Comparison of two Methods of Preoxygenation in Elderly Patients Undergoing Surgery under General Anaesthesia

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Abstract
Background and Aims: Preoxygenation is performed routinely as an integral part of induction for general anaesthesia to reduce the risk of hypoxia for planned or unplanned periods of apnoea during the induction of anaesthesia. 3-5 minutes of tidal volume preoxygenation is the most preferred technique of choice for preoxygenation. This technique may not be feasible in emergency situations. Faster method of preoxygenation may be more useful and a better alternative in certain clinical conditions like rapid sequence induction, life saving emergencies etc. This study aims to compare the effectiveness between two methods of preoxygenation i.e. 8 vital capacity breath (VCB) in 1 minute and Tidal volume breathing (TVB) for 3 minutes and in elderly patients, by assessing changes in arterial oxygen tension (PaO₂) and apnoea induced desaturation time.

Material and Methods: This prospective randomised controlled double blind study was conducted with 60 elderly patients (age 60-70 yrs) and were divided into 2 groups-Group VCB and Group TVB. After preoxygenation with either method, anaesthesia was induced in all patients. Mask ventilation was not done and following intubation endotracheal tube was kept open to atmosphere. The time taken for the patients to desaturate to 90% was noted and immediately ventilation was resumed. Arterial blood gas samples were taken while patients were breathing room air, immediately after preoxygenation and at 90% desaturation.

Results: After preoxygenation group VCB had significant higher PaO₂ values than group TVB( 451.50 ± 68.57 vs 400.19 ± 63.69). The apnoea induced desaturation time for group VCB was 494.77 ± 18.92 secs and group TVB was 506.67 ± 22.70 secs, which was statistical insignsignificant.

Conclusion: The eight vital capacity breath technique may be used as an alternative to the traditional technique of preoxygenation in elderly patients undergoing rapid-sequence induction of anaesthesia, as well as in other emergency situations.

Keywords: Preoxygenation, Vital capacity breath, Tidal volume breath.

Introduction
General anaesthesia enables surgery to be performed by inducing reversible loss of consciousness. A significant and feared problem during induction of anaesthesia is failure to secure the airway thereby not being able to oxygenate the
Oxygen desaturation, is often a harbinger of serious complications such as cardiovascular collapse, anoxic brain injury, and death. It is imperative to attenuate this risk by optimizing preoxygenation prior to intubation. Hence pre-operative oxygenation is a time honoured technique and to prevent apnoea-induced hypoxia during induction of anaesthesia it has become a standard practice of care.

The principle is to maintain haemoglobin saturation despite ongoing oxygen consumption during apnoea by providing a reservoir of 95% oxygen (assuming an obligatory 5% alveolar carbon dioxide) in the patient's lungs for planned or unplanned periods of apnoea during the induction period.

Several studies have demonstrated that most subjects are optimally oxygenated after 3 minutes of normal tidal volume breathing of 100% O₂ using O₂ flow of 5 L/min through standard breathing systems, which is the traditional method of preoxygenation. However, in certain clinical conditions like rapid sequence induction, full stomach, uncooperative patients who cannot tolerate face mask, life saving emergencies, etc where it may be impractical to preoxygenate for longer duration, faster method of preoxygenation may be more useful and better alternative.

Time sparing methods of preoxygenation have also been described. Using a series of four vital capacity breaths of 100% O₂ over a 30 second period, a high arterial PaO₂ (339 mmHg) can be achieved, but the time to desaturation remains shorter than with traditional techniques. A modified vital capacity technique, wherein the patient is asked to take eight deep breaths in a 60-second period, shows promise in terms of prolonging the time to desaturation. But this technique is yet to be studied in elderly age group. Hence we studied to compare 8 vital capacity breath in 1 min with 3 min tidal volume breathing to provide comparable protection against hypoxia in elderly patients when apnoea follows the induction of anaesthesia, by assessing changes in arterial partial pressure of oxygen (PaO₂) and apnoea induced desaturation time (DAWD).

