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A Comparision of Nitroglycerine and Dexmedetomidine for Controlled Hypotension in Endoscopic Resection of Juvenile Nasopharyngeal Angiofibroma

P. Indira[1], Seema[2], Deepraj Singh[3], Rajola Raghu[4]

Author's Affiliation:

[1,4]Associate Professor, [2]Post graduate, [3]Professor & Head of the Department, Department of Anaesthesiology, Osmania Medical College/Hospital , Hyderabad, Telangana 500095, India.

Abstract

Background: Juvenile nasopharyngeal angiofibroma(JNA) is a locally invasive benign vascular tumor, controlled hypotension is used to facilitate endoscopic resection. Nitroglycerine and Dexmedetomidine were compared as hypotensive agents in this study.

Methods : Ethics Committee has approved the study which was conducted at Government ENT hospital, Hyderabad during 2016 – 2018.Study included 40 patients divided into two groups, Group D (Dexmedetomidine) n=20,1µg/kg over 15 min followed by a maintenance infusion at 0.5µg/kg/hr,GroupN(Nitroglycerine) n=20- Nitroglycerine @ 0.5µg/kg/min and titrated in doses 0.5-5µg/kg/min for target blood pressure respectively.

Results : A statistically significant difference was observed in the study in mean pulse rate between two groups Group D-66.09±2.83, Group N-86.59±4.24, p value 0.0001. Blood loss was lower in Group D-310.71±140.58 compared to Group N 482.61±141.42 , and is statistically significant (p value 0.0004). SBP, DBPand MAP were comparatively lower in Group D throughout the surgery.

Conclusions: Dexmedetomidine is a better hypotensive agent with favourablehemodynamics and significantly less blood loss when compared to Nitroglycerine .

Key words: Controlled Hypotension; Dexmedetomidine; Endoscopy; Juvenilenasopharyng ealangiofibroma; Nitroglycerine; Massive Hemorrhage.

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Introduction

Juvenile nasopharyngeal angiofibroma (JNA) is a highly vascular, locally invasive benign tumor exclusive of male adolescents, incidence of 1:150,000 it was first described by Hippocrates in 5th century B.C., Friedberg (1940) called it juvenile angiofibroma[1] JNA arises from posterolateral wall of nasal cavity, anatomical location of tumor is readily accessible for transnasal endoscopic resection[2,3] which is gold standard of care. The tumor has vascular supply from external carotid system, potential risks exist throughout

Corresponding Author: Rajola Raghu, Associate Professor, Department of Anaesthesiology, Osmania Medical College/ Hospital, Hyderabad, Telangana 500095, India.
E-mail: raghu.rajula@gmail.com

anesthesia- intraoperative massive bleeding, bronchoaspiration and airway obstruction after extubation.[4] Intraoperative bleeding[5] interferes with surgical field visibility, poses a challenge for both surgeon and anaesthesiologist. Controlled hypotension[6] provides a relatively bloodless field improves visibility of anatomical landmarks, minimizes blood loss and facilitates safe, rapid and effective tumor resection.Various pharmacological agents are used to achieve controlled hypotension. In this study we compared nitroglycerine (NTG) versus dexmedetomidine for inducing controlled hypotension with primary objective-monitoring haemodynamic parameters and secondary objective amount of intraoperative blood loss.

Patients and Methods

A prospective randomized single blinded study was conducted at Governement ENT Hospital, Osmania Medical College, Hyderabad. Institutional ethics committee has approved the study which included 40 patients in age group of 10-20 years posted for transnasal endoscopic resection of NPA. At pre-anaesthetic evaluation all patients were explained about surgery and written informed consent was obtained and were thoroughly investigated for comorbid conditions and cleared for surgery.

Patients were randomized into two groups-Group D(n=20), Dexmedetomidine -200µg in 100 ml of normal saline (2µg/ml), administered as iv infusion in pediatric volumetric burette set, dose 1µg/kg over 10 mins followed by maintenance infusion 0.5µg/kg/hr.

Group N(n=20)-Nitrogly cerine-25mg in 100ml normalsaline, infusion dose 5µg-1kg-1min-1 and titrated in the dose 0.5-5µg-1kg-1min-1to achieve desired mean arterial pressure.

Intraoperatively effects of both agents on hemodynamic parameters and blood loss during surgery were noted in a preset proforma.

