

COMPARISON BETWEEN INTRAVENOUS DEXMEDETOMIDINE AND LOCAL LIGNOCAINE INFILTRATION TO ATTENUATE THE HAEMODYNAMIC RESPONSE TO SKULL PIN HEAD HOLDER APPLICATION DURING CRANIOTOMY

Dr. Anandh P.* and Dr. Selvamani S.

¹Junior Resident, Department of Anaesthesia, Sree BALAJI Medical College, Chennai.

²Professor, Department of Anaesthesia, Sree Balaji Medical College, Chennai.

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*Corresponding Author

Dr. Anandh P.

Junior Resident, Department
of Anaesthesia, Sree BALAJI
Medical College, Chennai.

ABSTRACT

Background and Aims: Skull pin application is intensely painful and can be accompanied by detrimental haemodynamic changes. We compared intravenous (IV) dexmedetomidine with local infiltration of lignocaine at pin application sites to attenuate haemodynamic changes associated with pin application. **Methods:** Fifty-two patients undergoing craniotomy were randomised to either group dexmedetomidine (received 1 µg/kg dexmedetomidine over 10 min starting at induction of anaesthesia) or group lignocaine (received 3 ml of 2% lignocaine infiltration at pin application sites before pin

application). Anaesthetic protocol was standardised. Heart rate (HR) and mean arterial pressure (MAP) were recorded at the following time intervals, pre-induction baseline, pre-infiltration, post-infiltration, pre-pin application and post-pin application at 1, 2, 3, 4, 5, 10 and 15 min. Statistical analysis was done using independent samples t-test, Fisher exact test and Chi-square test. **Results:** HR and MAP were comparable between the groups at all the study intervals. The incidence of adverse haemodynamic effects attributable to pin application (tachycardia and/or hypertension) was comparable between the groups (2 patients in group dexmedetomidine and 5 in group lignocaine). However, the incidence of hypotension and/or bradycardia was significantly greater in the dexmedetomidine group (19 patients in group dexmedetomidine and 5 patients in group lignocaine; $P = 0.0007$). **Conclusion:** IV dexmedetomidine 1 mcg/kg over 10 min is comparable to local infiltration of 2% lignocaine at pin application sites to attenuate the haemodynamic response associated

with skull pin application. However, use of dexmedetomidine is associated with significantly higher incidence of hypotension and bradycardia.

KEYWORDS: Dexmedetomidine, Haemodynamic Changes, Lignocaine, Skull Pin Head Holder.

INTRODUCTION

Application of skull pin head holder during neurosurgical procedures is intensely painful and accompanied by an abrupt increase in heart rate(HR) and arterial blood pressure. These may lead to brain oedema, increased intracranial pressure or intracranial haemorrhage.^[1] Different techniques have been used to blunt these deleterious haemodynamic changes with variable success. Dexmedetomidine, a α_2 -agonist, may be used to attenuate the haemodynamic response during skull pin application due to its sympatholytic and analgesic properties. In this study, we compared intravenous (IV) dexmedetomidine with the conventionally used method of local infiltration with lignocaine to attenuate haemodynamic response to skull pin head holder application.

METHODS

This prospective, randomised, double-blinded study commenced after obtaining Hospital Ethics Committee approval (IEC 440/2011) and registration with Clinical Trials Registry, India (CTRI/2012/04/002595). Fifty-two consenting patients were recruited. Patients of either gender aged between 18 and 70 years, belonging to American Society of Anesthesiologists physical status 1 or 2 and scheduled for elective craniotomy in a supine or lateral position under general anaesthesia were included. Exclusion criteria were patients having hypertension, ischaemic heart disease, heart block, pregnancy or lactation, signs and symptoms of raised intracranial pressure, head injury, previous craniotomy and tumours of hypophysis. Patients were evaluated and written informed consent was obtained. All patients received fasting instructions of 6 and 2 h for solids and clear fluids, respectively. On the day of the surgery, patients were randomised to group dexmedetomidine or group lignocaine (computer generated random numbers table). Patient allocation was concealed in sealed envelope. Study drugs were prepared by the anaesthesia technician/resident not involved in the study in unlabelled 10 ml syringes. Patients randomised to group dexmedetomidine received 1 $\mu\text{g}/\text{kg}$ of dexmedetomidine diluted to 10 ml with 0.9% saline over 10 min through a syringe pump, after recording pre-induction baseline haemodynamic parameters. Before pin application, these patients received infiltration of the pin sites with 0.9% saline (3 ml for each

