"A Study of Effects of Varying Durations of Pre-**Oxygenationon Peripheral Oxygen Saturation in Patients** Undergoingelectivesurgeries under General Anaesthesia''

Dr MUDE NAGE RAMESH KUMAR¹, Dr MOHANA KRISHNA REDDY EGA BOJJIREDDY GARI²

¹(Department of Anesthesiology, S.V. Medical College/Dr N. T. R. University of Health Sciences, India) ²(Department of Anesthesiology, S.V. Medical College/Dr N. T. R. University of Health Sciences, India)

Abstract:

Background: In clinical practice, anesthesiologists face unexpected challenges with tracheal intubation. Preoxygenation is essential for all patients before general anesthesia induction to help with such unanticipated problems. (A.S.A.) on difficult airways, the algorithm did not mention pre-oxygenation. Despite this, the A.S.A Task Force on Difficult Airway Management (2003) recommends facemask pre-oxygenation before induction as a prerequisite.

Materials and Methods: Study Design: A tertiary care hospital-based Prospective, Randomized double-blind Controlled study. Study Subject: 75ASAI and 2 physical status patients posted for elective surgeries, of age GROUP of 20- 50 years. Study Setting: Department of Anesthesiology and critical care medicine at SVRRGGH.StudyPeriod: 1 yearduration from the time of IECA pproval. Source of Data: Patient admitted at SVRRG.G Hospital for various elective surgeries Sample size: 75 cases were divided into 3 Groupings, as follows. GROUP A: patients Receiving pre-oxygenation for one min. GROUPB: patients receiving preoxygenation for three minutes GROUP C: patients Receiving pre-oxygenation for five mins.

Results The demographic data were analyzed using the ANOVA test. The observed datahavebeen analyzed usingtheUnpaired student t-test. All data are presented as mean +standard deviation. Based on the p-value, the result is stated as significant or not significant.

Key Word: Trachéal intubation, pré-oxygénation, General Anesthésia.

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I. Introduction

The process of Preoxygenation before anesthetic induction and intubation is widely accepted. It raises the lungs oxygen reserve and delays the beginning of oxygen desaturation during apnea,¹ because the nitrogen in functional residual capacity (F.R.C.) is replaced by oxygen, this method has dubbed de-nitrogenation. Therefore the duration of apnoea without de-saturation (D.A.W.D.) is increased. For the anesthesiologist it is precious time while securing the airway. The process of Preoxygenation is complete when the alveolar, arterial, tissue, and venous compartments are saturated with oxygen.

II. **Material And Methods**

Study design: A Tertiary care hospital based Prospective, Randomized double blind Controlled study. StudySubject: 75 ASA I and 2 physical status patients posted for elective surgeries, of age GROUP of 20- 50 years

Study Setting: Department of Anesthesiology and critical care medicine at SVRRGGH. Study Period: 1 year duration from the time of IEC Approval.

Source of Data: Patient admitted at SVRRGG Hospital for various elective surgeries Sample size: 75 cases were divided into 3 Groupings, as follows.

GROUP A: patients receiving pre-oxygenation for one minutes. GROUPB: patients receiving pre-oxygenation for three minutes GROUP C: patients receiving pre-oxygenation for five minutes. **Inclusion criteria:**

Agebetween20and50yearswithA.S.A.Grading1&2elective surgical procedures under general anesthesia.

Exclusion criteria:

- Compromised respiratory, cardiac and renal functions.
- Mallampattigrades3and 4
- Obese patients in whom difficult intubation is anticipated.
- Hemoglobin less than 10g/dl
- History of smoking and
- Respiratory tract infection<3monthsof surgery. Pregnant and lactating women.

Procedure methodology

In an operating theater on the day of surgery, after securing an intravenous line A.S.A. standard monitors such as Pulse oximeter, electrocardiograph (E.C.G.), non- invasive blood pressure monitor (N.I.B.P.) were attached, and baseline pulse rate, SpO2, and systolic & diastolic blood pressure values were recorded on participants breathing room air.

