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A STUDY OF EFFECTS OF VARYING DURATIONS OF PREOXYGENATION ON PERIPHERAL OXYGEN SATURATION IN PATIENTS UNDERGOING ELECTIVE SURGERIES UNDER GENERAL ANESTHESIA

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Abstract

Background: In this study, we wanted to study the comparison of the effects of different periods of pre-oxygenation on peripheral oxygen saturation (SpO2), to find out the optimal duration of pre-oxygenation.

Methods: This was a prospective, randomised, double-blind, controlled study that was carried out over a period of one-year in the Department of Anesthesiology and Critical Care Medicine at ACSRGMC, Nellore, involving 75 ASA I and 2 physical status patients who were posted for elective surgeries. The patients were in the age range of 20 to 50 years. The study was approved by the institutional ethics committee, and the participant's gave written informed consent.

Results: All of the GROUPs had similar demographics. The mean SpO2 levels after one minute of apnoea were 97.44±2.95 in GROUP A, 98.20±2.59 in GROUP B, and 99.96 ± 0.20in GROUP C, comparable between GROUPs A, B but revealed a significant difference between GROUPs A and B. The difference in SPO2 between GROUP B, C and GROUP A, C suggests that the drop in saturation in GROUP A, B was significant compared to GROUP C. The mean PR in GROUP A was 107.16 ±10.71, which was significantly higher than the mean pulse rates in GROUP B, C which were 101.04 ± 7.55and 99.24 ± 9.02, respectively and, GROUP B, C scores were substantially closer to baseline levels. Based on our findings, pre oxygenation for five minutes protects against saturation drops better than pre oxygenation for three minutes or 1 minute.

Conclusion: The best way to pre-oxygenate is to use the Magill circuit and tidal volume breathing technique for five minutes. Moreover, pre-oxygenation significantly postpones by three and a half

minutes the commencement of apnea-induced decrease in peripheral oxygen saturation during tidal volume breathing.

Keywords: Pre-oxygenation, Peripheral Oxygen Saturation, Elective Surgeries, General Anesthesia.

INTRODUCTION

It is generally acknowledged that preoxygenation occurs prior to anaesthetic induction and intubation. It increases the oxygen reserve in the lungs and postpones the onset of oxygen desaturation during apnea.[1] This process is known as denitrogenation since oxygen replaces the nitrogen in F.R.C (Functional Residual Capacity). As a result, the duration of desaturation-free apnea (D.A.W.D.) is prolonged. While securing the airway, the anesthesiologist is working with little time. When oxygen is saturated in the tissue, venous, arterial, and alveolar compartments, preoxygenation is finished.[2] Patients can endure a longer duration of apnoea with pre-oxygenation, which raises the safety margin between the induction of anaesthesia and the time the airway is secured. This extra time is critical in instances where patients are not or cannot be manually ventilated, such as during rapid sequence induction of anaesthesia or when difficult tracheal intubation and difficult ventilation are predicted. Anesthesiologists encounter unforeseen difficulties while performing tracheal intubations in clinical settings. For all patients, preoxygenation is necessary prior to the induction of general anaesthesia in order to address such unforeseen issues. (A.S.A.) on the challenging airway algorithm, pre-oxygenation was not mentioned. However, the A.S.A. task force on difficult airway management (2003) recommends facemask pre-oxygenation before induction as a prerequisite.[3]

Aims and Objectives

- To study the comparison of the effects of different periods of pre oxygenation on peripheral oxygen saturation (SpO2).
- To find out the optimal duration of pre-oxygenation.

METHODS

This was a prospective, randomised, double-blind, controlled study that was carried out over a period of one-year in the Department of Anesthesiology and Critical Care Medicine at ACSRGMC, Nellore, involving 75 ASA I and 2 physical status patients who were posted for elective surgeries. The patients were in the age range of 20 to 50 years. The study was approved by the institutional ethics committee, and the participant's written informed consent was obtained.

