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Original Research Article.

A Comparative Study of Single Breath Induction of Anaesthesia Using a Vital Capacity Breath of Halothane Nitrous Oxide and Oxygen Mixture with Intravenous Sodium Pentothal

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ABSTRACT

Background: Induction of anaesthesia is accompanied by intravaneous sodium pentothal or inhalation of an appropriate mixture of Halothane, Nitrous oxide and oxygen.

Aims and Objectives: 1) To study the vital capacity single breath induction using Halothane, Nitrous oxide and Oxygen Mixture. 2) To compare the efficiency and acceptance of above method with i/v Sodium Pentothal induction.3) To access any complication arising from above technique.

Methods: Patients were randomly divided in two equal of 25 each belonging to ASA grade 1.

Group A: Patient was given intravenous sodium Pentothal with an intravenous drip of 5% dextrose solution.

Group B: Patients were given a mixture of 4% Halothane in 66%N_2o and 33% oxygen by using mapleson. A breathing system (Vital Capacity Breath)

Results: It has been observed that there was no clinically significant fluctuation in pulse rate and arterial pressure at any time during induction. 1) No patient developed arrhythmia during induction in thiopentone group while one patient in halothane group developed transient ventricular ectopic 1 minute after starting the induction. 2) Except one patient who had coughing during induction with thiopentone, induction was smooth in all. Nausea and vomiting were more with thiopentone group as compared to halothane group. 3) The induction time using the single breath technique is slightly longer than that seen with thiopentone group.4) Recovery from halothane is significantly quicker than thiopentone following which patients were more drowsy for long periods.

Conclusion: On comparing the data of group-A with group-B, It was seen that a single breath induction technique using 4% halothane in nitrous oxide and oxygen is a safe, acceptable and practical alternative to thiopentone induction in cooperative unsedated adult patients.

Keywords: Single Breath Induction, Vital Capacity Breath, Halothane, Sodium Pentothal.

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INTRODUCTION

Thiopentone: In 1934 Lundy et al introduced thiopentone in anaesthetic practice and quickly superseded all other intravenous agents (Dundee 1956).[1]

Thiopentone is sodium 5 -ethyl 5'(methylbutyl)-2-thiobarbiturate. It is sulphur analogue of pentobarbitone.

Brooke and colleagues (1948) were the first to describe the haemodynamic concept of distribution of thiopentone followed by Brodie et el (1950)[2] and price(1960).[3]

The central nervous system, heart, lungs, liver and kidneys take up 90% of drug within one minute, the drug having a special affinity for the nervous tissue(price 1960).[3] It has been observed that within certain limits the higher the initial dose of thiopentone the greater will be increments needed to maintain a constant degree of cerebral depression, this being the phenomenon of acute tolerance (Dundee, Price and Dripps,1956).[4] Saidman and Eger (1966)[5] have shown that metabolism is of major importance in the early rapid reduction of arterial level of thiopentone.

Halothane: Halothane (2- Bromo-2 Chloro-1-1-1Trifluroethane) is potent non inflammable volatile anaesthetic agent.[6] Lowrence J Saidman and Edmond Eger (1964) studied the effect of nitrous oxide and of narcotic premedication on the alveolar concentration of Halothane required for anaesthesia and concluded that nitrous oxide is very effective in lowering the alveolar concentration required for anaesthesia.

Halothane like other inhalation anaesthetic agents is a potent respiratory depressant. In surgical anaesthesia the oxygen

consumption is reduced by about 20%.[7] Severinghaus and Cullen (1958). Halothane is known to cause a fall in heart rate (Jhonstone et al 1956 and [8] Burnap et al 1958) and is associated with a greater incidence of arrhythmias due to vagal inhibition of, specially in combination with hypercarbia and hypotension. Low concentration of halothane have no cardiovascular action [9](Price et al 1966). [10]Eger et al (1970) and [11]Morse et al (1963) found an initial decrease in heart rate and later increase, due to increased sympathetic activity or due to decreased parasympathetic activity, or due to increased sensitivity of myocardium to catecholamines.

