Efficacy of Adding Dexmedetomidine to Bupivacaine on Attenuating Hemodynamic Response to Skull Pin Placement for Performing Scalp Block

Anne Kiran Kumar[1], Mohammad Rahmathullah[2], Dilip Kumar Kulkarni[3], Gopinath Ramachandran[4]

ABSTRACT

Introduction: Acute increases in heart rate and arterial blood pressure can be deleterious in the neurosurgical patient with increased ICP or with cerebral aneurysms. So present study was done to evaluate the efficacy of adding dexmedetomidine to bupivacaine for performing scalp block on attenuating hemodynamic response to skull pin placement.

Material and Methods: This was a prospective, randomised, double-blinded study conducted from November 2009 to September 2010 in which 50 patients of either sex were selected. Patients were randomly allocated to one of the two groups(25 each), Group 1 received scalp block with a 20 ml solution having Dexmedetomidine 1mcg/kg and 0.25% bupivacaine. Group 2 received scalp block with 20 ml of 0.25% bupivacaine. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure(DBP) and mean blood pressure (MBP) were recorded at base line (pre induction), before block, before pin placement, during pin placement and later at 1, 3, 5, and 8 minute intervals. Hemodynamic changes in response to pin fixation were compared with the baseline in each group. Also the hemodynamic parameters at each step of recording were compared between the two groups.

Results: There is a significant difference between the two groups, with the mean heart rate of G2 (bupivacaine only) group showing a significant increase during pin placement and one minute after that. In G1 (dexmedetomidine + bupivacaine 0.25%) group , there was no increase in mean heart rate during and 8 minutes after pin placement. In group 1 (dexmedetomidine + bupivacaine 0.25% group) pinning did not result in an increase in MAP from 91.49 ±10.96mmHg to 89.42 ± 16.56 mmHg (P<0.01). Inter-group comparison showed significant increase in MAP in group 2 at each step of recording. In group 1 (dexmedetomidine + bupivacaine 0.25% group) pinning did not result in an increase in SBP and DBP. Inter-group comparison showed significant increase in SBP and DBP in group 2 at each step of recording. There was no statistically significant difference in oxygen saturation between the two study groups during the study period.

Conclusion: This study demonstrates that adding dexmedetomidine to bupivacaine while performing scalp block is very efficient in obtunding hemodynamic response to skull pin placement.

Keywords: Dexmedetomidine, Bupivacaine, Hemodynamic, Skull Pin Placement, Scalp Block

INTRODUCTION

Intracranial surgery on patients with increased intracranial pressure (ICP) poses a unique challenge to the anaesthesiologist. Although much intracranial surgery is not particularly stimulating, certain aspects of the procedure, including laryngoscopy, insertion of skull pins, incision, and periosteal-dural contact induce noxious stimulation. These noxious events can result in sudden increases in blood pressure and heart rate (HR), which can cause potential morbidity due to further increases in ICP in patients with intracranial pathology, and a greater risk for rupture in patients with intravascular aneurysms. Therefore, a method to blunt these noxious stimuli would be valuable. Blockade of the nerves that supply the involved region of the scalp may be effective in reducing hypertension and tachycardia, the requirement for vasodilators, and the requirement for an increased depth of anesthesia early in the surgical procedure, all of which may cause increased cerebral blood flow and an increase in ICP.[1] The scalp is innervated by the following; Supratrochlear nerve and the supraorbital nerve from the ophthalmic division of the trigeminal nerve. Zygomaticotemporal nerve from the maxillary division of the trigeminal nerve supplying the hairless temple. Auriculotemporal nerve from the mandibular division of the trigeminal nerve. Greater occipital nerve (C2) posteriorly up to the vertex. Lesser occipital nerve (C2) behind the ear. The supraorbital and supratrochlear nerves are blocked as they emerge from the orbit with a 24-gauge needle introduced above the eyebrow perpendicular to the skin. The zygomaticotemporal nerves are blocked as they emerge lateral to the orbit. The auriculotemporal nerves are blocked bilaterally anterior to the ear at the level of the tragus. Care to be taken to inject above the zygoma to avoid entering the parotid gland and blocking the facial nerve. The post auricular branches of the greater auricular nerves are blocked ~ 1.5 cm posterior to the ear at the mastoid process. The greater, lesser, and third occipital nerves are blocked using a 24-gauge needle, with infiltration along the superior