Inclusion Criteria

- Age between 20 and 50 years with A.S.A. Grading 1 and 2 elective surgical procedures under general anesthesia.

Exclusion Criteria

- Compromised respiratory, cardiac and renal functions.
- Mallampati grades 3 and 4.
- Obese patients in whom difficult intubation is anticipated.
- Hb < 10 g/dl.
- Pregnant and nursing women.
- History of smoking and respiratory tract infection within three months of surgery.

Sample Size

The three Groups comprised of 75 cases were as follows:
Patients in Group A received one minute of pre-oxygenation.
Group B: Three minutes of pre-oxygenation for the patients
Group C: Five minutes of pre-oxygenation for the patients.

Statistical Methods

Data was entered in MS Excel and analyzed using SPSS software. The results were presented as tables.

RESULTS

Demographic Data	**Group A**	**Group B**	**Group C**	**P-Value**
No. of Patients	25	25	25	-
Age (Yrs.)	35.32 ±9.41	39.12 ± 7.60	34.56 + 8.02	F= 2.122@ P=0.127
Gender (M : F)	14 : 11	12 : 13	12 : 13	$\chi 2$ = 0.427 p=0.808
ASA (I : II)	14 : 11	16 : 9	17 : 8	$\chi 2$ = 0.798 p=0.671
Mean Age, Gender, and A.S.A.				
	Height (in cms)			**Weight (in kgs)**
	Mean ± S.D.			**Mean ± S.D.**
Group A	162.96 ± 10.35			60.04 ± 8.56
Group B	158.92 ± 7.71			59.04 ± 7.48
Group C	162.92 ± 6.12			56.76 ± 5.81
F-value (p-value)	1.980@ (0.145)			1.260@ (0.289)
Mean Height and Weight				
Table 1				

Inference: Homogeneity among the means of three Groups concerning age, gender, and A.S.A. was observed.

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Pulse Rate	**Group**			**F-Value (P-Value)**
	Group A	**Group B**	**Group C**	
	Mean ± S D	**Mean ± S D**	**Mean ± S D**	
Baseline	81.16 ± 8.07	80.96 ± 8.92	84.16 ± 10.21	0.968@ (0.385)
End of Pre-oxygenation	90.24 ±6.23	86.96 ± 7.73	93.52 ± 9.64	4.212*(0.019)
End of 1 Min APNOEA	107.16 ±10.71	101.04 ± 7.55	99.24 ± 9.02	5.110*(0.008)
a. Comparison of Mean Pulse Rate in Groups A, B, and C				
@- Not significant; *significant at 0.05 level;				
Pulse Rate	**Group**		**Mean Difference**	**T-Value (P-Value)**
	Group A	**Group B**		
	Mean ± S D	**Mean ± S D**		
Base line	81.16 ± 8.07	80.96 ± 8.92	0.20	0.083@(0.934)
End of Pre-oxygenation	90.24 ±6.23	86.96 ± 7.73	3.28	1.904@ (0.069)
End of 1 Min APNOEA	107.16 ±10.71	101.04 ± 7.55	6.12	2.168*(0.035)
@- Not significant				
b. Comparison of Mean Pulse Rate in Groups A and B				
Pulse Rate	**Group**		**Mean Difference**	**T-Value (P-Value)**
	Group A	**Group C**		
	Mean ± S D	**Mean ± S D**		
Baseline	81.16 ± 8.07	84.16±10.21	3.00	1.153@ (0.255)
End of Pre-oxygenation	90.24 ±6.23	93.52 ± 9.64	3.28	1.145@ (0.258)
End of 1 Min APNOEA	107.16 ±10.71	99.24 ± 9.02	7.92	2.804**(0.007)
@ - Not significant; *significant at 0.05 level				
c. Comparison of Mean Pulse Rate in Groups A and C				
Pulse Rate	**Group**		**Mean Difference**	**T-Value (P-Value)**
	Group B	**Group C**		
	Mean ± S D	**Mean ± S D**		

