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Efficacy of Dexmedetomidine Versus Clonidine in Attenuating Hemodynamic Response to Laryngoscopy and Intubation

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ABSTRACT
Attempts were made to differentiate between effect of laryngoscopy and those of tracheal intubation and their individual contribution to hemodynamic changes. Prys Roberts *et al*. (1971) observed that a majority of patients produced reflex tachycardia and hypertension well before the act of intubation. So it is laryngoscopy rather than endotracheal intubation which generates the stimulus. A correlation between pressor response and plasma catecholamine concentration is implicated in causation of this hemodynamic response. All the patients were visited the day before surgery and pre anaesthetic counselling done. All the patients received Tab alprazolam 0.5 mg night before the surgery. On the day of surgery intravenous line was secured. On entering the OT, pulse oximeter, non-invasive BP, ECG monitors were connected. A baseline heart rate, systolic and diastolic blood pressure, SpO2 were recorded (BL-baseline). After giving the loading dose of dexmedetomidine there was more fall in SBP, also at the time of laryngoscopy the rise in SBP was lesser in dexmedetomidine group keeping parameters closer to baseline values. It is also observed that the return to baseline values was faster in dexmedetomidine group than in clonidine group.

INTRODUCTION

Airway management is very important during delivery of general anaesthesia. Patients who have been anaesthetized are unable to maintain an adequate airway and artificial airway maintenance devices are needed.

Traditionally, laryngoscopy and endotracheal intubation has been the mainstay in providing adequate airway, delivering anaesthesia and avoiding aspiration in anaesthetized patients[1].

Endotracheal intubation is translaryngeal placement of endotracheal tube, via the nose or mouth. It is one of the most commonly performed procedures, where the role of anesthesiologists in patient care is supreme. Laryngoscopy and tracheal intubation are noxious stimuli which provoke a transient but marked sympathetic response manifesting as hypertension and tachycardia[2].

Hypertension and tachycardia have been recognized since 1950's as commonly associated with intubation under light anesthesia. It has been observed that the mechanical stimulation of four areas of the upper respiratory tract, the nose, the epipharynx, the laryngopharynx and tracheobronchial tree, induce the reflex cardiovascular response, associated with enhanced neuronal activity in cervical sympathetic fibers[3].

Attempts were made to differentiate between effect of laryngoscopy and those of tracheal intubation and their individual contribution to hemodynamic changes. Prys Roberts *et al*. (1971) observed that a majority of patients produced reflex tachycardia and hypertension well before the act of intubation.

So it is laryngoscopy rather than endotracheal intubation which generates the stimulus. A correlation between pressor response and plasma catecholamine concentration is implicated in causation of this hemodynamic response.

These changes are of little consequence in healthy patients but in susceptible patients particularly those with systemic hypertension, coronary heart disease, cerebrovascular disease and intracranial aneurysm, even these transient sympathetic changes can result in potentially deleterious effects like left ventricular failure, arrhythmias, myocardial ischemia, cerebral haemorrhage and rupture of cerebral aneurysm. Convulsions may be precipitated in eclamptic patients. Almost all types of dysrhythmias have been reported in addition to sinus tachycardia and sinus bradycardia. The common abnormalities are nodal rhythm, atrial and ventricular extra systoles and pulsus alternans, less commonly multifocal extra systoles, pulsus bigeminy and atrial fibrillation have been reported.

Radionucleotide studies have shown the stress response to laryngoscopy and endotracheal intubation produce a rapid decline in global left ventricular function (ejection fraction) within seconds, often exceeding that seen following exercise in patients with symptomatic coronary artery disease[4].

MATERIALS AND DISCUSSIONS

Inclusion Criteria:

- Age between 18-60 years of both sexes
- ASA grades I and II
- Mallampatti classes I and II
- Weight between 40-90 kgs

Exclusion Criteria:

- Heart rate <70/min
- Basal systolic BP<100 mm Hg
- H/o of asthma, cardiac disease and presence of heart block ASA > II
- Anticipated difficult intubation
- Anxious or apprehensive patients
- Intubation time more than 15 seconds

Patients were selected after thorough pre anesthetic assessment and investigations.

Investigations:

- Hb%, Complete Blood count
- Random blood sugar
- Blood urea and serum creatinine
- ECG
- X ray chest - AP View

Group - 1 Clonidine Group: Comprising of 25 patients, received intravenous Clonidine 2 µg/kg, 10 mins before induction.

Group - 2 Dexmedetomidine Group: Comprising of 25 patients, received Dexmedetomidine diluted in 10 cc of normal saline, 0.5 µg/kg, over 10 minutes as intravenous infusion 10 min before induction.