[1]Associate Professor, [3]Professor, [4]Senior Professor and Head, Department of Anaesthesiology and Intensive Care, Nizam's Institute of Medical Sciences, Panjagutta, Hyderabad, India
[2]Consultant, Intensive Care and Anesthesia, Star Hospitals, Banjara Hills, Hyderabad, India

Corresponding author: Dr. Dilip Kumar Kulkarni, MD, Professor, Department of Anaesthesiology and Intensive Care, Nizam's Institute of Medical Sciences, Panjagutta, Hyderabad, India

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nuchal line, approximately halfway between the occipital protuberance and the mastoid process. This study evaluates the efficacy of adding dexmedetomidine to bupivacaine for performing scalp block by attenuating hemodynamic response to skull pin placement.

MATERIAL AND METHODS

This was a prospective, randomised, double-blinded study conducted from from November 2009 to September 2010 in which 50 patients of either sex were included. The institutional ethics committee approval, informed and written consent was obtained.

Inclusion criteria were patients of either sex, belonging to ASA I and II status undergoing elective craniotomy were enrolled.

Exclusion criteria were patients belonging to ASA III and ASA IV, who were of age less than 18 years and age of more than 60 years, patients with GCS less than 8, patients who had allergy or intolerance to study drugs, refusal by the patient.

Premedication consisted of tablet Ranitidine 150 mg and alprazolam 0.25 mg. After securing intravenous lines under local analgesia, a left radial artery and right cubital vein were cannulated for monitoring direct arterial blood pressure and central venous pressure simultaneously. Transducers were placed at mid axillary level and readings noted. All patients were preoxygenated and induced with fentanyl 1.5µgm/kg and thiopentone 4-6mg/kg, tracheal intubation was facilitated with Rocuronium bromide 0.6-1.2mg/kg. Anaesthesia was maintained with 60% nitrous oxide in oxygen and isoflurane 0.4-0.6%. Patients were randomly allocated to one of the two groups, Group 1 received scalp block with a 20 ml solution having Dexmedetomidine 1mcg/kg and 0.25% bupivacaine. Group 2 received scalp block with 20 ml of 0.25% bupivacaine. Both, the anaesthesiologist giving the block and the surgeon applying the pins were blinded, pin placement was done 5 minutes after scalp block was given. Data was recorded from before induction till 8 minutes after pin placement. During pin placement, since BP and HR increase abruptly over a period of few seconds during tightening of pins, to determine the peak increase in these parameters each reading of increase in their values was stored manually in the monitor until such time there was no more increase, the peak heart rate and peak systolic and diastolic blood pressures were noted. A Sujitha head holder was used for all of the craniotomies in the study. The Sujitha head holder used pointed pins that are inserted simultaneously through the dermis engaging in the periosteum to secure the head in a stable position for surgery. The following variables were recorded; heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure(DBP) and mean blood pressure (MBP). The variables were recorded at the following time intervals: Base line (pre induction), before block, before pin placement, during pin placement and later at 1, 3, 5, and 8 minute intervals. Hemodynamic changes in response to pin fixation were compared with the baseline in each group. Also the hemodynamic parameters at each step of recording were compared between the two groups. Descriptive statistics for MAP, SBP and DBP in both the groups were done by mean and standard deviation. Since the baseline values were statistically different in the two groups, percentage changes were calculated at each of the subsequent times. In order to detect a significant change from baseline within the same group, Wilcoxon sign rank test was applied. To compare average MAP,SBP and DBP changes in between the groups, at each point of time, Wilcoxon rank sum (Mann-Whitney) test was applied due to non-normality of distribution of percent change in values. P value of less than 0.05 was taken as statistically significant. Paired 't' test was applied to compare changes in mean HR from baseline in each group separately. Wilcoxon rank sum test (Mann-Whitney), was used to compare differences in HR in the two groups at each point of time. P value of less than 0.05 was taken as statistically significant. Chi square test and student's 't' test were applied to compare the demographic data of the two groups. P value of less than 0.05 was taken as significant.

