

# Nasopharyngeal oxygen insufflation following pre-oxygenation using the four deep breath technique

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## Summary

This paper evaluates the effectiveness of nasopharyngeal oxygen insufflation following pre-oxygenation using the four deep breath technique within 30 s, on the onset of haemoglobin desaturation during the subsequent apnoea. Thirty ASA I or II patients were randomly allocated to one of two groups. In the study group ( $n = 15$ ), pre-oxygenation was followed by insufflation of oxygen at a flow of  $5 \text{ l.min}^{-1}$  via a nasopharyngeal catheter commenced at the onset of apnoea. In the control group, pre-oxygenation was not followed by nasopharyngeal oxygen insufflation ( $n = 15$ ). In the control group,  $S_{\text{pO}_2}$  fell to 95% within a mean (SD) apnoea time of 3.65 (1.15) min, whereas in the study group,  $S_{\text{pO}_2}$  was maintained in all patients at 100% throughout the 6 min of apnoea, at which point apnoea was terminated and positive pressure ventilation commenced. We conclude that nasopharyngeal oxygen insufflation following pre-oxygenation using the four deep breath technique can delay the onset of haemoglobin desaturation for a significant period of time during the subsequent apnoea.

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Using the Nottingham Physiology Simulator, McNamara *et al.* [1] studied the effect of varying ambient oxygen fraction on  $P_{\text{aO}_2}$  following pre-oxygenation, during subsequent apnoea in the presence of an open airway. Increasing ambient  $\text{O}_2$  fraction from 0.9 to 1.0 more than doubled the time before haemoglobin desaturation fell to 50% and extended the period of apnoea to a greater extent than when the ambient  $\text{O}_2$  fraction was increased from 0.21 to 0.9. During brainstem death testing, only one study has compared the effects of 100% oxygen and air as alternative ambient gases on  $P_{\text{aO}_2}$ ; patients who had been pre-oxygenated with 100%  $\text{O}_2$  developed little or no hypoxaemia when compared to those receiving air [2].

The clinical application of this concept was reported by Teller *et al.* [3] who conducted an investigation in surgical patients under general anaesthesia to determine whether pharyngeal oxygen insufflation following pre-oxygenation using tidal volume breathing for 3 min could prolong the period of subsequent apnoea during which they remain adequately oxygenated. Their results demonstrated that patients pre-oxygenated for 3 min with

tidal volume breathing followed by pharyngeal  $\text{O}_2$  insufflation at the onset of apnoea sustained 10 min of adequate oxygenation with  $S_{\text{aO}_2}$  remaining equal to or  $> 97\%$ . In contrast, in the absence of  $\text{O}_2$  insufflation, the  $S_{\text{aO}_2}$  fell to 91% within a mean apnoea time of 6.8 (0.6) min [3].

Gold *et al.* [4] demonstrated that pre-oxygenation using four deep breaths in 30 s produced increases in arterial oxygen tension comparable to those obtained using tidal volume breathing for 5 min. However, subsequent investigations showed that haemoglobin desaturation during apnoea was more rapid following pre-oxygenation using the four deep breath technique than following pre-oxygenation either by the traditional tidal volume breathing for 3 min [5] or by the eight deep breath technique for 60 s [6]. The present study investigates the effectiveness of nasopharyngeal  $\text{O}_2$  insufflation following pre-oxygenation with the four deep breaths in 30 s as compared to the four deep breaths alone in delaying the onset of haemoglobin desaturation during subsequent apnoea.

