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CHANGES IN ARTERIAL OXYGEN SATURATION DURING INDUCTION OF ANAESTHESIA AND ENDOTRACHEAL INTUBATION WITH AND WITHOUT PREOXYGENATION

Dr. Geeta Bhavesh Patel M.D.(Anaesthesiology) General Hospital, Palanpur, Gujarat, India

ABSTRACT Preoxygenation is a technique used by anaesthetists who are about to carry out the procedure on the upper airway that necessitates the patients remaining apnoeic for a while and at a risk of hypoxia.Study of changes in arterial oxygen saturation during induction of anaesthesia and endotracheal intubation was carried out with and without preoxygenation in 100 ASA risk I &II patients in 4 different groups.In my study, I found there was 16 % incidence of hypoxaemia(SPO 2 < 94 %) when 33% O2 was used(group IV) & 4 % incidence of hypoxaemia with 50% O2 (Group :III) as against the groups where some form of preoxygenation was performed .I observed 10 % incidence of hypoxaemia in patients who received less than 100 % O2 during suxamethonium apnopa as against none in patients who received 100% O2.

KEYWORDS : Preoxygenation,pulse oximeter,SPO2(arterial oxygen saturation),Hypoxaemia,apnoea

INTRODUCTION: Pre-anesthetic or Preoperative oxygenation, usually known as pre oxygenation, is a technique used by anesthetists who are about to carry out the procedure on the upper airway that necessitates the patient remaining apnoeic for a while and thus at risk of hypoxia. In most cases, the procedure is insertion of an endotracheal tube. The standard intubation technique is to bring about anaesthesia with an induction agent, give a muscle relaxant, ventilate the lungs for a few breaths with an oxygen-enriched mixture by use of a face mask and manual compression of a reservoir breathing bag and then insert the tube. Various factors may increase the dangers of mask ventilation :

1) Difficulties with maintaining the patency of airway:
2) A full stomach, when pressure to the upper airway might induce regurgitation;
3) Anticipated difficulties with intubation such that the patient may be apnoeic for longer than usual;
4) Gross obesity, when unusually high pressures are required to ventilate the lungs by bag and mask;
5) Later stage of pregnancy when in addition to the potential dangers of a full stomach, there is a large incompressible mass in the abdomen and decreased competence of the gastro-oesophageal junction;
6) Also in pregnancy, enlarged breasts and an increased antero-posterior diameter of the chest wall add to the technical difficulties of intubation.

Under these circumstances the onaesthetist may consider preoxygenation. Main goal of preoxygenation is to denitrogenate the functional residual capacity (FRC) of the lungs and thus to increase substantially the reservoir of 0_2 available to an apneic patient. For example, an average adult breathing room air has about 1.5 L of available 0_2 (0.45 L in the lung, with the remainders dissolved or bound in blood and other tissues). In contrast, complete denitrogenation provides about 4.25 L 0_2 storage, 3 L of which occupies the FRC. This 6 fold increase of 0_2 storage in the FRC may provide sufficient 0_2 to prevent hypoxemia after 5 minutes of apnea. During preoxygenation, the patient breathes 100% oxygen via a face mask before induction and while fully Conscious, nitrogen is washed out of the lungs and oxygen is washed in. Haemoglobin becomes fully saturated and some oxygen dissolves in the plasma. The recommended duration of preoxygenation varies between 2 and 5 minutes. Preoxygenation is especially important in old, anaemic or shocked patients, in patients with cardiac and respiratory problems and in small children. The deleterious effect of hypoxaemia under anaesthesia or otherwise are wellk nown. The introduction of pulse oximeters has now made it possible to recognise episode of arterial desaturation occuring unexpectedly during induction and maintainance of even an uncomplicated case. Incidence of hypoxaemia during induction of anaesthesia in those patients who were not preoxygenated is more as compared to who were pre oxygenated. Hence, preoxygenation has been recommended even in ASA Grade I patients undergoing elective surgery. Increasing the oxygen reserve of lungs results in decreased risk of hypoxia when mechanical ventilation has to be avoided prior to tracheal intubation. Hence, in this study, we intended to compare various preoxygenation techniques and find out the incidence of hypoxia with or without preoxygenation.

AIMS OF STUDY : A study of changes in arterial oxygen saturation during induction of anaesthesia & endotracheal intubation has been done by means of pulse oximetry with the main aims:

To observe the effect of preoxygenation and/or ventilation with different percentages of oxygen on incidence and degree of hypoxaemia by measuring and comparing in all four groups, the following parameters.

(1) Preinduction $Sp0_2$ measurement: $Sp0_2$ R
(2) Lowest $Sp0_2$ from Inj Sodium pentothal to intubation . $Sp0_2$ I
(3) Lowest $Sp0_2$ during 3 minutes after tracheal intubation : $Sp0_2$ P
(4) Cardiovascular changes (Pulse, BP)
(5) Intubation time
(6) Any complication.

