Print ISSN: 2321-6379
Online ISSN: 2321-595X

Original Article

Dexmedetomidine Versus Labetalol in Controlling Emergence Hypertension in Craniotomy

Naveen R S[1], Neeraj Narang[2], Ashish Sethi[3]

[1]Senior Resident, Department of Anesthesiology and Critical Care, Netaji Subhash Chandra Bose Medical College, Jabalpur, Madhya Pradesh, India, [2]Associate Professor, Department of Anesthesiology and Critical Care, Netaji Subhash Chandra Bose Medical College, Jabalpur, Madhya Pradesh, India, [3]Professor and Head, Department of Anesthesiology and Critical Care, Netaji Subhash Chandra Bose Medical College, Jabalpur, Madhya Pradesh, India

Abstract

Introduction: Emergence hypertension is a poorly understood yet notorious phenomenon, especially in neurosurgical cases. This may pave the way for deleterious complications leading to increased patient morbidity. The aim of this study was to compare dexmedetomidine and labetalol in controlling emergence hypertension in patients undergoing craniotomy.

Materials and Methods: We conducted a prospective, randomized, comparative study of 60 patients who underwent craniotomy. These patients were randomized into two groups. At the start of dural closure, Group D received intermittent i.v boluses of dexmedetomidine 0.1 mcg/kg every 3 min and Group L received intermittent i.v boluses of labetalol 0.04 mg/kg every 3 min if systolic BP was >20% of baseline or more than 140 mm of Hg. Hemodynamic parameters, cumulative dose of the drugs and sedation scores were determined.

Results: Significant effect in attenuating pulse rate, systolic, diastolic, and mean arterial blood pressure was observed in both groups with better reduction of blood pressures by dexmedetomidine and that of pulse rate by labetalol. Patients who received dexmedetomidine were found to have lower Richmond Agitation and Sedation Scores (RASS) than those who received labetalol. The mean dose of drugs used to achieve this was 18 mcg of dexmedetomidine and 12 mg of labetalol.

Conclusion: Dexmedetomidine was more effective than labetalol in controlling the blood pressure associated with the emergence phenomenon in patients undergoing craniotomy but caused more sedation.

Key words: Dexmedetomidine, Labetalol, Emergence hypertension

INTRODUCTION

Rapid and smooth emergence after craniotomy is a common practice, the goal of which is to permit early neurological examination. The most feared complication after intracranial surgery is the development of post-operative hematoma, the incidence being 0.8%–2.2%.[1] The surgical stress often leads to the emergence hypertension and may predispose to intracranial hematoma and cerebral edema.[2] Both may lead to cerebral hypoperfusion and brain injury.

Access this article online

www.ijss-sn.com

Month of Submission : 09-2020
Month of Peer Review : 09-2020
Month of Acceptance : 10-2020
Month of Publishing : 11-2020

Recovery from general anesthesia and extubation is a period of intense physiological stress for patients. This is mainly caused by pain associated with the surgery and discomfort caused by the presence of endotracheal tube during awakening.

There are several post-operative physiological responses to stress, which include an increase in oxygen consumption, catecholamine levels in blood, blood pressure, and heart rate. Blood pressure and heart rate increase gradually toward pre-operative values or higher as the patient recovers from general anesthesia. Extubation causes a transient increase in these parameters due to tracheal and laryngeal stimulation. Shivering and pain are also among the main causes of these metabolic and hemodynamic changes and changes in cerebral blood flow and intracranial pressure. Pain is a key stress factor which increases post-operative oxygen consumption and release of catecholamines.

Corresponding Author: Neeraj Narang, Flat no 402, RABS Heights, Near Second Bridge, South Civil Lines, Jabalpur - 482 001, Madhya Pradesh, India.

Thus, anesthetic emergence should include the maintenance of respiratory and cardiovascular parameters. Emergence hypertension has an incidence of more than 90% in neurosurgical patients.[3] Hypertension during recovery from anesthesia and extubation is partly reduced by analgesia through a decrease in catecholamine release.[2] Some of the factors like pre-operative patient status and the surgical procedure does not remain under the control of the anesthesiologist. However, conditions such as post-operative hypothermia, severe pain, restlessness, coughing, and fighting the endotracheal tube seen during recovery are preventable.