## Method

The study was approved by the institutional review board, and informed consent was obtained from all patients. The study was performed on 30 ASA I–II patients with an age range of 20–52 years scheduled for elective surgery under general anaesthesia. Patients were non-bearded with normal airway anatomy and free from cardiovascular, pulmonary and gastro-intestinal reflux disease. Patients were assigned to one of two groups using a computer-generated table of random numbers. All patients received diazepam 5 mg orally 1 h prior to induction of anaesthesia. An infusion of lactated Ringer's solution was commenced in the operating room. A standard anaesthesia machine (Datex ADU AS/3 anaesthesia monitor; Helsinki, Finland) with an absorber system and a 2 litre reservoir bag was used. Patients were allowed to become familiar with facemask breathing and the four deep breath pre-oxygenation technique. Baseline oxygen saturation was measured using a pulse oximeter (Novamatrix pulse oximeter, Wallingford, CT).

Prior to pre-oxygenation, the anaesthesia circuit was flushed with O<sub>2</sub> and the reservoir bag was filled to capacity. In the supine position, patients were pre-oxygenated with 10 l.min<sup>-1</sup> O<sub>2</sub> using four deep breaths in 30 s using a properly sized, tight fitting facemask to ensure that there was no leak. Patients were asked to take four maximal inspirations slowly and expire fully within 30 s. Throughout the study period, patients were routinely monitored using electrocardiography, non-invasive blood pressure measurement, pulse oximetry and end-tidal capnography. When the patient was starting their fourth deep breath of the pre-oxygenation sequence, anaesthesia was induced using propofol 2 mg.kg<sup>-1</sup>, fentanyl 2 µg.kg<sup>-1</sup>, and rocuronium 0.6 mg.kg<sup>-1</sup>. To avoid the possibility of awareness during the apnoeic period, a bolus dose of 10 mg of propofol was administered 2 min following the induction dose and then every minute until the completion of the study. At the onset of apnoea, as indicated by loss of chest movement and disappearance of end-tidal CO<sub>2</sub> waveform, the facemask was removed. A 10 Fr catheter was inserted nasally into the nasopharynx in all patients; the length of catheter inserted was equal to the distance between the angle of the mouth and the tragus of the ear. In the study group (*n* = 15), we insufflated O<sub>2</sub> at a rate of 5 l.min<sup>-1</sup> via the nasopharyngeal catheter; no oxygen was used in the control group (*n* = 15). The oxygen delivery tubing was connected to an independent flowmeter, which was out of view of the investigator, who was unaware of whether oxygen or no oxygen had been administered. The time from the onset of apnoea until S<sub>p</sub>O<sub>2</sub> fell to 95% was recorded. Apnoea was allowed to continue until S<sub>p</sub>O<sub>2</sub> either fell to 95% or

apnoea of 6 min duration had occurred, at which point the patient's lungs were ventilated using 100% O<sub>2</sub>, and tracheal intubation was performed. Inspired O<sub>2</sub> fraction (F<sub>i</sub>O<sub>2</sub>), end-tidal O<sub>2</sub> (ETO<sub>2</sub>) after four deep breaths, as well as end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) after four deep breaths and following initiation of positive-pressure ventilation were recorded using a gas monitor (Datex ADU AS/3). Side-stream respiratory gases were sampled from a sampling port placed between the facemask and the Y-piece of the anaesthetic circuit. Calibration with a known gas mixture was carried out according to the manufacturer's specifications prior to each patient investigation. The time from the onset of apnoea until S<sub>p</sub>O<sub>2</sub> fell to 95% or until 6 min had elapsed was compared between the two groups.

Postoperatively, the patients in both groups were interviewed concerning the possibility of intra-operative awareness.

## Statistical analysis

A power analysis was performed to determine the number of subjects required for the study. For data analysis, we considered that a 5% change in oxygen saturation was clinically significant. We also considered Type I and Type II errors of 5% and 20%, respectively (power of 80%). From a previous pilot study, we determined that the standard deviation of oxygen saturation was 1.82%: as such, the power analysis indicated that at least 15 patients were needed in each group. Student's *t*-test was used for statistical analysis (SPSS 13.0 for Windows Software). A *p*-value of <0.05 was considered significant.

