ORIGINAL RESEARCH PAPER VOLUME-6 | ISSUE-8 | AUGUST - 2017 | ISSN No 2277 - 8179 | IF : 4.176 | IC Value : 78.46

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

EVALUATION OF HAEMODYNAMIC CHANGES AFTER INTRAVENOUS DEXMEDETOMIDINE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION.

Anesthesiology

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ABSTRACT

Background-our aim was to study the effectiveness of intravenous dexmedetomidine (1 ug/kg) in attenuation of haemodynamic response to laryngoscopy and endotracheal intubation.

Material and Method – study was conducted on 100 patients divided randomly into 2 groups{ each 50} comparable in demographic parameters. Group D Patients were given Inj Dexmedetomidine 1ug/kg IV diluted in 100 ml normal saline and administered over 20 min before induction & Group C Patients were given intravenous 100 ml normal saline administered over 20 min before induction.

RESULT -there was significant reduction in heart rate, systolic blood pressure, diastolic blood pressure, mean blood pressure in group D as compared to group C at 0 minute, 5 minute & 10 minute after intubation.

Conclusion :

Dexmedetomidine at a dose of 1µg/kg body weight diluted in 100 ml Normal saline given over 20 minutes before induction significantly attenuates the haemodynamic responses to laryngoscopy and endotracheal intubation.

KEYWORDS:

dexmedetomidine;heart rate; MAP;intubation response.

Introduction -Laryngoscopy and endotracheal intubation is an integral and essential component of general anesthesia. It is a procedure that results in sympathetic stimulation resulting in hemodynamic changes like hypertension and tachycardia due to increase in catecholamine activity.

Although these hemodynamic changes may be well tolerated by healthy individuals, they may be detrimental in hypertensive patients leading to life threatening complications such as myocardial ischemia, cardiac arrhythmias and even cerebrovascular hemorrhage.

We studied the haemodynamic effects of intravenous dexmedetomidine in adult patients posted for various surgeries under general anaesthesia with single intravenous bolus dose of 1µg/kg body weight dexmedetomidine when given prior to laryngoscopy and endotracheal intubation.

Material and method-Prospective randomized double blind clinical comparative study carried out in our institute after ethical committee clearance and obtaining written informed valid consent from 100 patients.

Patients aged 20-60 yrs of both sexes , ASA grade I & II , MPC Grade I & II posted for elective surgical procedure requiring general anaesthesia included in study.

Patients of ASA Grade III & IV , on beta blocker drugs ,with cardiovascular disorders , having COPD , recent history of URTI, Patients with anticipated difficult intubation (MPC Grade III & IV) & Pregnant patients were excluded from study.
Patients were randomly assigned

into two groups i.e Group D (50) and Group C (50). Preloading was done with Ringer Lactate 10ml/kg.

All patients received premedication with tab.Alprazolam 0.5mg and tab. Ranitidine 150 mg at bedtime prior to surgery.

Group D - Patients were given Inj Dexmedetomidine 1ug/kg IV diluted in 100 ml normal saline and administered over 20 min before induction.

Group C - Patients were given intravenous 100 ml normal saline administered over 20 min before induction.
Pre oxygenation was done for 5 min.

Patient induced with inj.Thiopentone Sodium 5 mg/ kg i.v until loss of eye lash reflex. Intubation facilitated with inj. vecuronium 0.1 mg/kg i.v.

All intubations were done by expert anesthesiologists within 15 sec with

proper size of endotracheal tube.
Anaesthesia was maintained with oxygen 50% ,nitrous oxide 50%, and isoflurane.

EVALUATION OF PARAMETERS

Haemodynamic responses were compared in both groups by measuring HR, SBP, DBP, MAP, SPO2 ECG and lead V5.

Sequence	recording
Basal reading when the patient is shifted to OT	T0
5 mins after securing iv line and applying monitors.	T1
During drug infusion [0 min]	T2
During drug infusion [5 min]	T3
During drug infusion [10 min]	T4
During drug infusion [15 min]	T5
During drug infusion [20 min]	T6
Induction	T7
After Intubation [0 min]	T8
After Intubation [5 min]	T9
After Intubation [10 min]	T10