**Method**

After obtaining approval from the institutional ethics committee, this prospective randomized double blind study was carried out on a total of 60 patients of either sex between the age of 60 – 70 years, belonging to American Society of Anesthesiologists (ASA) physical status I and II with normal preoperative spirometry, scheduled for undergoing elective surgery under Surgery department. The study period was 2 year from November 2017 to October 2019. The patients were subjected to detailed clinical examination and routine investigations to exclude any systemic disorder. Patients with ASA grade III and IV, with associated co-morbidities like respiratory, cardiovascular, hepatic, renal disease and endocrine disease, with psychiatric disorder, obesity (BMI >35) or patients anticipated for difficult ventilation or intubation were excluded from the study.

Patients were randomly allocated to one of the two groups, group VCB and group TVB, by another team of anaesthesiologists using computer generated sequence of random numbers. During the pre-anesthetic check up, patients were properly evaluated and their written and informed consents were obtained. All patients received premedication with Tab. Diazepam 0.2 mg kg⁻¹ body weight and Tab Ranitidine 3 mg kg⁻¹ body weight orally on the night before. Patients were asked to stay nil per oral as per the guidelines.

In the operation theatre, a peripheral venous access was achieved using an 18 Gauge catheter. A finger probe pulse oximeter, non invasive blood pressure and 5 channel electrocardiogram were connected to the patient. All patients received premedication with Inj. Glycopyrrolate 0.005 mg kg⁻¹. Baseline heart rate, peripheral oxygen saturation, systolic and diastolic blood pressure of all patients before preoxygenation were recorded. The radial artery was cannulated (22G) under local anaesthesia for arterial blood sampling.
Patients were preoxygenated by 2nd observer with proper sized tight fitting mask with help of magill circuit with a 2 L capacity reservoir bag which was flushed with 100 % O₂ for 30 sec. After the anaesthesia circuit was washed out with oxygen, the right-size anaesthesia face mask was placed on his/her face to fit correctly without air leakage. Reflection of the respiratory status and volumes on the balloon reservoir and display of significant capnograph traces were assured.

In Group VCB patients were asked to take 8 deep breaths in 60 s with maximal inspiration and maximum exhalation (8DBs/60 s), with an oxygen flow of 10 L min⁻¹ and in Group TVB, patients were told to breathe normally with 100% oxygen with normal tidal volume for 3 min with a flow rate of 5 L min⁻¹ (TVB/3 min).

Following preoxygenation with either method, induction of anaesthesia was achieved with Inj Propofol 2 mg kg⁻¹ and Inj Nalbuphine 0.2 mg kg⁻¹ body weight followed by Inj. Rocuronium 0.8-1 mg kg⁻¹ intravenously. Positive pressure ventilation was not administered, and the time at which patients became apnoeic was noted, that is, time at which there was the cessation of bag movement and/or EtCO₂ showing a flat line. A quick and gentle laryngoscopy was done at 90 sec following administration of Inj. Rocuronium and trachea was intubated with 7-8 mm ID cuffed endotracheal tube, which was kept open to atmosphere without administering positive pressure ventilation.

Hemodynamic parameters like SpO₂, heart rate (HR), systolic BP (SBP), diastolic BP (DBP) and mean arterial pressures (MAP) were closely monitored by 3rd observer. The time at which saturation reached the study end point of 90% was noted, and the patients were immediately bag ventilated with 100% O₂ till saturation became 98-100%. Then, anaesthetic gases were added, and patients were put on a ventilator. All the patients included in the study were ventilated using the same anaesthesia machine.

Arterial blood gas samples were taken by the 3rd observer before preoxygenation while patients were breathing room air (baseline), immediately after preoxygenation and also at 90% desaturation and gave to the 4th observer (assessor) who was outside the operating room for sample collection and ABG analysis. All samples were numbered and samples were analysed accordingly and was tabulated in observation charts. At these three points of time i.e before preoxygenation while patients were breathing in room air, immediately after preoxygenation and also at 90% desaturation HR, SpO₂, SBP, DBP and MAP were also documented. In this way a double blind study was conducted.

