is effective in alleviating the sympathoadrenal response to skull pin placement and in providing postoperative analgesia, scalp block has been found to be superior in blocking this response [14]. Scalp block also removes the requirement for additional anaesthesia or vasoactive drugs during the period of head pinning. Therefore, addition of a scalp block to the anaesthetic plan for patients undergoing craniotomy will successfully prevent the hyperdynamic response to head pinning without increasing the requirement for volatile anaesthetics or antihypertensive drugs [4].

The present study demonstrated that the addition of dexmedetomidine to bupivacaine in scalp blocks provided superior attenuation of the haemodynamic response to skull pin placement compared to bupivacaine alone. Significant reductions in HR and SBP were observed at 10 and 15 minutes after pin placement in the dexmedetomidine-bupivacaine group, indicating a delayed but more effective block. This suggests that a soak time of approximately 10 minutes is necessary for optimal efficacy of dexmedetomidine when used peripherally. This delay may also be attributed to the slower peripheral onset of dexmedetomidine compared to its rapid i.v. action.

These findings correlate well with those of Vallapu S et al., who also found significantly prolonged analgesia and reduced pain scores (p-value <0.05) when dexmedetomidine was used as an adjuvant to bupivacaine for nerve blocks and scalp infiltration [15]. Similarly, Anyapu P et al., compared fentanyl and dexmedetomidine with bupivacaine as adjuvants to scalp block and noted better control of sympathetic response to Mayfield pin insertion in patients who received dexmedetomidine as the adjuvant. They noted this as lower requirements of propofol boluses to maintain MAP at 90 mmHg. Their results support the role of dexmedetomidine in modulating pain through central and peripheral mechanisms, including α2-adrenoceptor-mediated analgesia and anti-inflammatory effects [16].

Both studies used bupivacaine as the local anaesthetic, enabling a direct comparison of the impact of dexmedetomidine as an adjuvant. Moreover, in present study use of a scalp block rather than infiltration ensured targeted and effective blockade of superficial sensory branches of the scalp as it offers enhanced precision, reduces the risk of nerve damage and helped in ease of repositioning the pins

	Change in SBP from baseline to different time-points				Change in DBP from baseline to different time-points				Change in MAP from baseline to different time-points			
	Group D		Group C		Group D		Group C		Group D		Group C	
	Mean difference	p-value	Mean difference	p-value	Mean difference	p-value	Mean difference	p-value	Mean difference	p-value	Mean difference	p-value
1 min	-4.886	0.004*	-9.257	<0.001*	-3.43	0.018*	-4.37	<0.001*	-3.200	0.077	-3.914	<0.001*
3 mins	-3.657	0.173	-7.771	<0.001*	-2.6	0.290	-2.60	0.217	-2.543	0.640	-2.686	0.068
5 mins	-0.629	1.000	-5.114	<0.001*	-1.83	1.000	-1.20	1.000	-0.514	1.000	-1.314	1.000
10 mins	4.943	<0.001*	-1.914	1.000	-0.83	1.000	1.20	1.000	0.486	1.000	1.657	0.83

15 mins	6.086	<0.001*	-0.086	1.000	0.40	1.000	2.91	0.033*	1.543	0.077	2.686	0.036*
	F (3.46, 117.65)=38.54 p<0.001*		F (3.5, 119.08)=42.18 p<0.001*		F (2.125, 72.234)=9.209 p<0.001*		F (3.28, 111.42)=23.079 p<0.001*		F (3.00, 102.04)=9.154 p<0.001*		F (2.34, 79.08)=33.149 p<0.001*	

[Table/Fig-5]: Variation of SBP, DBP and MAP within the groups.
*Significant difference at p≤0.05, p-value for significance in each group is calculated using unpaired t-test

