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A STUDY ON HAEMODYNAMIC RESPONSE ATTENUATION DURING HEAD HOLDER APPLICATION FOR CRANIOTOMY COMPARING IV DEXMEDITOMIDINE AND LOCAL LIGNOCAINE

Jayadheer.D*

Associate Professor Department of Anaesthesiology Guntur Medical College. Guntur. A.P. India.*Corresponding Author

ABSTRACT **Background;** One of the goals of neuro anesthesia is to ensure stable perioperative cerebral hemodynamics, thus avoiding a sudden rise in intracranial pressure and prevent acute brain swelling.1 The intense surgical stimuli associated with craniotomy frequently causes sympathetic activation, which results in changes in heart rate (HR), blood pressure (BP), and cerebral blood flow (CBF). These changes may increase intracranial pressure (ICP) and a reduction in cerebral perfusion pressure (CPP). Finally, it leads to cerebral ischemia, especially in patients with impaired autoregulation and compromised cerebral compliance2,3. Thus, it is essential to preserve cerebral homeostasis and to prevent abrupt changes in hemodynamics. Smooth and rapid recovery from anesthesia allows immediate neurological assessment. Application of skull pin head holder is a necessity for stabilizing the head during craniotomy. Mayfield device or head holder is a clamp thatconsists of a C-shaped metal piece with three sharp interchangeable metal pins arranged triangularly to one another.4 These pins forced through the layers of scalp and periosteuminto the external lamina of the skull. Skull pins support the head without allowing any direct pressure on the face, allow access to the airway, and hold head firmly in one position that can finely be adjusted for optimal neurosurgical exposure.**Methods:** This study was conducted in fifty ASA grade 1 or 2 patients who were admitted at Government General Hospital, Guntur affiliated to Guntur Medical College, Guntur, to undergo elective craniotomies under general anesthesia. After getting Ethical committee approval, a total of fifty patients were allocated into two groups of 25 each. They were connected to the non-invasive monitors, and the basal heart rate and mean arterial pressure were recorded.Patients randomized to group dexmedetomidine received 1mcg/kg of dexmedetomidine diluted to 10ml with 0.9% saline over 10 min through a syringe pump, after recording pre-induction baseline hemodynamic parameters. Before the pin application, these patients received infiltration of the pin sites with 0.9% saline (3ml for each site). Patients randomized to group lidocaine received infusion of 10ml of 0.9% saline over 10min, after recording baseline hemodynamic parameters. They then received infiltration of the pre-marked pin sites with 2% lidocaine (without adrenaline), 3ml for each site. Heart rate and mean arterial pressure were recorded at various time intervals.Baseline, preinfiltration, post infiltration, pre pin, 1 minute after post pin, 2minutes after post pin, 3 minutes, 5 miutes, 10 minutes and 15 minutes after post pin application. The result was analyzed using student t-test, and a P value of less than 0.05 was taken as significant. **Results**With patients matched for demographic data, the results showed there was no significant difference in baseline values between the two groups. Heart rate and mean arterial pressure were comparable between the groups at various time intervals in the study. Thus both dexmedetomidine and lidocaine are equally effective in controlling the hemodynamic response to skull pin application. Despite being comparable to lidocaine infiltration, dexmedetomidine causes significantlymore episodes of hypotension and bradycardia, which could be detrimental in neurosurgical patients. **Conclusion** Dexmedetomidine 1mcg/kg infusion and 2% lignocaine infiltration both are equally effective in controlling the hemodynamic response to skull pin placement. Despite being comparable to lignocaine infiltration, dexmedetomidine causes significantly more episodes of bradycardia and hypotension, so they need rescue medication, which could be detrimental in a neurosurgical patient. We conclude that 2% of local lignocaine infiltration is better in controlling hemodynamic responses to skull pin head holder application and does not cause any adverse effects in any of the patients.