Inclusion Criteria:	Exclusion Criteria:
ASA Grade I or II	ASA Grade III or IV H/o Coagulopathy
Age 10-20 yrs	Anticoagulants, H/o Drug sensitivity

Standard General anaesthesia technique was used in both groups, Glycopyrrolate 0.04mg/kg, Ondansetron 0.08mg/Kg, Thiopentone @ 3-5mg/kg and vecuronium 0.08mg/kg, Intubated with appropriate sized cuffed ET tube and throat packed. IPPV, 33:66 ; O2/N2O, desflurane 4-6%, Ventilation adjusted to maintain EtCO2: 30-35 mm Hg. Two 16 G venflon were secured on forearms, one for fluids, one for blood and another 20G venflon on dorsum of hand for infusion of hypotensive agents. A 15° Head up position used to facilitate venous drainage.

Monitoring: ECG-V5 lead with ST segment analysis to detect is chemia[7] NIBP, SPO_2, $EtCO_2$ Urine output. Before start of surgery, MAP was decreased to achieve a target MAP of 60-70 mmHg in both groups, baseline SBP, DBP, MAP, HR and SPO_2 were recorded and every 10min till end of surgery. Fluid therapy included–fasting fluid deficit was replaced during first 1 or 2 hr. maintenance, 5 to 6 mL/kg-hr of Ringer's lactate. Intraoperatively blood loss was measured by blood volume in suction bottles and swabs, if blood loss exceeds 20% to 25% of patient's total blood volume it was replaced with blood. Total duration of surgery was 150 mins in all patients. Severe hypotension (MAP<55mmHg) corrected by stopping inhalation agent, Mephentermine 6mgiv bolus.Bradycardia(HR<50bpm) corrected by Atropine 0.5mg iv. Hypotensive agent was stopped 10 minutes before anticipated end of surgery, residual neuromuscular block antagonized with neostigmine 50µg/kg & glycopyrrolate 10µg/kg. After recovery, patients were transferred to post anesthesia care unit (PACU).

Statistical Analysis was done using :

1. Mean
2. Standard deviation
3. Independent t test (for hemodynamic parameters)

Data was entered using MS Excel software and analysed using SPSS 16 version software for determining statistical significance. p values < 0.05 are considered statistically significant.

Observations and Results

Study included 40 patients posted for endoscopic resection of NPA, divided into two groups, Group N(n=20) received NITROGLYCERINE, Group D(n=20) received Dexmedetomidine respectively for controlled hypotension. Intraoperatively hemodynamics (HR, SBP, DBP, MAP) were observed in both groups and recorded in a tabular form for statistical analysis, amount of blood loss between two groups was compared.

Table 1: Distribution of mean age & weight of study groups.

	GroupN (Mean±S.D.)	GroupD(Mean±S.D.)	'p' value
Age	15.33±2.64	15.94±2.26	0.4373
Weight	45.29±9.75	45.29±9.75	0.8192

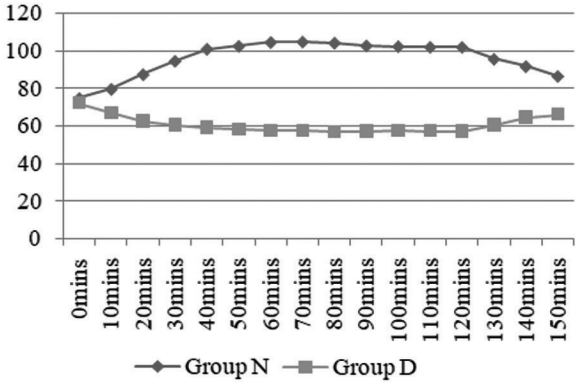
All patients were males in the age group 10-20 years and weight in the range of 28-60Kg and no statistically significant difference was noted between both groupss (p>0.05) (Table.1).

Hemodynamic Variables

Table 2: Intraoperative Pulse Rate variations.