The design of the study ensured airway protection allowing for rapid oxygenation once the saturation had decreased to 90% or if any deterioration in patient condition occurred. By leaving the endotracheal tube open to air and keeping the patient anesthetized and apneic, the O₂ reserve was measured by observation of the time required for arterial desaturation to occur after induction of anaesthesia. At the end of surgery, patients were reversed with Inj. Glycopyrrolate 0.01 mg/kg and Inj. Neostigmine 0.05 mg/ kg and were shifted to post anaesthesia care unit (PACU).

During the study period if there was tachycardia (HR >100/min) or hypertension (SBP >160 mmHg), plane of anaesthesia was deepened with Inj Propofol 30 mg bolus IV. For bradycardia (an HR under 50 beats/min), the patients were administered 0.5 mg of iv atropine. In the case of hypotension (decrease in systolic arterial pressure of more than 20% from the baseline) during induction, the patient was first placed in the Trendelenburg position. In the case of sustained tachycardia, hypertension or development of arrhythmias, even with saturation >90%, patients were immediately ventilated and excluded from the statistical analysis.

Sample size calculation was done based on the previous study by Rajan et al¹⁸ results using Master 2.0 software. For a projected difference of 10% between the groups for a type I error of 0.05 and a power of 0.8, 26 patients were required in each group. Calculating for a 10 % drop out rate...
we needed at least 30 patients per group to be able to say with any degree of confidence whether a difference exists between both the groups.

Statistical analyses were performed using IBM SPSS version 21. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. The association between genders were assessed by comparison of percentages. Chi square test was used to test statistical significance. All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution.

The association between categorical age, weight, height, BMI, HR, SBP, DBP, MAP, SpO$_2$, PaO$_2$, PaCO$_2$, pH and study group were assessed by comparing the mean values. Independent sample t test or unpaired t test (2 groups) was used to assess statistical significance. P value < 0.05 was considered statistical significant.

**Results**

As there were no dropouts and none of the patients experienced adverse events during the process of preoxygenation. Hence, all the 60 patients completed the study and included in the statistical analysis with 30 patients in each group.

Both the groups were comparable in demographic parameters in terms of age, gender, weight, height, and BMI, as the P value was found to be statistical insignificant. (Table 1)

In terms of hemodynamic parameters, SBP (Table 3), DBP (Table 4), MAP (Table 5) and SpO$_2$ (Table 6) were comparable as there was no statistical difference in both the groups. But HR(Table 2) showed statistical increase in group VCB after preoxygenation.

Baseline values before preoxygenation, when the patients were breathing in room air for arterial blood gas parameters i.e. PaO$_2$, PaCO$_2$ and pH were comparable for both the groups as the P value was found to be statistical insignificant. But after preoxygenation with 100 % O$_2$, PaO$_2$ value was increased in both the groups but there was statistical significant increase in group VCB than in group TVB. (Figure 2)

Similarly, after preoxygenation PaCO$_2$ value increased in both groups but just after preoxygenation there was statistical significant increase in group TVB than in group VCB. Whereas the increase in PaCO$_2$ value at 90 % desaturation were comparable in both the groups. (Figure 3) The pH value was significantly decreased in group VCB than in group TVB after preoxygenation and at 90 % desaturation. (Figure 4)