Baseline	80.96 ± 8.92	84.16±10.21	3.20	1.180@ (0.244)
End of Pre-oxygenation	86.96 ± 7.73	93.52 ± 9.64	6.56	2.627*(0.012)
End of 1 Min APNOEA	101.04 ± 7.55	99.24 ± 9.02	1.80	0.910@ (0.367)
d. Comparison of Mean Pulse Rate in Groups B and C				
@ - Not significant; *significant at 0.05 level				
Table 2: Mean Pulse Rate (Beats min^{-1})				

It was examined that the pulse rate at baseline and the end of pre-oxygenation were comparable in all three Groups. But at the end of the 1-minute apnoea period, the pulse rate was much higher in Group A than in Groups B and C, which was statistically significant.

SPO2	**Group**			**F-Value (P-Value)**
	Group A	**Group B**	**Group C**	
	Mean ± SD	**Mean ± SD**	**Mean ± SD**	
Baseline	98.28±1.49	97.88±1.81	98.20±1.55	0.425@ (0.655)
End of Preoxygenation	99.56±0.65	99.16±0.75	99.88 ± 0.33	8.954** (0.000)
End of 1 Min APNOEA	97.44±2.95	98.20±2.59	99.96 ± 0.20	8.060** (0.001)
a. Comparison of Saturation between Groups A, B, and C				
@- Not significant; *significant at 0.05 level; **significant at 0.01 level				
SPO2	**Group**		**Mean Difference**	**T-Value (P-Value)**
	Group A	**Group B**		
	Mean ± SD	**Mean ± S D**		
Base line	98.28±1.49	97.88 ± 1.81	0.40	0.854@ (0.397)
End of Preoxygenation	99.56±0.65	99.16 ± 0.75	0.40	2.020* (0.050)
End of 1 Min APNOEA	97.44±2.95	98.20±2.59	0.76	0.964@ (0.334)
b. Comparison of Saturation between Groups A and B				
@- Not significant				
SPO2	**Group**		**Mean Difference**	**T-Value (P-Value)**
	Group A	**Group C**		
	Mean ± SD	**Mean ± SD**		
Base line	98.28±1.49	98.20 ± 1.56	0.08	0.186@ (0.853)
End of Preoxygenation	99.56±0.65	99.88 ± 0.33	0.33	2.191* (0.033)
End of 1 Min APNOEA	97.44±2.95	99.96 ± 0.20	2.52	4.202**(0.000)
c. Comparison of Saturation between Groups A and C				
SPO2	**Group**		**Mean Difference**	**T-Value (P-Value)**
	Group B	**Group C**		
	Mean ± SD	**Mean ± SD**		
Base line	97.88±1.81	98.20 ± 1.55	0.32	0.671@ (0.506)
End of Preoxygenation	99.16±0.75	99.88 ± 0.33	0.72	4.409** (0.000)
End of 1 Min APNOEA	98.20±2.59	99.96 ± 0.20	1.76	3.377** (0.002)
d. Comparison of Saturation between Groups B and C				
Table 3: Mean of the Differences between Measured Saturation and the Cut of Point Saturation (percentage)				

The baseline saturation values were found to be similar in all three Groups. Additionally, Group A and Group B's readings at the conclusion of pre-oxygenation were comparable. They did, however, demonstrate a statistically significant difference between Groups A and C as well as between Groups B and C. Compared to Group C, there was a statistically significant decrease in saturation throughout the one-minute apnea phase in Groups A and B. Five individuals in Group A had an accidental, statistically insignificant drop in saturation below 90%.

DISCUSSION

Random events may spur research on basic questions that are critical to patient safety, very complex topics like pre-oxygenation, and more contentious ones like the optimal pre-oxygenation duration. The necessity of pre-oxygenation being thought of as necessary to avoid adverse effects in settings with insufficient ventilation, as well as the difficulty the profession faces in establishing and advising a standard length of pre-oxygenation, served as the driving forces behind this study.

