

Comparison of Intravenous Dexmedetomidine versus Labetalol for Their Effectiveness in Suppression of Haemodynamic Response during Extubation

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ABSTRACT

Background: Tracheal extubation causes autonomic nervous system disturbances which causes tachycardia, hypertension which are harmful in susceptible patients. so we conducted a study between iv Dexmedetomidine and iv Labetalol to assess their effectiveness in decreasing haemodynamic disturbances during extubation.

Materials and Methods: we included 100 participants of age of 18-55 yrs with ASA grading I & II and divided them into 2 groups. Group D was given injection Dexmedetomidine 0.6mcg/kg iv and Group L was given injection Labetalol 0.25mg/kg body weight. We recorded Heart rate, systolic and diastolic blood pressure at baseline, 2,5,8 minutes post drug injection, at extubation and 1,3,5,8,10 and 15 minutes after extubation.

Results: Group D had better decreased heart rate, systolic and diastolic blood pressure at the time of extubation, and also 15 minutes post extubation in comparison to Group L.

Conclusion: Injection Dexmedetomidine 0.6µg/kg has showed a better attenuating effect on sympathoadrenal system during extubation than injection Labetalol 0.25mg/kg

Keywords: Dexmedetomidine, labetalol, extubation, hemodynamics

INTRODUCTION

Endotracheal intubation and extubation are associated with various cardiovascular and airway responses that lead to hemodynamic instability due to sympathetic discharge caused by epipharyngeal and laryngeal stimulation. [1,2]

All these transitory responses might lead to unpredictable and hazardous effects in patient with pre-existing co-morbidities. [3]

Different strategies have been employed to control emergence hypertension such as extubation in a deep plane of anesthesia, drugs as lidocaine, labetalol, esmolol, and intravenous opiates such as morphine, fentanyl. [4]

Preemptive therapy is another such effort used now a days to avoid occurrence of such effects Dexmedetomidine is a selective α_2 agonist that provides sedation, hypnosis, analgesia and sympatholysis. It maintains intraoperative haemodynamics by decreasing levels of catecholamines during surgery [5]

Labetalol is a unique antihypertensive adrenergic antagonist having an effect on both selective α_1 and nonselective β_1 and β_2 , with rapid onset of action and reaches its peak effect at 5-15 min after i.v. injection [6,7]

This study was planned primarily to compare iv Dexmedetomidine 0.6µg/kg and Labetalol 0.25mg/kg in suppression of sympathoadrenal response to evaluate haemodynamic responses at extubation.

MATERIAL AND METHODS

On approval of institutional ethics committee and written informed consent from each patient This prospective, randomized, controlled study was conducted. 100 patients included in the study were of age 18-50 years of either sex, belonging to ASA grade I-II posted for elective surgery of duration less than two hours under general anaesthesia with endotracheal intubation. Patients excluded were those with known hypersensitivity to study drugs, pregnant females, patients with cardiovascular, respiratory, hepatic or renal diseases, patients on β blockers, patients with anticipated difficult intubation, heart blocks, bradycardia (heart rate <60bpm) and those patients in whom intubation was attempted for more than 30 seconds.

Patients were assessed for pre anaesthesia checkup, all required investigations were done and were instructed to maintain nil by mouth for 8 hours.

The patients were randomly allocated with the help of computer generated coded envelopes based on study drugs into two groups of 50 each as per protocol given below:

Group D received injection Dexmedetomidine 0.6 µ g/kg body weight diluted upto 10ml with normal saline intravenously over 10 minutes using a syringe pump before extubation.

Group L - received injection Labetalol 0.25mg/kg body weight diluted upto 10ml with normal saline given intravenously over 10 minutes using a syringe pump before extubation.

In the pre-operative room, under all aseptic and antiseptic precautions a peripheral intravenous line was secured by 18 G canula. The patients were then

preloaded with 500 ml ringer lactate solution.

On shifting to the operating room, all patients were monitored for baseline vital parameters like non-invasive blood pressure, heart rate (HR), pulse oximeter (SpO₂) and electrocardiograph (ECG). The patients were premedicated with glycopyrrolate 5mcg/kg, ondansetron 0.1mg/kg and fentanyl 1mcg/kg intravenously and were preoxygenated with 100% oxygen.

Anaesthesia was induced with propofol 2mg/kg followed by suxamethonium 1.5mg/kg. Ventilation of lungs was manually assisted till muscles were relaxed satisfactorily. Then laryngoscopy was carried out and patient's airway was secured with an endotracheal tube of appropriate size and was fixed after checking bilateral equal air entry.