Pre oxygenation was performed utilizing face mask and a Magill breathing circuit during the period allotted based on the participant's GROUP. At the end of pre-oxygenation, the pulse rate,SpO2, and systolic and diastolic blood pressure data were recorded.AlltheGROUP'spatientsreceivedpre-medicationofInj.Glycopyrrolate0.005mgkg¹,Inj.Midazolam0.04mgkg¹ and Inj. Fentanyl 1to2mg/kg¹ Induction was done with the Thiopentone sodium3-5mg/kg¹ followed by intubation with depolarizing muscle relaxant Suxamethonium1.5mg/kg-

The participants were examined while kept apnoeic for 1 minute, with oxygen saturation, HR changes, and systolic and diastolic blood pressure. A saturation fall less than 90% was considered significant. When the saturation falls below 75%, rescue breaths with100% oxygen given. A laryngoscopy was done. Attend of one minute, participants were intubated with an adequate-sized cuffed endo-tracheal tube, and the lungs were ventilated with100%02untilperipheraloxygensaturationreached100%.

Statistical analysis

The demographic data were analyzed using the ANOVA test. The observed data have been analyzed using the Unpaired student t-test. All data are presented as mean + standard deviation. Based on the p-value, the result is stated as significant or not significant.

Table 10.1. Mean Age, Ochuer, and A.S.A.					
Demographic Data	Group A	Group B	Group C	P-value	
No. of Patients	25	25	25		
Age (Yrs)	35.80+9.98	37.44+10.17	34.52 +8.04	F=0.600 P=0.551	
Gender (M:F)	14:11	12:13	12:13	x2=0.427 p=0.808	
ASA (I :II)	14 11	16:9	17:8	x ² =0.798 p=0.671	

III. Result Table No.1: Mean Age, Gender, and A.S.A.

Graph No.1:Age wise distribution







Table No.2: Mean Height and Weight

	Height(in Cms)	Weight(in kgs)
	Mean + S.D.	Mean + S.D.
Group A	163.3619.59	59.7218.55
Group B	159.12+7.94	58.60+7.38
Group C	163.64+4.86	56.24+5.43
F-value (p-value)	2.695@ (0.074)	1.508 (0.228)

Graph No.3: Height

Graph No.4: Weight



Table No.3: Mean pulse rate (Beats/min¹)

		Group		
Pulse Rate	Group A	Group B	Group C	F-value (p-value)
	Mean + SD	Mean + SD	Mean + S I	5
Baseline	81.16 + 8.07	80.961 8.92	84.16110.2	0.968 (0.385)
End of Pre- oxygenation	90.16 + 6.28	87.281 8.19	93.7219.77	3.863* (0.025)
End of 1 Min APNOEA	107.80+ 11.71	101.72+8.60	99.32+9.4	4.783* (0.011)



Graph No.5: Comparison of mean pulse rate in groups A, B, and C

Comparison of mean pulse rate in group A and B

		Group	Mean Difference	t-value (p-value)
Pulse Rate	Group A	Group B		, and the second s
	Mean + SD	Mean + S D	_	
Baseline	81.16+8.07	80.96+8.92	0.20	0.083
				(0.934)
End of Pre- oxygenation	90.16+6.28	87.28+8.19	2.88	1.395
	,			(0.169)
End of 1 Min APNOEA	107.80+ 11.71	101.72+ 8.60	6.08	2.092*
			0.00	(0.042)

GraphNo.6: Comparison of mean pulse rate in group A and B



Comparison of mean pulse rate in group A and C

	Group			
Pulse Rate	Group A	Group C	Mean Difference	t-value (p-value)
i uise ivae	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	90.16+6.28	93.72+9.78	3.56	1.532 (0.132)
End of 1 Min APNOEA	107.80+ 11.71	99.32+9.41	7.56	2.823** (0.006)



Comparison of mean S.B.P. in groups A, B, and C

	Group A	Group B	Group C	F-value (p-value)
S.B.P.	Mean + SD	Mean + S D	Mean + S D	
Baseline	114.00 +6.06	113.20+6.25	116.88 +6.71	2.326
End of Pre- oxygenation	120.72+5.53	120.60+6.00	123.88 +6.58	2.361 (0.102)
End of 1 Min APNOEA	127.36+5.35	127.00+9.15	124.92 +6.05	0.874

