Clinical Investigation

Effect of positive airway pressure during pre-oxygenation and induction of anaesthesia upon safe duration of apnoea

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ABSTRACT

Background and Aims: Induction of general anaesthesia *per se* as also the use of 100% oxygen during induction of anaesthesia, results in the development of atelectasis in dependent lung regions within minutes of anaesthetic induction. We aimed to assess the effect of application of a continuous positive airway pressure (CPAP) of 5 cm H_2O during pre-oxygenation and induction of anaesthesia on the period of apnoea before the occurrence of clinically significant desaturation. **Methods:** In this prospective, randomised, and double-blind study, 40 patients posted for elective surgery were enrolled. Duration of apnoea was measured as the time from the administration of succinylcholine hydrochloride to the time when oxygen saturation fell to 93%. Student's *t*-test was used for comparing the duration of apnoea. **Results:** The safe duration of apnoea was found to be significantly longer in patients receiving CPAP of 5 cm H_2O (Group P; $n = 16$) compared to the group receiving no CPAP (Group Z; $n = 20$), that is, 496.56 ± 71.68 s versus 273.00 ± 69.31 s ($P < 0.001$). **Conclusion:** The application of CPAP of 5 cm H_2O using a Mapleson "A" circuit with a fixed positive end-expiratory pressure device during 5 min of pre-oxygenation with 100% oxygen prior to the induction of anaesthesia provides a clearly longer duration of apnoea before clinically significant arterial desaturation occurs.

Key words: Positive airway pressure, pre-oxygenation, safe duration of apnoea

INTRODUCTION

Induction of general anaesthesia *per se* as also the use of 100% oxygen during induction of anaesthesia results in the development of atelectasis in dependent lung regions within minutes of anaesthetic induction.[1,2] Application of continuous positive airway pressure (CPAP) during pre-oxygenation and induction of anaesthesia[3-5] helps to increase the duration of apnoea before the development of significant arterial desaturation by preventing atelectasis, increasing oxygen stores, and decreasing intrapulmonary shunt.

Difficulties in airway management during induction of anaesthesia may result in mortality of patients. Difficulty in airway management is not easily anticipated. Therefore, avoiding the formation of atelectasis during induction of general anaesthesia may increase the duration of non-hypoxic apnoea which in turn will increase the margin of safety.

The study was designed to assess the effect of application of a positive airway pressure of 5 cm H_2O during pre-oxygenation and induction of anaesthesia on the period of apnoea before the occurrence of clinically significant desaturation that is, an arterial saturation of haemoglobin as measured by a pulse oximeter (oxygen saturation [SpO_2]) of <93%.

METHODS

The study commenced after obtaining approval from the Institutional Ethics Committee. A written and informed consent was obtained from the patients to participate in the study. The study

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design was prospective, randomised, and double blind. A previous study by Herriger *et al*.[1] found that non-hypoxic apnoea duration was longer in the positive end-expiratory pressure (PEEP) group compared to zero end-expiratory pressure (ZEEP) group (599 ± 135 s vs. 470 ± 150 s). Assuming 30% difference, at two-sided type 1 error of 0.05 and power of 90%, a sample size of 20 per group was arrived at. 40 adult patients of either gender (20 in each group) participated in the study. Patients belonged to the age group of 18–70 years and underwent elective surgery requiring endotracheal anaesthesia for major surgical procedures which required an invasive blood pressure monitoring, during the period from December 2004 to March 2006. All of them belonged to physical status I or II as outlined by the American Society of Anesthesiologists. Patients with an SpO_2 of <97% while breathing room air, those who had been non-ambulant in hospital for >24 h, patients with a body mass index (BMI) >25 kg/m^2, patients with haemoglobin <8 g%, those with medical conditions that preclude even a short period of arterial desaturation or rise in arterial partial pressure of carbon dioxide ($PaCO_2$) such as patients with coronary artery disease, cerebrovascular disease, intracranial hypertension, epilepsy or severe pulmonary disease, and patients in whom difficulty in maintaining airway or obtaining an effective mask fit was anticipated were excluded from the study.

Patients were pre-medicated with oral diazepam (5 mg if they weighed <50 kg; 10 mg if they weighed >50 kg) on the night before and on the morning of surgery.

They were randomly allocated to one of the two groups using random number draws, Group Z (ZEEP group) (control group) and Group P (PEEP group) (study group).