Premedication: All the patients were visited the day before surgery and pre anaesthetic counselling done. All the patients received Tab alprazolam 0.5 mg night before the surgery. On the day of surgery intravenous line was secured. On entering the OT, pulse oximeter, non-invasive BP, ECG monitors were connected. A baseline heart rate, systolic and diastolic blood pressure, SpO2 were recorded (BL-baseline). All the patients were given injection glycopyrrolate 0.01 mg/kg IV, and one of the study drugs was administered. Intravenous infusion of Normal Saline was started.

RESULTS AND DISCUSSIONS

Table showing the Systolic Blood Pressure in the two groups, there was significant difference between the groups ($p<0.05$). After giving the loading dose of

Table 1: Comparison of SBP (mm Hg) in two groups of patients studied

SBP (mm Hg)	Group I	Group II	P value
BL	127.72±8.55	125.08±8.12	0.269
PI	125.72±8.22	120.92±9.21	0.003**
AL	132.96±8.09	128.72±8.27	0.073*
1 min	137.16±8.12	133.92±8.00	0.162
3 min	133.80±8.38	131.76±8.64	0.401
5 min	127.48±8.26	127.16±8.14	0.891

Table 2: Comparison of DBP (mm Hg) in two groups of patients studies

DBP (mm Hg)	Group I	Group II	P-value
BL	80.20±5.58	78.12±5.83	0.204
PI	79.08±5.51	74.56±7.01	0.003**
AL	83.24±5.70	80.64±6.54	0.140
1 min	86.28±5.58	83.92±6.14	0.162
3 min	83.96±5.73	82.08±6.14	0.269
5 min	78.96±5.39	78.60±6.36	0.830

Table 3: Comparison of MAP (mm Hg) in two groups of patients studied

MAP (mm Hg)	Group I	Group II	P-value
BL	95.88±6.09	94.08±6.50	0.319
PI	94.29±6.28	90.08±7.53	0.003**
AL	99.60±6.57	96.76±7.00	0.146
1 min	103.20±6.28	100.40±6.42	0.126
3 min	101.06±6.64	98.20±7.04	0.145
5 min	95.13±6.17	94.92±6.61	0.907

dexmedetomidine there was more fall in SBP, Also at the time of laryngoscopy the rise in SBP was lesser in dexmedetomidine group keeping parameters closer to baseline values. It is also observed that the return to baseline values was faster in dexmedetomidine group than in clonidine group.

Table showing the Diastolic Blood Pressure in the two groups, there was significant difference between the groups ($p<0.05$). After giving the loading dose of dexmedetomidine there was more decrease in diastolic blood pressure than the clonidine group. Also at the time of laryngoscopy the rise in DBP was lesser in dexmedetomidine group keeping parameters closer to baseline values. After laryngoscopy rise of the values in dexmedetomidine group was less and returned to baseline faster than the clonidine group.

Table showing the Mean Blood Pressure in the two groups, there was significant difference between the groups ($p<0.05$). After giving the loading dose of dexmedetomidine there was a greater decrease in Mean blood pressure than the clonidine group. Also at the time of laryngoscopy the rise in MAP was lesser in dexmedetomidine group keeping parameters closer to baseline values.

Clonidine is an imidazoline compound with the molecular formula C9H9Cl2N3. It is the prototype of α-2 adrenoceptor agonists that has been studied with an α-2: a-1 ratio of 200:11. The drug is licensed for the treatment of hypertension, migraine and menopausal flushing.

It is also an analgesic, sedative and anxiolytic. These properties along with its ability to maintain peri-operative haemodynamic stability make clonidine a useful agent in anaesthesia and intensive care[5].

Clonidine inhibits norepinephrine release from the peripheral prejunctional nerve endings and causes bradycardia. There are no post junctional a2 receptors in myocardium. Hence a direct effect on heart is unlikely. It causes hypotension due to centrally mediated reduction in sympathetic outflow. Clonidine exerts vagomimetic effect on heart by stimulating nucleus tractus solitarius which can be attenuated completely by highly selective muscarinic M receptor antagonists. It can cause bradycardia and reduction in cardiac output without affecting the cardiac contractility and peripheral vascular resistance. It enhances baroreflex sensitivity. In higher doses it depresses the atrioventricular nodal conduction with slight prolongation of PR interval. It has antiarrhythmic action mediated via imidazoline receptors and vagus[6].

Dexmedetomidine is now being used off-label outside of the ICU in various settings, including sedation and adjunct analgesia in the operating room, sedation in diagnostic and procedure units, and for other applications such as withdrawal, detoxification amelioration in adult and pediatric patients[7].