Conventionally, non-anesthetic drugs such as β-blockers, calcium channel blockers, and lidocaine have been tried successfully to blunt the hemodynamic response during emergence and extubation.[4] These drugs have their own disadvantages. Labetalol, a combined selective alpha-1 adrenergic receptor blocker and non-selective beta-adrenergic blocker given by i.v bolus or continuous infusion, has been studied for the treatment of emergence hypertension. The overall response rate is 85–100%,[5] making labetalol an effective option to control emergence hypertension. However, its contraindications in asthma and congestive heart failure and significant bradycardia must be considered.[6]

Intravenous infusions of dexmedetomidine, a highly selective alpha-2 adrenergic agonist with sedative, anxiolytic, analgesic and hypotensive properties, have also been evaluated as an adjuvant to anesthesia for neurosurgery with desirable effects on hemodynamic parameters.[7,8] However, studies with intermittent boluses of dexmedetomidine rather than continuous infusion are yet to be documented.

In this study, we compare the effect of intermittent i.v boluses of dexmedetomidine and labetalol in attenuating emergence hypertension in patients undergoing craniotomy.

MATERIALS AND METHODS

The study was planned as a prospective, randomized, and double-blind study. After getting approval from the Institutional Ethical Committee and obtaining informed consent from the patients enrolled in the study, 60 patients of ASA I and II scheduled for craniotomy under general anesthesia were randomly allocated to one of the two following groups using the sealed envelope technique. Any patient who refused to give consent, who was of the age <18 or >65 years, ASA physical status >II, with impaired renal functions, uncontrolled hypertension, bronchial asthma, arrhythmias, congestive cardiac failure, Glasgow coma scale <12, requirement of post-operative ventilation, and history of allergy to the study drugs were excluded from the study.

At any point from the start of dural closure, should emergence hypertension occur, we planned to give intermittent boluses of i.v dexmedetomidine to patients in Group D and intermittent boluses of i.v labetalol for patients in Group L till the hemodynamic parameters normalized.

A careful pre-anesthetic evaluation was done. The patients were randomized on the day of surgery into two groups. Patients were kept nil per oral for 8 h before surgery. After shifting the patient to the operation theatre, two large-bore (18 G) intravenous cannulae were secured and normal saline was started at the rate of 4 ml/kg/h. Standard monitors such as pulse oximeter, noninvasive blood pressure, 3-lead electrocardiogram were connected, and pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and oxygen saturation (SpO_2) were recorded at that time.

Patients were premedicated with intravenous glycopyrrolate (0.2 mg) and fentanyl (1.5 mcg/kg). After adequate preoxygenation for 5 min, induction was achieved with i.v propofol (2 mg/kg) followed by i.v vecuronium bromide (0.1 mg/kg) to facilitate direct laryngoscopy and endotracheal intubation (high-volume/low-pressure cuffed endotracheal tube). The internal diameter of the endotracheal tube was 7 mm for women and 8 mm for men. All the patients were mechanically ventilated at a fresh gas flow of 1 L/min and anesthesia was maintained with isoflurane (minimum alveolar concentration 0.8–1.0) and vecuronium (0.01 mg/kg every 20–30 min) throughout the surgical procedure. Intraoperative analgesia was supplemented with incremental doses of fentanyl (1 mcg/kg) every hour. Intraoperatively, end-tidal CO_2 and urine output were also monitored. During mechanical ventilation, a respiratory rate of 12–16/min and tidal volume of 4–6 ml/kg were used, which were adjusted to maintain normocapnia and normoxia with oxygen saturation ≥ 98%. Ringer lactate and normal saline were used for the maintenance of fluid balance. Colloids, blood, and blood products were used as and when required.

From the start of dural closure, if at any point, the patient's systolic BP rose to > 20% of baseline value or 140 mm of Hg (whichever is higher), study drugs were administered over 20 s. Group D and Group L received intermittent boluses of i.v. Dexmedetomidine 0.1 mcg/kg and i.v labetalol 0.04 mg/kg, respectively, every 3 min until the desired hemodynamic response was achieved (reduction of Systolic BP to <140 mm of Hg or <20% of baseline value). Both the study drugs were prepared in identical

syringes and administered by a senior anesthesiologist not involved in the study.