After shifting the patients into the operating room, the following monitoring was established: Electrocardiography, non-invasive blood pressure, pulse oximetry (SpO_2) (in two locations). The additional precaution of monitoring SpO_2 simultaneously at two sites was taken for reasons of patient safety. We used the same two instruments throughout the study, viz., the Welch Allyn PIC50™ biphasic defibrillator monitor and the BCI Capnocheck™ pulse oximeter-with-capnograph.

An intravenous line was secured on the upper limb using an 18-gauge cannula. Under strict aseptic precautions, the radial artery was cannulated after local infiltration with 2% lignocaine and the arterial line flushed with heparinised saline.

Baseline values of heart rate, blood pressure and SpO_2 were recorded, and a sample of arterial blood gas (ABG1) was taken with the patient breathing room air.

Application and maintenance of position of the facemask were done by the anaesthesia consultant in charge of the case (Observer 1). In both the groups, patients were pre-oxygenated with 100% oxygen for 5 min using an oxygen flow of 5 LPM.

In Group P, pre-oxygenation was undertaken using a Mapleson "A" circuit with a fixed PEEP device attached to the expiratory port that maintained a CPAP of 5 cm H_2O [Figure 1]. In Group Z, no CPAP was used.

A sample of arterial blood was obtained after 5 min of pre-oxygenation (ABG2) and the SpO_2 recorded at this point. Anaesthesia was induced by Observer 2 using fentanyl 2 μg/kg (rounded off to the nearest multiple of 25 μg) and propofol 2.5 mg/kg (rounded off to the nearest multiple of 10 mg). Patients were paralysed with succinylcholine hydrochloride (SCh) 2 mg/kg and tracheal intubation performed 60 s later by the anaesthesia consultant in charge of the case.

Following intubation, positive pressure breaths were not administered. The correct position of the tracheal tube was confirmed by Observer 1 using a modified oesophageal detector device [Figure 2]. This would supplement the fact that tracheal intubation was performed under direct vision by Observer 1.

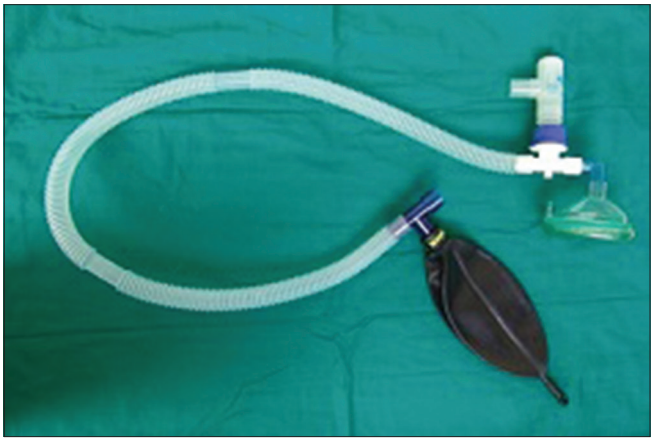

Figure 1: Fixed positive end expiratory pressure device attached to Mapleson A circuit

Patients were administered intravenous vecuronium bromide 0.08 mg/kg immediately after tracheal intubation to maintain muscle relaxation and anaesthesia maintained using an infusion of propofol at 50 μg/kg/min. Following tracheal intubation, the cuvette of a mainstream capnograph was connected to the tracheal tube to detect any spontaneous breaths that could possibly negate study conditions.

The tracheal tube was left open to air at atmospheric pressure and the patient allowed to remain apnoeic until SpO_2 fell to 93% at either of the two sites of pulse oximetry monitoring or a period of 10 min had elapsed, whichever was earlier. An ABG was drawn at this point (ABG3). Following this, patients were ventilated with 100% oxygen until the SpO_2 returned to baseline values. Observer 2 who performed anaesthetic induction also observed capnograph (for evidence of breathing), pulse oximeter (for evidence of clinically significant desaturation as indicated by an SpO_2 <93%, duration of apnoea (in seconds) measured as the time from the administration of SCh to the time when SpO_2 fell to 93% or a period of 10 min had elapsed, whichever occurred earlier. Observer three took samples of arterial blood at designated intervals.

