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Does apneic oxygenation prevent desaturation during emergency airway management? A systematic review and meta-analysis

Tan E, Loubani O, Kureshi N, Green RS

Abstract

Purpose The purpose of this study was to examine previous studies of apneic oxygenation via nasal cannula during intubations in the ICU, emergency department, and out of hospital to determine whether apneic oxygenation delayed arterial desaturation.

Background Oxygen desaturation occurs not uncommonly during difficult or emergency intubations. Severe hypoxia to a saturation below 80% has been reported to occur in 25% of these intubations. Statistics from the United Kingdom have reported hypoxia as the cause of death in 50% of ICU patients and 27% of emergency department patients who required intubation. Apneic oxygenation is the passive mass movement of oxygen into the lungs without ventilation. It has been described in both animals and humans as a technique to slow oxygen desaturation during apnea. Apneic oxygenation has been used successfully to prevent desaturation during intubations in the OR. Apneic oxygenation in the OR has delayed desaturation by up to 17 minutes.

Methodology This was a systematic review and meta-analysis of cohort studies and randomized controlled trials. Inclusion criteria were: adult patients undergoing emergency intubation in the ICU, emergency department, or out of hospital. Control patients were intubated in the usual manner. Experimental patients received oxygen via nasal cannula at various flow rates depending upon the original study. Patients were excluded if they received bag-valve-mask ventilation before intubation.

Result After examining 544 potential publications, 10 studies were selected for inclusion in the meta-analysis. The 10 studies included 2,322 subjects. Study locations were the ICU (4), emergency department (3), out of hospital (2), and mixed ICU & emergency department (1). All subjects were paralyzed for intubation and most were rapid sequence. Mean time for intubation among the studies ranged from 34 sec. to 150 sec. Preoxygenation with a non-rebreather mask was used in over 80% of patients. Overall, there was a 24% reduction in the risk of desaturation (P=0.02, 95% CI for risk reduction 5% to 39%).

Conclusion Apneic oxygenation via nasal cannula was associated with a 24% reduction in the likelihood of oxygen desaturation during emergency intubation.

Comment We should all be taking advantage of apneic oxygenation during challenging airway management. I’m not sure why this isn’t a routine
part of practice. It has virtually no risk. But I’m a little ahead of myself. Allow me to back up and provide, what to me at least, was remarkable information.

We’ve known for some time that apneic oxygenation works. Basically as long as there is a patent airway and a supply of oxygen at the entry to the airway (glottis) the oxygen will be sucked into the trachea to the alveoli as the oxygen in the alveoli is absorbed into the body. When oxygen is absorbed out of the alveoli the volume, and thus pressure, of gas in the lungs decreases. Since the pressure is lower in the lungs than outside the body, whatever gas is present at the entry to the airway will enter the lungs by mass movement. The alveoli don’t fill with CO$_2$ because it isn’t being eliminated, and therefore the CO$_2$ is distributed equally throughout the body. A 1959 study in humans who had an elective surgical procedure demonstrated just how well apneic oxygenation can work.¹ Eight patients were kept anesthetized and paralyzed with IV agents. They had a (reusable) ETT in place attached to an anesthesia circuit. They had been denitrogenated with 100% oxygen. Next, ventilation was ceased. The patients had 100% oxygen at the entry to their airway but were not ventilated at all. The duration of apnea ranged from 18 minutes to 55 minutes. The lowest arterial oxygen saturation by ABGs was 98%. The highest P$_a$CO$_2$ at the end of the period of apnea was 250 mm Hg in one patient and 160 mm Hg or less in the other patients. The lowest arterial pH was 6.88.

There is no doubt that the 1959 study was a best case scenario. But what it showed was that the phenomenon of apneic oxygenation is real and can work for us. Is it going to work this well when there is no ETT and the oxygen source is a nasal cannula or a catheter placed in the pharynx? No, but it will still work and work better than room air. Of course, patients with less than a best case delivery of oxygen to the airway won’t have a sat of 98% after 30 or 40 or 55 minutes of apnea. But they will usually have a higher oxygen sat for a longer time than if they weren’t receiving apneic oxygenation.