Graph No.9:Comparison of mean S.B.P.in groups A, B, and C



Comparison	of mean	S.B.P.	in group	A and B
			O F	

		Group		t ()
S.B.P.	Group A	Group B		t-value (p-value)
	Mean + SD	Mean + S D		
	114.00+	113.20+		0.460
Baseline			0.80	
	6.06	6.25		(0.648)
End of Pre- oxygenation	120.72+	120.60+		0.074
			0.12	
	5.53	6.00		(0.942)
End of 1 Min	127.36+	127.00+ 9.15		0.170
			0.36	
APNOEA	5.35			(0.866)



Graph No.10: Comparison of mean S.B.P .in group A and B



	-		-	
	Group			
	Group A	Group C	Mean Difference	t-value (p-value)
S.B.P.	Mean + SD	Mean + SD		
	114.001 6.06			1.593
Baseline		116.88+6.71	-2.88	
				(0.118)
End of Pre- oxygenation	120.721 5.53			1.838
		123.88+6.58	-3.16	
				(0.072)
End of 1 Min APNOEA	127.36+ 5.35			1.511
		124.92 +6.05	2.44	
				(0.137)

Graph No.11: Comparison of mean S.B.P. in group A and C



Com	oarison	of mean	S.B.P.in	groups	B and C
r				8r-	

	G	roup		
CDD	Group B	Group C	Mean Difference	t-value (p-value)
5.D.F.	Mean + SD	Mean + SD	Difference	
Baseline	113 2016 25	116 8816 71	-3.68	2.006
Busenne	115.2010.25	110.0010.71	5.00	(0.060)
End of Pre- oxygenation	100 (0) (00	102.00.050	2.00	1.842
	120.60+ 6.00	123.88+6.58	-3.28	(0.072)
End of 1 Min APNOEA	107.00.015	124.02 6.05	2.00	0.948
	127.00+9.15	124.92 +6.05	2.08	(0.348)



Graph No.12: Comparison of mean S.B.P. in groups B and $\,C$

		F (()		
DBP	Group A	Group B	Group C	
	Mean + SD	Mean + SD	Mean + SD	
Baseline	72.32+6.07	71.32+4.23	73.40+3.61	1.197
				(0.308)
End of Pre-oxygenation	78.48+5.72	80.16+8.18	78.24+5.11	0.653
				0.502
End of 1 Min APNOEA	79.56+16.43	82.20+6.43	79.68+3.50	(0.607)

 Table5: Mean for the Diastolic blood pressure (mm of Hg)

Graph No.13: Comparison of mean DBP in groups A, B, and C



	Group			
חחת	Group A	Group B	Mean Difference	t-value (p-value)
DBP	Mean + SD	Mean + S D		
	50.00 6.05	51.00.1.00	1.00	0.676
Baseline	72.32+6.07	71.32+4.23	1.00	(0.503)
End of Pre- oxygenation	50.40.5.50	00.15.0.10	1.60	0.842
	78.48+5.72	80.16+8.18	-1.68	(0.404)
End of 1 Min APNOEA				0.738
	79.56+16.43	82.20+6.43	-2.64	(0.464)

Comparison of mean DBP in group A and B



Comparison of mean DBP in group A and C

	I	<u> </u>		
	Group		D 100	
DBP	Group A	Group C	Mean Difference	t-value (p-value)
	Mean + SD	Mean + S D		
Baseline	72.3216.07	73.4013.61	1.08	0.764
				(0.448)
End of Pre-oxygenation	78.4815.72	78.2415.11	0.24	0.157
				(0.876)
End of 1 Min	79.56+ 16.43			0.036
		79.68+3.50	-0.12	
APNOEA				(0.972)

Graph No.15: Comparison of mean DBP in group A and C



	comparison of n	ican DD1 in grou	ips D and C	
	Gre	oup		
	Group B	Group C	Mean	t-value (p-value)
DBP	Mean + S D	Mean + S D	Difference	
				1.871
Baseline	71.3214.23	73.4013.61	2.08	
				(0.067)
End of Pre- oxygenation				0.995
	80.16+8.18	78.24+5.11	1.92	
				(0.324)
End of 1Min APNOEA				1.601
	82.20+6.43	79.68+3.50	2.52	
				(0.116)