Time (mins)	Group N	Group D	't' value	'p' value
	Mean±S.D.	Mean±S.D.		
0	74.86±8.39	72.05±6.07	1.2135	0.2324
10	79.74±7.81	67.05±5.85	5.8159	0.0001
20	87.53±7.11	62.62±5.49	12.4015	0.0001
30	94.83±5.69	60.49±4.49	21.1878	0.0001
40	100.98±4.94	59.10±3.44	31.1132	0.0001
50	102.53±4.96	58.11±3.23	33.5618	0.0001
60	104.56±6.64	57.47±2.92	29.0325	0.0001
70	104.81±6.6	57.45±3.42	28.4928	0.0001
80	104.20±6.69	57.05±3.36	28.166	0.0001
90	102.72±6.23	57.34±3.45	28.4977	0.0001
100	102.40±5.8	57.48±3.6	29.1922	0.0001
110	102±5,66	57.39±3.54	29.8841	0.0001
120	101.99±5.98	57.35±3.34	29.146	0.0001
130	95.69±5.59	60.63±2.92	24.8613	0.0001
140	91.77±3.8	64.49±2.72	26.1065	0.0001
150	86.59±4.24	66.09±2.83	17.9844	0.0001

Graph 1: Intraoperative Heart rate variations.

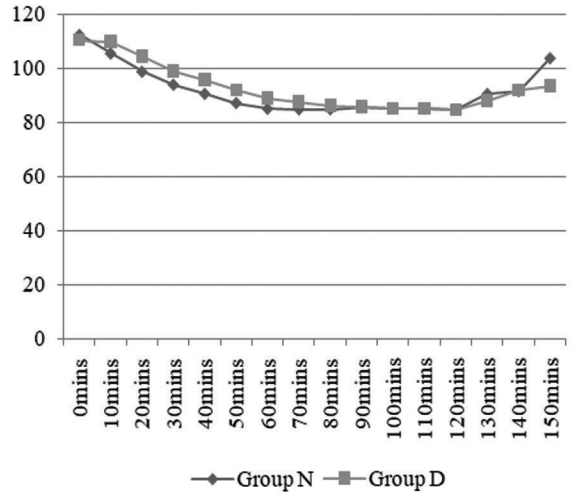


Heart rate was observed in both study groupsat specified intervals and the difference was highly significant (p<0.0001) statistically (Table. 2), with lower pulse rates recorded in group D (Graph.1) from 10 minutes of starting test drug infusions to stopping infusion (57.05±3.36 and 67.05±5.85) when compared with nitroglycerine group (79.74±7.81 and 104.81±6.6).

Table 3: Systolic Blood Pressure variations.

Time (mins)	Group N	Group D	t value	P value
	Mean±S.D.	Mean±S.D.		
0	112.47±10.02	110.37±11.11	0.6277	0.5339
10	105.62±9.65	109.79±9.72	1.3615	0.1814
20	98.91±8.95	104.30±9.33	1.8644	0.07
30	94.11±7.86	99.02±8.33	1.8241	0.07
40	90.74±7.24	95.85±8.28	2.0017	0.0525
50	87.37±6.15	91.96±7.17	2.0145	0.0511
60	85.24±5.02	89.01±6.08	1.9961	0.0531
70	85.03±4.52	87.71±5.48	1.6127	0.1151
80	85.05±4.42	86.33±5.17	0.8336	0.0497
90	85.73±4.39	85.89±4.65	0.1122	0.9113
100	85.34±3.99	85.33±4.53	0.007	0.9945
110	85.26±4.18	85.23±4.69	0.0222	0.9824
120	84.95±5.98	84.87±3.34	0.0569	0.9549
130	90.71±3.94	88.10±4.22	2.0217	0.0503
140	91.77±3.8	91.99±4.42	0.1688	0.8669
150	103.89±5.88	93.56±4.14	6.4241	0.0001

Graph 2: Intraoperative Systolic blood pressure variations.



The mean systolic blood pressure, mean diastolic blood pressure and mean arterial pressure recorded in group D were marginally less when compared with Group N from 90mins of starting test drug infusion to stopping infusion.

The mean SBP, DBP and MAP recorded were significantly lower in Group D (Table.6) after stopping test drug infusion ('p' value 0.0001)

Table 4: Diastolic Blood Pressure variations.

Time (mins)	Group N	Group D	t value	'p' value
	Mean±S.D.	Mean±S.D.		
0	74.91±6.14	71.88±7.01	1.4493	0.1555
10	69.14±5.53	72.25±7.26	1.524	0. 1358
20	64.22±4.9	67.98±6.75	2.016	0.0509
30	60.04±4.52	63.27±6	1.9229	0.062
40	57.85±3.41	59.12±3.12	1.2288	0.2267
50	54±3.38	55.25±3.34	1.1764	0.2467
60	52.75±3.23	53.74±2.6	1.0678	0.2924
70	52.51±3.15	52.85±2.38	0.3851	0.7023
80	52.82±2.82	53.25±3.26	0.4461	0.658
90	52.98±2.79	52.92±2.95	0.76	0.452
100	52.58±2.83	52.57±2.87	0.00111	0.9912
110	52.48±2.83	52.42±2.89	0.0663	0.9475
120	52.79±2.43	52.54±2.41	0.3267	0.7457
130	57.72±3.01	56.49±2.54	1.3967	0.1706
140	62.56±3.36	60.96±2.12	1.8011	0.0769
150	70.75±4.8	63.02±1.95	6.6724	0.0001

Graph 3: Intraoperative Diastolic Blood pressure variations.

