

Comparison of heart rate and mean arterial pressure between single dose intravenous dexmedetomidine and intravenous clonidine in patient undergoing elective surgery under general anesthesia

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Abstract

The sympathetic system in response to stress acts to increase heart rate, blood pressure, cardiac output, dilates bronchial tree and shunts blood away from skin and viscera to muscles. A powerful noxious stimulus like laryngoscopy and tracheal intubation induces hypothalamic activity and results in an increased outflow in the sympathetic tracts. Consequently, norepinephrine is released by post ganglionic sympathetic fibers and increased secretion from adrenal medulla. A preanesthetic evaluation of history of surgical and medical illness, drug allergies previous anaesthetic exposure and Baseline investigations of blood, ECG, radiograph of chest and airway examination was done. Patient was kept nil by mouth for at least 8hrs prior to surgery. All patients were premedicated with injection Pantoprazole 40mg (IV) one hour prior to surgery. Preoperative vital parameters like baseline pulse, blood pressure were noted. Mean arterial pressure (MAP) significantly lower in Group D patient at 4 minutes, 6 minutes, 8 minutes, 10 minutes, 15 minutes, 20 minutes after intubation compared to Group C. Heart rate was on lower side in group D compared to Group C but the difference was significant statistically after 6 minutes of intubation till 25 minutes post intubation.

Keywords: Heart rate, mean arterial pressure, intravenous dexmedetomidine and intravenous clonidine

Introduction

Autonomic nervous system does the biological house-keeping of the internal environment of the body. Sympathetico-adrenal system regulates the body response to combat any stress. Neurotransmitters of sympathoadrenal system are noradrenaline and adrenaline. Normal basal secretion by adrenal medulla of adrenaline is 0.2µg/kg/min and that of noradrenaline is 0.05µg/kg/min which is adequate to maintain the body physiology. In situations of stress the sympatho-adrenal system is stimulated by hypothalamus resulting in an increase in the catecholamine secretion. This reaction is closely con-elated with endocrine system in

combating stress ^[1, 2].

The sympathetic system in response to stress acts to increase heart rate, blood pressure, cardiac output, dilates bronchial tree and shunts blood away from skin and viscera to muscles. A powerful noxious stimulus like laryngoscopy and tracheal intubation induces hypothalamic activity and results in an increased outflow in the sympathetic tracts. Consequently norepinephrine is released by post ganglionic sympathetic fibers and increased secretion from adrenal medulla ^[3].

Attempts have been made to assess sympathetic activity directly by measurement of plasma catecholamine concentrations with the use of radio enzymatic assays and high pressure liquid chromatography, by various worker ^[4].

It was concluded by study of changes of plasma catecholamine concentration during laryngoscopy and endotracheal intubation by Russell WJ and Moltis RG ^[5] that a positive correlation existed between arterial pressure and plasma noradrenaline concentration. The magnitude of increase in blood pressure paralleled the increase in plasma noradrenaline concentration. Plasma adrenaline did not change significantly.

This was further confirmed by Derbyshire ^[6] and Smith who showed that the plasma noradrenaline concentration increased by 34% in samples obtained from central venous line and by 74% in samples obtained from radial artery. This can be explained by uptake of noradrenaline in lungs.

The adrenergic responses were maximum by one minute and had diminished by 5 minutes. This hemodynamic response due to activation of sympathico-adrenal system, increases heart rate, blood pressure. These serve as indirect indices to measure the response. Thus, heart rate and blood pressure have been used as indirect indices to measure levels of sympathetic activity clinically. In addition to activation of the autonomic nervous system, endotracheal intubation also stimulates central nervous system activity as evidenced by increase in electroencephalographic activity and basal metabolic rate. Inpatients with compromised intracranial compliance, the increase in CBF may result in elevated intra cranial pressure which in turn may result in herniation of brain contents and severe neurologic compromise.

Methodology

Source of data

Data from the patients admitted and undergoing major elective surgeries at Medical College and Hospital.

Study design: Prospective Double blind randomized comparative study.

Place of study: Patients admitted and undergoing major elective surgery at Dr. B.R. Ambedkar medical college and hospital, Bangalore.

Sample size: Hospital based study of 60 patients who fulfilled the inclusion criteria.

Inclusion criteria

- Patient aged between 18 to 60 years.
- ASA Physical status 1 and 2.
- Both sexes.
- Undergoing major elective surgery under general anaesthesia.

Exclusion criteria

- Patients with cardiac disease.
- Severe pulmonary disease.

- Psychiatric illness.
- Severe renal derangement.
- Uncontrolled hypertension.
- Diabetes mellitus.
- Pregnancy.
- Liver failure.

After obtaining approval from the institutional ethical committee patients fulfilling the inclusion/exclusion criteria were included in the study after obtaining informed consent.

A preanesthetic evaluation of history of surgical and medical illness, drug allergies previous anaesthetic exposure and Baseline investigations of blood, ECG, radiograph of chest and airway examination was done. Patient was kept nil by mouth for at least 8hrs prior to surgery. All patients were premedicated with injection Pantoprazole 40mg (IV) one hour prior to surgery. Preoperative vital parameters like baseline pulse, blood pressure were noted.

Study was undertaken in patients planned for elective surgeries under General Anaesthesia. Patient were selected between 18 to 60yrs of Age with ASA 1 and ASA 2 grades. They were divided into 2 groups of 30 each and allocated randomly. All patients were explained about the procedure and its complication and informed consent obtained.