MATERIALS & METHODS

The study was carried out in fifty patients of both sexes ranging in age from 25 to 45 years and scheduled for planned short surgical procedures. The cases were divided randomly into two equal groups of 25 each belonging to ASA grade 1.Each patient was induced either by sodium pentothal or by inhalation of an appropriate mixture of halothane, O_2 and Nitrous oxide mixture.

Pre-anaesthetic Medication

All the patients were given injection atropine 0.6mg intramuscularly 30-45 minutes before operation. No other pre-medicant was used.

Group A: An assessed patient was given an intravenous drip of 5% dextrose solution by using a 20 gauge needle. The injection sodium pentothal was injected slowly. It was followed by inhalation of nitrous oxide and oxygen, through facemask with magill's circuit in a flow of 5 litres and 2 litres respectively.

Group B: Prior to induction a mapleson-A breathing system with a 4 litres reservoir bag to be prepared and filled with a mixture of 4% halothane in 66% N_2O and 33% Oxygen, supplied by 6 litres /minute of gas flow.

The patient will then be instructed to breath in deeply and breath out as far as possible to exhale to residual volume. The anaesthetic system mask will then be gently applied to patient's face and patient instructed to breath as far as possible and hold his breath ie: vital capacity breath.

OBSERVATIONS TAKEN

The following parameters were recorded

1) Onset of action: This was recorded from the time of starting the injection (zero time) to the loss of eye-lash reflex or interval between the beginning of the vital capacity breath and disappearance of eye-lash reflex.

2) Cardiovascular changes: Pulse rate and blood pressure were recorded by auscultation method at first, third, and fifth minutes and starting after zero time.

3) Recovery: Recovery time was recorded after the end of procedure to the appearance of eye lash reflex and consciousness. Consciousness was judged by the ability of the patient to open his eye spontaneously. The time when patient was fully oriented and able to sit and stand were recorded.

Ambulation Time: It was judged when there was :

(a)Cessation of dizziness and drowsiness.

(b)Ability to stand and walk along a straight line without support.

4) Side effects: Presence or absence of excitatory phenomenon like: respiratory upset like cough, hicough laryngospasm, bronchospasm, salivation were recorded during anaesthetic period. Nausea and vomiting were recorded during the emergence and recovery period.

Table 1: Showing the Onset of Action (induction time)

No.	Group	No of Patients	Induction time in seconds	't'	P
1	'A'	25	37.68 ± 3.52	17.3	< .001
2	'B'	25	79.2± 10.61		

Table 2: Showing duration of apnoea

No	Group	No of Patients	Induction time in seconds	't'	P
1	'A'	25	30.52± 4.93	1.05	>.05
2	'B'	25	28.88± 6.05		

Table 3: Showing changes in Pulse rate in control group

N=25	Mean S.D.	't'	'p'
Preoperative	97.36 ± 17.69		
Pre-Induction	99.52 ± 20.59	.65	>.05
1 min .after Induction	99.64 ±18.75	.72	>.05
3 min .after Induction	101.76 ± 16.77	.88	>.05
5 min .after Induction	99.28 ± 19.89	.075	>.05

Table 4: Changes in Systolic blood pressure in control group

N=25	Mean S.D.	't'	'p'
Preoperative	114 ± 11.77		
Pre-Induction	119.36 ± 10.81	1.83	>.05
1 min .after Induction	110.16 ± 7.32	5.29	<.01
3 min .after Induction	107.68 ± 13.63	5.1	<.01
5 min .after Induction	107.68 ± 13.63	5.1	<.01

Table 5: Changes in diastolic blood pressure in control group

N=25	Mean S.D.	't'	'p'
Preoperative	80.29 ± 9.51		
Pre-Induction	84.5 ± 6.73	1.85	>.05
1 min .after Induction	83.2 ± 5.92	1.92	>.05
3 min .after Induction	78.9 ± 6.10	3.47	<.05
5 min .after Induction	78.9 ± 6.10	3.47	<.05

Table 6: Changes in pulse rate in patients of group 'B'

N=25	Mean S.D.	't'	'p'
Preoperative	90.68 ± 9.57		
Pre-Induction	92± 9.86	1.85	>.05
1 min .after Induction	97.76 ± 9.47	17.30	<.001
3 min .after Induction	92.8 ± 9.24	1.67	>.05
5 min .after Induction	89.2 ± 8.71	5.43	<.01