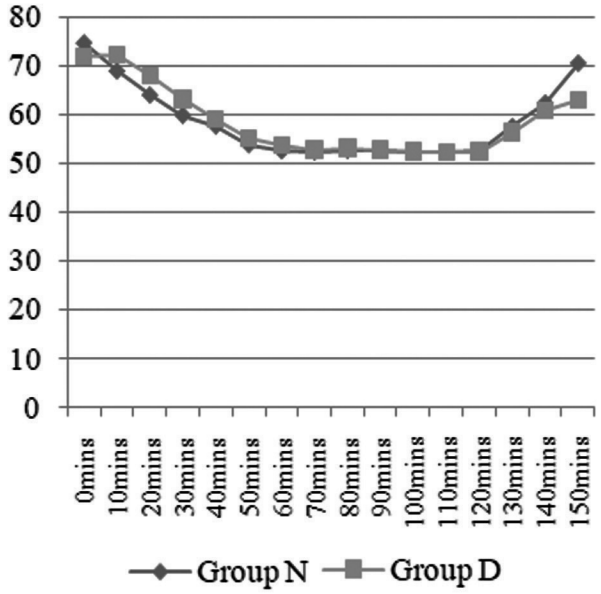
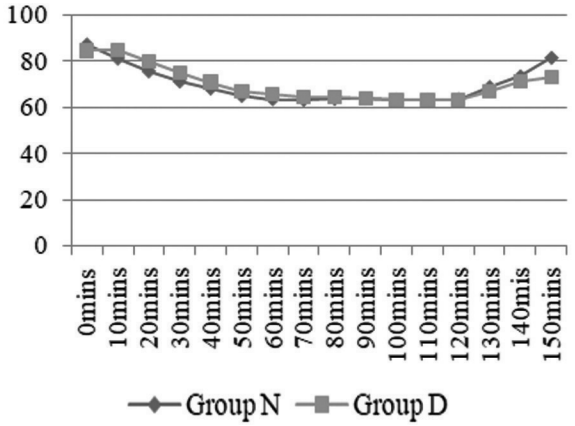


Table 5: Intraoperative Mean Arterial Pressures between study groups.

Time (mins)	Group N	Group D	t value	P value
	Mean±S.D.	Mean±S.D.		
0	87.48±7.04	84.73±8.12	1.1444	0.2596
10	81.51±6.4	84.92±7.72	1.5208	0. 1366
20	75.88±5.89	79.89±7.23	1.923	0.062
30	71.41±5.32	75.08±6.5	1.954	0.0581
40	68.33±4.87	71.05±3.75	1.979	0.0551
50	65.11±4.27	67.18±4	1.5822	0.1219
60	63.64±3.09	65.61±4.27	1.737	0.0905
70	63.36±3.34	64.48±3.01	1.114	0.2723
80	63.67±3.3	64.42±3.3	0.4461	0.4767
90	63.92±3.16	63.87±3.3	0.7187	0.9612
100	63.52±3.16	63.52±3.28	0	1
110	63.48±3.05	63.47±3.18	0.0101	0.992
120	63.58±2.97	63.38±2.99	0.2122	0.8331
130	68.89±2.91	67.14±2.82	1.9313	0.0609
140	73.62±3.34	71.35±2.66	2.3776	0.0226
150	81.7±5.01	73.2±2.55	6.762	0.0001

Graph 4: Intraoperative Mean arterial pressure variations.



(Graph. 5) Attributed to spectrum of effects of dexmedetomidine extending into postoperative period.

Table 6: Blood pressure variations after stopping the study drug infusion.

Time -150 mins	Group N	Group D	't' test	'p' value
SBP	103.89±5.88	93.56±4.14	6.4241	0.0001
DBP	70.75±4.8	63.02±1.95	6.6724	0.0001
MAP	81.7±5.01	73.2±2.55	6.762	0.0001

Graph 5: Blood pressure variations after stopping test drug infusion in both study groups.