RESULTS

A total of 50 patients were studied. Group I and group II had 25 patients each. The two groups were well matched for age, sex and weight. All the data are reported as Mean ± SD.

Table 1 shows age in years in group I was 37.12±11.391 and in group II, it was 42.12±13.179. Male to female was 14:11 in group I, it was 11:14 in group II. Weight in kgs was 58.4±10.939 in group I and it was 55.28±7.486 in group II.

Table 2 shows that in group 2 pinning resulted in significant increase in HR from 80.76 ± 15.41 to 87.72 ± 14.75 beats per min (P<0.01). This increase in HR lasted only upto 1 min post pins application and subsequently it started to decline, being significantly less (P<0.05) compared to the baseline at 8 min. In group 1 there was no significant increase in HR during pinning from 80.48 ± 14.02 to 74.20 ± 14.44 beats per min (P<0.01), and HR started to decrease after this being significantly less compared to the base line at 8 min. There was no increase in mean heart rate during and 8 minutes after pin placement. Inter-group comparison during and after

Demographics	Group I	Group II
Age in years	37.12±11.391	42.12±13.179
Sex (Male:Female)	14:11	11:14
Weight (Kgs)	58.4±10.939	55.28±7.486

Table-1: Demographic distribution in the study.

Heart Rate (Beats/min)	Group I	Group II
Pre-induction	80.48±14.02	80.76±15.41
Pre-Block	76.76±13.98	78.48±14.05
Before pin placement	70.84±12.39	78.80±13.88
During pin placement	74.20±14.44	87.72±14.75
1 min after pins	71.16±14.01	117.8±18.46
3 mins after pins	70.44±13.80	78.68±14.68
5 mins after pins	68.28±10.96	77.40±14.11
8 mins after pins	69.72±10.97	75.60±13.45

Table-2: Heart rate changes at various stages in two groups (beats/mins)

pinning of HR showed comparable changes at each step of recording.

Table 3 shows that in group 2 (Bupivacaine 0.25% plain) skull pinning increased MAP significantly from 92.42 ± 9.22 mmHg to 110.37 ± 17.39 mmHg (P<0.001) and this significant increase in MAP was observed at each point of recording throughout the study period. In group 1 (dexmedetomidine + bupivacaine 0.25% group) pinning did not result in an increase in MAP from 91.49 ±10.96mmHg to 89.42 ± 16.56 mmHg (P<0.01). Inter-group comparison showed significant increase in MAP in group 2 at each step of recording.

Table 4 shows that in group 2 (Bupivacaine 0.25% plain) skull pinning increased SBP significantly from 129.92±17.98 mmHg to 148.24±20.83 mmHg (P<0.001) and this significant increase in SBP was observed till 1 minute after pinning. In group 1 (dexmedetomidine + bupivacaine 0.25% group) pinning did not result in an increase in SBP from 131.68±15.93mmHg to 120.04±20.23 mmHg (P<0.01). Inter-group comparison showed significant increase in SBP in group 2 at each step of recording. In group 2 (Bupivacaine 0.25% plain) skull pinning increased DBP significantly from 73.68±8.0 mmHg to 91.44±16.87mmHg (P<0.001) and this significant increase in DBP was observed till 1 minute after pinning. In group 1 (dexmedetomidine + bupivacaine 0.25% group) pinning did not result in an increase in DBP from 71.40±9.36mmHg to 74.12±14.98mmHg (P<0.01). Inter-group comparison showed significant increase in DBP in group 2 at each step of recording.

