Original Article

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Effects of Propofol and Sevoflurane on Haemodynamic Changes

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Abstract

Introduction: Heart rate does not change after induction dose of propofol because it resets or inhibits the baroreceptor reflex, thus reducing tachycardia response to hypotension. Propofol decrease the sympathetic activity to a greater extent than parasympathetic activity.

Methodology: A thorough pre-anaesthetic evaluation was done to assess the general condition and status of cardiovascular, respiratory and central nervous system. Routine investigations like hemoglobin percentage, total leucocyte counts, differential leucocyte counts, bleeding time, clotting time and chest X-ray was done and checked. A written informed consent was taken from parents.

Results: The systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were compared between the two groups following induction and intubation using unpaired student's t-test; statistically there were no significant differences between the two groups.

Conclusion: propofol and sevoflurane effectively blunted the systolic, diastolic and mean arterial pressure

Keywords: Propofol; Sevoflurane; Hemodynamic.

Introduction

Propofol is primarily a hypnotic and action is mediated by binding to the (3 subunit of $GABA_A$ receptor, thereby potentiating the γ-aminobutyric acid (GABA) induced chloride current conductance resulting in hyperpolarization and inhibition of postsynaptic neurons. During induction, 2-2.5 mg/kg of propofol produces 25-40% reduction in systolic BP, 20% decrease in stroke volume and 15-20%, decrease in systemic vascular resistance. Decrease in systolic BP is due to vasodilatation and myocardial depressant effects [1].

Propofol effectively blunts the magnitude of pressor response during laryngoscopy and intubation of trachea. Heart rate does not change after induction dose of propofol because it resets or inhibits the baroreceptor reflex, thus reducing tachycardia response to hypotension. Propofol decrease the sympathetic activity to a greater extent than parasympathetic activity [2].

Sevoflurane was first described in North America in 1971. It is halogenated fluorine (1,1,1,3,3,3, hexafluoroisopropyl fluoromethyl ether). It is non pungent and least airway irritant of all volatile anaesthetics.

Sevoflurane is an excellent choice for smooth and rapid inhalation induction in paediatric and adult patients because of its low blood solubility and non pungent odour. Similarly, upon discontinuation there will be rapid emergence due to rapid fall in alveolar anaesthetic concentration [3].

Sevofluratie mildly depresses myocardial contractility, decreases cardiac output. Systemic vascular resistance and arterial BP decline slightly. It causes [1] little rise in heart rate. It may prolong QT- interval.

Methodology

The study group consisted of 80 patients of both sexes, between the age of l-10years and belonging to ASA Physical status 1 and 2 who were scheduled for cleft lip/cleft palate/cleft alveolus surgery under general anaesthesia.

The following groups of patients were excluded from the study, if they had history of significant cardiac, respiratory,

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renal, hepatic or central nervous system diseases, children with history of sensitivity to the drugs used, children with anticipated difficult airway, children with active or recent upper respiratory tract infection.

Pre-Anaesthetic Evaluation and Preparation

A thorough pre-anaesthetic evaluation was done to assess the general condition and status of cardiovascular, respiratory and central nervous system. Routine investigations like hemoglobin percentage, total leucocyte counts, differential leucocyte counts, bleeding time, clotting time and chest X-ray was done and checked. A written informed consent was taken from parents.

Premedication

All the patients were made to fast for 6 hours for solids and milk and 3 hours for clear fluids and premedicated with combination of midazolam 0.5 mg /kg and atropine 20/ μ g/kg orally 45 minutes prior to surgery.

Patients were randomly allocated using envelope method into 2 groups:

Group A (Propofol) and Group B (Sevoflurane). Patient was shifted to OT and i.v access was established. Sedation score was noted in the OT.

Preoperative baseline values of heart rate, blood pressure and oxygen saturation were recorded and infusion of crystalloid lactated ringer's solution was started according to "4-2-1" formula (based on body weight and hours of fasting).

Both the groups received 2/g/kg of fentanyl, over 30 seconds. After 5 minutes, Group A patients received 3 mg /kg of propofol. Lignocaine 0.2mg/kg was added to propofol solution to abolish pain on injection; speed of injecting propofol was about 30 mg/10 seconds. Group B patients received 8% sevoflurane via a face mask connected to, Mapelson F breathing circuit after priming the circuit with 8% sevoflurane.

Heart rate, blood pressures and oxygen saturation were monitored continuously and recorded at baseline, after propofol / sevofluraneinduction, during intubation, 1 min, 2 min, 5 min & 10 minutes after intubation.