Comparison of mean DBP in groups B and C

Graph No. 16: Comparison of mean DBP in groups B and C



Comparison of saturation between groups A, B, and C

		Group		
SPO2	Group A	Group B	Group C	F-value (p-value)
	Mean + SD	Mean + SD	Mean + SD	
Baseline	98.2811.49	97.8811.81	98.2011.55	0.425
End of Dragonation				(0.655)
End of Pre- oxygenation	99.56+0.65	99.16+0.75	99.88+0.33	(0.000)
End of 1 Min APNOEA	97.48+2.96	98.20+2.60	99.88+0.33	7.283** (0.001)

Graph No.17: Comparison of saturation between groups A, B, and C



	G	Group		
SDOJ	Group A	Group B	Mean Difference	t-value
SP02	Mean + SD	Mean + SD		(p-value)
Baseline	98.2811.49	97.8811.81	0.40	0.854
End of Pre- oxygenation	99 5610 65	99 161 0 75	0.40	2.020*
			0.10	(0.050)
End of 1 Min APNOEA	97.48+2.96	98.20+2.60	0.72	0.914 (0.365)

Comparison of saturation between group A and B





Comparison of saturation between group A and C

	Group			
(The s	Group A	Group C	Mean Difference	t-value (p-value)
SPO2	Mean+ SD	Mean+ SD		
Baseline	98.28+1.49	98.20+1.56	0.08	0.186 3 (0.853)
End of Pre- oxygenation	99.56+0.65	99.88+0.33	0.33	2.191* (0.033)
End of 1 Min APNOEA	97.48+2.96	99.88+0.33	-2.40	4.029** (0.000)

Graph No.19: Comparison of saturation between group A and C



	Group			
	Group B	Group C	Mean Difference	t-value (p-value)
SPO2	Mean + SD	Mean + SD		
				0.671
Baseline	97.88+1.81	98.20+1.55	-0.3	2
				(0.506)
End of Pre- oxygenation				4.409**
	99.16+0.75	99.88+0.33	-0.7	2
				(0.000)
End of 1 Min APNOEA				3.207**
	98.20+2.60	99.88+0.33	-1.6	8
				(0.002)







It was observed that the baseline saturation values were comparable among the three groups. Furthermore, the values at the end of pre-oxygenation were similar between Group A and Group B. Still, they showed a statistically significant difference between Group B and Group C and between Group A and Group C. Fall in saturation during one minute apnoea period was statistically significant in Group A and B in comparison to Group C. In group A, five participants showed a fall in saturation below 90%, which was incidental and statistically insignificant.

IV. Discussion

Random events can lead to the study of primary concerns that are both vital to patient safety and extremely complex topics like pre-oxygenation, as well as more contentious issues like the best pre-oxygenation duration. The call for pre- oxygenation to be regarded crucial in avoiding adverse occurrences in settings with difficulties in ventilation, as well as the profession's issue in deciding and prescribing a standard period of pre-oxygenation, were the driving forces behind this research.

Induction of general anesthesia is associated with variable periods of apnoea. In this scenario, preoxygenation is a principle step of providing general anesthesia safely.

Many factors must be considered in the assessment of replenishment of oxygen stores and amount of de-nitrogenation needed. These include the patient's oxygen consumption, the F.R.C., the FGF rate, inspired oxygen concentration, and the type of breathing system used²⁰. Oxygen intake of adults differs greatly depending on their age and body size, although it is unlikely to exceed 250 ml min-1. Posture, sex, body size, and pregnancy all affect F.R.C. In reclined adults, values of around 2 liters are typical.²⁰

Breathing circuits, the Magills ystem and Bain's modification of Mapleson-D used for spontaneous breathing. The Magill system is having minimal re-breathing under spontaneous ventilation than Mapleson-D system as the adjustable pressure limiting valve isolated distally at the patient end, and the FGF is delivered proximally near the reservoir bag of the Magill system, resulting in less re-breathing of CO2.^{*8}