At the end of the surgery, all anesthetic agents were discontinued, and patients were ventilated with 100% oxygen. Neuromuscular blockade was reversed with i.v neostigmine 0.05 mg/kg and i.v glycopyrrolate 0.01 mg/kg. Once the patient became conscious and responded to verbal commands, extubation was performed. After extubation, the patients were oxygenated with 100% oxygen for 5 min. The values of pulse rate, systolic BP, diastolic BP, and mean arterial pressure were obtained just before administration of the study drug, every 3 min from the time of administration of the first dose till extubation and every 3 min until 15 min after extubation. After extubation, all these patients were observed for sedation using Richmond Agitation Sedation Score (RASS).

Statistical Analysis

The data were analyzed with the help of IBM SPSS 22 for windows. Appropriate univariate and bivariate analysis for comparing two means were applied and Student's *t*-test was used to test the null hypothesis for continuous variables. Test of normality was done for all the continuous variables before applying parametric statistical methods. All means were expressed as mean ± standard deviation and proportions in percentage (%). $P < 0.05$ was considered statistically significant.

RESULTS

There were no significant differences in demographic data between the two groups with respect to the patients' age, body weight, ASA physical status, and duration of surgery. The pre-operative hemodynamic parameters such as Glasgow Coma Score, pulse rate, systolic, diastolic, and mean arterial blood pressures were comparable in both the groups [Table 1]. However, as far as pulse rate was concerned, at 9 min, 6 min, 3 min before extubation and 12 min, and 15 min after extubation, a significant difference was noted between these two groups, with labetalol achieving better control of pulse rate than dexmedetomidine [Table 2] [Figure 1].

With respect to systolic blood pressure, at 9 min, 6 min, 3 min before extubation, and for the entire 15 min after extubation, a significant difference was noted with dexmedetomidine achieving a better control than labetalol [Table 3] [Figure 2].

Significant differences in diastolic blood pressure were noted at 6 min and 3 min before extubation, with dexmedetomidine achieving a better control than labetalol [Table 4] [Figure 3].

Dexmedetomidine achieved better control of mean arterial pressure than labetalol, with significant differences at 9 min, 6 min, 3 min before extubation, and for the entire 15 min after extubation [Table 5] [Figure 4].

The median RAS score for patients in Group D was 0 (alert and calm) and for Group L was 2 (agitated) [Figures 5 and 6]. This shows that patients of Group D were more sedated than those of Group L. This can be attributed to the central mechanism of action of dexmedetomidine.

The median dose to achieve favorable hemodynamic control was only 18 mcg of dexmedetomidine and 12 mg of labetalol [Figure 7].

DISCUSSION

Emergence hypertension refers to the systemic surge of catecholamines that accompany a patient when he/she shifts from deeper to lighter plane of anesthesia. The chief instigator of this cataclysmic event is the activation of the sympathetic system, although Renin Angiotensin Aldosterone system activation and surgical manipulation of the brain have also been cited as causative factors. These are commonly ascribed causes of post-operative intracranial hematoma, which is a catastrophic complication that paves the way to increased morbidity and mortality of these patients. A pool of

Table 1: Demographic characteristics of patients

Characteristics	Group D Mean±SD	Group L Mean±SD	*P* value	Significance
Age (In years)	37.33±13.69	40.279.45	0.338	Not significant
Weight(In kg)	63.17±9.61	67.77±9.45	0.067	Not significant
Pre-Op Parameters				
GCS	13.77±1.19	4.27±1.01	0.086	Not significant
Pulse rate (per min)	79.47±10.10	82.00±8.92	0.307	Not significant
Systolic blood pressure (mmHg)	117.63±9.59	118.37±9.37	0.765	Not significant
Diastolic blood pressure (mmHg)	76.70±7.47	78.63±5.62	0.262	Not significant
Mean arterial pressure (mmHg)	90.54±7.31	91.88±5.40	0.359	Not significant
Duration of surgery (min)	204.17 ± 8.64	212.47 ± 6.39	0.781	Not significant