Varying durations of apnea are linked to the induction of general anaesthesia. Pre-oxygenation is a fundamental step in successfully administering general anaesthesia in this situation. When determining how much denitrogenation and oxygen storage replenishment are required, several aspects must be taken into account. These comprise the oxygen consumption of the patient, the breathing system type, the F.R.C., the FGF rate, and the inspired oxygen concentration.[4]

Adults' oxygen consumption varies widely based on their age and body size, although it usually doesn't go over 250 ml/min. Pregnancy, body size, sex, and posture all have an impact on FRC. Values of about 2 litres are common for individuals who are reclining.

The Magill system, breathing circuits, and Bain's adaptation of Mapleson D are utilized for spontaneous breathing. There is less CO2 rebreathing during spontaneous ventilation in the Magill system than in the Mapleson D system because the adjustable pressure limiting valve is detached at the patient's end and the FGF is administered proximally to the reservoir bag of the Magill system.[5]

Metrics that demonstrate its effectiveness have been the focus of pre-oxygenation monitoring strategies. To determine how successful pre-oxygenation is, one may use a variety of methods, including mass spectrometry, pulse oximeter observations, blood gas analysis to monitor PaO2, and just observing the reservoir bag move on the breathing system.[6] But the main indicator of pre-oxygenation efficacy is the drop in haemoglobin's oxygen saturation during apnea.[7]

The purpose of pre-oxygenation is to increase the body's O2 levels.

By the following equation the arterial blood O2 content is depicted:

$CaO2 = SaO2 \times Hb\% \times 1.31 + 0.003 \times PaO2.$

0.003 = Solubility co-efficient of oxygen.

1.31 = Oxygen binding capacity of hemoglobin.

PaO2 is partial pressure of O2 in mm hg.

CaO2 is blood oxygen content.

SaO2 is hemoglobin saturation.

It is evident that saturation has a more significant role in determining blood oxygen content than does PaO2.

A better indicator of the effectiveness of preoxygenation is the degree of saturation reduction.

In order to prevent fast desaturation of arterial haemoglobin during apnea induction, it is essential to pre-oxygenate as much as possible.

Maximum pre-oxygenation occurs when the tissues, as well as the arterial, venous, and alveolar systems, are fully saturated. Two important but manageable criteria that restrict maximum pre-oxygenation are not reaching an FAO2 of 0.87 and FiO2 of 1. Secondly, inadequate pre-oxygenation duration.[2]

The main reason for not reaching a FiO2 of 1 and a FAO2 of 0.87 is a leak beneath the mask that allows inspiratory entrainment of ambient air. According to Caroline Gagnon et al.[8] even a little leak beneath the face mask lowers preoxygenation. When FiO2 is achieved by a sealed breathing system, the duration plays a crucial role in establishing the maximum level of preoxygenation.

The effects of one, three, and five minutes of preoxygenation on SPO2 were studied in order to establish the optimal preoxygenation interval.

At the end of pre-oxygenation in this investigation, GROUP A had a mean SPO2 of 99.56 ± 0.65, GROUP B had a mean SPO2 of 99.16 ± 0.75, and GROUP C had a mean SPO2 of 99.88 ± 0.33. Statistically significant differences were noted across the three GROUPs. Patients with 1 minute of apnoea had a mean value of 97.44±2.95 at the end of the minute, while those with 3 minutes pre-oxygenation had a similar SPO2 value of 98.20±2.59.
With a mean SPO2 value of 99.96 ± 0.20, the patient who was preoxygenated five minutes earlier than the other two GROUPs showed statistical significance.

According to the statistical analysis, there was a statistically significant difference between the decrease in SPO2 in Groups A and B and the decrease in SPO2 in Group C. It illustrates why Group C's five-minute TVB pre-oxygenation methodology is better than the three- and one-minute methods. The time needed for proper pre-oxygenation depends on the rate at which oxygen replaces nitrogen in the alveolar compartment and the rate at which oxygen is delivered to the tissue.
The primary cause of the early increases in stored oxygen during pre-oxygenation was the removal of N2 from the lungs, which was followed by the replenishment of oxygen. This process, which follows an exponential curve, reaches 80% competitiveness after one minute and completes denitrogenation in around seven minutes; the more F.R.C., the more oxygen that is stored.