Pre-oxygenation monitoring techniques have centered on metrics that reflect its efficacy. The efficacy of pre oxygenation can be measured using end-tidal CO2 concentration. alone or more important gases (oxygen, nitrogen, or both), pulse oximeter observations, blood gas analysis to monitor PaO2, mass spectrometry, or simply observing movement of reservoir bagon the breathing system.⁴⁷

However, the primary measure of pre-oxygenation efficiency is the decrease in oxygen saturation in hemoglobin during apnoea.³⁶

Goals of Pre oxygenation is to elevate the O2 content in the body. By the following equation thearterialblood02contentisdepicted: CaO2=SaO2•Hb%•1.31+0.003 PaO2 0.003=Solubility co-efficient of Oxygen

1.31= Oxygen binding capacity of Hemoglobin PaO2is partial pressure of 02in mm hg

CaO2isblood oxygen content

SaO2ishemoglobinsaturation.

It is clear that the contribution of saturation is important than the contribution of PaO2 in determining the 02content of the blood.

The degree of decrease in saturation is a more suitable measure of the efficacy of pre oxygenation.

Various factors can cause rapid de-saturation in arterial hemoglobin at the time of apnoea it's crucial to preoxygenate as much as possible before induction.

When the alveolar, arterial, and venous systems, as well as the tissues, are totally saturated, maximum preoxygenation is achieved. Failure to reach an FiO2of 1 and FAO2of 0.87, as well as insufficient pre oxygenation time, are two significant yet controllable factors that limit maximal pre-oxygenation.²

A leak behind the mask, permitting inspiratory entrainment of room air, is the primary cause of failure to attain aFAO2 of 0.87 and FiO2 of 1.Caroline Gagnon et al.³⁷ discovered even a minor leak behind the face mask leads reduced pre oxygenation. Once FiO2 is obtained using a sealed breathing system, the time length becomes an important factor in determining maximal pre oxygenation.

In order to determine the ideal pre-oxygenation period, the effects of 1 minute, 3 minutes, and 5 minutes of pre oxygenation on SPO2.

In this study the end of pre-oxygenation, in GROUP-A a mean SPO2of 99.56+0.65andGROUPB99.16+0.75andGROUPC99.88+0.33was

observed and a statistically significant difference among 3 GROUPs. At the end of one minute of apnoea, patients with 1 min had a mean value of 97.48 +2.96, and 3 minutespreoxygenationhadaSPO2valueof98.20+2.60 and were comparable.

Patient pre oxygenated with 5 minutes had a mean SPO2 value of 99.88 +0.33, superior to the other two GROUPs and significant statistically.

The statistical analysis revealed, decrease in SPO2 in GROUPs A and B when compared to the decrease in SPO2 in GROUP C, is statistically significant. It demonstrates that the five-minute TVB pre-oxygenation technique used in GROUP C is superior to the three-minute and one-minute procedures.

The pace at which alveolar compartment Nitrogen displacement by oxygen, as well as the rate at which tissue oxygen stores are supplied, determines the time required for appropriate pre-oxygenation.

Early gains in stored oxygen during pre-oxygenation are mostly due to N2 displacement from the lungs, which is followed by oxygen replacement. Following an exponential curve, this process is80percent competitive after one minute and takes around seven minutes to accomplish total de-nitrogenation; the larger the F.R.C., the more oxygen is stored.³⁶

The Magill system's functional properties include the retention of early expiration gas. The 'alveolar component' of late expiration, on the other side, is expelled from the breathing circuit.

In terms of CO2 removal, it is efficient but there is re-inhalation of dead space gas (N2) at the time of pre oxygenation phase. Due to the mixing of fresh and dead space gas, this remains during multiple breaths. In spite of using higher O2flow rates than recommended to avoid re breathing and to ensure that reservoir bag was pre filled with O2 before starting of pre oxygenation, this phenomena happens.⁴⁸

A zero N2 tension in the inhaled gas is necessary for optimal de-nitrogenation which represents the in efficiency of pre oxygenation by 1 minute. Even- though lengthy pre-oxygenation over1minute or few VC breaths appears to have little effect on arterial saturation or lung de-nitrogenation.