Table 2: Comparison of Pulse Rate

Time	Group Mean±SD	Group L Mean±SD	*P* value	Significance
9 min before	96.17±12.24	73.73±9.2	<0.0001	Significant
6 min before	97.37±11.24	72.47±5.13	<0.0001	Significant
3 min before	93.93±8.75	72.77±49	<0.0001	Significant
Extubation	90.4±7.98	91.7±7.68	0.523	Not significant
3 min	90.03±8.97	89.73±7.31	0.888	Not significant
6 min	89.83±8.27	88.07±663	0.315	Not significant
9 min	88.27±7.53	84.9±6.46	0.068	Not significant
12 min	86.87±7.64	81.83±6.05	0.006	Significant
15 min	85.77±7.24	77.9±5.1	<0.0001	Significant

Table 3: Comparison of systolic BP

Time	Group D Mean±SD	Group L Mean±SD	*P* value	Significance
9 min before	128.9±16.32	138.63±9.33	0.006	Significant
6 min before	119.13±16.49	134.3±8.45	<0.0001	Significant
3 min before	112.2±7.13	131.73±7.44	<0.0001	Significant
Extubation	126.8±6.4	128.7±6.69	0.266	Not significant
3 min	112.73±11.68	125.67±5.68	<0.0001	Significant
6 min	110.2±11.1	124.1±6.27	<0.0001	Significant
9 min	109.43±10.55	122.13±5.18	<0.0001	Significant
12 min	109.17±9.4	120.1±5.31	<0.0001	Significant
15 min	110.37±9.36	119.47±5.6	<0.0001	Significant

Table 4: Comparison of diastolic BP

Time	Group D Mean±SD	Group-L Mean±SD	*P* value	Significance
9 min before	79.17 ±11.77	83.57±5.54	0.069	Not significant
6 min before	72.83±10.81	82.87±5.79	<0.0001	Significant
3 min before	71±6.52	81.1±5.45	<0.0001	Significant
Extubation	80.13±5.32	78.97±5.18	0.39	Not significant
3 min	80.13±5.03	79.07±5.23	0.196	Not significant
6 min	79.2±5.01	78.5±3.61	0.537	Not significant
9 min	78.33±5.45	78.1±4.71	0.860	Not significant
12 min	77±4.68	75.9±3.98	0.330	Not significant
15 min	75.8±5.32	75.13±4.06	0.587	Not significant

Table 5: Comparison of mean arterial pressure

Time	Group D Mean±SD	Group L Mean±SD	*P* value	Significance
9 Min before	95.74±12.18	101.92±5051	0.014	Significant
6 Min before	88.27±11.58	100.01±5.43	<0.0001	Significant
3 Min before	84.73±4.95	97.98±4.79	<0.0001	Not significant
Extubation	95.69±4.2	95.54±4.24	0.805	Significant
3 Min	91.44±4.89	94.6±4.36	0.01	Significant
6 Min	89.53±4.92	93.7±2.96	<0.0001	Significant
9 Min	88.87±4.35	92.78±3.66	<0.0001	Significant
12 Min	87.72±3.9	90.63±3.28	0.003	Significant
15 Min	87.32±4.48	89±3.24	0.013	Significant