	SBP (mmHg)			DBP (mmHg)			MAP (mmHg)		
	Group D Mean±SD	Group C Mean±SD	p-value	Group D Mean±SD	Group C Mean±SD	p-value	Group D Mean±SD	Group C Mean±SD	p-value
Baseline	126.5±11.9	125.8±10.8	0.801	71.3±8.2	69.7±7.4	0.383	86.5±7.1	89.5±8.7	0.130
1 min	131.4±12.3	135.1±10.1	0.176	74.8±8.9	74.1±6.9	0.72	90.5±7.5	92.7±9.0	0.272
3 mins	130.2±10.5	133.6±11.1	0.189	73.9±8.6	72.3±7.3	0.396	89.2±6.5	92.0±7.9	0.114
5 mins	127.1±12.1	130.9±10.9	0.172	73.2±8.2	70.9±6.5	0.204	87.9±6.0	90.0±9.2	0.259
10 mins	121.6±11.6	127.7±10.4	0.022*	72.2±8.0	68.5±5.5	0.029*	84.9±5.8	88.9±8.8	0.025*
15 mins	120.4±12.1	125.9±9.7	0.04*	70.9±8.0	66.8±5.0	0.011*	83.9±5.7	87.9±8.8	0.026*

[Table/Fig-6]: Variation of BP between the groups.
*Significant difference at p≤0.05, p-value for significance calculated using unpaired t-test

intraoperatively without additional interventions if the surgeon needed to do so.

Dexmedetomidine is the dextro-isomer of medetomidine, which exerts sedative, analgesic and neuroprotective effects by α2 receptor-mediated tyrosine kinase phosphorylation [17]. It promotes the release of various growth factors by agitating astrocytes to participate in neural protection. It also activates survival-promoting enzymes through the α2 adrenoceptor and exerts cardioprotective effects by adjusting the protein kinase, protein kinase B and endothelial nitric oxide synthase pathways extracellularly [18].

Dexmedetomidine activates presynaptic α2-receptors, suppressing norepinephrine release, while postsynaptic activation modulates pain and causes vasoconstriction. This can induce hypotension and bradycardia in some patients, as suggested in previous research papers [18,19]. Like clonidine, dexmedetomidine also inhibits hyperpolarisation-activated cation currents. In present study group, however, authors did not observe any patients having bradycardia or hypotension necessitating treatment. Furthermore, haemodynamic trends observed in the current study mirror those reported by Kumar AK et al., who noted a transient rise in HR following skull pin insertion, followed by stabilisation in the dexmedetomidine-bupivacaine group [4].

In present study, the dexmedetomidine group showed a rise in MAP and HR during the initial minutes but displayed a statistically significant reduction by 10 and 15 minutes. This similarity reinforces the understanding of dexmedetomidine's pharmacodynamic profile when administered peripherally, as opposed to its rapid central action when administered intravenously. Furthermore, the sustained MAP elevation in the bupivacaine-only group observed in present study matches Kumar AK et al., and Praisontarangkul V et al., indicating less effective attenuation of the haemodynamic response in the absence of dexmedetomidine [4,20]. These studies underscore the importance of timing in achieving full block efficacy [4,20].

In contrast, Sahana BN et al., reported no significant haemodynamic advantage when dexmedetomidine was added to ropivacaine for skull pin insertion [21]. This discrepancy may be attributed to the different local anaesthetic used-ropivacaine, which is less lipid-soluble and approximately 30% less potent than bupivacaine [22]. Present study used bupivacaine, which may have enhanced the efficacy of dexmedetomidine by ensuring deeper and more sustained neural blockade. This highlights the importance of considering the physicochemical properties of the local anaesthetic when evaluating the synergistic effects of adjuvants like dexmedetomidine.

Present study supports the integration of dexmedetomidine into scalp blocks for neurosurgical procedures, given its contribution to haemodynamic stability, prolonged analgesia and reduced intraoperative anaesthetic demand. Unlike other α2 agonists, such as clonidine, dexmedetomidine has a more selective α2 activity, offering sedative and neuroprotective benefits without significant adverse effects. Notably, none of the patients experienced bradycardia or hypotension requiring intervention, despite these effects being documented in prior research. This suggests that careful dosage titration and patient selection are crucial. Future studies should investigate optimal dosing strategies to maximise benefits while minimising potential side-effects.

Limitation(s)

Present study included only ASA I and II patients. Those with cardiac disease may be at a higher risk of developing significant bradycardia and hypotension due to multiple drug interactions, which could exacerbate the undesirable side effects of dexmedetomidine. Although haemodynamics were measured, direct measurements of ICP would have been an ideal way to show any elevation of ICP following skull pin placement. Also, plasma catecholamine levels for assessing sympathoadrenal response were not measured.