Any stimulus including surgical stimuli was avoided for 10 minutes after tracheal intubation. Complications if any were noted down.

E_tCO_2 was maintained between 30 - 35 mmHg during the procedure. After intubation anaesthesia was maintained with 66% nitrous oxide in oxygen with 0.2% halothane. Muscle relaxant vecuronium 0.1 mg/kg i.v was given after 10 minutes of tracheal intubation.

Intubation times i.e. the time taken from insertion of laryngoscope into the oral cavity till the removal of laryngoscope, number of attempts of tracheal intubation was noted.

After completion of surgery with resumption of spontaneous respiratory attempts, neostigmine 0.05mg/kg and glycopyrrolate 0.01 mg/kg was given to reverse the residual neuromuscular blockade. Patients were extubated after adequate recovery from muscle power, reflexes and spontaneous respiration.

Statistical analysis was performed using Student's unpaired t-test to analyze for time taken for intubation, number of attempts for intubation, hemodynamic parameters between two groups and Chi-square test was used to analyze intubating conditions, complications between the two groups. A p-value less than 0.05 was regarded as significant.

Results

This shows the distribution of baseline heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP).

Table 1: Baseline HR

Group	Mean	Standard deviation	p- value
A	136.98	40.755	0.138*
B	149.28	32.178	

Table 2: Baseline SBP

Group	Mean	Standard deviation	p- value
A	109.70	14.936	0.871*
B	110.30	17.764	

Table 3: Baseline DBP

Group	Mean	Standard deviation	p- value
A	68.83	12.653	0.236*
B	**64.93**	16.315	

Table 4: Baseline MAP

Group	Mean	Standard deviation	p- value
A	75.75	11.399	0.226*
B	71.20	17.704	

*not significant

Table 5: Intergroup comparison of heart rate

Heart rate	Group	Mean	Standard Deviation	P-Value
Base line	A	136.98	40.755	0.138
	B	149.28	32.178	
After induction	A	114.18	24.365	0.001
	B	135.32	28.389	
After intubation 0 min	A	122.35	27.653	0.002
	B	141.65	25.304	
After intubation 1 min	A	119.78	24.627	0.003
	B	137.73	27.605	
After intubation 2 min	A	114.08	22.481	0.002
	B	132.25	27.519	
After intubation 5 min	A	109.03	19.197	0.003
	B	126.40	27.915	
After intubation 10 min	A	104.03	19.197	0.003
	B	119.90	26.500	

Table 6: Intergroup comparison of Systolic blood pressure

SBP	Group	Mean	Standard Deviation	P-Value
Base line	A	109.70	14.936	0.871
	B	110.30	17.764	
After induction	A	88.00	14.408	0.442
	B	90.73	17.037	
After intubation 0 min	A	98.10	15.546	0.087
	B	91.45	18.634	
After inbutation 1 min	A	95.88	12.847	0.977
	B	95.78	17.298	
After intubation 2 min	A	91.83	12.310	0.990
	B	91.78	21.000	
After intubation 5 min	A	86.68	16.448	0.307
	B	89.98	11.907	
After intubation 10 min	A	87.25	11.899	0.466
	B	89.25	12.512	

Table 7: Intergroup comparison of diastolic blood pressure

DBP	Group	Mean	Standard Deviation	P-Value
Base line	A	68.83	12.653	0.236
	B	64.93	16.315	
After induction	A	47.70	11.543	0.683
	B	48.90	14.486	
After intubation 0 min	A	53.18	13.160	0.222
	B	49.35	14.568	
After inbutation 1 min	A	51.13	9.809	0.623
	B	49.65	16.143	
After intubation 2 min	A	48.83	9.145	0.958
	B	48.70	11.732	
After intubation 5 min	A	44.98	6.941	0.681
	B	45.80	10.586	
After intubation 10 min	A	45.90	10.470	0.628
	B	47.18	12.860	

Table 8: Intergroup comparison of mean arterial pressure

MAP (Hg)	Group	Mean	Standard Deviation	P-Value
Base line	A	75.75	11.399	0.226
	B	71.20	17.704	
After induction	A	59.65	13.014	0.939
	B	59.43	13.387	
After intubation 0 min	A	65.28	12.918	0.234
	B	31.58	14.765	
After intubation 1 min	A	66.50	10.884	0.185
	B	63.05	12.165	
After intubation 2 min	A	63.05	9.871	0.926
	B	62.05	11.531	
After intubation 5 min	A	57.40	7.503	0.464
	B	58.78	9.127	
After intubation 10 min	A	57.35	10.163	0.429
	B	59.28	11.480	