Statistical analysis was performed using SPSS 16.0 (Statistical Package for the Social Science for windows; Version 16.0, SPSS Inc., Chicago, USA). Three statistical methods were employed in the study. Chi-square test and for Student's *t*-test used comparing demographic variables, duration of apnoea and the haemodynamic variables between the two groups. The Pearson product-moment correlation coefficient was used for seeing any correlation between the duration of non-hypoxic apnoea with a partial pressure of arterial oxygen (PaO_2) after pre-oxygenation, BMI, smoking, age and gender.

RESULTS

The age, gender, weight, height, BMI, and history of smoking of the 40 patients were noted. There was no significant difference between the two groups [Table 1].

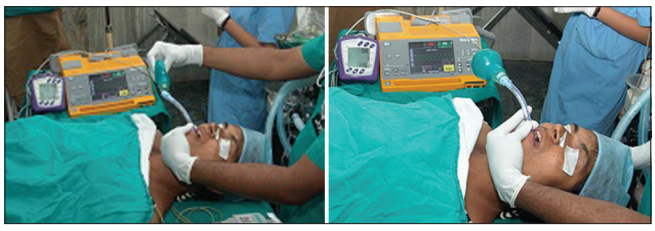

Figure 2: The correct position of the tracheal tube confirmation using a modified esophageal detector device

The arterial SpO_2 dropped to 93% within the study period of 10 min in all 20 patients in the ZEEP group and only 16 out of 20 patients in the PEEP group. The mean and standard deviation of the safe duration of apnoea was found to be significantly longer [Table 2] in patients receiving positive airway pressure of 5 cm H_2O (Group P; $n = 16$) compared to the group receiving no positive airway pressure (Group Z; $n = 20$), that is, 472.50 ± 71.46 s versus 249.20 ± 64.82 s. This difference was statistically very highly significant.

The time taken for arterial SpO_2 to drop to 95% was also noted. This event also occurred in all 20 patients in the ZEEP group and only 16 out of 20 patients in the PEEP group. The mean and standard deviation of the time taken for the SpO_2 to drop to 95% was also significantly longer [Table 2] in patients receiving positive airway pressure of 5 cm H_2O (Group P; $n = 16$) compared to the group receiving no positive airway pressure (Group Z; $n = 20$), that is, 472.50 ± 71.46 s versus 249.20 ± 64.82 s. This difference was statistically very highly significant. The time taken to return to baseline SpO_2 was also less in the PEEP group [Table 2], but this difference was

Table 1: Demographic data

Parameter	Group Z (*n*=20)	Group P (*n*=20)	*P*
Age (years)			
Mean (SD)	45.65 (12.22)	42.75 (11.97)	
Gender			
Male/female	11/9	11/9	
Weight (kg)			
Mean (SD)	58.65 (9.85)	55.60 (8.73)	
Height (cm)			
Mean (SD)	166.65 (7.14)	161.55 (9.36)	
BMI (kg/m^2)			
Mean (SD)	21.01 (2.38)	20.97 (2.29)	
History of smoking			
Yes/no	6/14	7/13	0.736

SD – Standard deviation; BMI – Body mass index

Table 2: Comparison of duration of apnoea and time to return to baseline SpO_2 among the two groups

Endpoints	Group Z	Group P	*P*
Duration of apnoea (in seconds) for desaturation to 93% mean (SD)	273.00 (69.31)	496.56 (71.68)	<0.001
Duration of apnoea (in seconds) for desaturation to 95% mean (SD)	249.20 (64.82)	472.50 (71.46)	<0.001
Time (in seconds) for return to baseline SpO_2 mean (SD)	43.20 (14.88)	36.69 (14.96)	0.202

SpO_2 – Oxygen saturation; SD – Standard deviation

not statistically significant. The PaO_2 and $PaCO_2$ at the start of pre-oxygenation were comparable in both the groups [Table 3].

The PaO_2 measured following pre-oxygenation was 355.65 ± 96.55 mmHg in the ZEEP group and 433.44 ± 75.11 mmHg in the PEEP group. This difference was statistically significant. However, the $PaCO_2$ in the two groups at this point was comparable. The haemoglobin SpO_2 in four patients in the PEEP group remained above 96% during the 10 min of apnoea that we had defined to be the end point of our study.

Influence of demographic variables on the duration of non-hypoxic apnoea are depicted in Table 4. No correlation was found between the duration of non-hypoxic apnoea with PaO_2 after pre-oxygenation, BMI, smoking, age or gender in both the ZEEP group and PEEP group.

The heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were comparable in the ZEEP group and PEEP group at various time intervals, baseline, after 5 min of pre-oxygenation and at the point of desaturation (at SpO_2 93%).

DISCUSSION

During apnoea, oxygenation depends on the oxygen reserves available in the body. The principal stores of oxygen in an individual breathing air are in the lungs (450 ml), in blood (850 ml), dissolved in tissue fluids (50 ml) and in combination with myoglobin (200 ml). These stores are small while breathing air. Very little of this small store of oxygen can be released without an unacceptable reduction in PaO_2. According to the oxygen dissociation curve, blood will not release substantial quantities of oxygen until the PaO_2 falls below 40 mmHg. Myoglobin is even more reluctant to part with oxygen, and very little can be released above a PaO_2 of 20 mmHg.

Breathing 100% oxygen causes a nearly three-fold increase in oxygen stores to around 4250 ml. Most of this additional oxygen is accommodated in the alveolar gas where 80% can be withdrawn without causing the PaO_2 to fall below the normal value. However, breathing 100% oxygen also results in near-complete denitrogenation of the alveoli. While breathing room air, the low solubility of nitrogen in the blood causes the unabsorbed nitrogen to act as a pneumatic splint and prevent atelectasis from developing.

During general anaesthesia, up to 85–90% of patients develop atelectasis in dependent lung regions within 5 min of anaesthetic induction.[6] This results in a decrease in functional residual capacity (FRC) which in turn causes a reduction in the oxygen stores in the body. In addition, it also increases the intrapulmonary shunt which will hasten desaturation to hypoxic levels.[7]

Positive end-expiratory pressure increases FRC to or above a lung volume greater than closing capacity (CC), thereby restoring a normal FRC to CC relationship, so that no airways are closed at any time during the tidal respiration. Application of PEEP during induction of anaesthesia increases the duration of non-hypoxic apnoea by preventing atelectasis formation, increasing oxygen stores, and decreasing intrapulmonary shunt.

The development of atelectasis is a consistent finding with the use of 100% oxygen for pre-oxygenation before induction of anaesthesia.[8,9] The use of lower fractional concentration of inspired oxygen may increase this risk of hypoxaemia, especially when difficulty in airway management and/or ventilation is encountered.[10] Some investigators have found that during routine induction of general anaesthesia, pre-oxygenation with 80% oxygen caused minimal atelectasis, but the time duration before desaturation occurred was significantly shortened compared to a situation when 100% oxygen was inhaled.[11]

Table 3: Arterial blood gases at various time intervals of measurement

Arterial blood gas values	Group Z (*n*=20)	Group P (*n*=16)	*P*
Baseline PaO_2 mean (SD)	93.25 (5.09)	93.94 (5.50)	0.700
Baseline $PaCO_2$ mean (SD)	35.85 (6.14)	34.19 (5.24)	0.396
PaO_2 following pre-oxygenation mean (SD)	355.65 (96.55)	433.44 (75.11)	0.012
$PaCO_2$ following pre-oxygenation mean (SD)	36.05 (8.74)	33.06 (6.54)	0.265

SD – Standard deviation; $PaCO_2$ – Partial pressure of carbon dioxide; PaO_2 – Partial pressure of arterial oxygen

Table 4: Correlation between demographic variables and duration of non-hypoxic apnoea

Parameters	Group Z		Group P	
	Pearson correlation	*P*	Pearson correlation	*P*
PaO_2 after pre-oxygenation	0.332	0.153	−0.216	0.361
BMI	−0.247	0.294	−0.095	0.691
Smoking	0.132	0.578	−0.380	0.099
Age	−0.082	0.732	0.338	0.145
Gender	−0.294	0.208	0.070	0.770

BMI – Body mass index; PaO_2 – Partial pressure of arterial oxygen

A clinical study[7] found that atelectasis formation can be effectively prevented by the use of PEEP applied during induction of anaesthesia despite the use of 100% oxygen. They noted that applying PEEP during the entire anaesthetic induction with 100% oxygen provided the clinical benefit of an increased duration of non-hypoxic apnoea.