site). Patients randomised to group lignocaine received infusion of 10 ml of 0.9% saline over 10 min, after recording pre-induction baseline haemodynamic parameters. They then received infiltration of the pre-marked pin sites with 2% lignocaine (without adrenaline), 3 ml for each site. Infiltration of pin sites was done by the same neurosurgeon blinded to group allocation with 24-gauge needle. Pins were applied 2 min after infiltration. Observer 1 recording the haemodynamic variables was blinded to group allocation. In the operating room, monitoring was established with 5 lead electrocardiogram, blood pressure, pulse oximetry and end-tidal carbon dioxide. (ETCO₂) pre-induction baseline values were recorded. IV access was secured. General anaesthesia was induced with IV propofol and fentanyl 2 mcg/kg. After confirming the ability to ventilate, IV vecuronium 0.1 mg/kg was given. Ventilation was assisted with 2% isoflurane in 100% oxygen for 3 min followed by tracheal intubation. For the next 10 min, it was ensured that there was no stimulus to allow the haemodynamic parameters to settle. Anaesthesia was maintained with isoflurane in 50% oxygen and nitrous oxide targeting 1–1.3 minimum alveolar concentration and ETCO₂ of 33–36 mmHg. Subsequently, pin site infiltration and pin application were done as detailed above. HR and mean arterial pressure (MAP) were recorded by the blinded observer at the following time intervals: Pre-induction baseline value (immediately before IV administration of dexmedetomidine or saline), pre-infiltration (just before infiltration of pin sites), post-infiltration (just after infiltration of all pin sites), pre-pin application (just before pin application) and post-pin application T1, T2, T3, T4, T5, T10, T15 (1, 2, 3, 4, 5, 10 and 15 min respectively after pin insertion). Up to 15 min following skull pin application, no other stimulus was applied. Bradycardia (HR 30% increase from baseline HR), hypertension (>30% increase from baseline MAP) and hypotension (>30% decrease from baseline in MAP) were treated. IV propofol in increments of 10 mg up to 1 mg/kg was administered to treat hypertension and tachycardia. Bradycardia was treated by administration of IV atropine 0.5 mg. Hypotension was initially treated by decreasing the inspired isoflurane concentration to 0.5% and if persistent by administration of IV mephentermine in 3 mg boluses. The number of patients who received rescue medications was recorded. An increase in HR to more than 15 beats and MAP by 20% from the pre-pin application time to 1 min after pin application was used as the primary outcome for sample size estimation. The sample size was calculated in consultation with statistics department. Considering the analysis of variance for this clinically detectable difference, at 5% level of significance and 80% power, the required sample size was 26 in each group.

RESULTS

Table 1: Patient characteristics.

| Patient characteristics | Group dexmedetomidine | Group lignocaine |
|---------------------------------------|-----------------------|------------------|
| Age in years Mean (SD) | 43.38 (14.93) | 42.62 (14.27) |
| Weight in kg Mean (SD) | 60.5 (6.68) | 61.08 (13.12) |
| Gender Male/female (absolute numbers) | 12/14 | 18/8 |

Table 2: Comparison of incidence of adverse haemodynamic effects during the study period.

| Group (number of patients) | Adverse haemodynamic effect present | Adverse haemodynamic effect absent | P value |
|----------------------------|-------------------------------------|------------------------------------|---------|
| Group dexmedetomidine | 22 | 4 | 0.001 |
| Group lignocaine | 10 | 16 | |

Table 3: Comparison of incidence of adverse haemodynamic effects due to application of skull pins.

| Group (number of patients) | Adverse effect (pinning) | No adverse effect (pinning) | P value |
|----------------------------|--------------------------|-----------------------------|---------|
| Group dexmedetomidine | 2 | 24 | 0.22 |
| Group lignocaine | 5 | 21 | |