KEYWORDS : Craniotomy E04.525.190,Arterial Pressure G09.330.380.076.347, Blood Circulation G09.330.100

2)Introduction

Skull pin insertion is intensely painful and causes an abrupt increase in heart rate and blood pressure due to sympathetic stimulation. These hemodynamic changes can lead to cerebral edema, an increase in intracranial pressure and rupture of an intracranial aneurysm, which is deleterious, especially in patients with intracranial pathology.An acute increase of blood pressure may cause rupture or rerupture of intracranial vascular lesions (cerebral aneurysms or arterio venous malformations) and present with subarachnoid or intracerebral hemorrhage (ICH). Arterial hypertension can lead to acute cerebral edema and herniation of the brain within minutes.In susceptible patients, an acute rise in BP might disrupt the intracranial milieu and can also cause extra cranial complications such as myocardial ischemia and pulmonary edema. Hence it is necessary to attenuate the hemodynamic response to skull pin application to avoid harmful effects.

Various anesthetic and pharmacologic techniques, like infiltration of local anesthesia at the site of skull pin application and intravenous drugs like opioids, esmolol, clonidine, gabapentin, and dexmedetomidine are used to attenuate the hemodynamic response to skull pin application and to avoid harmful effects. Dexmedetomidine (DEX) is a highly specific potent and selective α-2 adrenoreceptor agonist.It produces dose-dependent sedation, anxiolysis, and analgesia without respiratory depression. These characteristics make this drug a potentially attractive adjunct for neuroanesthesia and in the neurological intensive care unit (ICU). Dexmedetomidine decreases heart rate, mean arterial pressure, and sympathetic nervous system activity in a dose-dependent fashion. Dexmedetomidine has shown analgesic effects without significant respiratory depression.It also provides excellent intraoperative hemodynamic stability with decreased intraoperative opioid requirements. Local anesthetic agents are infiltrated into the skull before the application of pins to attenuate the hemodynamic responses. Local anesthesia uses a small volume of the drug, with rapid onset of analgesia, no additional increase in depth of anesthesia, and attenuation of hemodynamic perturbation. Local lidocaine is a widely used local anesthetic. It is readily available, inexpensive, and despite being an antiarrhythmic, when used as a local anesthetic, it has minimal side effects.

In this study, the effects of Dexmedetomidine infusion and local Lidocaine infiltration on hemodynamic responses to skull pin insertion in patients undergoing Craniotomies were studied.

3)Aim and objectives

Aims;

To assess the effects of Dexmedetomidine in attenuating hemodynamic response to skull-pin head-holder application during craniotomy.

To assess the effects of Lidocaine infiltration in attenuating hemodynamic response to skull-pin head-holder application during craniotomy.

To assess the intraoperative requirements of rescue medications like vasopressors and atropine.

Objective

To compare the effectiveness of IV dexmedetomidine infusion 1mcg/kg and local 2% lidocaine infiltration (3ml in each pin site) in attenuating heart rate and blood pressure response to skull pin insertion in patients undergoing craniotomies.

4)Material and methods

This study was conducted in Government General Hospital, Guntur, which is a tertiary hospital affiliated to Guntur Medical College, Guntur. After obtaining approval from the institutional ethical committee patients' consent was taken before hand. Surgeon was

informed of the study A total of 50 patients of (25 in each group) of both sexes in the group between 18-70 years belonging to ASA 1 or 2 undergoing elective craniotomies under general anesthesia were included in this double blinded, randomized, clinical study.

methodology

Patients were evaluated, and written informed consent was obtained. All patients received fasting instructions of 6 and 2 hrs for solids and clear fluids, respectively. On the day of the surgery, patients were randomized to group dexmedetomidine or group lignocaine (computergenerated random numbers table). The patient allocation was concealed in a sealed envelope.In the operating room, monitoring was established with five lead electrocardiogram, blood pressure, pulseoximetry, and end-tidal carbon dioxide (ETCO2). The baseline values heart rate and mean arterial pressure recorded. IV access was secured with 18 G cannula. General anesthesia was induced with IV Thiopental 5mg/kg and fentanyl 2mcg/kg.