The apnoea induced desaturation time was comparable in both the groups as there was no statistical significant difference. (Figure 5)
Table 1: Comparison of demographic parameter between groups

| Demographic parameter                      | Group | Unpaired t test P value |
|--------------------------------------------|-------|-------------------------|
| **Group** VCB (N=30)                       |       |                         |
| Age (Years) (Mean ± STD)                   | 63.83 ± 3.28 |                         |
| Gender                                     |       |                         |
| Male                                       | 13 (43.3%) |                         |
| Female                                     | 17 (56.7%) |                         |
| Weight (kg) (Mean ± STD)                   | 62.83 ± 6.96 |                         |
| Height (cm) (Mean ± STD)                   | 164.43 ± 8.85 |                         |
| BMI (kg/m^2) (Mean ± STD)                  | 23.19 ± 0.84  |                         |
| **Group** TVB (N=30)                       |       |                         |
| Age (Years) (Mean ± STD)                   | 63.17 ± 3.27 |                         |
| Gender                                     |       |                         |
| Male                                       | 14 (46.7%) |                         |
| Female                                     | 16 (53.3%) |                         |
| Weight (kg) (Mean ± STD)                   | 65.40 ± 7.11  |                         |
| Height (cm) (Mean ± STD)                   | 168.57 ± 6.75 |                         |
| BMI (kg/m^2) (Mean ± STD)                  | 22.93 ± 0.93  |                         |

Table 2: Comparison of mean HR between groups

| HR                          | Group | Unpaired t test P value |
|-----------------------------|-------|-------------------------|
| **Group** VCB (N=30)        |       |                         |
| Before preoxygenation       | 86.40 ± 13.90 |                         |
| Just after preoxygenation   | 97.83 ± 11.23  |                         |
| At 90% desaturation         | 107.57 ± 9.51  |                         |
| **Group** TVB (N=30)        |       |                         |
| Before preoxygenation       | 87.87 ± 14.29  |                         |
| Just after preoxygenation   | 90.30 ± 13.66  |                         |
| At 90% desaturation         | 99.77 ± 11.08  |                         |

Table 3: Comparison of mean Systolic Blood Pressure between groups

| Systolic Blood Pressure     | Group | Unpaired t test P value |
|-----------------------------|-------|-------------------------|
| **Group** VCB (N=30)        |       |                         |
| Before preoxygenation       | 123.80 ± 7.45  |                         |
| Just after preoxygenation   | 123.80 ± 4.94  |                         |
| At 90% desaturation         | 122.47 ± 5.84  |                         |
| **Group** TVB (N=30)        |       |                         |
| Before preoxygenation       | 122.87 ± 7.45  |                         |
| Just after preoxygenation   | 126.63 ± 6.78  |                         |
| At 90% desaturation         | 118.37 ± 5.85  |                         |

Table 4: Comparison of mean Diastolic Blood Pressure between groups

| Diastolic Blood pressure     | Group | Unpaired t test P value |
|-----------------------------|-------|-------------------------|
| **Group** VCB (N=30)        |       |                         |
| Before preoxygenation       | 78.70 ± 6.01  |                         |
| Just after preoxygenation   | 78.07 ± 5.07  |                         |
| At 90% desaturation         | 73.73 ± 4.96  |                         |
| **Group** TVB (N=30)        |       |                         |
| Before preoxygenation       | 77.57 ± 5.97  |                         |
| Just after preoxygenation   | 77.93 ± 6.86  |                         |
| At 90% desaturation         | 72.80 ± 4.54  |                         |

Table 5: Comparison of mean MAP between groups

| MAP                         | Group | Unpaired t test P value |
|-----------------------------|-------|-------------------------|
| **Group** VCB (N=30)        |       |                         |
| Before preoxygenation       | 93.27 ± 5.60  |                         |
| Just after preoxygenation   | 93.07 ± 3.98  |                         |
| At 90% desaturation         | 89.97 ± 4.43  |                         |
| **Group** TVB (N=30)        |       |                         |
| Before preoxygenation       | 92.83 ± 5.77  |                         |
| Just after preoxygenation   | 94.0 ± 6.33   |                         |
| At 90% desaturation         | 87.97 ± 4.37  |                         |
Table 6: Comparison of mean SpO\textsubscript{2} between groups

| SpO\textsubscript{2} | Group VCB (N=30) | Group TVB (N=30) | Unpaired t test P value |
|----------------------|------------------|------------------|------------------------|
| Before preoxygenation | 99.60 ± 0.77     | 99.73 ± 0.52     | 0.435                  |
| Just after preoxygenation | 100 ± 0.00      | 100 ± 0.00       | -                      |
| At 90% desaturation    | 90 ± 0.00        | 90 ± 0.00        | -                      |