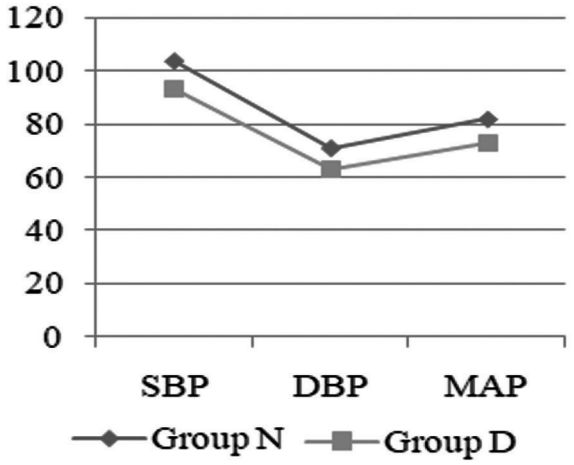
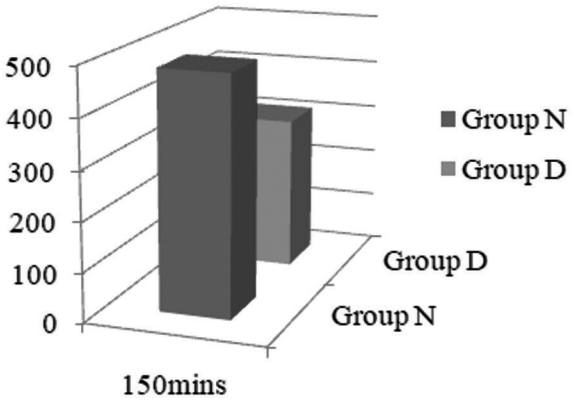


Table 7: Intraoperative Blood Loss.

Mean ± S.D.	Blood loss	't'test	'p' value
482.61±141.42	Group N	3.8553	0.0004
310.71±140.58	Group D		

Graph 6: Comparision of intraoperative blood in both study groups.



Intraoperatively blood loss was less in Group D when compared with Group N (Table. 7) and difference is statistically significant ('p'value0.0004) (Graph. 6), because of favourable hemodynamic profile induced by dexmedetomidine.

Discussion

In 1918, Canon and his colleagues introduced concept of permissive hypotension[8] as a resuscitation strategy used in acute phase of traumatic hemorrhagic shock and its variation known as controlled or induced hypotension (IH) were used in various specialities to create a clearer surgical view, reduce intraoperative blood loss and facilitate surgery.

The etiology of JNA is unknown, anaesthesia management[8] of these tumors is a challenge owing to vascularity, adjacent anatomical structures and intraoperative bleeding, transnasal endoscopic[53] resection is gold standard of care, with controlled hypotension tumor removal is easier, quicker and satisfactory.

Definition of controlled hypotension takes into account level of hypotension required toproduce effect, same time limited by safety(ECKENHOFF & RICH'1966).

Harvey Cushing(1917) proposed deliberate hypotension to provide a bloodless field and better operative conditions for neurosurgery.[9]

Enderby[10] used ganglionic blocking drugs for controlled hypotension in maxillofacial surgery. Schalberg, 1976[11] reported using sodium nitroprusside for hypotensive anaesthesia and blood loss in orofacial corrective surgery.

A mean arterial pressure 30% below a patient's usual MAP, with a minimum of 50 mm Hg in ASA Class I patients and 80 mm Hg in elderly, is clinically acceptable[12], hypotension should be considered satisfactory when bleeding is minimal with adequate organ perfusion (urine out put>0.5ml/kg/min). In theory, as long as mean arterial pressure exceeds sum of colloid osmotic pressure and venous pressure, circulation should be adequate for tissue needs, theoretically a pressure of 32 mm Hg should be sufficient, in practice it is below the safe limit due to specific blood flow requirements of different organs. Controlled hypotension rarely results in vital organ damage as autoregulation maintains their perfusion over a wide range of blood pressures.

The use of dexmedetomidine for providing hypotensive anaesthesia during septoplasty and tympanoplasty[13,14] was studied by Durmus M et al and Ayoglu H et al.

Hypotensive anesthesia should be induced in relation to patient's preoperative blood pressure rather than specific target pressure and limited to level necessary to provide a bloodless field , within safety limits of cerebral and coronary blood flow.