Anaesthesia was maintained with oxygen, nitrous oxide, sevoflurane with intermittent dose of injection atracurium.

Group D received injection Dexmedetomidine 0.6 µ g/kg diluted upto 10ml with normal saline intravenously over 10 minutes using a syringe pump before extubation and Group L - received injection Labetalol 0.25mg/kg diluted upto 10ml with normal saline given intravenously over 10 minutes before extubation.

At the end of the procedure, neuromuscular blockade was reversed with Inj neostigmine 0.05mg/kg body weight and Inj glycopyrrolate 0.01mg/kg body weight.

Haemodynamic parameters such as heart rate, systolic and diastolic blood pressure were recorded

- at baseline
- 2, 5, 8 minutes after drug infusion
- at the time of extubation
- at 1, 3, 5, 8, 10 and 15 minutes postextubation.

STATISTICAL METHOD

Demographic data of the patients were expressed as mean ± standard deviation. The statistical data were analysed by mean, standard deviation and p values

were calculated by using SPSS trial software version 26.

RESULT

Demographic variables among 2 groups were comparable with respect to age, sex, height, weight and duration of surgery.

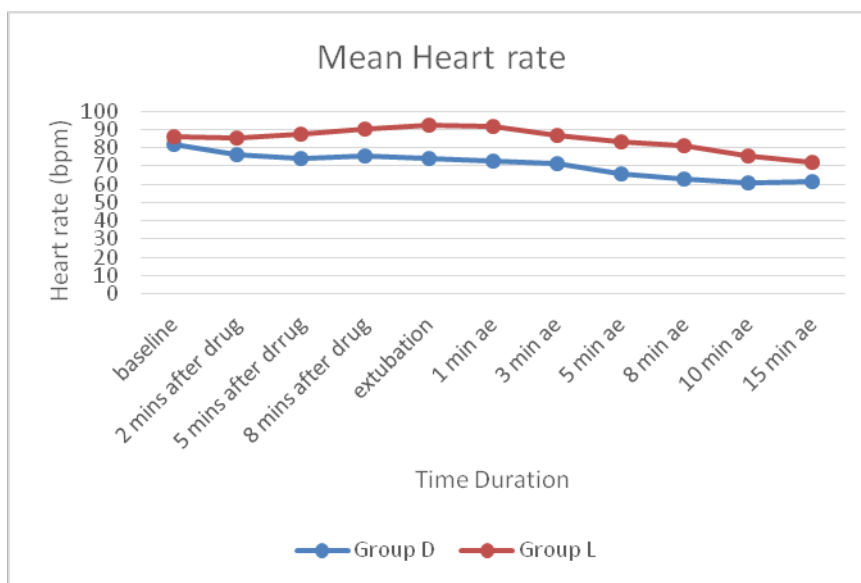


Figure 1: Changes in mean heart rate between Dexmedetomidine and Labetalol group

The baseline mean heart rate in patients of both groups was comparable and statistically insignificant

In both groups, there was decrease in mean heart rate after drug infusion and at 1, 3, 5, 8, 10 and 15 mins post extubation (Figure 1)

However, the decrease in mean heart rate was more significant in group D at 2, 5, 8 mins after drug administration, at

extubation and 1, 3, 5, 8, 10 15 mins post extubation as compared to group L

At extubation there was decrease in mean heart rate by 8 bpm from baseline in group D and increase by 6 bpm in group L from baseline. The changes were statistically significant p value <0.0001 as compared to group L (figure 1)

Mean heart rate was below baseline values even at 15th minutes post extubation in both groups.