Figure 2: Trend line diagram of comparison of mean PaO\textsubscript{2} between groups

Figure 3: Trend line diagram of comparison of mean PaCO\textsubscript{2} between groups

Figure 4: Trend line diagram of comparison of mean pH between groups
Discussion
Preoxygenation increases physiological stores of oxygen in order to prolong the time to desaturation during a period of apnoea, a situation that is usual following induction of anaesthesia. This is particularly important if difficulty with manual ventilation of the lungs or intubation of the trachea is anticipated or the case during a rapid sequence induction, when positive pressure ventilation is avoided prior to intubation of the trachea. This, in turn, means that the patient relies on his/her oxygen reserve during the entire intubation procedure.

The functional goal for preoxygenation is to prolong the safe apnoea time (or duration of apnoea without desaturation [DAWD]). Safe apnoea time is typically defined as the time from cessation of breathing or ventilation until the peripheral arterial oxygen saturation (SpO₂) declines to 90 percent, after which it falls precipitously.

There are several changes that occur during the process of aging that may influence the period of preoxygenation that should be used in the elderly and the subsequent time to desaturate to a given level. Aging is associated with significant structural and physiologic changes in the respiratory system. The changes include weakened respiratory muscles and parenchymal alterations within the lungs accompanied by a decrease in the elastic recoil. Lung volumes are decreased with increased closing volume, resulting in ventilation—perfusion mismatch, a reduced pulmonary reserve, and an impaired O₂ uptake at the lung. Even though basal oxygen consumption (VO₂) decreases with aging, the impaired O₂ uptake produces a more rapid desaturation during apnoea under anaesthesia. Patients with a decreased capacity of oxygen loading or an increased oxygen extraction, or both, are desaturated during apnoea much faster than healthy patients. In elderly patients, tidal volume breathing for 3 minutes or longer has been shown to be more effective than the 4 deep breathing technique. As the older patients are at more risk for apnoea induced desaturation, preoxygenation is must. But we need a faster and better technique of preoxygenation which can be used in emergency situations.

Techniques of monitoring preoxygenation have focused on measurements of indices reflecting its efficacy or efficiency. The decline of haemoglobin saturation of oxygen during apnoea is the only indicator of efficacy of preoxygenation. Pre-oxygenation is done to increase the dissolved oxygen content in the body. The equation which depicts oxygen content of the arterial blood is:

\[ \text{CaO}_2 = \text{SaO}_2 \times \text{Hb} \times 1.31 + 0.003 \times \text{PaO}_2 \]

Where
- \( \text{CaO}_2 \) = oxygen content of the blood
- \( \text{SaO}_2 \) = Haemoglobin saturation
- \( \text{PaO}_2 \) = partial pressure of O₂ in mmHg

0.003 : Solubility co-efficient of oxygen
1.31 : oxygen binding capacity of haemoglobin

If we observe this equation, it is clear that the contribution of \( \text{PaO}_2 \) is more important in
determining dissolved oxygen content of the blood, thus indicating a more appropriate measure of efficiency of preoxygenation.

In our study we have assessed both efficiency and efficacy in terms of arterial blood gas measurements and apnoea induced desaturation time.