One of the functional characteristics of the Magill system is its early expiration gas retention. On the other hand, the "alveolar component" of late expiration is eliminated from the respiratory circuit.
It is effective at removing CO2, but during the pre-oxygenation phase, dead space gas (N2) is inhaled again. This persists over several breaths because new and dead space gases are mixed together. This issue occurs even when higher than recommended O2 flow rates are used to prevent rebreathing and to make sure the reservoir bag is pre-filled with O2 before beginning pre-oxygenation.[9]

To achieve optimum denitrogenation, which indicates the inefficiency of preoxygenation by one minute, the breathed gas must have zero N2 tension. Long preoxygenation lasting more than a minute or a few forced ventilation breaths don't seem to have much of an impact on lung denitrogenation or arterial saturation. In one research of older people (spO2 93%), it took 3 minutes over 5 minutes to keep spo2 steady in the presence of persistent apnea; in another, it took 7 minutes (spO2 90%). This suggests that the duration of time it took appears to be much longer. Finally, it was said that tissue storage was most likely necessary.[10]
To maintain metabolic VO2 during the apnea phase, O2 is taken from FRC and supplied to the circulation at a flow rate of 250 ml/min. The blood's greater solubility of CO2 causes it to expand the alveolar space at a rate of 10 ml/min. Consequently, the blood's net gas flow rate was 240 ml/min. The alveoli generate an atmospheric pressure that is lower than usual, which causes room air, which is mostly composed of 21% O2 and 79% N2, to be drawn into the lungs.
The F.R.C will quickly absorb nitrogen by diffusion from bodily tissues in addition to nitrogen, which dilutes O2. This is how the relative importance of tissue storage is explained.[11]
Hb becomes completely saturated at the beginning of the oxygenation process, and spo2 rises to 100% in 15 seconds. Since monitoring SpO2 only supplies 36 milliliters of oxygen to arterial blood, it is an inadequate endpoint for full preoxygenation. There is more dissolved oxygen in the plasma and physiological tissues, which accounts for the increases in o2 storage in the last few minutes before oxygenation.
Even if Henry's rule is valid and the gas partition coefficient is close to the gas water coefficient, it is challenging to estimate the tissue's oxygen storage capacity. For example, breathing in oxygen for three minutes will raise tissue oxygen reserves from 25 ml to 377 ml.

Since oxygen stays in various organs for different lengths of time-4 seconds in the thyroid and 165 seconds in skeletal muscle-and assuming a blood volume of 5.4 litres and 6.5 litres of CO, only 77% of the blood is exposed to high levels of oxygen in the lungs during a 3-minute breathing session when

inhaling 100% oxygen. After just three minutes, saturation of venous blood finally increases o2 storage by 216 ml, with the assumption that the metabolic VO2 of various organs remains constant. This explains why preoxygenation for five minutes is far more effective than for three or one minute. Our results agreed with those of Sanjay et al.[12] They found that in terms of four V.C.Bs, one minute of apnea during the induction phase decreased SPO2 to 77 percent 3.26 in those who did not get preoxygenation, compared to 87 percent 1.1 in those who did. Similarly, following three minutes of pre-oxygenation, an individual's saturation levels decreased. 96 percent 0.75 in the Group that received 5 minutes of pre-oxygenation, and 99 percent 0.67 in the Group that did not.