CONCLUSION(S)

Regional anaesthesia techniques have gained significant importance in perioperative management and in enhancing postoperative recovery. Scalp blocks are commonly used to attenuate haemodynamic responses during neurosurgical procedures. This study demonstrates that using 0.25% bupivacaine with 1 μg/kg of dexmedetomidine provides superior haemodynamic stability compared to bupivacaine alone during skull pin placement in craniotomy patients. The addition of dexmedetomidine significantly blunts stress responses, thereby improving patient stability during surgery. Adjuvants like dexmedetomidine can enhance the quality and duration of regional blocks. Therefore, their inclusion can lead to improved perioperative outcomes in neurosurgical patients.

REFERENCES

[1] Shapiro HM. Intracranial hypertension: Therapeutic and anaesthetic considerations. Anaesthesiology. 1975;43(4):445-71. PMID: 1101754.
[2] Dong J, Wang D, Sun H, Zeng M, Liu X, Yan X, et al. Effect of anaesthesia on the outcome of high-grade glioma patients undergoing supratentorial resection: Study protocol for a pragmatic randomized controlled trial. Trials. 2022;23:816. Doi: 10.1186/s13063-022-06716-9.
[3] Nijs J, Malfliet A, Nishigami T. Nociplastic pain and central sensitization in patients with chronic pain conditions: A terminology update for clinicians. Braz J Phys Ther. 2023;27(3):100518. Doi: 10.1016/j.bjpt.2023.100518. PMID: 37348359; PMCID: PMC10314229.
[4] Kumar AK, Rahmathullah M, Kulkarni DK, Ramachandran G. Efficacy of adding dexmedetomidine to bupivacaine on attenuating hemodynamic response to skull pin placement for performing scalp block [Internet]. Int J Contemp Med Res. 2017;4(12):09-13.
[5] Pinosky ML, Fishman RL, Reeves ST, Harvey SC, Patel S, Palesch Y, et al. The effect of bupivacaine skull block on the hemodynamic response to craniotomy. Anaesth Analg. 1996;83(6):1256-61. Doi: 10.1097/00000539-199612000-00022. PMID: 8942596.