In other studies,[1] higher arterial oxygen tensions and longer duration of non-hypoxic apnoea (>2 min), was observed in patients who had received CPAP or PEEP during anaesthetic induction as compared to those who had not received such manoeuvres. However, as pointed out by the authors, the limitation of their study was the inability to distinguish between the effects of CPAP applied during pre-oxygenation and PEEP applied during the initial phase of ventilation with mask prior to tracheal intubation. Keeping this limitation in mind, we designed our study to specifically look at the effect of applying PEEP only during the period of pre-oxygenation. In our study, patients who received a PEEP of 5 cm H_2O during pre-oxygenation had higher arterial oxygen tensions following pre-oxygenation than patients who did not receive such a manoeuvre (433.44 ± 75.11 mmHg vs. 355.65 ± 96.55 mmHg). In addition, they also tolerated longer durations of apnoea (496.56 ± 71.68 s vs. 273.00 ± 69.31 s) which was statistically very highly significant. As our patients did not receive any PEEP following muscle paralysis nor were any positive pressure breaths administered after tracheal intubation, we can attribute the observed differences between the groups to be the result of application of CPAP during the 5 min of pre-oxygenation.

Our study was designed to avoid any manoeuvres that could cause alveolar recruitment and reinflate any atelectatic areas that might have occurred during anaesthetic induction. To this end, we intentionally kept our patients apnoeic following neuromuscular blockade and also avoided positive pressure breaths following tracheal intubation. We also closely watched the capnograph waveform as detected by a mainstream capnograph for evidence of spontaneous breathing which could also open up previously atelectatic areas of the lung.

Earlier studies have used fibreoptic endoscopy to confirm correct tracheal tube position,[1,12] which is not only the gold standard for such confirmation but also avoids alveolar recruitment. The mainstream capnograph has also been used to detect the presence of carbon dioxide following the administration of one single small breath of 100% oxygen.[13] As mentioned earlier, such a manoeuvre could open up atelectatic areas of the lung and negate study conditions. We therefore confirmed correct placement of the tracheal tube with the help of the modified oesophageal detector device as this technique does not rely on the use of positive pressure breaths for its performance. Thus, our study conditions were controlled to avoid alveolar recruitment. Hence, the differences observed in the PEEP group as compared to the ZEEP group could be directly attributed to the beneficial effects of PEEP.

This technique of application of positive airway pressure during pre-oxygenation and induction of anaesthesia[14] may have advantages especially in those patients in whom difficulty in airway management is anticipated, those who are more at risk of desaturation such as morbidly obese patients and when assisted ventilation is not applied such as during rapid sequence induction. The application of CPAP or PEEP can have potentially deleterious effects on the cardiovascular system. An earlier study using a CPAP of 7.5 cm H_2O has not reported any adverse effects on systolic and diastolic blood pressure.[13] As we have used a PEEP of 5 cm H_2O during pre-oxygenation, it is unlikely to have produced any significant effects on the heart rate and blood pressure. We observed no changes in heart rate, systolic blood pressure, diastolic blood pressure or mean arterial pressure both in the PEEP group and in the ZEEP group.

We attempted to evaluate any correlation between the period of non-hypoxic apnoea and parameters such as arterial oxygen tension after pre-oxygenation, BMI, age, gender, and smoking. There was no correlation between any of the above parameters and the duration of non-hypoxic apnoea. This was in agreement with a previous study[1] which also found that other than gender, the other parameters showed no correlation to the period of non-hypoxic apnoea. With regard to correlation with gender the same study[1] found that females had a tendency towards shorter duration of non-hypoxic apnoea as compared with males in the PEEP group, but this difference was not observed in the ZEEP group.

Thus, in summary, our study showed that the use of 5 cm H_2O CPAP during pre-oxygenation with 100% oxygen for 5 min provided higher arterial oxygen tensions at the end of pre-oxygenation when compared to patients who breathed spontaneously at ZEEP. In

addition, these patients also had a significantly longer duration of apnoea before they developed arterial desaturation to a SpO_2 of <93%.

This technique could afford some degree of protection against the development of hypoxia during tracheal intubation, especially when one is confronted with difficulty in airway management.

CONCLUSION

In comparison to pre-oxygenation at ambient pressures (zero positive airway pressure), the application of CPAP of 5 cm H_2O using a Mapleson "A" circuit with a fixed PEEP device during 5 min of pre-oxygenation with 100% oxygen prior to the induction of anaesthesia is a safe and simple technique, well-tolerated by patients, provides higher arterial oxygen tensions at the end of pre-oxygenation and a clearly longer duration of apnoea before clinically significant arterial desaturation.

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