So how can we make this work for us clinically? First, understand that apneic oxygenation works best when patients have been well denitrogenated. Keep in mind that oxygen will not flow through the nasal passages in everyone even if it is running at 15 L/min via a nasal cannula. If you can verify ahead of time that the nasal passages are wide open and/or perhaps administer a decongestant such as oxymetazoline that’s fine. Also, remember that cricoid pressure can compress the trachea. If this happens apneic oxygenation will be impeded to the extent that the trachea is compressed. Consider placing an oxygen cannula through the nasal passage or placing the end of an oxygen cannula through the mouth right into the pharynx. If you are attempting nasal intubation, once the nasal ETT is in the nasal passage blow as much oxygen as possible through it by whatever means available. Some laryngoscope blades have a channel for oxygen built in. If you’ve got one, connect an oxygen source to it. The bottom line is this; pushing oxygen into the hypopharynx during a prolonged intubation attempt is not a wasted step. It provides oxygen to the trachea that will be sucked into the lungs and prolong the time until oxygen desaturation.

Michael A. Fiedler, PhD, CRNA

THE RELATIONSHIP BETWEEN PRE-OPERATIVE HYPERTENSION AND INTRA-OPERATIVE HAEMODYNAMIC CHANGES KNOWN TO BE ASSOCIATED WITH POSTOPERATIVE MORBIDITY

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DOI: 10.1111/anae.14239

Abstract

Purpose The purpose of this study was to determine whether preoperative hypertension was independently associated with perioperative hemodynamic instability and adverse perioperative events. Specifically, the aim was to determine any association between preoperative BP and intraoperative hypotension < 55 mm Hg.

Background Hypertension is thought to affect 30% of the population. It is associated with preventable morbidity and mortality. A prominent European guideline recommends proceeding with elective surgery when the BP is <180/110. This recommendation is based upon limited evidence. However, complications associated with hypertension are known perioperative risk factors, including: coronary artery disease, congestive heart failure, renal failure, cerebrovascular disease, and diabetes. Cardiovascular disease is associated with about a 6% risk of major adverse events in the first 30 days postoperatively. A mean arterial pressure (MAP) < 55 mm Hg for ≥ 1 min. has been associated with myocardial injury and renal injury postoperatively. Hypotension to a MAP of 55 mm Hg or less is not uncommon, occurring in about 18% of elective cases. When MAP was < 55 mm Hg for ≥ 20 min. mortality increased. Similarly, a heart rate (HR) > 100 bpm has been associated with adverse events. However, previous studies have not controlled adequately for other major cardiovascular event risk factors, such as:

• ASA Physical Status
• functional status
• major surgery
• duration of surgery
• blood transfusion

Methodology This was a prospective, multi center, observational study. Data was collected at seven government funded hospitals. Eligibility criteria included adult inpatients scheduled for elective surgery. Exclusion criteria were: ophthalmic, cardiac, obstetric, or pediatric surgery. Data was collected by an attending anesthesiologist unaware of the study purpose.

Preoperative vital signs were recorded the day before surgery. BP was measured three times to establish the baseline. A baseline EKG was assessed for evidence of left ventricular hypertrophy. Immediately preoperatively BP and HR were measured. Intraoperatively the duration and severity of hypotension and tachycardia was recorded along with
the type of anesthesia, class of surgery, duration of surgery, and fluids administered. Sample size needed was estimated to be 500 participants. Intraoperative hypotension and tachycardia were adjusted for ASA physical status, functional status, duration of surgery, transfusion, and major surgery. Statistical analysis was appropriate.

**Result** Data was analyzed from 324 patients. Preoperative hypertension was present in 48% of patients (N=164). Duration of surgery was longer in patients with preoperative hypertension (108 min. vs. 86 min., P<0.01) and these patients also had statistically significantly greater blood loss (183 mL vs. 92 mL, P<0.01). Maintenance antihypertensive medications included diuretics in 62% of patients, ACE inhibitors in 48% of patients, and β-blockers in 33%. BP immediately before induction was >140/90 in 24% of patients (N=81). Pre-induction BPs were significantly higher than baseline BPs the day before.