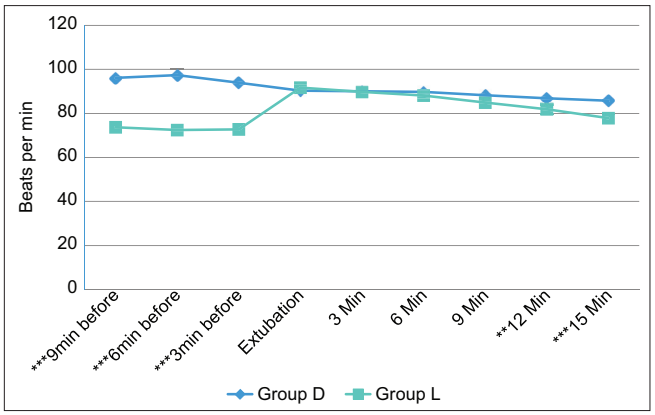

Figure 1: Comparison of Pulse Rate

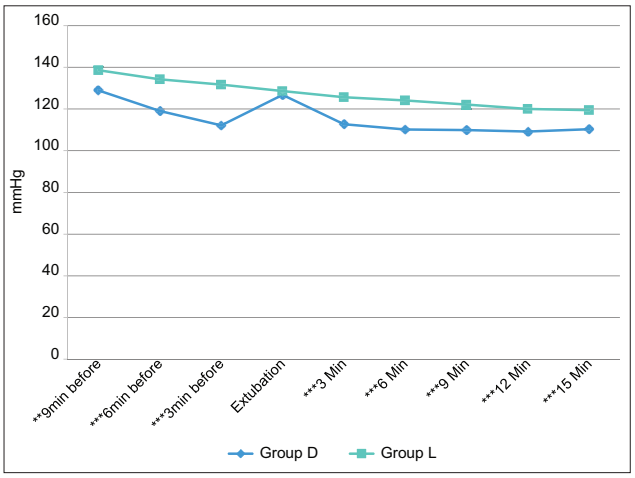

Figure 2: Comparison of systolic BP

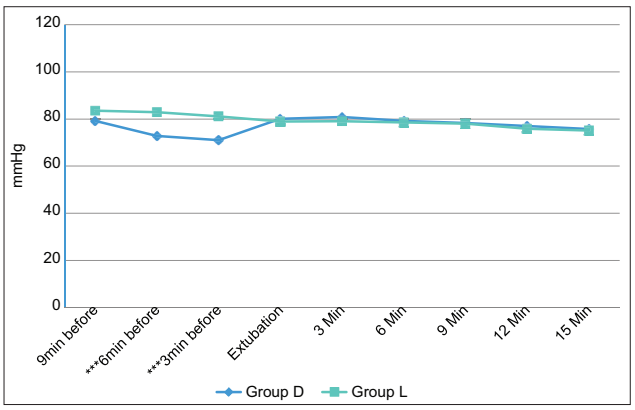

Figure 3: Comparison of diastolic BP

drugs is available for the anesthesiologists to control or prevent this notorious event, some examples of which are nicardipine, dexmedetomidine, labetalol, esmolol, lignocaine, and verapamil. Numerous alterations to anesthetic techniques, especially during emergence, have been proposed to overcome the periextubation hypertensive response. These include titrated doses of short-acting opioids, continuing volatile anesthetics, and nitrous oxide until head dressing is over, extubating patients undergoing otherwise uncomplicated surgeries in deeper planes of anesthesia, etc. Besides pharmacological intervention, methods such as Bailey's maneuver are also handled by anesthesiologists to control emergence hypertension.