After confirming the ability to ventilate,IV vecuronium 0.1mg/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 hemodynamic parameters to settle. Anesthesia was maintained with isoflurane in 50% oxygen and nitrous oxide.Study drugs were prepared by the postgraduate who did not involve in the study Patients randomized to group dexmedetomidine received 1mcg/kg of dexmedetomidine diluted to 10ml with 0.9% saline over 10 min through a syringe pump, after recording pre-induction baseline hemodynamic parameters. Before the pin application, these patients received infiltration of the pin sites with 0.9% saline (3ml for each site).

Patients randomized to group lignocaine received infusion of 10ml of 0.9% saline over 10min, after recording baseline hemodynamic parameters. They then received infiltration of the pre-marked pin sites with 2% lignocaine (without adrenaline), 3ml 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.

HR and mean arterial pressure (MAP) were recorded at the following time intervals : Baseline, Pre-infiltration,Post-infiltration, Pre-pin application Post-pin application, One minute (T1), Two minutes (T2), Three minutes (T3), Five minutes (T5), Ten minutes (T10),Fifteen minutes (T15). Up to 15 min following skull pin application, no other stimulus was applied Bradycardia (HR<55 beats/min), tachycardia (>30% increase from baseline MAP), Hypertension (>30% increase from baseline MAP) and hypotension (>30% decrease from baseline MAP) were treated. Bradycardia was treated by administered IV atropine. Hypotension was initially treated by decreasing the inspired isoflurane concentration and if persistent, by the administration of phenylephrine. The number of patients who received rescue medications was recorded.

Statistical methods

The present study was undertaken in 50 ASA grade 1 or 2 patients of both genders between the age group of 18 to 70 years scheduled for elective craniotomy under general anesthesia. The patients were categorized into two groups Dexmedetomidine group (GROUP- D) and the Lidocainegroup(GROUP-L). We used the students' independent "t" test to compare various factors between the two groups. Results were expressed as mean, and standard deviation (mean±SD) Chi-square test was done to compare proportions, ad values less than 0.05 were considered as statistically significant. The groups were matched for demographic data, and there was no statistically significant difference found between the groups in age, sex, and weight.

Inclusion criteria

ASA grade 1 and grade 2
Both sexes
Age 18-70 years
Elective craniotomy in a supine or lateral position under general anesthesia.

Exclusion criteria

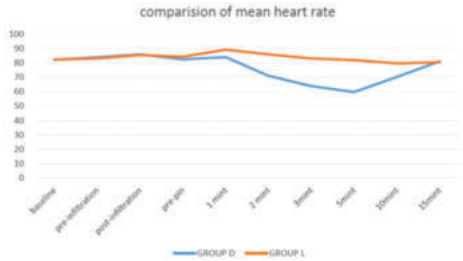
Ischaemic heart disease
Heart block
Hypertension
Pregnancy or lactation
Previous craniotomy

5)Results

In the study, nearly half of the study population was in the age group 30-40 years in both groupsi.e.group D and group L, followed by the age group 40-50 years.The mean age of the study population who receivedDexmedetomidine was 39.04±9.91, and who received lignocaine infiltration was 37.96±9.75. The difference was not statistically significant(P-value, 0.6994).The mean weight of the study population who received Dexmedetomidine was 54.28±4.72 kg and who received lignocaine infiltration was 54.76±4.58kg. The difference was not statistically significant(P-value 0.7171). Out of 50 patients, males constitute 25 in number, of which 12 were administered dexmedetomidine, and 13 were administered local lidocaine. Females represent 25, of which 13 were administered dexmedetomidine, and 12 were administered local lidocaine.There was no significant statistical difference in heart rate and mean arterial pressure between the two groups. The baseline mean heart rate of dexmedetomidine group 82.32±4.50 and lidocaine group 82.32±6.83. The P-value wasP=1.00, which was not statistically significant.