## Results

Patients in both groups had similar demographic data (Table 1). The F<sub>i</sub>O<sub>2</sub>, ETO<sub>2</sub>, ETCO<sub>2</sub>, and S<sub>p</sub>O<sub>2</sub> were not significantly different in both groups following pre-oxygenation with the four deep breaths within 30 s (Table 2).

In the control group, S<sub>p</sub>O<sub>2</sub> decreased from 100% to 95% after a mean (SD) apnoea time of 3.65 (1.15) min,

**Table 1** Characteristics of patients not receiving nasopharyngeal oxygen insufflation after pre-oxygenation with four deep breaths (control group) or receiving nasopharyngeal oxygen insufflation after pre-oxygenation with four deep breaths (study group). Values are mean (SD).

|            | Control group ( <i>n</i> = 15) | Study group ( <i>n</i> = 15) |
|------------|--------------------------------|------------------------------|
| Age; years | 37.4 (9.8)                     | 37.8 (11)                    |
| Weight; kg | 69 (15)                        | 64 (11)                      |
| Height; cm | 167 (11)                       | 164 (9)                      |
| Sex; M : F | 6 : 9                          | 5 : 10                       |

**Table 2** Fraction of inspired oxygen ( $F_{iO_2}$ ), end-tidal oxygen ( $ETO_2$ ), end-tidal carbon dioxide ( $ETCO_2$ ), and oxygen saturation ( $S_pO_2$ ) following pre-oxygenation with four deep breaths in the control group and in the study group prior to nasopharyngeal oxygen insufflation. Values are mean (SD).

|                 | Control group (n = 15) | Study group (n = 15) |
|-----------------|------------------------|----------------------|
| $F_{iO_2}$ ; %  | 98 (1)                 | 98 (1)               |
| $ETO_2$ ; mmHg  | 82 (3)                 | 81 (3)               |
| $ETCO_2$ ; mmHg | 31 (3)                 | 30 (2)               |
| $S_pO_2$ ; %    | 100 (0)                | 100 (0)              |

and the  $ETCO_2$  increased from 31 (3) mmHg to 39 (2) mmHg. When ventilation with 100%  $O_2$  started there was an initial transient decline in  $S_pO_2$  from 95 (0)% to 92 (1)%, followed within 5–10 s by an increase of  $S_pO_2$  up to 100%.

In the study group of patients who received supplemental nasopharyngeal oxygen insufflation following pre-oxygenation, the  $S_pO_2$  was maintained at 100% throughout the apnoeic period up to 6 min when apnoea was terminated and ventilation with 100%  $O_2$  commenced. Following the 6 min of apnoea in this group, the  $ETCO_2$  increased from 30 (2) to 44 (3) mmHg, which is significantly higher ( $p = 0.001$ ) than the  $ETCO_2$  after 3.65 min of apnoea in the control group (39 (2) mmHg).

The blood pressure and heart rate of all patients remained within 20% of baseline values during the apnoeic period. No patient reported any episodes of awareness.

## Discussion

Gold *et al.* demonstrated in awake patients that pre-oxygenation using four deep breaths in 30 s produced comparable increases in arterial  $O_2$  tension to that obtained following 5 min of tidal volume breathing [4]. Although rapid pre-oxygenation using the four deep breath technique can denitrogenate the functional residual capacity (FRC), it might not adequately denitrogenate the tissue body stores, as the tissue and venous compartments need >30 s to fill with oxygen [7, 8]. Subsequent studies have shown that the four deep breath technique is less effective for pre-oxygenation than the 3 min of tidal volume breathing [5] and the eight deep breath technique in 60 s [6, 9]. This is particularly the case in parturients [10, 11], and the elderly [12, 13], as shown by the more rapid haemoglobin desaturation following apnoea.

Our results have demonstrated that apnoea following pre-oxygenation with four deep breaths in 30 s without nasopharyngeal oxygen insufflation resulted in a reduction in  $S_pO_2$  95% within a mean time of 3.65 (1.15) min. In contrast, when the four deep breaths were followed

during the subsequent apnoea by nasopharyngeal  $O_2$  insufflation,  $S_pO_2$  was maintained at 100% for up to 6 min, when the apnoea was terminated.