RESULT-

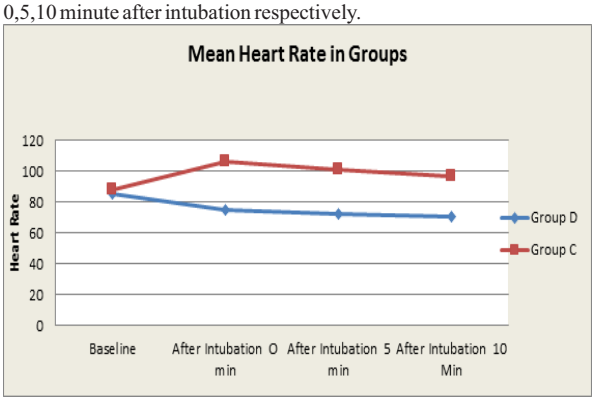
Both the groups were comparable and there was no statistically significant difference with regards to mean age, weight, gender distribution.

The basal mean HR in the present STUDY group and CONTROL Group were 85.30 bpm and 87.44 bpm respectively. There was increase in heart rate in group

C, 105.92±10.67, 100.94±7.58 , 96.56±8.12 at 0 min. , 5 min., & 10 min. after intubation respectively, Whereas decrease in heart rate in group D was 74.92±13.11, 72.18±10.14, 70.06±8.27 at 0 min , 5min & 10 min after intubation respectively.

In group D there was significant decrease in heart rate by 11, 13,15 beats per

minute at at 0 min , 5min & 10 min respectively after intubation. , where as in control group HR increased by 18, 13, 9 beats per minute at 0,5,10 minute after intubation respectively.

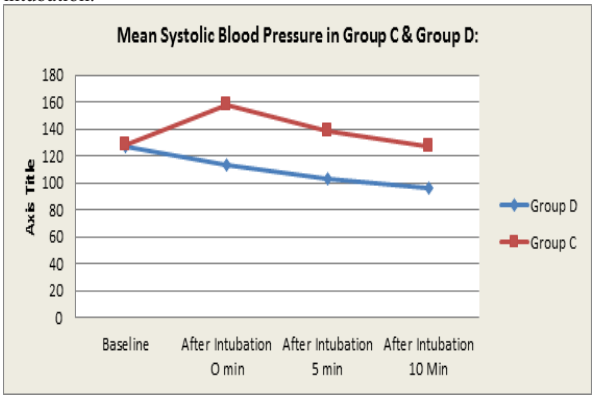


In the present study, the basal mean SBP in STUDY Group and CONTROL

group were 128.10 mmHg and 127.20 mmHg respectively. 0 min after intubation in STUDY group there was 14 mmHg decreases in SBP as compared to basal value. Where as in CONTROL group there was 29 mmHg increases in SBP as compare to basal value. this was statistically significant.

In the STUDY group, fall in SBP when compared to basal level was of 14 mmHg, 24 mmHg and 31 mmHg at 0, 5, and 10 min after intubation respectively.

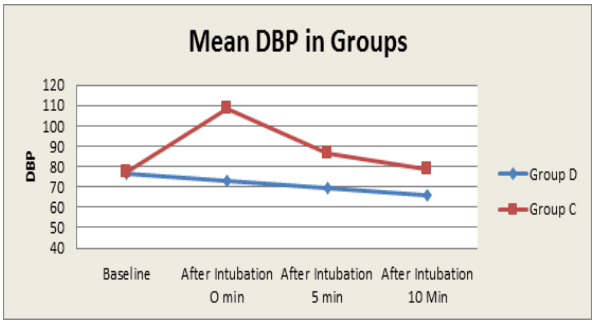
In CONTROL group, there was increase in SBP at 0 and 5 min by 29 and 11 mmHg respectively and fall in SBP by 1 mmHg at 10 min after intubation.



The basal mean DBP in the present study in STUDY group and CONTROL group were 76.68 mmHg and 77.04 mmHg respectively.

In STUDY group there was decrease in DBP by 4 mmHg, 7 mmHg and 11 mmHg At 0, 5 and 10 min after intubation respectively as compared to basal value.

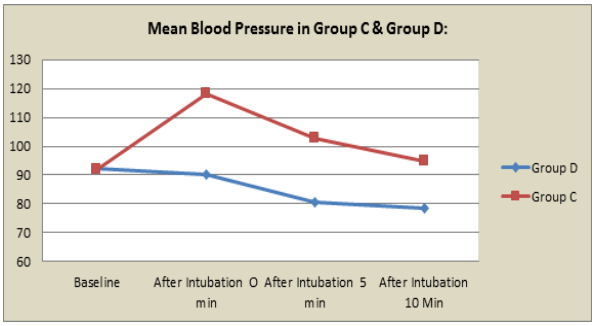
In control group there was increase in DBP by 31 mmHg, 9 mmHg, 1 mmHg at 0 ,5 ,10 min after intubation respectively as compared to basal value .