Hypotension to a BP < 55 mm Hg for > 1 min occurred in 18% of patients (N=59); of these 42% were hypertensive preoperatively. Intraoperatively, only about 1% were hypotensive for more than 20 minutes. None of the patients who were hypotensive for more than 20 minutes had been hypertensive preoperatively. Preoperative hypertension was not independently associated with intraoperative hypertension or tachycardia. Furthermore, none of the cardiovascular risk factors were associated with intraoperative hypertension or tachycardia.

Patients with preoperative hypertension were more likely to have received a vasopressor (P=0.04) and a larger volume of IV fluid (P=0.04) during surgery.

**Conclusion** Preoperative HYPERtension was not associated with intraoperative HYPOtension or tachycardia. Furthermore, this study did not identify an association between any known cardiovascular risk predictors and intraoperative hypotension.

**Comment**
I think we all encounter patients who are hypertensive in the holding area and ask ourselves, “should this surgery proceed?” This is, of course, a team decision, not ours alone. Surgical judgment may deem the risk of waiting to outweigh the possible benefits of better preoperative BP control. Still, none of us wants to go forward and have our patient stroke during emergence because their BP couldn’t be controlled.

<table>
<thead>
<tr>
<th>Systolic BP</th>
<th>Diastolic BP</th>
<th>% of participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;140</td>
<td>&lt;90</td>
<td>51%</td>
</tr>
<tr>
<td>140-159</td>
<td>90-99</td>
<td>34%</td>
</tr>
<tr>
<td>160-179</td>
<td>100-109</td>
<td>12%</td>
</tr>
<tr>
<td>≥180</td>
<td>≥110</td>
<td>3%</td>
</tr>
</tbody>
</table>

**Notes:** BP = blood pressure.
So the study asks a relevant question, “is a preoperative BP below the level of a hypertensive emergency (180/110-120) associated with perioperative adverse events?” I’d love to have a good evidence based answer to this question. To my surprise the investigators focused not on perioperative HYPERtension, but on HYPOtension. But they also focused on “adverse perioperative events,” which is still helpful.

While this study has some value, it definitely has its plusses and minuses. On the plus side, it has (mostly) sound methodology. It was prospective, drew data from multiple centers, had the foresight to obtain baseline preoperative BPs the day before surgery, and the statistical analysis was sound. On the minus side they appeared to have guessed that they needed 500 patients in the study but then only included 324 in the analysis, 65% of what they thought they needed. And, this, without comment or explanation. I think the number of patients included hampered a strong answer to their question because only 15% (N= 49) of their patients had a baseline BP with a systolic greater than 160 and diastolic greater than 100. To study whether or not a higher BP was an independent risk factor for adverse perioperative events one probably needs to include a larger number of patients who were HYPERtensive preoperatively.

So what did I learn from this study? Well, at the very least the study did not produce evidence that a baseline BP pushing 180/110 is, in itself, a risk factor for perioperative adverse events. That evidence may come along later on, but it isn’t in this study. Maybe the surgeons have been right all along and being hypertensive is no reason to cancel the case.

The other thing I picked up on was not studied so we must be careful about how much weight we give it. But I noticed that while there was no association between BP or cardiac risk factors and perioperative adverse events, there was a correlation between baseline hypertension and two other factors; blood loss was approximately doubled in patients who were hypertensive preoperatively, from an average of 92 mL in normotensive patients to 183 mL in hypertensive patients. And, likewise, the duration of surgery was about 25% longer in patients who were hypertensive preoperatively. Perhaps because there was more blood in the field? That, at least, is information useful to us.

Michael A. Fiedler, PhD, CRNA

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**POSITIVE END-EXPIRATORY PRESSURE ALONE MINIMIZES ATELECTASIS FORMATION IN NONABDOMINAL SURGERY**

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DOI: 10.1097/ALN.0000000000002134

**Abstract**

**Purpose** The purpose of this study was to test the hypothesis that PEEP alone was sufficient to prevent atelectasis and to maintain a high ratio of \( P_AO_2 : F_IO_2 \) during nonabdominal surgery.

**Background** Protective ventilation has been conceptualized as the combination of low tidal volume, positive end-expiratory pressure (PEEP), and alveolar recruitment maneuvers during general anesthesia. Whether each of these interventions must be used together or, conversely, one or more provides most or all of the benefits is unknown. Previous studies have produced seemingly conflicting results. Previous work by these investigators have shown little atelectasis in healthy patients who received only moderate PEEP during general anesthesia.