[6] Chen Z, Liu Z, Feng C, Jin Y, Zhao X. Dexmedetomidine as an adjuvant in peripheral nerve block. Drug Des Devel Ther. 2023;17:1463-84. Doi: 10.2147/DDDT.S405294. PMID: 37220544; PMCID: PMC10200118.
[7] Kaur M, Singh PM. Current role of dexmedetomidine in clinical anaesthesia and intensive care. Anaesth Essays Res. 2011;5(2):128-33. Doi:10.4103/0259-1162.94750. PMID: 25885374; PMCID: PMC4173414.
[8] Panchal SM, Gosavi KS, Kudre KR. Comparison of dexmedetomidine and clonidine as an adjuvant to bupivacaine in scalp block for supratentorial craniotomy surgery: A prospective, randomised Indian study. Int J Adv Res. 2023;11(6):1054-62.
[9] Martín MF, Álvarez SL, Aldecoa C. Less is better: Role of adjuvants. Curr Opin Anaesthesiol. 2025. Doi: 10.1097/aco.0000000000001495.
[10] Singh N, Gupta S, Kathuria S. Dexmedetomidine vs dexamethasone as an adjuvant to 0.5% ropivacaine in ultrasound-guided supraclavicular brachial plexus block. J Anaesthesiol Clin Pharmacol. 2020;36(2):238-43. Doi: 10.4103/joacp.JOACP_176_19. PMID: 33013041; PMCID: PMC7480314.
[11] Bao N, Shi K, Wu Y, He Y, Chen Z, Gao Y, et al. Dexmedetomidine prolongs the duration of local anaesthetics when used as an adjuvant through both perineural and systemic mechanisms: A prospective randomized double-blinded trial. BMC Anaesthesiol. 2022;22:176. Doi: 10.1186/s12871-022-01716-3.
[12] Nagappa S, Kalappa S, Sridhara R. Evaluation of the hemodynamic response of intravenous clonidine versus ropivacaine scalp block to insertion of scalp pins in neurosurgical patients. Anaesth Essays Res. 2018;12(1):213.
[13] Stachtari C, Stergiouda Z, Koraki E, Sifaki F, Bagntasarian S, Chatzopoulos S. Dexmedetomidine as an adjuvant to scalp block in patients undergoing elective craniotomy: A prospective randomized controlled trial. Clin Neurol Neurosurg. 2023;227:107669. Doi: 10.1016/j.clineuro.2023.107669. PMID: 36924695.
[14] Yang X, Ma J, Li K, Chen L, Dong R, Lu Y, et al. A comparison of effects of scalp nerve block and local anaesthetic infiltration on inflammatory response, hemodynamic response, and postoperative pain in patients undergoing craniotomy for cerebral aneurysms: A randomized controlled trial. BMC Anaesthesiol. 2019;19(1):91. Doi: 10.1186/s12871-019-0760-4. PMID: 31153358; PMCID: PMC6545200.
[15] Vallapu S, Panda NB, Samagh N, Bharti N. Efficacy of dexmedetomidine as an adjuvant to local anaesthetic agent in scalp block and scalp infiltration to control postcraniotomy pain: A double-blind randomized trial. J Neurosci Rural Pract. 2018;9(1):73-79.
[16] Anyapu P, Abhiruchi YP, Rao KP, Durga P. Dexmedetomidine vs fentanyl in scalp nerve block for blunting response to skull pin insertion and postoperative pain: A randomised double-blinded study. Indian J Anaesth Analg. 2019;6(6 Pt-II):2089-94.
[17] Dahmani S, Rouelle D, Gressens P, Mantz J. Effects of dexmedetomidine on hippocampal focal adhesion kinase tyrosine phosphorylation in physiologic and ischemic conditions. Anaesthesiology. 2005;103(5):969-77. Doi: 10.1097/00000542-200511000-00011. PMID: 16249671.
[18] Zhang X, Wang R, Lu J, Jin W, Qian Y, Huang P, et al. Effects of different doses of dexmedetomidine on heart rate and blood pressure in intensive care unit patients. Exp Ther Med. 2016;11(1):360-66.
[19] Singh G, Arimanikam G, Lionel KR, Smita V, Yadav B, Arulvelan A, et al. Comparison of dexmedetomidine infusion versus scalp block with 0.5% ropivacaine to attenuate hemodynamic response to skull pin insertion in craniotomy: A prospective, randomized controlled trial. J Neuroanaesth Crit Care. 2021;8(3):180-86.
[20] Praisontarangkul V, Saringcarinkul A, Pin-on P, Chuasuwan S, Wongchanma N. 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. Biomed Sci Clin Med [Internet]. 2024;63(4):242-50. [cited 2025 May 17]. Available from: https://he01.tci-thaijo.org/index.php/CMMJ-MedCMJ/article/view/269608.
[21] Sahana BN, Radhapuram SD, Samantaray A, Hemanth N, Pasupuleti H, Mangu HR. Comparison of effects of dexmedetomidine added to ropivacaine versus ropivacaine alone infiltration scalp block for attenuation of the haemodynamic response to skull pin placement in neurosurgical procedures: A double-blind, randomised clinical trial. Indian J Anaesth. 2021;65(11):782-88. Doi:10.4103/ija.ija_450_21. PMID: 35001949; PMCID: PMC8680418.
[22] Arthur RG, Feldman HS, Covino BG. Comparative pharmacokinetics of bupivacaine and ropivacaine, a new amide local anaesthetic. Anaesth Analg. 1988;67(11):1053-58.

PARTICULARS OF CONTRIBUTORS:
1. Senior Resident, Department of Anaesthesiology, St. John's Medical College Hospital, Bengaluru, Karnataka, India.
2. Associate Professor, Department of Anaesthesiology, St. John's Medical College Hospital, Bengaluru, Karnataka, India.
3. Assistant Professor, Department of Anaesthesiology, St. John's Medical College Hospital, Bengaluru, Karnataka, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Nayanthara Joachim,
Assistant Professor, Department of Anaesthesiology, St. John's Medical College Hospital, Sarjapur Road, Bengaluru, Karnataka, India.
E-mail: nayanthara1st@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Mar 27, 2025
- Manual Googling: May 17, 2025
- iThenticate Software: May 19, 2025 (19%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Mar 13, 2025**
Date of Peer Review: **Apr 10, 2025**
Date of Acceptance: **May 21, 2025**
Date of Publishing: **Jun 01, 2025**