In 1956, Holmdahl introduced the term apnoeic diffusion oxygenation, which is achieved by pre-oxygenation with 100%  $O_2$  followed by  $O_2$  insufflation during subsequent apnoea [14]. During the apnoeic period,  $O_2$  is extracted from the FRC into the blood at a rate of 250 ml.min<sup>-1</sup> to maintain metabolic  $O_2$  consumption. However, because of the greater solubility of  $CO_2$  in blood, it is only added to the alveolar space at a rate of 10 ml.min<sup>-1</sup>, resulting in a net gas flow from the alveoli to the blood at about 240 ml.min<sup>-1</sup>. Hence, a subatmospheric pressure is established in the alveoli, and the ambient oxygen is drawn 'en masse' into the lungs and maintains oxygenation [14]. When air, which contains 79%  $N_2$  and 21%  $O_2$  is used as the ambient gas, it will be drawn into the lungs during apnoea;  $N_2$  will rapidly accumulate in the FRC, in addition to  $N_2$  diffusing from body tissue [14, 15]. The rate of nitrogen accumulation in the FRC is inversely proportional to the FRC body weight ratio [15].

Following pre-oxygenation using the four deep breath technique, the mean  $ETCO_2$  averaged 31 (3) mmHg in the control group and 30 (2) mmHg in the study group. Following the subsequent apnoea, the average  $ETCO_2$  increased to 39 (2) mmHg in the control group following an average apnoea time of 3.65 min, and to 44 (3) mmHg in the study group following an apnoea time of 6 min. The rate of increase of the  $ETCO_2$  following the apnoeic periods was 2.4 mmHg min<sup>-1</sup> in the control group and 2.3 mmHg min<sup>-1</sup> in the study group.

As demonstrated by the physiological simulator, McNamara *et al.* [1] showed that increasing ambient  $O_2$  fraction from 0.9 up to 1.0 more than doubled the time to haemoglobin desaturation and extended the apnoea longer than when the ambient  $O_2$  fraction was increased from 0.21 to 0.9, suggesting that even a small concentration of  $N_2$  in the ambient gas may decrease the effectiveness of apnoeic diffusion oxygenation. When air is used as the ambient gas, the  $P_aO_2$  is reduced after about 3 min to a level which inhibits  $O_2$  uptake, resulting in quite inadequate blood  $O_2$  saturation [14]. This may explain why, when patients were pre-oxygenated with four deep breaths in 30 s without nasopharyngeal  $O_2$  insufflation, we found rapid desaturation during subsequent apnoea to an  $S_pO_2$  of 95% after a mean time of 3.65 min. In contrast, when pre-oxygenation was followed by nasopharyngeal oxygen insufflation,  $S_pO_2$  was maintained at 100% in all patients throughout the entire 6 min period of apnoea.

To optimise pre-oxygenation, different techniques have been recommended, such as using the eight deep

breath technique instead of the four deep breath technique [6], application of positive airway pressure during pre-oxygenation [16] and using the head-up position [17, 18]. This study suggests that nasopharyngeal oxygen insufflation which provides apnoeic diffusion oxygenation, is a simple technique that can follow pre-oxygenation by the four deep breath technique within 30 s and significantly increase the period of subsequent apnoea without desaturation. The technique can also optimise pre-oxygenation by tidal volume breathing within 3 min [3]. Pre-oxygenation followed by nasopharyngeal oxygen insufflation may be particularly indicated in patients who are liable to develop rapid desaturation during apnoea such as the morbidly obese and patients who are predicted to have a difficult airway [19].

In conclusion, our study demonstrates that nasopharyngeal oxygen insufflation following pre-oxygenation by the four deep breath technique can delay the onset of haemoglobin desaturation for a significant period during the subsequent apnoea.

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