Table 5 shows that there was no statistically significant difference in oxygen saturation between the two study groups during the study period.

Rescue analgesia: In group 2 (Bupivacaine 0.25% plain) skull pinning resulted in an increase in SBP above 180 mmHg in 4 (16%) patients and rescue analgesia with 0.5 mcg/kg of fentanyl and increasing concentration of isoflurane from 0.4% to 1% till SBP reached acceptable levels had to be used. In group 1 (dexmedetomidine + bupivacaine 0.25% group) skull pinning did not result in such drastic increase in SBP as in group 2 and the need for rescue analgesia did not arise.

MAP (mmHg)	Group I	Group II
Pre-induction	91.49±10.96	92.42±9.22
Pre-Block	83.22±11.13	86.37±10.55
Before pin placement	83.08±15.44	93.65±13.41
During pin placement	89.42±16.56	110.37±17.39
1 min after pins	84.65±14.53	98.78±14.14
3 mins after pins	79.73±12.16	93.64±11.78
5 mins after pins	76.56±10.08	89.04±14.69
8 mins after pins	78.36±10.40	87.92±13.56

Table-3: Mean Arterial Pressure changes at various stages in two groups (mmHg)

DISCUSSION

Acute arterial hypertension can further increase ICP with a risk for herniation, and may also result in pulmonary edema and ruptured cerebral aneurysms.[2,3] Therefore, prevention of acute hypertension in the neurosurgical patient due to noxious stimuli such as head pinning would be desirable.

There have been many studies that demonstrate stable hemodynamics during head pinning with a scalp block using bupivacaine (0.25-0.5%).

Scalp block also removes the requirement for additional anesthesia or vasoactive drugs during the period of head pinning. Therefore, addition of a scalp block to the anesthetic plan for patients undergoing craniotomy will successfully prevent the hyperdynamic response to head pinning without increasing the requirement for volatile anesthetics or antihypertensive drugs.

	SBP (mmHg)		DBP (mmHg)	
Variables	**Group I**	**Group II**	**Group I**	**Group II**
Pre-induction	131.68±15.93	129.92±17.98	71.40±9.36	73.68±8.00
Pre-Block	114.80±16.93	119.36±16.88	67.44±9.39	69.88±9.48
Before pin placement	111.64±18.80	126.08±17.59	68.80±14.18	77.44±12.32
During pin placement	120.04±20.23	148.24±20.83	74.12±14.98	91.44±16.87
1 min after pins	111.48±16.98	131.64±19.35	71.24±13.75	82.36±12.29
3 mins after pins	106.64±14.33	126.04±16.00	66.28±11.78	77.44±10.30
5 mins after pins	102.88±12.94	120.00±20.87	63.40±9.50	73.56±12.12
8 mins after pins	106.20±13.17	118.48±19.20	64.44±9.84	72.64±11.55

Table-4: Systolic (SBP) and diastolic blood pressure (DBP) at various stages in two groups.

SpO_2	Group I	Group II
Pre-induction	97.16±0.55	97.16±0.62
Pre-Block	98.96±0.2	98.88±0.33
Before pin placement	98.96±0.2	98.88±0.33
During pin placement	98.96±0.2	98.88±0.33
1 min after pins	98.96±0.2	98.88±0.33
3 mins after pins	98.96±0.2	98.88±0.33
5 mins after pins	98.96±0.2	98.88±0.33
8 mins after pins	98.96±0.2	98.88±0.33

Table-5: SpO_2 at various stages in two groups (mmHg) in mean±SD.