But Baraka et al. observed that preoxygenation by 8 D.B.T. for 1 minute delays the onset of apnea-induced arterial/venous Hb oxygen desaturation for 3 minutes, while TVB does not.[13]
Despite the fact that their study did not measure arterial pressure or end tidal CO2 concentrations, it is reasonable to assume that the hyperventilation brought on by 8 D.B.T. caused CO2 levels to drop below the normal range, which in turn produced nitrogen washout and respiratory alkalosis.
Therefore, rather than being the result of the patients' inherent defences, the extended onset of desaturation in the preceding study may have been caused by a shift to the LEF The current study comes to the conclusion T in the ODC curve.[14]

Numerous compensatory mechanisms can be used when arterial oxygenation drops. These include a rightward shift in the ODC to maintain venous PvO2 stability and modifications in CO to provide oxygenated blood to vital organs. In humans, assuming maximal cerebral vasodilation, the lowest tolerable PaO2 is around 30.4 mmHg. Consequently, we selected a 90% SpO2 study endpoint, which causes a negligible shift in PaO2, even if PaO2 makes up a very tiny portion of the total oxygen content.[15]

CONCLUSION

The current study comes to the conclusion that the most efficient way of pre-oxygenation is to use the Magill circuit for five minutes of tidal volume breathing technique. Moreover, pre-oxygenation significantly postpones by three and a half minutes the commencement of apnoea-induced decreases in peripheral oxygen saturation during tidal volume breathing.

REFERENCES

1. Baraka A, Ramez Salem M, Benumof's airway management. 2nd edn. Mosby Elsevier Publications 2007:303-12.
2. Benumof JL. Pre-oxygenation: best method for both efficacy and efficiency? The Journal of the American Society of Anesthesiologists 1999 Sep 1;91(3):603-5.
3. Practice guidelines for management of difficult airway. An updated report by American Society of Anaesthesiologists Task Force on management of difficult airway. Anaesthesiol 2003;98:1269-77.
4. Berthoud M, Read DH, Norman J. Pre-oxygenation-how long? Anaesthesia 1983;38:103-7.
5. Taha S, Khatib MEI, Sayyid SS, Dagher C, Chehade MJ, Baraka A. Pre-oxygenation with the Mapleson D system requires higher oxygen flows than Mapleson A or circle systems. Can J Anesth 2007;54(2):141-5.
6. Campbell IT, Beatty PC. Monitoring pre-oxygenation. Br J Anaesth 1994;72(1):3-4.
7. Lane S, Saunders D, Schofield A, Padmanabhan R, Hildreth A, Laws D. A prospective, randomized controlled trial comparing the efficacy of pre- oxygenation in 20 degree head-up v/s supine position. Anaesthesia 2005;60(11):1064-7.
8. Gagnon C, Fortier LP, Donati F. When a leak is unavoidable, pre-oxygenation is equally ineffective with vital capacity or tidal volume breathing. Can J Anesth 2006;53(1):86-91.
9. Ooi R, Joshi P, Soni N. A high flow semi-open system for pre- oxygenation: an evaluation. Br J Anaesth 1992;68(1):39-42.

10. Pre-oxygenation: physiology and practice. Lancet 1992;339(8784):31-2.
11. Taha SK, Siddik-Sayyid SM, El-Khatib MF, Dagher CM, Hakki MA, Baraka AS. Nasopharyngeal oxygen insufflation following pre-oxygenation using the four deep breath technique. Anaesthesia 2006;61(5):427-30.
12. Sanjay OP, Raipet MA. Use of pulse-oximeter to study the effects of varying durations of pre-oxygenation. Indian J Anaesth 2004;48(3):201-3.
13. Baraka AS, Taha SK, Aouad MT, El-Khatib MF, Kawkabani NI. Pre-oxygenation: comparison of maximal breathing and tidal volume breathing techniques. Anesthesiology 1999;91(3):612-6.
14. Nimmagadda U, Chiravuri SD, Salem MR, Joseph NJ, Wafai Y, Crystal GJ et al. Pre-oxygenation with tidal volume and deep breathing techniques: the impact of duration of breathing and fresh gas flow. Anesth Analg 2001;92(5):1337-41.
15. McCarthy G, Elliott P, Mirakhur RK, McLoughlin C. A comparison of different pre-oxygenation techniques in the elderly. Anaesthesia 1991;46(10):824-7.