Table 7: Changes in Systolic blood pressure in group 'B'

N=25	Mean S.D.	't'	'p'
Preoperative	116 ± 9.71		
Pre-Induction	118.24 ± 6.72	1.19	>.05
1 min .after Induction	114.8 ± 8.37	2.97	<.05
3 min .after Induction	108 ± 7.82	6.96	<.01
5 min .after Induction	102 ± 8.36	9.23	<.001

Table 8: Changes in diastolic blood pressure in group 'B'

N=25	Mean S.D.	't'	'p'
Preoperative	84.0 ± 10.62		
Pre-Induction	86.0 ± 11.14	1	>.05
1 min .after Induction	85.4 ± 5.19	1.4	>.05
3 min .after Induction	83.5 ± 6.64	1.88	>.05
5 min .after Induction	82.80 ± 7.87	2.6	<.05

Table 9: Showing the comparison of pulse rate in different groups(n=25 in each group)

Group		Preoperative 1	Preinduction 2	Change 1-2	After starting induction change in pulse rate from pre induction		
					1 min	3 min	5 min
'A'	Mean	97.36	99.52	2.16	+.12	+2.24	-.24
	S.D.	±17.69	±20.59	±16.59	±6.14	±12.73	±16.05
	t=	1.91	1.65	1.35	5.23	.56	.79
	P	>.05	>.05	>.05	<.01	>.05	>.05
'B'	Mean	90.68	92	+1.32	+5.76	-.8	-2.8
	S.D	±9.57	±9.86	±1.6	±1.67	±2.38	±2.58

Table-10: Showing the comparison of systolic blood in different groups(n=25 in each group)

Group		Preoperative 1	Preinduction 2	Change 1-2	After induction change		
					1 min	3 min	5 min
'A'	Mean	114	119.36	+5.36	-9.2	- 11.68	- 11.68
	S.D.	±11.77	±10.81	±14.6	± 8.72	± 11.44	± 11.44
	t=	.66	.44	.9	2.76	.56	1.56
	P	>.05	>.05	>.05	<.05	>.05	>.05
'B'	Mean	116	118.24	2.24	-3.44	- 10.16	- 16.16
	S.D	±9.71	±6.72	±9.44	± 5.79	± 7.32	± 8.77

Table-11: Showing the comparison of Diastolic blood in different groups(n=25 in each group)

Group		Preoperative 1	Preinduction 2	Change 1-2	After induction change		
					1 min	3 min	5 min
'A'	Mean	80.20	84.5	+4.3	-1.3	-5.6	-5.6
	S.D.	±9.51	±6.73	±10.44	± 3.38	± 8.06	± 7.32
	t=	1.33	.57	.77	.08	1.58	2.01
	P	>.05	>.05	>.05	>.05	>.05	>.05
'B'	Mean	84.00	86.00	+2	-1.4	-2.5	-3.2
	S.D	±10.62	±11.14	±10.62	±5	± 6.64	±10

Table 12: Comparison of ECG changes in patients of both the groups during induction

	Groups		Percentage
	'A'	'B'	
Ventricular ectopics	0	1	4.00

Table 13: Side effects in different groups (n=25 in each group)

S. No		Group - 'A' No of Patients	(%)	Group- 'B' No of Patients	(%)
1	Salivation	0	0	1	4.00
2	Laryngospasm	0	0	0	0
3	Coughing	1	4.00	0	0
4	Hiccough	0	0	0	0
5	Nausea & vomiting	2	8.00	0	0

Table 14: Results from Questionnaire

No	Question	Answer	
		Y	N
1	Do you remember holding your breath?	25	0
2	Do you remember for smell of gas?	20	5
	-Did you find it pleasant 3		
	-No comment 12		
	-Unpleasant 5		
3	Did you get any ringing or buzzing in your years?	2	23
4	Would you have this form of anaesthetic again?	21	4
5	Have you had an anaesthetic before?	15	10