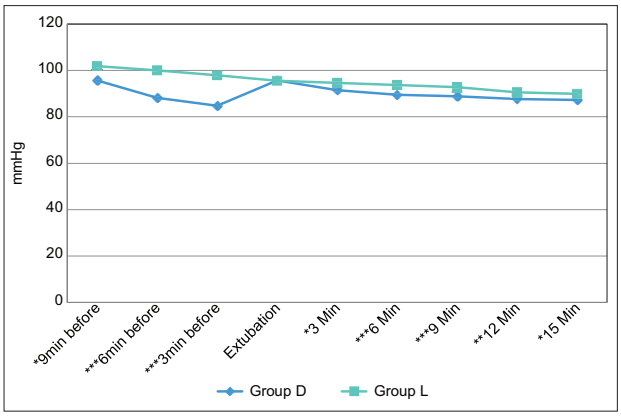

Figure 4: Comparison of mean arterial pressure

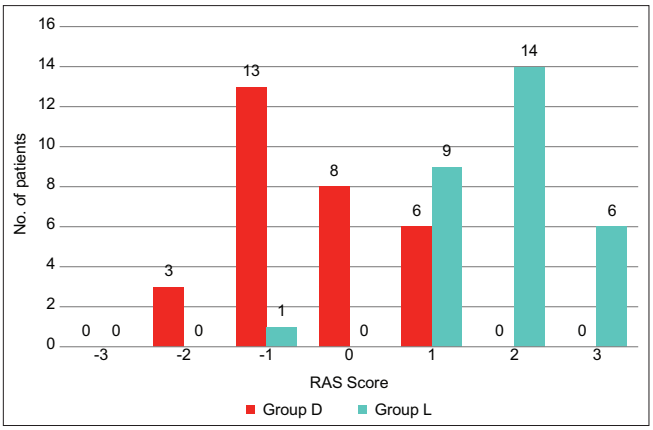

Figure 6: RAS score comparison

Richmond Agitation-Sedation Scale

	Target RASS Value	RASS Description
+4	Combative	Combative, violent, immediate danger to staff
+3	Very Agitated	Pulls or removes tube(s) or catheter(s); aggressive
+2	Agitated	Frequent non-purposeful movement, fights ventilator
+1	Restless	Anxious, apprehensive but movements are not aggressive or vigorous
0	Alert and Calm	
-1	Drowsy	Not fully alert, but has sustained awakening to voice (eye opening & contact **greater** than 10 seconds)
-2	Light Sedation	Briefly awakens to voice (eye opening & contact **less** than 10 seconds)
-3	Moderate Sedation	Movements or eye opening to voice (but **NO** eye contact)
-4	Deep Sedation	No response to voice, but has movement or eye opening to physical stimulation
-5	Unarousable	No response to voice or physical stimulation

Figure 5: Richmond agitation sedation scale

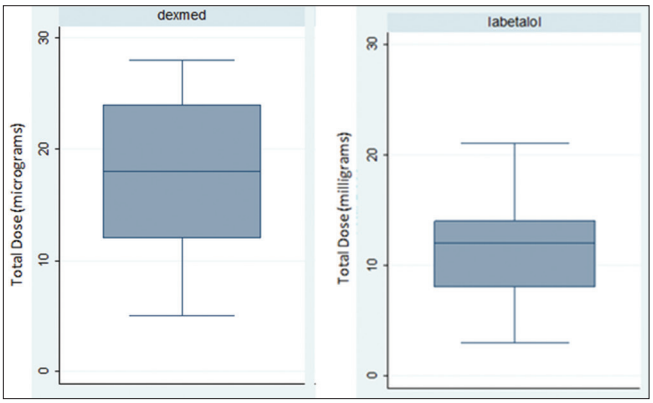

Figure 7: Total dose of the drug given

The present study was done to demonstrate the efficacy of dexmedetomidine and labetalol in controlling emergence hypertension in craniotomy. To the best of our knowledge, no comparative study between the hemodynamic effects of labetalol and dexmedetomidine (using intermittent boluses) on emergence from anesthesia during craniotomies is yet available.

The results of the present study were in accordance with those of Radwan *et al.*[9] who compared the efficacy of infusions of dexmedetomidine and labetalol in preventing emergence hypertension in patients undergoing craniotomy for supratentorial tumors. They studied 27 patients of ASA I-II who were scheduled for supratentorial craniotomy under general anesthesia, who were randomly allocated to one of three groups. At the time of dural closure, the group "dex" received dexmedetomidine infusion in a rate of 1 mcg/kg/h, group "labetalol" received labetalol infusion in a rate of 0.5 mg/kg/h, and group "control" received saline infusion at the same rate of dexmedetomidine and labetalol. Hypertensive episodes were managed with nitroglycerin at a dose starting from 1 mcg/kg/min if systolic blood pressure exceeded 25% of its preinduction value. It was concluded that dexmedetomidine infusion in a dose of 1 mcg/kg/h and labetalol infusion in a dose of 0.5 mg/kg/h started at dural closure had a significant effect in reducing the incidence and the extent of emergence hypertension without prolonging the time needed for extubation. There was more reduction of the systolic, diastolic, and mean arterial pressure in the dexmedetomidine group and of the pulse rate in the labetalol group in comparison with the other two groups. It should, however, be noted that our study used intermittent boluses of these drugs rather than continuous infusion. Furthermore, we differed from this study in such a way that the pharmacological intervention was started not as prophylaxis but as a treatment, meaning, the drugs were administered only when the emergence hypertension occurred. Another striking inference was that our median dose to achieve comparable hemodynamic control was only 18 mcg of dexmedetomidine and 12 mg of labetalol, which was notably lower than their median dose. The nil incidence of sudden hypotension and bradycardia can be attributed to the strikingly lower doses of both these drugs. Moreover, complications such as severe hypertension due to the peripheral alpha 2 receptor stimulation[10] in patients who received dexmedetomidine

were not observed as we refrained from giving the conventional initial bolus dose.