MATERIALAND METHOD

The study was carried out in 100 ASA Risk I and II patients aged 10-60 years in both sexes undergoing elective ENT surgery and urological surgery. The patients with history of smoking, recent respiratory infection, obesity and anticipated difficult intubation were excluded from the study. The patients were randomly divided into four equal groups. All the patients were premedicated with anticholinergic drug (either Atropine, 20-25 Microgram/Kg. Or Glycopyrolate 6-8 Microgram/Kg. i.v.). Sedative premedication was avoided. Informed consent taken from all the patients. Study was done in following four groups :

Group I was preoxygenated for 3 minutes before induction with oxygen at 6 litre/min and ventilated with 100% oxygen after administration of Suxamethonium. This was changed to 66% Nitrous Oxide immediately after intubation for 3 minutes.

Group II was not preoxygenated and ventilated with 100% 02 after administration of suxamethonium. This was changed to 66% N20 immediately after intubation for three minutes.

Group III was not preoxygenated and was ventilated with 50% N20 in 0_2 prior to intubation, which was increased to 66% N20 immediately after intubation.

Group IV was also not preoxygenated and was ventilated with 66% N20 in 0_2 both prior to and after intubation.

Anaesthesia was induced with intravenous sodium pentothal (3-5mg/kg). The selected anaesthetic gas mixture was supplimented immediately after

loss of eyelash reflex using Magill's open circuit or Pain circuit (for paediatric age group & weight below 25 kg.) and a face mask. All the patients were ventilated with the selected gas mixture for one minute prior to intubation. Tracheal intubation was facilitated with suxamethonium. (1-1.5 mg/kg. i..v.).

An Ohmeda Biox 3700e/Simed pulse oximeter with a finger probe to monitor the areterial 0_2 saturation by pulse oximetry ($Sp0_2$) was used.

0_2 saturation was continuously monitored from patient's arrival in the opeartion room till 3 minutes after intubation.

The $Sp0_2$ values used for analysis were :

- Preinduction $Sp0_2$ when patient was breathing room air ($Sp0_2R$)
- The lowest $Sp0_2$ from injection of thiopentone to tracheal intubation ($Sp0_2I$)
- The lowest $Sp0_2$ during 3 minutes after tracheal intubation ($Sp0_2P$)

The anaesthetist was blinded to $Sp0_2$ values during intubation. Hypoxia was prospectively defined as $Sp0_2$ of less than 94% for more than 20 seconds.

It was defined as ……Mild : 91-93 %
Moderate : 86-90 %
Severe : <=85%

If the $Sp0_2$ fell below 94% for more than 20 seconds, the observer registered the fact. If the fall was below 85%, the study was terminated and the patient was ventilated with 100% 0_2. The time taken for tracheal intubation from cessation of mask ventilation to the resumption of IPPV was also noted. After 3 minutes of intubation, anaesthesia was maintained with standard technique as required for the type of surgery.

OBSERVATION AND RESULTS

In my study, 100 patients were anaesthetized. Patients were randomly divided into four groups of 25 each, depending on preoxygenation techniques. Sedative premedication was avoided in all the patients.

Table 1 : Patient Characteristics

		Group I	Group II	Group III	Group IV
Age (years)	Mean±	22.32	25.56	21.84	24.12
	S.D.	2.92	2.65	3.02	2.91
	Range	10--55	10--60	10--60	1Q—58
Weight (Kgs.)	Mean±	38	37.56	32.64	47.48
	S.D.	2.48	2.38	2.4	2.4
	Range	20--GO	20--60	20-58	22—60
Sex Ratio	M:F	10:15	12:13	15:10	21:04

Table : 2 Age and Sex Distribution of the patients.

Age		Female	%	Male	%
10-20 yrs.	57	26	45.61	31	54.38
21-30 yrs.	22	9	40.9	13	59
31-40 yrs.	6	1	16.6	5	83.33
41-50 yrs.	9	5	55.55	4	44.44
51-60 yrs.	6	1	16.6	5	83.33
Total	100	42		58	

There was no signficant difference among the groups as regards the age, weight and sex. Most of the patients were in younger age group from 10-30 years age group (79%). There were 42 female and 58 male patients in our study.

Table 3: Duration of intubation time

		Group I	Group II	Group III	Group IV
Intubation	Mean±	26.68	26.56	27.16	31.44
Time	S.D.	1.03	0.65	1.03	1.39
(seconds)	Range	20--40	20--30	20—39	20—40

There was no significant difference among the groups as regards the intubation time.

Table 4: Intubation Time distribution of the patients

	Group I	Group II	Group III	Group IV
20-25 3ec.	19	18	12	13
26-30 sec.	5	7	9	2
31-35 sec.	0	0	2	7
36-40 sec.	1	0	2	3

The intubation time was in the range of 20-40 sec, among the various groups. In the most of the patients (62%) in all the four groups, intubation was finished within 20-25 seconds.