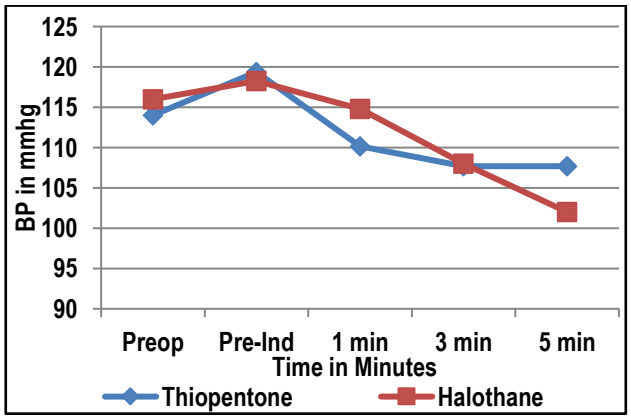

Fig 1: Changes In Systolic Blood Pressure

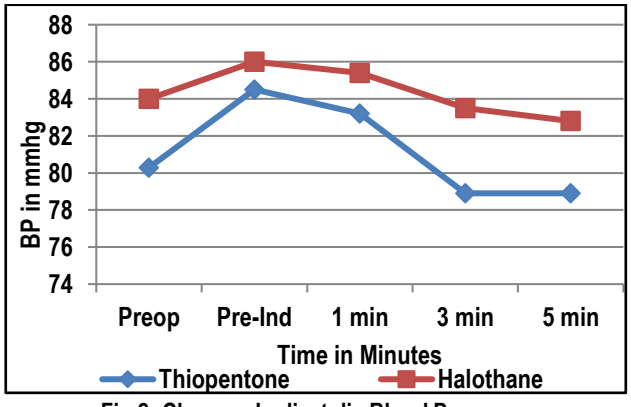

Fig 2: Changes In diastolic Blood Pressure

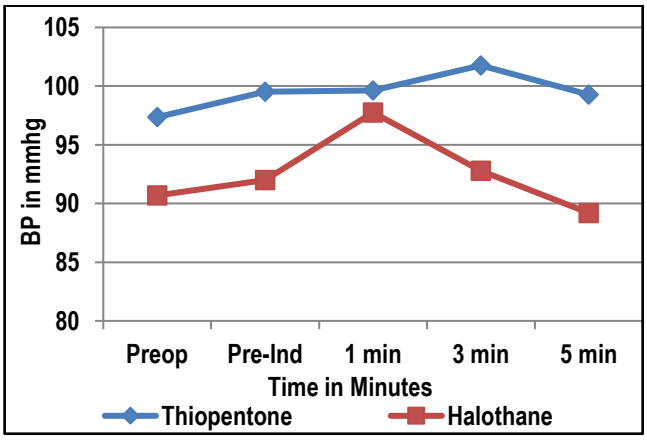

Fig 3: Changes In Pulse Rate

DISCUSSION

Group A: In thiopentone group I patient (4%) had coughing during induction. This had also being shown by studies of[12] Dundee & Moore (1961) whereas incidence of cough was 0-8%.

Two (8%) patients had nausea and vomiting in recovery period, which was comparable with the studies of[12] Moore & Dundee (1961) whereas nausea and vomiting (8.9 to 36 %) had been reported during recovery period.

We did not observe any incidence of laryngospasm, bronchospasm, hicough and sweatness.

Group B: [13]Black et al (1959) found that by decreasing the concentration of halothane, the susceptibility of myocardium to arrhythmias is decreased in response to increasing $paco_2$ and is mediated via release of catecholamines.

As far as recovery is concerned, no patients had restlessness after being given halothane.

Spontaneous eye opening, sitting etc are concerned; full recovery was significantly quicker in the group of patients where halothane was used.

CONCLUSION

Relative cardiovascular stability was a notable finding with the single breath induction, despite the high initial concentration of halothane. The mild decrease in blood pressure although statistically significant is of doubtful clinical significance and is of similar magnitude to that seen with slow intravenous induction using thiopentone. The relative bradycardia observed after 5 minutes did not cause any clinical problem. There was no evidence of excitatory phenomena, vomiting, or laryngospasm.

We suggest that a single breath induction technique using 4% halothane in nitrous oxide and oxygen is a safe acceptable and practical alternative to thiopentone induction in cooperative unsedated adult patients.

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