Techniques of Hypotensive Anaesthesia: The key equation is Mean Arterial Pressure=Cardiac Output x Systemic Vascular Resistance (SVR).Thus MAP can be decreased by reducing SVR, CO or both. A reduction in cardiac output for hypotensive anaesthesia is not ideal because maintenance of tissue blood flow is essential. SVR can be reduced by peripheral vasodilation whilst cardiac output can be reduced by lowering venous return, heart

rate, myocardial contractility or a combination of these.

Strategies for Inducing Controlled Hypotension.

Category	Strategy	Examples
Decrease cardiac output	Reduce blood volume	Arteriotomy
	Dilate capacitance vessels	Nitroglycerin
	Decreasecardiac contractility	Inhalation anesthetics (eg, halothane), β-blockers (eg, esmolol)
	Decrease heart rate	Inhalational agents, β-adrenergic blockers
Decrease peripheral vascular resistance	Block autonomic ganglia	Methonium compounds, Pentolinium, Trimethaphan
	α-adrenergic receptor blocker	Phentolamine, Labetalol
	Vascular smooth muscle relaxation	Direct-acting vasodilators (eg, nitroprusside), Calcium-channel blockers (eg, nicardipine), Purines (eg, adenosine), prostaglandin E1, Inhalational anesthetics (eg,Isoflurane)

Measures such as changing position of patient, adjusting airway pressure, adding other drugs to complement activity of primary hypotensive agent(eg., N-acetylcysteine) facilitate induced hypotension. Various drugs are used to achieve controlled hypotension-eg., Nitroglycerine, α-adrenergic receptor blockers, volatile anaesthetics, autonomic ganglion-blockers, β-adrenergic blocking agents, prostaglandin E1, $MgSO_4$ and Ca2+channel blockersand alpha2 agonists eg., dexmedetomidine[15]. Drugs used to produce controlled hypotension must be easy to administer, have a short onset time, an quick offset time on discontinuation, a rapid elimination without toxic metabolites, negligible effects on vital organs, and predictable dose-dependent effects.

Nitroglycerine (NG) was discovered in 1847 by Ascanio Sobrero in Turin, following work with Theophile-Jules Pelouze. Nitrovasodilators[16], (NTG) predominantly dilate capacitance vessels , reduce venous return with concomitant decrease in stroke volume and cardiac output, resulting in hypotension. NTG is frequently used to produce controlled hypotension as it is easily titratable, onset of action is rapid within 1 min and t½ is 2 min. Intravenous infusion is started at 1-2µg/kg/min, there is no upper limit to rate of infusion no toxic metabolites are reported, reversal is spontaneous after stoping infusion. Disadvantages are tachyphylaxis, reflex tachycardia and venous congestion leading to increased blood loss and dose related increase in bleeding time.

Dexmedetomidine is a dextrorotatory S-enantiomer of racemate medetomidine (50:50),((+)-4- (2,3-dimethyle phenyl) ethyl-1 H-imidazole monohydrochloride) (Kuusela et al. 2001)[7]. Itis a highly selective α2-adrenoceptor agonist , with spectrum of beneficial effectslike anxiolysis sedation, analgesia,central sympatholytic effect with a decrease in serum norepinephrine[17,18,19] levels which makes it a good agent for hypotensive anaesthesia. It enhances anaesthesiaby stimulating central α2 and imidazoline receptors, has opioid sparing effects.[20] A biphasic cardiovascular response has been described after i.v.bolus of 1µg/kg dexmedetomidine, initially a transient hypertension and reflex bradycardia are noticed ,due to peripheralα2β-adrenoceptor stimulation of vascular smooth muscle , this is attenuated by a slow infusion over 10 or more minutes.[21,22] Dexmedetomidine exhibits linear pharmacokinetics distribution phase is rapid, with a t½ of distribution 6 min(approx.).[23] Advantages of dexmedetomidine are no reflex tachycardia and no rebound hypertension.