Results

Table 1: Mean Arterial Blood Pressure Comparison in Two Groups of Patients Studied

Map (mm Hg)	Group C	Group D	Total	P Value
Base line BP	93.31±4.85	91.12±4.57	92.51±4.74	0.198
before intubation	92.38±6.04	89.73±5.98	91.05±6.11	0.094
2 min	88.73±5.62	89.88±6.41	89.31±6.01	0.464
4 min	94.29±5.19	86.79±5.17	90.34±6.38	<0.001"
6 min	93.34±4.50	84.81±4.43	89.07±6.17	<0.001 **
8 min	93.20±4.90	84.47±3.97	88.83±6.24	<0.001 **
10 min	97.69±6.21	90.65±5.49	94.17±6.81	<0.001"
15 min	94.35±6.20	85.88±4.19	90.11±6.95	<0.001 **
20 min	94.18±8.83	83.34±7.86	88.86±9.87	<0.001 **
25 min	84.38±5.75	81.97±5.01	83.17±5.48	0.090+
30 min	87.12±7.02	87.04±7.48	87.08±7.19	0.965

Mean arterial pressure (MAP) significantly lower in Group D patient at 4 minutes, 6 minutes, 8 minutes, 10 minutes, 15 minutes, 20 minutes after intubation compared to Group C.

Table 2: Heart Rate: Comparison in Two Groups of Patients Studied

Herat rate	Group C	Group D	Total	P Value
Base line HR	81.16±7.51	77.40±7.96	79.28±7.91	0.100
before intubation	77.40±6.64	73.86±6.30	75.63±6.66	0.039*
2min	73.80±6.26	74.86±5.53	74.33±5.88	0.487
4min	75.90±6.70	73.26±6.67	74.58±6.76	0.133
6min	76.13±5.60	69.40±5.38	72.76±6.42	<0.001**
8min	77.63±6.22	68.66±4.28	73.15±6.96	<0.001**
10min	83.06±6.16	74.46±4.89	78.76±7.01	<0.001**
15min	82.73±8.16	71.26±4.57	77.00±8.74	<0.001**
20min	82.60±8.28	74.93±8.23	78.76±9.05	<0.001**
25min	77.46±7.07	70.36±6.46	73.19±7.61	<0.001**
30min	74.80±8.69	70.96±8.68	72.88±8.83	0.093+

Heart rate was on lower side in group D compared to Group C but the difference was significant statistically after 6 minutes of intubation till 25 minutes post intubation.

Discussion

In our study, we found that dexmedetomidine group was superior to clonidine group in attenuation of cardiovascular and catecholamine response with MAP decreased significantly with dexmedetomidine group from 89.73 ± 5.98 as baseline to 84.4 ± 3.97 at 8 minute compared to clonidine group which was 92.38 ± 6.04 to 93.20 ± 4.90 and heart rate decreased significantly in dexmedetomidine group from 73.86 ± 6.30 at baseline to 68.66 ± 4.28 at 8 min compared to clonidine group which was 77.40 ± 6.64 at baseline to 77.63 ± 6.22 at 8 min.

In a study [7] in the year 2017 did a comparative study between dexmedetomidine, clonidine and Magnesium Sulphate in attenuating hemodynamic response to laryngoscopy and intubation using 1 TNCG/kg IV dexmedetomidine, 1 mcg/kg of clonidine and 30 mg/kg of Magnesium Sulphate and found that dexmedetomidine is far more effective in blunting the haemodynamic response starting from just after intubation to 2 min, 3 min, 4 min, 5min, 10 min after intubation. At 2 min after intubation in dexmedetomidine group mean pulse rate and DBP remain similar but SBP and MBP decreased as compared to Baseline but in clonidine and Magnesium sulphate group there was an increase in DBP, SBP, MAP and pulse rate.

In our study, we found that Mean HR and MAP remain similar at 2 min after intubation but MAP, SBP and DBP decreased significantly at 4th min and Mean HR at 6th min as compared to the clonidine group. We found that MAP from 92.38 ± 6.04 at intubation time to 94.29 ± 5.19 at 4 minutes in clonidine group as compared to 89.73 ± 5.98 to 86.79 ± 5.17 in dexmedetomidine group at 4 mins.

Arindam Sarkar [8] *et al.* in the year 2014 did a comparative study on blunting presser response during laryngoscopy and intubation using intravenous clonidine 3 TNCG/kg, intravenous dexmedetomidine 0.5 mcg/kg and placebo (0.9% NS) was found that mean systolic blood pressure in group D and group C was significantly lower after intubation and all subsequent intervals when compared to a placebo group, but in our study.

We found that MAP, Mean systolic pressure and mean HR in dexmedetomidine group were significantly lower when compared to clonidine group.

Sallaantha Ray [9] *et al.* in the year 2016 did a study with IV clonidine and IV dexmedetomidine to see for efficacy on hemodynamic response to laryngoscopy and tracheal intubation using 21 mcg/kg of clonidine and 1 mcg/kg of dexmedetomidine as premedication and found that fall in heart rate over time in both groups but reached statistical significance only in dexmedetomidine group at 1 min post intubation Compared to clonidine group and maximum percentage of SBP, DBP and MAP were significantly higher in clonidine group than in dexmedetomidine group (26% vs 22%, 29.4% vs 26% and 28.7% vs 24%) respectively. In our study using 1mcg/kg of IV clonidine and 1mcg/kg IV dexmedetomidine group found that the fall in heart rate observed at 6 min in the group which was statistically significant in dexmedetomidine group compared to clonidine group. Maximum percentage of fall in SBP, DBP and MAP were significantly lower in clonidine group compared to dexmedetomidine group in view of low dose of clonidine [10].

Conclusion

In our study we found that HR and MAP were significantly less if premedication given with Dexmedetomidine after Laryngoscopy and endotracheal intubation for 1st 25 minutes period when compared to premedication given with clonidine.

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