Scalp block with bupivacaine can significantly influence HR and BP after skull pin placement in adult patients undergoing craniotomy. However, the procedure is not always successful in obtunding the haemodynamic response. Various drugs have been tried as additives of which α 2 agonist clonidine has shown good results.

Clonidine premedication was reported to attenuate the haemodynamic responses to pin head-holder application during craniotomy.[4] Clonidine, the protypical alpha2-adrenoceptor agonist, has been widely studied in humans. Dexmedetomidine is a novel alpha2-agonist with potent anxiolytic and sedative properties. The haemodynamic profile of Dexmedetomidine was found to be similar to clonidine.

Several studies have found Dexmedetomidine to be well tolerated and effective in various neuraxial and regional anaesthetics in humans, including during the delivery of intrathecal,[5] caudal,[6] and intravenous (i.v.) regional anaesthesia.[7]In an animal study, Dexmedetomidine was reported to be well tolerated and it prolonged the duration of sensory blockade effectively when injected perineurally in the peripheries.[8]

In the present study, the Dexmedetomidine-bupivacaine group showed the maximum attenuation of haemodynamic responses to skull pin placement when compared to bupivacaine only group. The need for rescue analgesia also did not arise in the dexmedetomidine-bupivacaine group, where as 16% of the study patients needed rescue analgesia due to sudden increase in blood pressure during pin placement in bupivacaine only group. The mechanism of action of α2-adrenoceptor agonists in peripheral nerve blocks is not understood fully. Proposed mechanisms include central analgesia, vasoconstriction, and anti-inflammatory effects.[9] However, none of these mechanisms can explain fully the synergistic effect of α2-adrenoceptor agonists when added to a local anaesthetic in peripheral nerve blocks.

The direct action of α2-adrenoceptors on the peripheral nerve may be mediated through an increase in hyperpolarization of the after-potential that follows a single compound action potential.[10]

It is well known that in peripheral myelinated and nonmyelinated fibres, membrane hyperpolarization develops during and after stimulation and mainly results from the activation of the sodium–potassium pump after the transient influx of sodium ions.[11]

Pinosky ML et al;[12] conducted a study in which the placement of pointed cranial pins into the periosteum is a recognized acute noxious stimulation during intracranial surgery which can result in sudden increases in blood pressure and heart rate, causing increases in intracranial pressure. A skull block (blockade of the nerves that innervate the scalp, including the greater and lesser occipital nerves, the supraorbital and supratrochlear nerves, the auriculotemporal nerves, and the greater auricular nerves) may be effective in reducing hypertension and tachycardia. Twenty-one patients were allocated in a prospective, double-blind fashion to a control group or a bupivacaine group. SAP, DAP, MAP & HR were noted 5 min after the induction of anesthesia, during performance of the skull block, during head pinning, and 5 min after head pinning. Significant increases in SAP of 40 +/- 6 mm Hg, DAP of 30 +/- 5 mm Hg, MAP of 32 +/- 6 mm Hg, and HR of 22 +/- 5 bpm occurred during head pinning in the control group, while remaining unchanged in the bupivacaine group. These results demonstrate that a skull block using 0.5% bupivacaine successfully blunts the hemodynamic response to head pinning. Nethra H Nanjundaswamy et al;[13] conducted a randomized double blind study conducted with sample size - sixty patients, divided into two groups: Group C (n = 30) - clonidine i.v. dose 2 µg/kg; Group L (n = 30) - lignocaine i.v. dose 1.5 mg/kg. All patients posted for elective craniotomy belonging to American Society of Anesthesiologists (ASA) 1 and 2, age group 18–70 were included in the study, drugs were administered 10 min prior to induction in 10 ml syringes with infusion pump over 10 min. Standard anesthesia protocol followed. HR, noninvasive BP, mean arterial pressure (MAP), and IBP were recorded at baseline (BL), after study drug (AD), 1 min after intubation (AI), 1 min prior to pin insertion -pre pin (PP), and 5 min after pin insertion (AP). Groups were matched with respect to age (P = 0.7), gender distribution (P = 0.6), and weight (P = 0.67) There was no difference in BL HR in two groups. Significant difference in HR was noted after intubation P < 0.031 and pin insertion P < 0.001 stages with lower HR in Group C (76.03 ± 9.88) versus Group L (98 ± 60.89) MAP recordings showed no statistically significant difference in two groups at BL and after drug administration stages. A significant difference was seen in intubation (P < 0.014), very significant difference (P < 0.001) was noted in pre- and post-pin insertion stages with MAP being lower in Group C (76.03 ± 9.88) versus Group L (87.17 ± 8.90).