The amount of time it bought in terms of keeping spo2 stable in the presence of protracted apnea seems significantly longer ranging from 3 minutes over 5 minutes in a study, of elderly patients (spO2 93%), 7 minutes, in other study (spO2 90%). it finally stated that tissue storage was almost certainly required.⁴

At the time of apnea period, to maintain metabolic vo2, 02is extracted from F.R.C.in to the circulation at the flow rate of 250mI/min. as solubility of CO2 is higher in the blood itaddsathrateof10mI/min to the alveolar space. Therefore resulting in net gas flow rate 240mI/min in the blood. A below normal atmospheric pressure is created in the alveoli and the room air which contains 79%N2and21%⁰2 is pulled into the lungs.

In addition to nitrogen, the F.R.C. will rapidly acquire nitrogen diffusing from body tissues, which cause 0_{2in} the F.R.C. to be diluted The relative relevance of tissue storage is described in this way.⁵⁰

At the start of oxygenation process, Hb gets fully saturated and spo2 climbs to 100 % in 15 seconds. Monitoring SpO2 is an insufficient end goal for complete pre oxygenation because it only delivers 36 ml to arterial blood. The advances in o2 storage in the final pre-oxygenation minutes are due to more amount of dissolved o2 content in the plasma and physiological tissues.⁴⁷

It is difficult to determine tissue o2 storage even though Henry's rule holds true and the partition co efficient of gases approaches the gas water co efficient, inhaling oxygen for 3 minutes will increasetissueoxygenreservesfrom25 ml to 377 ml.

As o2 dwells in different organs for varying amounts of time, ranging from 4 seconds (thyroid)to 165 seconds(skeletal muscle), and assuming a6.5 liters CO& a5.4 liters of blood volume, when breathing 100% o2 for 3 minutes, only 77% of the blood that is exposed to high Fio2 levels in the lungs. Believing that metabolic vo2 of different organs remains constant, saturation of venous blood eventually raises o2 storage by 216 ml after only 3 minutes. This explains why 5 minutes of pre oxygenation is highly efficient than 3 minutes or 1 minute.⁴⁷

Our findings were comparable to those of Sanjayetal.³⁴.Theydiscovered that 1 min of apnoea at the time of the induction phase reduced SPO2to 77percent 3.26 in those who did not receive pre oxygenation, compared to 87 percent 1.1 in those who received in terms of four V.C.Bs. Similarly, individual's saturation levels dropped after 3 minutes of pre-oxygenation.

96percent 0.75 among those who had5min of pre-oxygenation, compared to 99 percent 0.67 in those who did not.

However, compared to TVB for three minutes, pre-oxygenation by 8D.B.T.in1 min prolongs the beginning of apnoea induced arterial/venous Hb oxygen de-saturation according to Baraka et al.³⁰.

Even though no end tidal co2 concentrations or arterial measurements were taken in their research, it is reasonable to assume that HYPERVENTILATION caused by 8 D.B.T, cause fall in co2 levels below normal range resulted and nitrogen washout, resulting in RESPIRATORY ALKALOSIS.

Thus, in the previous investigation, the prolonged onset of de-saturation could have been due to shift to LEFT in the ODC curve rather than any natural protection for the patients.³²

When arterial oxygenation falls, several compensation mechanisms occur. These include changes in CO to deliver oxygenated blood to important organs and a right-ward shift in the ODC to keep venous PvO2 constant. In humans, the lowest acceptable PaO2 is around 30.4 mmHg, assuming maximum cerebral vaso-dilation. As a result, we choose a research endpoint of 90% SpO2, that results in a minor change in Pao2, despite the fact that PaO2 is just a small part of overall content of oxygen.²⁸

LIMITATIONS

- 1. Sample size was a limiting factor as the duration of the study was limited to 12 months.
- 2. A single center based study
- 3. Based on single observer study

V. Conclusion

The present study concludes that pre-oxygenation for 5 minutes of tidal volume breathing technique using Magill circuit is the most effective method of pre- oxygenation. Furthermore, pre-oxygenation considerably delays the onset of apnoea- induced drops in peripheral oxygen saturation for three and one minutes of tidal volume breathing.

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