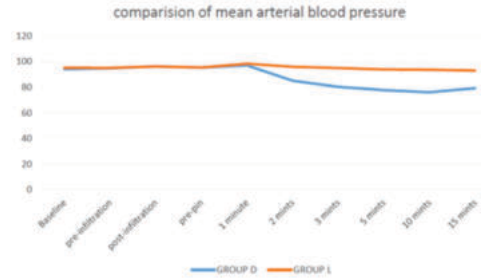
The baseline mean arterial pressure of dexmedetomidine group was94.08±4.34 and lidocaine group was 95.16±4.13. The P-value was P=0.3719, which was not statistically significant.After post pin placement (1 minute, 2 minute, 3minute, 5 minute,10minute, 15 minutes). The mean heart rate of the baseline, pre infiltration, post infiltration, and pre-pin application of dexmedetomidine and lidocaine group the Pvalue was greater than 0.05 and not statistically significant. After 1 minute of pin application, both groups were equally attenuating the hemodynamic response to skull pin but maximum attenuation was seen in dexmedetomidine group P<0.0018. The mean heart rate hadmaximum fall in the dexmedetomidine group when compared to the lidocaine group with a P value <0.0001, which was statistically significant. Dexmedetomidine group required rescue medication.(graph-1)

Graph -1 COMPARISON OF MEAN HEART RATE AT DIFFERENT TIME INTERVALS



The mean MAP of baseline, pre-infiltration, post infiltration, and prepin application of dexmedetomidine and lidocaine group the P-value was greater than 0.05, so P-value was not statistically significant. After 1 minute of pin application, both groups were equally attenuating the hemodynamic response to skull pin P=0.2137 (not statistically significant). As the mean MAP showed a maximum fall in the dexmedetomidine group when compared to lidocaine group with a P value <0.0001, which was statistically significant. Dexmedetomidine group was not given any rescue medication for the fall in MAP.(Graph -2)

Graph -2 COMPARISON OF MEAN MEAN ARTERIAL PRESSURE (MAP) AT DIFFERENT TIME INTERVALS



Out of the 50 patients, five patients in the dexmedetomidine group required atropine due to bradycardia (HR less than 55 per min), and none of the patients in the lidocaine group required atropine.

6)Discussion

Uyar et al.,comparedDexmedetomidine (1 µg/kg over ten mints) with a placebo and its effects on hemodynamic response to skull pin application during craniotomy. Pin attachment significantly increased heart rate and mean arterial pressure in the placebo group compared with both the baseline and the dexmedetomidine group at one and five minutes after pin insertion (P<0.05). Although heart rate and mean arterial pressure values were similar to baseline in the placebo group, they weresignificantly decreased in the dexmedetomidine group from 10 to 60 minutes after pin insertion. They found that dexmedetomidine attenuates the hemodynamic response to pin placement. They did not find hypotension and bradycardia requiring rescue medication in both groups[1].

Paul et al.,reported significant adverse effects such as bradycardiaand hypotension with dexmedetomidine in their study. They compared IV dexmedetomidine infusion (1 mcg/kg) and lignocaine infiltration at the pin site and found that both were comparable in attenuating the response to skull pin insertion. Similar to our study, dexmedetomidine causes more incidence of hypotension and bradycardia. Other studies on Dexmedetomidine did not show this effect[2].