Table 9: Intergroup comparison of SpO_2

SpO_2%	Group	Mean	Standard Deviation	P-Value
Base line	A	99.35	2.007	0 .465
	B	99.60	0.778	
After induction	A	99.95	0.221	0.306
	B	99.88	0.404	
After intubation 0 min	A	99.13	2.078	0.210
	B	96.55	12.718	
After intubation 1 min	A	99.40	1.257	0.220
	B	98.35	5.221	
After intubation 2 min	A	99.63	0.740	0.918
	B	99.65	1.331	
After intubation 5 min	A	99.80	0.516	0.581
	B	99.68	1.328	
After intubation 10 min	A	99.83	0.385	0.304
	B	99.93	0.474	

The statistical analysis of baseline heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure was done by student's unpaired t-test. The baseline heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were comparable between the groups.

(p- value =0.138, p- value =0.871, p-value =0.236, p-value=0.226).

There was a significant reduction in heart rate from baseline to post induction and post intubation, remained so throughout the study in group A, whereas in group B no much change in heart rate noted during post-0 induction and post – intubation.

The systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were compared between the two groups following induction and intubation using unpaired student's t-test; statistically there was no significant differences between the two groups.

SpO2 changes between the two groups

Both the groups were comparable with respect to S_pO_2. The children had oxygen desaturation in group B, S_pO_2 decreased to 30% in one patient and in the other two patients S_pO_2 decreased 60% and 70%. All were due to laryngospasm. Out of these, two patients required succinylcholine for intubation. In addition to this, one patienthad bronchospasm and one patient had excessive oral secretions. One patient in group B had desaturation upto 90% due to bronchospasm.

Discussion

In Group A, there was definite reduction in heart rate from baseline, post induction and post intubation. Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were decreased post induction and post intubation compared with baseline. Thus propofol decreased both heart rate and blood pressure which indicates there was decrease in cardiac output. So propofol effectively attenuated the hemodynamic response to intubation.

Similar results were found in other studies,Akhilesh Gupta et al [4] found a consistent and similar fall in MAP (16-18%) in all children receiving 2.5 mg/kg, 3.0mg/kg or 3.5mg/kg of

propofol preceded by fixed dose of fentanyl 3/g/kg. However children receiving 3.5mg/kg of propofol also had fall in HR (11%).

Uma Srivastavaet al [5] found significant decrease in HR and arterial pressure from baseline in children given propofol and fentanyl. Steyn et al [6]observed a no change in HR but found a significant fall in MAP after induction and following intubation with a dose combination of propofol 3mg/kg and alfentanil 15g/kg in children.

Blair et al [7] found a reduction in HR before intubation in children who received propofol 3mg/kg and alfentanil 10/g/kg. However, they did not mention about arterial blood pressure and HR changes after intubation.

Coghlanet al [8] compared propofol with or without alfentanil in healthy adult patients and found propofol (2.5mg/kg) alone caused significant increase in HR and MAP after intubation. The addition of alfentanil (20/g/kg) produced slight increase in MAP and no change in HR.

In the study by Davidson et al [9] HR and MAP was decreased after induction, and increased after intubation an all patients. However propofol-alfentanil-lignocaine combination attenuated MAP rise after intubation better compared to other groups. Alexander et al [10] found a significant reduction in HR and MAP in each group after remifentanil. However, no significant differences in MAP and HR were observed at any time in adult patients, who received propofol 2mg/kg with remifentanil either 2/g/kg, 3/g/kg, 4//kg respectively.

Similarly, McNeil et al [11] found that, post induction MAP reduced by 21% and 28% with remifentanil 2/g/kg or 4/g/kg when combined with propofol 2mg/kg respectively. Elvan Erhanet al [12] also found significant decreases in HR and MAP after induction and remained so even after intubation, when a combination of remifentanil 3/g/kg and propofol 2mg/kg was used in healthy adults. Taha et al [13] studied healthy adult patients after receiving propofol 2mg/kg- remifentanil 2/g/kg-lignocaine 1.5mg/kg combination and found significant reduction in HR and MAP post induction and post intubation. Aunetal [14] observed greater fall in MAP with propofol 2.5mg/kg (28-31%) than with thiopentone 5.0mg/kg (14-21%) post induction in children between 8months – 12 yrs.