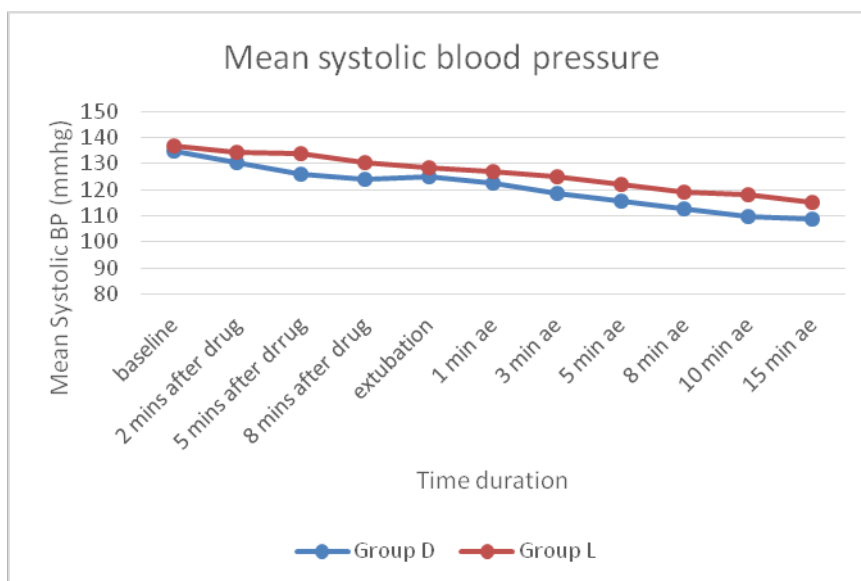


Figure 2: Changes in mean systolic blood pressure between Dexmedetomidine and Labetalol group

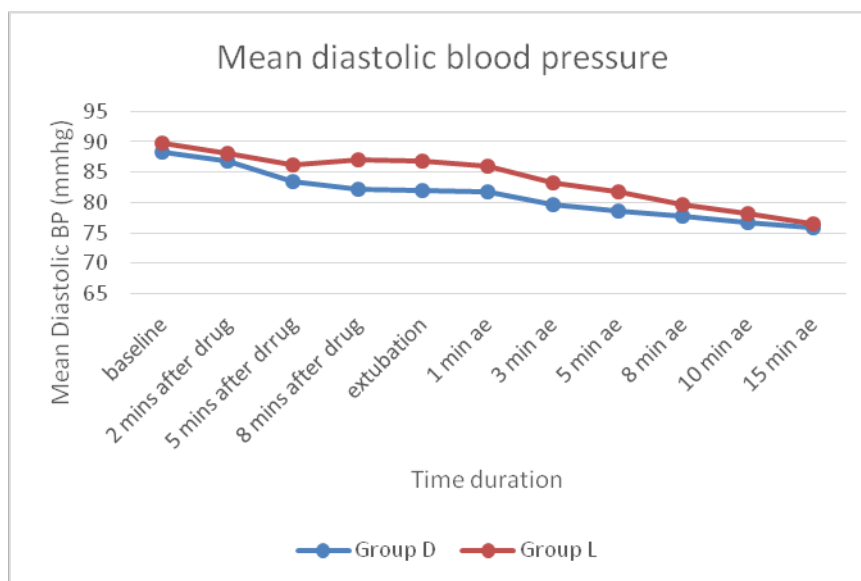


Figure 3: Changes in mean diastolic blood pressure between Dexmedetomidine and Labetalol group

The baseline mean systolic and diastolic blood pressure in patients of both groups was comparable and statistically insignificant

There was decrease in mean systolic and diastolic blood pressure in both groups after administration of drugs. Group D showed a greater decrease in mean systolic and diastolic blood pressure at 2, 5, 8 mins after drug administration, at extubation and until 15 min post extubation as compared to baseline parameters. (figure 2,3)

At extubation in group D the decrease in mean systolic and diastolic blood pressure was 9 and 6 mm hg from baseline respectively with p value <0.0001 statistically when Compared to group L where the decrease in mean systolic and diastolic blood pressure was 8 and 3 mmhg from baseline respectively.

The mean systolic as well as diastolic blood pressure remained below the baseline parameters until 15 mins post extubation also. (figure 2,3)

DISCUSSION

Various theories have explained sudden increase in HR and BP during extubation such as a rise in catecholamine, airway irritation owing to suction, intense pain from surgical wounds and emergence. [8]

This evokes noxious stress responses and causes disturbance in hemodynamic parameters like tachycardia, hypertension, change in heart rhythm and evokes coughing, bronchospasm, raised intraocular, intracranial pressure and thus hazardous consequences in vulnerable patients [9]

Dexmedetomidine, an alpha-2 adrenergic agonist possesses properties of sympatholysis, titratable sedation without respiratory depression, analgesia, benefit of reduced dosage of opioids. [10]

It is a small molecule possessing an imidazole ring with distribution half-life of 6-8 minutes on intravenous administration [11,12] it possesses eight times more affinity for alpha-2 adrenoceptors as compared to clonidine, Hence a preferred agent as a full alpha-2 adrenoceptor agonist. [13] Activation of these presynaptic alpha 2 receptors inhibits release of norepinephrine and terminates transmission of noxious stimuli. Thus, it attenuates sympathoadrenal response by changes in haemodynamic parameters -decrease in heart rate and blood pressure by inhibiting the sympathetic activity through activation of postsynaptic alpha-2 receptors [14]

Labetalol, an antihypertensive drug used widely as an alpha-1, nonselective beta-1 and beta-2 adrenergic antagonist. R isomer of labetalol has potent vasodilatory

effect with intrinsic sympathomimetic effect on beta adrenergic receptors. [15]

On intravenous administration it reaches peak effect by 5-15minutes and attenuates sympathoadrenal response by decreasing heart rate by blocking beta adrenergic receptor and blood pressure by blocking alpha 1 adrenergic receptors. [16,17]

As very few researchers have conducted study comparing role of dexmedetomidine and labetalol for suppression of sympathoadrenal stress response to extubation we conducted a study their effect on hemodynamic parameters during extubation.