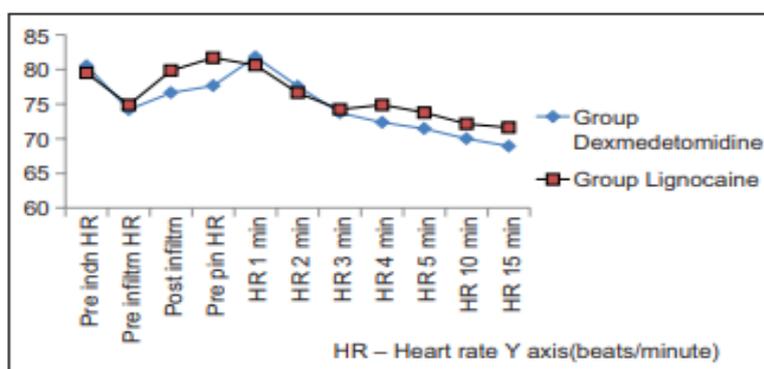


Figure 1: Comparison of mean heart rate between the groups

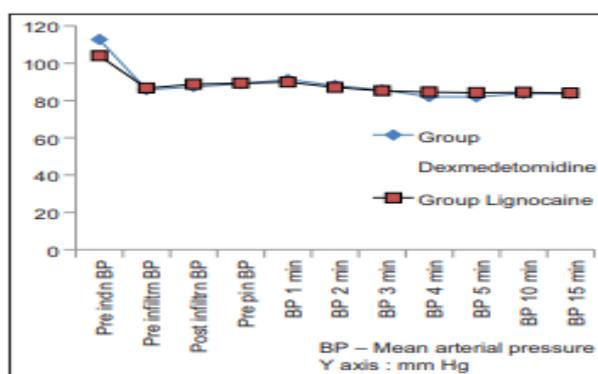


Figure 2: Comparison of mean arterial pressure between the groups

The patient characteristics are given in Table 1. The mean (standard deviation) time interval between induction of anaesthesia and the application of the skull pin was 34.04 (15.62) min and 31.96 (12.22) min in group dexmedetomidine and lignocaine, respectively ($P = 0.596$; independent samples t-test). The HR and MAP at various study periods were comparable between the groups [Figures 1 and 2, independent samples t-test]. The overall incidence of adverse haemodynamic effects (bradycardia, hypotension, tachycardia and hypertension) was significantly greater in the dexmedetomidine group [Table 2]. The incidence of adverse haemodynamic effects attributable to pin application (tachycardia and/or hypertension) was comparable between the groups [Table 3]. However, 19 patients in group dexmedetomidine and 5 patients in group lignocaine had hypotension and/or bradycardia. This difference was significant ($P = 0.0007$).

DISCUSSION

Different modalities have been tried to reduce the haemodynamic response to skull pin application, the most commonly studied method being local lignocaine infiltration at the pin application sites.^[2-9] However, this method was not always successful because of improper infiltration, head movement during fixation, changes in pin sites and inadequate dosage of local anaesthetic. This resulted in comparison of lignocaine infiltration with other modalities such as oral clonidine or gabapentin pre-medication, IV fentanyl, bupivacaine skull block and different IV anaesthetics.^[2-9] All the studies yielded varying results and thus, it was evident that further research would be required to identify the ideal modality. Dexmedetomidine, a selective α_2 adrenoceptor agonist, has sedative, analgesic and anaesthetic-sparing effect. It decreases the HR, MAP and sympathetic nervous system activity. We compared dexmedetomidine with the commonly used method of local lignocaine infiltration at pin sites for attenuating the haemodynamic responses to skull pin insertion. Anaesthetic technique was standardised in this study. Based on the standard recommended dose, group dexmedetomidine received 1 $\mu\text{g}/\text{kg}$ of dexmedetomidine diluted to 10 ml with 0.9% saline over 10 min at the time of induction.^[1] These patients received infiltration of the pin sites with 0.9% saline (3 ml at each site) to ensure blinding of the observer. Patients randomised to group lignocaine received 10 ml of 0.9% saline IV over 10 min at induction and then received infiltration of pin sites with 2% lignocaine 3 ml at each site. The timing of dexmedetomidine infusion was such that the peak effect of the drug would coincide with the time of pin application. We found that HR and MAP were comparable between the groups at various time intervals in the study. Thus, both dexmedetomidine and lignocaine is equally

effective in controlling the haemodynamic response to skull pin application. Despite being comparable to lignocaine infiltration, dexmedetomidine causes significantly more episodes of hypotension/ bradycardia, which could be detrimental in a neurosurgical patient. The bradycardia/hypotension may not be attributable to dexmedetomidine in all the cases because occasionally the haemodynamic response to skull pin application can manifest as bradycardia/hypotension. Few studies in literature have evaluated dexmedetomidine to control the haemodynamics during skull pin application. Uyar *et al.* compared dexmedetomidine (1 µg/kg over 10 min) with placebo and its effect on haemodynamics during pin application.^[1] They found that dexmedetomidine attenuated the haemodynamic response to pin application. Contrary to our findings, they did not find hypotension and bradycardia requiring rescue medication in both the groups. El Dawlatly *et al.* also conducted a study in which 28 patients were randomised to four groups as Dex group (0.25 µg/kg infusion of dexmedetomidine over 10 min), Ligno group (1% lignocaine infiltration at pin sites), Dex-Ligno group (combination of dexmedetomidine infusion and lignocaine infiltration) and placebo.^[10] They found that both dexmedetomidine and lignocaine were equally effective in attenuating the haemodynamic response to pin application. The combination of low dose dexmedetomidine infusion and local lignocaine infiltration maximally attenuated the haemodynamic response. They, too, reported no hypotension and/or bradycardia requiring treatment. Therefore, contrary to the above studies, although dexmedetomidine was useful in attenuating the haemodynamic response, the occurrence of hypotension and/or bradycardia was a limiting factor in its use. Considering that, both lignocaine and dexmedetomidine served the purpose of controlling the haemodynamic response to skull pin application with lignocaine having less drug-related side effects, it could be concluded that lignocaine is a better option in this regard. Further studies may be required to formulate a dosage of dexmedetomidine which will attenuate the haemodynamic response without causing significant hypotension or bradycardia.

CONCLUSION

IV dexmedetomidine 1 µg/kg over 10 min is comparable to local infiltration of 2% lignocaine at pin application sites to attenuate the haemodynamic response associated with skull pin application. The use of dexmedetomidine is associated with significantly higher incidence of hypotension and bradycardia.

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