A.Tripathi et al;[14] conducted a study on sixty American Society of Anesthesiologists Grades I and II patients scheduled for various orthopedic surgeries of the upper limb under supraclavicular brachial plexus block they were divided into two equal groups in a randomized, double-blind manner. Patients were assigned randomly to one of the two groups. In Group C (*n* = 30), 39 ml of 0.25% bupivacaine plus 1 ml (1 µg/kg) clonidine and in Group D (*n* = 30), 39 ml of 0.25% bupivacaine plus 1 ml (1 µg/kg) dexmedetomidine were given. There was no statistically significant difference in the onset of sensory and motor block in both the groups. The durations of sensory and motor block were 316.67 ± 45.21 and 372.67 ± 44.48 min, respectively, in Group C, whereas they were 502.67 ± 43.78 and 557.67 ± 38.83 min, respectively, in Group D. The duration of analgesia was 349.33 ± 42.91 min, significantly less in Group C compared to 525.33 ± 42.89 min in Group D (*P* < 0.001). The quality of anesthesia was significantly better in dexmedetomidine group compared to clonidine group (*P* < 0.001). This study concluded that the addition of dexmedetomidine prolongs the durations of sensory and motor block and duration of analgesia and improves the quality of anesthesia as compared with clonidine when injected with bupivacaine in supraclavicular brachial plexus block.

Sidhesh Bharne et al;[15] conducted a study on sixty-six patients undergoing elective neurosurgical procedures they were randomized into two groups, L (labetalol) and B (bupivacaine) of 33 each. After a standard induction sequence using fentanyl, propofol and vecuronium, patients were intubated. Baseline hemodynamic parameters and entropy levels were noted. Five minutes before application of the pins, group L patients received IV labetalol 0.25 mg/kg and group B patients received scalp block with 30 ml of 0.25% bupivacaine. Following application of the pins, heart rate (HR), systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), and response entropy (RE)/state entropy (SE) were noted at regular time points up to 5 min. HR increased by 19.8% in group L and by 11% in group B. SAP increased by 11.9% in group L and remained unchanged in group B. DAP increased by 19.7% in group L and by 9.9% in group B, MAP increased by 15.6% in group L and 5% in group B ($P < 0.05$). No adverse effects were noted. This study concluded that scalp block with bupivacaine is more effective than IV labetalol in attenuating the rise in hemodynamic parameters and entropy changes following skull pin application. Dalle et al;[16] found that clonidine increases the hyperpolarization that develops during low-frequency stimulation by inhibiting the hyperpolarization-activated cation (Ih) current. The Ih current is activated during the hyperpolarization phase of an action potential and acts to reset a nerve for subsequent action potentials. Thus, clonidine enhances the level of hyperpolarization by blocking the Ih current and thus inhibits subsequent action potentials. Dexmedetomidine is a selective α2-adrenoceptor agonist and it may enhance the sensory blockade in a manner similar to clonidine.

CONCLUSION

The results obtained in this study demonstrate that adding dexmedetomidine to bupivacaine while performing scalp block is very efficient in obtunding hemodynamic response to skull pin placement. This can be very useful in patients who might not tolerate even minimal fluctuations in hemodynamic variables like heart rate, blood pressure and intra cerebral pressure.

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