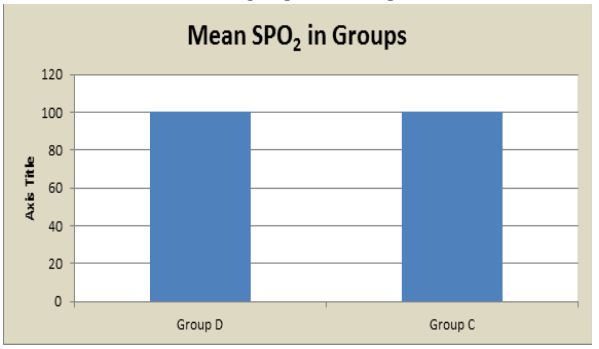
In the present study basal MAP in STUDY group and CONTROL group were 92.28 mmHg and 91.94 mmHg respectively.

In the STUDY group There was decrease in MAP by 2 mmhg,12mmhg and14 mmhg at 0 5 and 10 min after intubation respectively as compared to basal values.

In CONTROL group there was Increase in MAP by 17mmhg , 11 mmhg and 3 mmhg at 0, 5 and 10 min. after intubation respectively as compared to basal values.



There was no stastistically significant difference with regards to mean SPO2 values , hence both the groups were comparable.



ECG :
No significant ECG changes were observed in both the groups.

DISCUSSION-. Laryngoscopy and tracheal intubation provoke transient but marked sympathoadrenal response leading to hypertension and tachycardia.

These responses are transitory, variable and may not be significant in otherwise normal individuals, But in patients with cardiovascular compromises like hypertension, Ischemic heart disease, Cerebrovascular disease and patients with intracranial aneurysms these transient changes in haemodynamics can result in potentially harmful effects like left ventricular failure, pulmonary oedema, myocardial ischemia, ventricular dysrhythmias and cerebral haemorrhage. This is by far the most important indication for attenuation of haemodynamic response to laryngoscopy and tracheal intubation.

There was significant fall in mean HR after intubation compared to baseline value and that was statistically highly significant.
This effect is caused by the inhibition
of the central sympathetic outflow overriding the direct stimulant effect.

Another possible explanation for the subsequent heart rate decrease is the stimulation of presynaptic alpha-2 adrenoceptors leading to a decrease in norepinephrine release.

Dexmedetomidine provides dose dependant increase in anxiolysis and Sedation which leads to reduction in blood levels of catecholamines decreasing HR in the study group.

In the present study fall in SBP,DBP,MAP was observed as compmared to preinduction level and this fall continued till 10 min after intubation. This observation was found statistically significant.
Many authors have observed a transient increase of the blood pressure and a reflex decrease in heart rate, especially in young healthy patients

following dexmedetomidine bolus doses. The initial reaction can be explained by the peripheral alpha 2B adrenoceptors stimulation of vascular smooth muscles and can be attenuated by a slow infusion. We have not observed this transient increase in blood pressure probably because of slow infusion over 20 min and adequate preloading.

Even at slower infusion rates many have observed increase in mean arterial pressure over the first 10 minutes which was shown to be in the range of 7% .

The initial response lasts for 5-10 minutes and is followed by a decrease in blood pressure of approximately 10%-20% below baseline values; these effects are caused by the inhibition of the central sympathetic outflow overriding the direct stimulant effect. Another possible explanation for the subsequent blood pressure decrease is the stimulation of presynaptic alpha-2 adrenoceptors, leading to a decrease in norepinephrine release.

Control group has not received any analgesic as is the standard practice, before intubation which is noxious stimulus for hemodynamic response because our aim was to study effect of dexmedetomidine without any drug synergy or ineraction ,any distractions in result due to drugs like analgesics , which affects haemodynamic parameters were avoided. We induced patients after taking them in operation theatre without delay and nitrous oxide as an analgesics was started immediately after intubation

Conclusion :

Dexmedetomidine at a dose of 1µg/kg body weight diluted in 100 ml Normal saline given over 20 minutes before induction significantly attenuates the haemodynamic responses to laryngoscopy and endotracheal intubation.

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