Hypotensive anaesthesia in total hip arthroplasty significantly decreased intraoperative blood loss and operating times compared to normotensive group, Gale Thompson et.al., (Anesthesio -logy;48;91-96;1978), efficacy of dexmedetomidine in providing better surgical field and less blood loss during controlled hypotension was reported by Durmuset.,al,[24] intympanoplasty, septoplasty and maxillofacial surgeries. A single dose of dexmedetomidine 0.5µg/kg/min iv,10mins before induction produced a significant fall in MAP and Heart rate as reported by., Basaret., al,[25] In patients who received dexmedetomidine no other analgesic or anxiolytics were used because of inherent properties.[26,27] Controlled hypotension with dexmedetomidine in middle ear and maxillofacial surgeries provided an ideal surgical field with predictable hemodynamic effectsresults of this study concluded the same. Ulger et al.,[28] comparednitroglycerine with dexmedetomidine as hypotensiveagents(MAP65-75mmHg)inmiddleear surgery, conclude dhypotension and hemodynamic stability was better with dexmedetomidine, results in present study are in accordance with this study. In Group D dose of thiopentone used for induction was lower correlates with study of Peden et al.[29] Dexmedetomidine shows better hemodynamic

stability, clear surgical field, lower(VAS) pain scores and few side effects in FESS, Guven et al., and Goksu et al.[30,31] Cincikas and Ivaskevicius[32] used nitroglycerine infusion (0.79 ± 0.34 µg/kg/min) during FESS to maintain MAP-50-60 mmHg, observed reduced surgical bleeding and improved endoscopic vision. In this prospective randomized study, Dexmedetomidine and Nitroglycerine were equally effective as hypotensive agents (MAP of 60-70mmHg). It was observed dexmedetomidine ensured ideal surgical conditions and average blood loss was less when compared with NTG. In Group N, Fentanyl was used @ 2µg/kg, NTG infusion (0.5-5ug/Kg/min) started after intubation and before surgical incision and titrated dose to maintain mean pressure range of 60-70mmHg. Induction of controlled hypotension with NTG depends on intravascular fluid volume.

Excessive decreases in diastolic blood pressuredecreases coronary blood flow and mayevoke baroreceptor-mediated reflex increases in sympathetic nervous system activity result in tachycardia and increased myocardial contractility. Nitroglycerin produces a dose-related prolongation of bleeding time thatparallels hypotension, it may also be due to direct effect of nitroglycerin on vascular tone resulting in vasodilation. Karl-Erik Karlberg associates[33] assessed effect of iv NTG and concluded NTG inhibits plateletaggregationin higher doses due to glyceryl dinitrate formation.

In this study throughout surgery ,difference in mean heart rates in,GroupD: (60.61 ± 4.49) and Group-N(95.58 ± 9.41) (Table.2), and which is statistically significant from 10mins of starting the test drug infusions (p value < 0.0001) (Graph.1).

The mean SBP(GroupD-92.73 ±8.58, GroupN-91.86±8.77) (Table.3) (Graph.2), DBP (GroupD-58.40±7.03, Group N-58.33±7.48) (Table. 4) (Graph. 3), MAP(GroupD-69.85 ± 7.47, Group N-69.54 ± 7.91) (Table.5) (Graph. 4) between two groups shows no statistically significant difference.

Infusion of hypotensive agent was stopped 10mins before end of surgery. Intraoperatively mean blood loss in Group N-482 ± 141.42 and Group D-310.71±140.58 (Table. 7) (Graph. 6) respectively, the 'p'value was 0.0004.

Results in this study suggests that both Nitroglycerine and Dexmedetomidine are equally good for inducing controlled hypotension in endoscopic resection of NPA. Intraoperative blood loss in Group D, the increased blood loss in Group D was significantly less when compared with Group N" with nitroglycerin can be due to increased heart rate, prolongation of bleeding time by NTG due to inhibition of platelet aggregation partially off settingbeneficial effects of hypotension.

Limitations

1. Rare incidence of tumor.
2. Lack of Cardiac output monitoring.
3. Inability to measure bleeding tendencies with NTG.
4. Small sample size, cannot be concluded the results of present study are definitive.
5. More studies are required to conclude results.

Further recommendations

1. There is scope for further studies related to this topic.
2. Different doses of dexmedetomidine can be compared and studied.
3. Synergism of combination of dexmedetomidine with other drugs to be evaluated.

Conclusion

Dexmedetomidine was safe and equally effective in producing controlled hypotension when compared with Nitroglycerine, Dexmedetomidine has advantage of cardiovascular stability and less blood loss. The inherent properities of anxiolysis, sedation, analgesia, opioid and anaesthetic sparing effects, easy administration, predictability with anesthetic agents and lack of toxic side effects while maintaining adequate perfusion of vital organs makes dexmedetomidine a safe and near ideal agent for hypotensive anaesthesia."

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