Furthermore, Ilhan *et al*.[11] studied in a double-blind prospective clinical study, the effects of fentanyl and dexmedetomidine as adjuvant agents in supratentorial craniotomies on the hemodynamic changes during perioperative period. Thirty patients undergoing intracranial tumor surgeries were divided into two groups. In one group ($n = 15$), dexmedetomidine was infused as a 1 mcg/kg bolus 10 min before induction of anesthesia and maintained with 0.4–0.5 mcg/kg/min during the operation, and fentanyl was given as a 2 mcg/kg bolus at induction. In the other group ($n = 15$), patients were given fentanyl 4 mcg/kg before induction and 0.02 mcg/kg/min as an infusion for maintenance of anesthesia. They concluded that dexmedetomidine controlled the hemodynamic changes better than fentanyl perioperatively, which supports the results of our study. The time and method of administration of dexmedetomidine, however, differed from that of our study.

Regarding the hemodynamic effects of labetalol, in a recent study conducted by Busara *et al*.,[12] which was an equivalence trial comparing labetalol and diltiazem in controlling emergence hypertension after supratentorial tumor surgery, the drugs were administered at a fixed dosage of 2.5 mg every 2–3 min to maintain systolic BP lower than 140 mmHg with a cumulative dose within 20 mg. The success rate of treatment with labetalol was equivalent to diltiazem (87.1% and 80.2%, respectively) ($P = 0.003$, 95% CI = 6.88 [−2.06–15.8]). There was no statistically significant difference on the dosage of drugs used or incidence of side effect (hypotension, bradycardia, heart block, and bronchospasm). Median doses of labetalol and diltiazem were 10 mg (2.5–20 mg) and 10 mg (2.5–20 mg), respectively. The median dose of labetalol in the abovementioned study is in concordance with that of our study, where the median dose of labetalol was 12 mg. Labetalol at this dose was not only found to be efficient in achieving the desired hemodynamic control but also was devoid of complications of beta-blockade such as bronchospasm and bradycardia.

A study by Kross *et al*.[13] compared the efficacy of the combination of enalaprilat/labetalol with that of enalaprilat/nicardipine to prevent emergence hypertension in craniotomies. Forty-two patients received enalaprilat 1.25 mg i.v at dural closure followed by either multidose nicardipine 2 mg i.v or labetalol 5 mg i.v to maintain the systolic BP below 140 mmHg. In the labetalol group, at closure of dura, the systolic, diastolic, mean arterial pressure, and pulse rate were 104 ± 9 mmHg, 75 ± 8mm Hg, 60 ± 8 mm Hg, and 84 ± 11 beats per min, respectively. The labetalol group experienced a progressive decrease in HR (14%) which is consistent with our findings. In the nicardipine group, at closure of dura, the systolic, diastolic, and mean arterial pressures were 109 ± 9 mmHg, 76 ± 8 mmHg, and 60 ± 8 mmHg, respectively. The nicardipine group experienced a higher incidence of hypotension and tachycardia than the labetalol group. This study is in agreement with the present study except that labetalol in the present study was given as a sole antihypertensive agent starting at dural closure as intermittent boluses of 0.04 mg/kg.

Although not studied, we strongly believe that the incidence of rebound hypertension and tachycardia after discontinuing these drugs can be significantly cut down with the significantly smaller doses used in our study. Cost-effectiveness will also be in our favor. From these findings, it can be stated that dexmedetomidine was better in controlling the systolic, diastolic, and mean arterial pressures and labetalol in controlling the pulse rate. Due to its central action, dexmedetomidine caused sedation in these patients, which helped in smooth extubation and did not prolong the duration of extubation.

CONCLUSION

Both dexmedetomidine and labetalol had favorable effects on hemodynamics during emergence from anesthesia. Dexmedetomidine achieved a better reduction in systolic blood pressure, diastolic blood pressure, and mean arterial pressure and labetalol in pulse rate. A higher degree of sedation was produced by dexmedetomidine, which facilitated smooth extubation.

Limitations of Our Study

A sample size of 60 cannot be projected overall patients. This study included only patients undergoing craniotomy in the supine position. It did not include patients who were positioned prone. Furthermore, patients undergoing neuroendoscopic procedures for tumor removal were not included. Hemodynamic monitoring did not include invasive blood pressure monitoring and cardiac output monitoring. Surgeries lasting more than 4 h were not included in this study.

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How to cite this article: Naveen RS, Narang N, Sethi A. Dexmedetomidine versus Labetalol in Controlling Emergence Hypertension in Craniotomy. Int J Sci Stud 2020;8(8):74-80.

Source of Support: Nil, **Conflicts of Interest:** None declared.