The difference in the fresh gas flow is based on the concept by Nimmagadda et al in recent article while investigated the impact of fresh gas flow (FGF) on two methods of preoxygenation (Tidal volume and deep breathing) concluded that increase in FGF from 5 to 10 L min\(^{-1}\) does not enhance oxygenation in lungs with either tidal volume breathing or 4 deep breaths/30 sec however 8 deep breathing technique in 60 secs resulted in rise of EtO\(_2\) from 87% to 90% by increasing period of preoxygenation from 1 to 1.5 mins thus demonstrating 8 deep breathing technique with slight increase in duration of preoxygenation (1 to 1.5 min) further improves the oxygenation of lungs and thus may have important implication for patient’s safety. Hence we kept the FGF of 5 L min\(^{-1}\) in group TVB and FGF of 10 L min\(^{-1}\) in group VCB to reduce the duration of preoxygenation to 1 min.

The choice of paralytic agent may influence the time to desaturation during airway management. It is hypothesized that the Suxamethonium-induced fasciculation increases oxygen consumption during apnoea, which may become relevant in the event of airway obstruction. When used at a dose of greater than or equal to 1.2 mg kg\(^{-1}\), rocuronium provides intubating conditions identical to those of succinylcholine.\(^{19}\)

The baseline mean heart rate before preoxygenation for both the groups are comparable, as the P value was found to be insignificant. But just after preoxygenation with either method and at the time of 90 % desaturation there is increase in the mean heart rate. The P values were statistically significant, indicating that there was statistically more significant increase in mean heart rate in Group VCB than in group TVB. This is explained by the fact that in normal subjects, systemic haemodynamic changes mainly concern the heart rate (an effect related to both the barometric and the oxygen pressures).\(^{12}\) Arterial blood pressure tends to increase slightly. Cardiac output is maintained or moderately decreased due to compensating responses which may fail in patients with pre-existing cardiac failure. This finding was similar to Khandrani et al.\(^{17}\)

The PaO\(_2\) values before preoxygenation in both the groups were comparable as P value was statistically insignificant. But just after preoxygenation, there was increase in the PaO\(_2\) values for both the groups, but there was statistical significant increase in group VCB. This was similar to the study done by Rooney et al\(^{10}\), Gold and colleagues\(^{3}\), Gambee and Hertzka\(^{11}\), Valentine et al\(^{2}\), Sanjay et al\(^{14}\), Khandrani et al\(^{17}\), Ranjan et al\(^{18}\) and Taha et al\(^{16}\). In all these previous studies have shown that vital capacity breath technique in 30 sec produce greater increase in PaO\(_2\) value. Our study was similar to Baraka et al\(^{4}\), Ranjan et al \(^{18}\), and Singh et al\(^{15}\) who also observed that 8 vital capacity breath can produce higher PaO\(_2\) comparable to that achieved with tidal volume breathing for three minutes.

The comparison of the mean time to desaturate between the two groups were comparable. Our result was similar to the study of Baraka et al\(^{4}\) and Singh et al\(^{15}\) who carried out a trial to compare the adequacy of eight vital capacity breaths in comparison to four vital capacity breaths and traditional method for preoxygenation in younger patients. Their results showed that the apnoea following different techniques of preoxygenation was associated with slower Hb desaturation in 8 deep breaths technique as compared to both traditional and 4 deep breaths technique. This is supported by the study done by Drummond and Park\(^{13}\) who stated that age is not related to rapidity of desaturation.

**Conclusion**

In conclusion, preoxygenation using eight deep breaths within 60 secs at oxygen flow of 10 L min\(^{-1}\) is an excellent method of rapid
preoxygenation with regard to both efficacy and efficiency. This technique can provide oxygen saturation in arterial blood (SpO$_2$) values comparable and partial pressure of oxygen in arterial blood (PaO$_2$) significantly higher than the traditional preoxygenation technique. Also it provides a safe apnoea time (DAWD) as it delays the onset of subsequent apnoea induced Hb desaturation similar to the 3 min of tidal volume breath which is the traditional method of preoxygenation. Hence we recommend that eight vital capacity breath technique may be a better alternative to the traditional technique of preoxygenation in elderly patients undergoing rapid sequence induction, anticipated intubation/ventilation difficulty and life saving emergencies.

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