Rapid Intravenous administration of dexmedetomidine leads to transient increase in blood pressure and decrease in heart rate due to stimulation of peripheral alpha-2B adrenoceptor.

To overcome this hurdle, we administered dexmedetomidine 0.6µg/kg diluted upto 10ml with normal saline slowly using syringe pump.

Considering time of peak effect of labetalol, we administered intravenous labetalol 0.25mg/kg over 10 mins diluted in 10 ml normal saline with syringe pump.

Our study results on analysis show at extubation Dexmedetomidine group showed decrease in mean heartrate by 8 bpm, mean systolic by 9 mmhg and diastolic blood pressure by 6 mmhg In Group labetalol at extubation, the mean heart rate was increased by 6 bpm, and mean systolic and diastolic blood pressure was decreased by 8 mmhg and 3 mmhg respectively. These changes were statistically highly significant as compared to baseline (p value <0.0001)

In Both groups the heart rate, systolic blood pressure, diastolic blood pressure remained below the baseline within permissible limits 15 mins post extubation.

But the overall hemodynamic stability of dexmedetomidine was better than labetalol group at various time intervals and hence Dexmedetomidine maintains stable hemodynamic parameters as compared to Labetalol.

Sindhu S, V Y Srinivas et al conducted a study entitled “to study the effect of iv dexmedetomidine versus iv labetalol for the suppression of sympatho adrenal response to extubation” where they included 60 patients and concluded that administration of injection dexmedetomidine 0.6µg/kg resulted in more hemodynamic stable parameters as compared to labetalol administered 0.25mg/kg iv during extubation. At extubation in group D the decrease in heart rate, systolic blood pressure, diastolic blood pressure in their study was 3bpm, 13mm hg and 10 mmhg respectively. In labetalol group the increase in heart rate was 9 bpm, decrease in systolic and diastolic blood pressure was 5mm hg and 3 mmhg respectively [18]

The above study results are quite similar to ours and thus strengthen our findings.

In our study even though mean heart rate, systolic and diastolic blood pressure remained below the baseline value even at 15minutes postextubation in both groups.

We conclude that Dexmedetomidine was better in maintaining stable haemodynamic parameters compared to Labetalol at various time intervals that were monitored.

Kewalramani et al carried out a study entitled “Comparison of labetalol versus dexmedetomidine to assess the haemodynamic responses to laryngoscopy and intubation during induction of general anaesthesia “and compared Dexmedetomidine 0.5µg/kg with labetalol 0.25mg/kg iv for suppressing hemodynamic responses at intubation and extubation stated that dexmedetomidine has better attenuation than labetalol. At extubation dexmedetomidine heart rate was increased 2 bpm whereas in labetalol increase was 6bpm. [19]

The research conducted by D Single et al included borderline hypertensive patients undergoing laparoscopic cholecystectomy stated that labetalol group showed higher systolic blood pressure

(128.0±13.866 vs 123.2±10.672) and diastolic blood pressure (79.2±14.153 vs 73.1±9.683) compared to Dexmedetomidine group. This study results strengthen our result analysis. [20]

Kotak N et al compared Dexmedetomidine with esmolol and proved that dexmedetomidine showed more stable hemodynamic parameters at extubation. [21]

Many other studies have also been carried out comparing Dexmedetomidine with other agents and have similar conclusion.

CONCLUSION

In a nutshell, intravenous Dexmedetomidine at dose of 0.6microgram/kg is a better agent than labetalol 0.25mg/kg in supresses sympathetic response to tracheal extubation.

Acknowledgement: None

Conflict of Interest: None

Source of Funding: None

Ethical Approval: Approved

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How to cite this article: Shah PM, Kheskani D. Comparison of intravenous dexmedetomidine versus labetalol for their effectiveness in suppression of haemodynamic response during extubation. *International Journal of Research and Review.* 2021; 8(11): 286-292. DOI: <https://doi.org/10.52403/ijrr.20211137>