From the above studies, it is found that propofol definitely causes reduction in HR and blood pressure following induction and attenuates hemodynamic responses to laryngoscopy and intubation. The decreases in HR and blood pressure in our study was due to synergistic effects of fentanyl and propofol. Fentanyl blunted hemodynamic response to laryngoscopy and intubation whereas propofol decreased sympathetic nervous activity.

In Group B study, tracheal intubation was accomplished in 92.5% of children receiving fentanyl 2//kg and 8% sevoflurane; only 87.5% of those children had acceptable intubating conditions compared to 52.5% in group A, which is highly significant. Three patients developed laryngospasm, two of whom required succinylcholine for intubation. Laryngoscopy was easy in 100% of children. Vocal cords were open in 72.5% and moving in 20%, closing in 2.5% and closed in 5% of children. In Group B, 80% children had no coughing, 1% had slight coughing, 2.5% moderate coughing and severe coughing in 7.5% of children. Jaw relaxation was complete in 100% in group B. Limb movements were absent in 77.5% children, slight movement in 15%, moderate and severe limb movements in 5% and 2.5% of the children respectively.

In Thwaiteset al [15] study, all children could successfully be intubated with 8% sevoflurane in nitrous oxide and oxygen at 150s. 91% children had excellent intubating conditions and 9% had good intubating conditions. They demonstrated that 8% sevoflurane with nitrous oxide in oxygen can provide acceptable intubating conditions at 150s. Blair et al [7] found that 87.5% of children had acceptable intubating conditions, after administering 8% sevoflurane in 60% nitrous oxide in oxygen. Intubation was attempted at 180mins. Among these! 45% of children had excellent intubating conditions. The results of this study are similar to our study. Laryngoscopy, vocal cord position, coughing, jaw relaxation and limb movements were significantly better in propofol-succinylcholine group than 8% sevoflurane group, however it was not significant.

In Swadiaetal [15] study, anaesthesia was induced with 60% nitrous oxide in oxygen and incremental increase in concentration of sevoflurane from 1-7%? Time interval from application of facemask to intubation was 242±52.67s. 80% of children had excellent intubating conditions. None had fair or poor conditions. 16% had tachycardia, 8% had bradycardia and 80% had hypotension. Complications like laryngospasm, bronchospasm were not observed.

Parmod Kumar Bithalet al [16] found thai time to reach clinical end point for intubation was 325.93 ± 44.02s. The acceptable intubating conditions were achieved in 81.25% of patients. One patient had

moderate coughing. Jaw relaxation was complete. None had limb movements. There was no significant difference in the assessment of laryngoscopy and vocal cords between halothane and sevoflurane.

In Inomata etal [17] study, 5% sevoflurane was compared with 2.5% sevoflurane in oxygen. They found that Time El 50 and Time El 95 for sevoflurane was 147s and 194s respectively using modification of Dixon's up and down method. No patients demonstrated coughing or laryngospasm in this study. In the Cros et al study [18], acceptable intubating conditions were achieved with 2.5 ± 0.7% of sevoflurane preceded by remifentanil 1/g/kg and infusion 0.25/g/kg/min. In 21 patients intubation was possible without muscle relaxants. Failures were due to coughing or bucking after tracheal intubation. Vocal cords were either relaxed or moving but never closed.

In O'Brien et al [19] study, 8% sevoflurane with 60% nitrous oxide was compared with 5% halothane with 60% nitrous oxide in oxygen. Intubation was successful in all children at 1st attempt. Time to reach clinical end point was 243.5s for sevoflurane. One patient in sevoflurane had excessive vocal cord movement. 7out of 20 children had ideal intubating conditions in the sevoflurane group.

Iamaroon A et al [20] study, compared sevoflurane 8%- nitrous oxide with thiopentone-succinylcholine in adults. Intubation was successful in all patients. 16.7% of patients in sevoflurane group had excellent and 76.6% had goodintubating conditions. Jaw relaxation was similar in both groups. Vocal cords were widely open in 28.4% - 43.4%, midposition in 48.3% - 65%, 2 patients had closed vocal cords. 21.7% - 48.3% of patients in sevoflurane had diaphragmatic movement. 11.7% - 21.7% had mild to moderate coughing. One patient exhibited severe coughing.

Conclusion

Both propofol and sevoflurane effectively blunted the systolic, diastolic and mean arterial pressure following intubation. However, propofol caused a significant reduction in heart rate response during post intubation periods compared to sevoflurane.

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