Table 5 : Preinduction 02 Saturation : Sp02 R

		Group I	Group II	Group III	Group IV
Sp02 R	Mean±	99.32	99.4	99	98.68
	Median	99	99	98.6	99
	S.D.	0.48	0.58	0.5	0.74
	Range	99-100	98-100	99-100	97-100

Sp02 R was comparable in all the groups. No patient had Sp02 R of less than 97%.

Table 6 : $Sp0_2$ Values and change in $Sp0_2$ between $Sp0_2$ R, SpO_2 I and SpO_2P

		Group I	Group II	Group III	Group IV
SpO2R	Mean±	99.32	99.4	99	98.68
	Median	99	99	98.6	99
	S.D.	0.48	0.58	0.5	0.74
	Range	99--100	98--100	99-100	97-100
Sp02 I	Mean+	99.44	98.68	97.6	95.24
	Median	99	99	98	95
	S.D.	0.51	0.75	1.47	1.79
	Range	99--100	97--100	92--99	90--98
SpO2P	Mean±	99.48	98.76	99.76	97.88
	Median	99	99	99	98
	S.D.	0.51	0.6	0.75	0.93
	Range	99-- 100	98-- 100	97-- 100	96--99

The Sp02 I and Sp02 P in group I and II were comparable and there was no significant difference. Sp02 I in group IV was significantly lower as compared to group I and II. (95.24% against 99.44% & 98.68%). (Also see Graph No. 2). Sp02 I in group III was also significantly lower as compared to group I & group II. (97.6% against 99.44% & 98.68%). (Also see Graph No. 2). In group I and group II, the SP02 I and Sp02 P were significantly greater than the Sp02 R. (See Graph No. 1, 2 & 3,). In group III & group IV, SPO2 I was sign ficantly lower than SPO2 P.

Table 7 : Number and Degree of Hypoxaemia in patients

		Group I	Group II	Group III	Group IV
Hypoxia	Mi!d (91-93%)	0	0	1	3
	Moderate (86-90 %)	0	0	0	1
	Severe ('<85%,)	0	0	0	0
	Total	0	0	1	4

No patient in group 1 and 11 had hypoxaemia (Sp02 <94 %). One paient group III (4%) and four patients in group IV (16%) had hypoxaemia. Of these, only one patient in group IV had Sp02 of 90%. No patient in any group had a Sp02 <85% requiring the termination of the study.

Table 8: Pulse and BP changes in patients

			Group I	Group II	Group III	Group IV
Pulse Rate (_/min)	Preinduction Pulse Rate	Mean	91.28	95.04	94.4	92.84
		Range	78-I 10	80-140	77-124	72-I 18
	During Intubation Pulse Rate	Mean	89.64	89.96	88.84	89.04
		Range	80-108	80-120	71-I 10	70-I 04
	3 minutes after Intubation	Mean	90.92	94.88	94.48	93.12
		Range	79-106	78-134	78-125	73-120
Mean arterial Pressure (mm of Hg)	Preinduction Pulse Rate	Mean	86.56	89.48	90.4	93.36
		Range	78-95	80-98	80-96	80-1I6
	During Intubation Pulse Rate	Mean	87.64	90.2	91.08	93.2
		Range	80-96	82-1O0	81-96	80-120
	3 minutes after Intubation	Mean	86.68	89.4	90.48	93.12
		Range	80-95	80-98	83-96	80-1I8

There was not very significant pulse rate and blood pressure changes during study.

SUMMARY AND CONCLUSION

The study was undertaken with a view to evaluate the changes in arterial 0_2 saturation during induction of anaesthesia and endotracheal intubation with or without preoxygenation. $Sp0_2$ was measured with the help of pulse oximeter Ohmeda Biox 3700e/Simed.

I have studied 100 cases divided into four groups of 25 each according to the different methods of preoxygenation. I observed there was 16% incidence of hypoxaemia ($Sp0_2$ < 94%) when 33% 0_2 was used (group IV) and 4% incidence of hypoxaemia with 50% 0_2 (group III) as against the groups where some form of preoxygenation was performed. I observed 10% incidence of hypoxaemia in patients who received less than 100% 0_2 during suxamethonium apnoea as against none in patients who received 100% 02.

Summarizing my study and observation, I conclude that, patients be

preoxygenated to avoid hypoxaemia altogether and that ventilation with 100% 0_2 for 1 minute prior to intubation and preoxygenation for 3 minutes are equally effective in preventing hypoxaem Ia during induction.

Thus breathing 100% 0_2 before laryngoscopy and tracheal intubation leads to higher $Pa0_2$ and $Sa0_2$ values at the time of intubation than those seen after breathing air. 0_2 concentration intermediate between 100% and 21% are associated with lower value of $Pa0_2$ and $Sa0_2$ at the time of tracheal intubation.

I, therefore, suggest that serious consideration should be given to the routine use of preoxygenation before induction of any form of general anaesthesia.

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