α-2 agonists can produce either hypo- or hypertension. A biphasic cardiovascular response has been noted. At lower doses, their dominant action is sympatholysis (i.e. the ability to block sympathetic arm of the autonomic nervous system), which is mediated by the α-2A-adrenergic receptor subtype. There are several well documented mechanisms for this activity, including the inhibition of the firing of the locus coeruleus (the pivotal noradrenergic relay nucleus in the brain stem) and inhibition of norepinephrine release at neuro-effector junction. At higher doses, the hypertensive action dominates via activation of α-2B-adrenoceptors, which are located in smooth muscle cells in the resistance vessels. Among healthy volunteers given dexmedetomidine, a biphasic (low, then high) dose response relation for mean arterial pressure, pulmonary arterial pressure, and vascular resistance are observed[8].

The application of a single dose of dexmedetomidine reduced norepinephrine release by as much as 92% in young healthy volunteers. The release of epinephrine is also reduced by the same amount. This seems to be more important than the central a-adrenoceptor agonism.

The baroreceptor reflex is preserved in patients who received dexmedetomidine and the reflex heart rate response to a pressor stimulus is augmented. These results illustrate the cardiovascular response is evoked mainly by reverse mechanism in central sympathetic outflow[9].

Dexmedetomidine could result in cardiovascular depression i.e., bradycardia and hypotension. In retrospective study adverse drug reactions were reported in 30% of patients: hypotension was the most common one, occurring in 22.7% of patients, while bradycardia was reported in 1.4% of patients.

There is risk of bradycardia requiring intervention with a loading or a maintenance dose of more than 0.7 mcg/kg/hr. usually these temporary effects were successfully treated with atropine or ephedrine and volume infusions[10].

CONCLUSION

There was marked rise of HR, SBP, DBP and MAP following laryngoscopy and intubation in the clonidine group when compared to dexmedetomidine group. Hence, IV dexmedetomidine 0.5 µg/kg was effective in attenuating the pressor response to intubation. IV clonidine also had decreased the pressor response to laryngoscopy and tracheal intubation but dexmedetomidine showed better attenuation and hemodynamic control.

REFERENCES

1. Virtanen, R., J.M. Savola, V. Saano and L. Nyman, 1988. Characterization of the selectivity, specificity and potency of medetomidine as an a2-adrenoceptor agonist. Eur. J. Pharmacol., 150: 9-14.
2. Ahlquist, R.P., 1948. A study of the adrenotropic receptors. Am. J. Physiol. Legacy. Content., 153: 586-600.
3. Langer, S.Z., 1980. Presynaptic regulation of the release of catecholamines. Pharmacol. Rev., 32: 337-362.
4. Drew, G.M. and S.B. Whiting, 1979. Evidence for two distinct types of postsynaptic a-adrenoceptor in vascular smooth muscle In vivo. Br. J. Pharmacol., 67: 207-215.
5. Berthelsen, S. and W.A. Pettinger, 1977. A functional basis for classification of a-adrenergic receptors. Life Sci., 21: 595-606.
6. Anttila, M., J. Penttilä, A. Helminen, L. Vuorilehto and H. Scheinin, 2003. Bioavailability of dexmedetomidine after extravascular doses in healthy subjects. Br. J. Clin. Pharmacol., 56: 691-693.
7. Scheinin, H., S. Karhuvaara, K.T. Olkkola, A. Kallio, M. Anttila, L. Vuorilehto and M. Scheinin, 1992. Pharmacodynamics and pharmacokinetics of intramuscular dexmedetomidine. Clin. Pharmacol. Ther., 52: 537-546.
8. Lakhlani, P.P., L.B. MacMillan, T.Z. Guo, B.A. McCool, D.M. Lovinger, M. Maze and L.E. Limbird, 1997. Substitution of a mutant-adrenergic receptor via “hit and run” gene targeting reveals the role of this subtype in sedative, analgesic and anesthetic-sparing responses In vivo. Proc. Nat. Acad. Sci., 94: 9950-9955.
9. Makaritsis, K.P., C. Johns, I. Gavras and H. Gavras, 2000. Role of-adrenergic receptor subtypes in the acute hypertensive response to hypertonic saline infusion in anephric mice. Hyperten., 35: 609-613.
10. Ebert, T.J., J.E. Hall, J.A. Barney, T.D. Uhrich and M.D. Colinco, 2000. The effects of increasing plasma concentrations of dexmedetomidine in humans. Anes., 93: 382-394.