Kondavagilu et al.,comparedtwo Doses IV dexmedetomidine (1mcg/kg and 0.5 mcg/kg bolus) onattenuation of hemodynamic responses to skull pin insertion and found that dexmedetomidine in either dose (1mcg/kg or 0.5mcg/kg) was effective in attenuating hemodynamic response to skull pin insertion[3]. Dexmedetomidine 1mcg/kg showed a higher and sustained attenuation of mean arterial pressure. Dexmedetomidine in doses of 0.5 mcg/kg was as effective in attenuating the heart rate and mean arterial pressure response to skull pin insertion as compared to a dose of 1 mcg/kg similar to our study maximum attenuation of mean arterial pressure seen with dexmedetomidine at the dose of 1mcg/kg.El Dalwaltly et al.,conducted a study in which 28 patients were randomized to 4 groups as Dex group (0.25µg/kg) infusion of dexmedetomidine over 10 minutes. Lignocaine (1% lignocaine at pin sites), Dex-lignocaine group (a combination of dexmedetomidine infusion and lignocaine infiltration), and Placebo. They found that both dexmedetomidine and lignocaine were equally effective in attenuating the hemodynamic response to pin application. The combination of low dose Dexmedetomidine and local lignocaine infiltration maximally attenuate the hemodynamic response to pin application. Their study did not show any hypotension and bradycardia, which necessitated treatment[4]. This study corroborates with our study that attenuation of hemodynamic responses is seen with both dexmedetomidine and lidocaine infiltration.

Ozkose et al.,forty-five patients were allocated into three groups, namely fentanyl (2µg/kg infusion), a lidocaine group (1% lidocaineinfiltration at the pin sites) and the last group (combination of fentanyl and lidocaine infiltration). The result showed that the combination of fentanyl and lidocaine group was the best at controlling hemodynamic response to skull pin placement[5].Colley et al.,studied the prevention of blood pressure response to skull-pin head holder by local anesthesia. They divided group n1 (six patients), the skull -pins were placed without the use of local infiltration. In group 2(six patients), the skin, subcutaneous tissues, and periosteumwere infiltrated with 0.5% lidocaine without epinephrine at the pin site. The MAP in group 1 is 119±25.31, and group 2 is 88±8.8. They found that local infiltration of 3 to 5ml of 0.5% lidocaine without epinephrine at the pin site of pin application completely prevented the blood pressure response to the skull-pin head holder[6]. This study corroborates the evidence in our study that local anesthesia prevents acute hypertension.

Agarwal et al.,studied effect of a subanaesthetic dose of intravenous ketamine and/or local anaesthetic infiltration on hemodynamic response to skull pin placement .40 patients divided 4 groups ketamine (0.5mg/kg), placebo, lidocaine(1%), ketamine-lidocainegroup, mean arterial pressure response in the ketamine group was similarto the placebo group . The study demonstrates maximum attenuation of hemodynamic responses when a subanaesthetic dose of intravenous ketamine is administered with 1%lidocaine infiltration[7].

Arshadet al., studied how effective is the local anesthetic infiltration of pin sites prior to the application of head clamps. A prospective study was conducted in patients who were undergoing elective craniotomies. Divided into two groups, group A received lidocaineinfiltration containing adrenaline in concentration 1:1000, 2-3 ml at each pin site .while group B did not. Hemodynamic response to pin application was then studied at various intervals. The result has shown that lidocaineinfiltration of pin sites prior to skull pin application significantly blunts the rise in heart rate and mean arterial pressure (P≤0.001) without complimenting any systemic methods without causing any wound-related or systemic side effects[8]. This study corroborates with the evidence in our study local lignocaine infiltration blunts the hemodynamic response to skull pin head clamps.

7) Conclusion

Dexmedetomidine 1mcg/kg infusion and 2% lignocaine infiltration both are equally effective in controlling the hemodynamic response to skull pin placement. Despite being comparable to lignocaine infiltration, dexmedetomidine causes significantly more episodes of bradycardia and hypotension, so they need rescue medication, which could be detrimental in a neurosurgical patient. We conclude that 2% of local lignocaine infiltration is better in controlling hemodynamic responses to skull pin head holder application and does not cause any adverse effects in any of the patients.

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