**Methodology** This was a randomized, controlled, blinded study. Inclusion criteria were ASA Physical Status I and II non-obese outpatients 40 years to 75 years old undergoing nonabdominal surgery. Patients were excluded from the study if they had COPD, ischemic heart disease, were current or previous smokers, or had a baseline SpO2 <96% or Hb <10 g/dl. Those with a known or suspected difficult airway were also excluded.

Patients were not premedicated. Each had a radial arterial line to draw ABGs and monitor blood pressure. All patients were preoxygenated for three minutes with 100% oxygen with the head of the table elevated. General anesthesia was provided with an infusion of propofol and remifentanil. Rocuronium was used for paralysis. All patients were ventilated with an F\(_{I}\)O\(_2\) of 0.30 to 0.35 with volume control at 7 mL/Kg ideal body weight. The I:E ratio was set to 1:2 and rate was adjusted to maintain normocapnia. Alveolar recruitment maneuvers were not used. The control group received NO PEEP. The experimental group received 7 cm H\(_2\)O PEEP up to a BMI of 25 Kg/m\(^2\) and 9 cm H\(_2\)O PEEP if their BMI was 26 to 30 Kg/m\(^2\).

The primary outcome variable was the percentage of lung atelectasis at the end of surgery, before emergence. Atelectasis was assessed with computed tomography (CT scan). All CT scans were examined by a single radiologist who was blinded to the group the patient was in. A secondary measure was the efficiency of oxygenation assessed with the ratio of the P\(_A\)O\(_2\) : F\(_I\)O\(_2\). ABGs were drawn before induction, in the middle of surgery, and at the end of surgery. An F\(_{I}\)O\(_2\) of 0.30 to 0.35 was administered to all patients throughout surgery.
**Result**  Atelectasis was present in a median of 1.8% of lung area in the PEEP group (range 0.3% to 9.9%) and 4.6% of lung area in the No-PEEP group (range 1% to 10.2%) (P=0.002). The PEEP group also had a higher P\textsubscript{a}O\textsubscript{2} : F\textsubscript{I}O\textsubscript{2} ratio, indicating more efficient transfer of oxygen from the alveoli to the circulation (P=0.03). The PEEP group also had better lung compliance than the No-PEEP group (P=0.001). Lastly, the No-PEEP group had higher P\textsubscript{a}CO\textsubscript{2} values on ABGs (48 mm Hg vs. 41 mm Hg, P<0.001) despite a higher minute ventilation (5.8 L/min No-PEEP vs. 4.7 L/min PEEP, P=0.002).

**Conclusion**  PEEP reduced atelectasis, improved the efficiency of oxygenation, and improved lung compliance during nonabdominal surgery compared to using no PEEP at all. The PEEP group also had improved CO\textsubscript{2} elimination compared to the No-PEEP group. PEEP was necessary to minimize atelectasis. Using no PEEP resulted in significant atelectasis even in these healthy individuals.

**Comment**  One of the strengths of this study was the highly quantifiable methods used to assess the effects of PEEP; CT scans and ABGs. It would be easy to look at this study and say, “no big deal, these were all really healthy patients having minor procedures.” But it is precisely because the patients were healthy and undergoing minor procedures that the results are so striking. Even in the healthiest patients there were big differences between those who received relatively minimal PEEP and those who didn’t. Patients who didn’t receive PEEP developed atelectasis rather quickly. While the range of lung atelectasis is similar between groups, one look at the box plots in Figure 1 make it clear how large the difference in atelectasis was between groups. While both groups got the same percent inspired oxygen, P\textsubscript{a}O\textsubscript{2}s went UP throughout surgery in the PEEP patients but they went DOWN throughout surgery in the No-PEEP patients. Lastly, despite higher minute ventilation patients who did not receive PEEP had higher P\textsubscript{a}CO\textsubscript{2}s at the end of the case too. I’m left wondering how much worse the picture would have been in less healthy patients having much longer procedures.

Penny Benedict, PhD, CRNA, a respiratory physiologist, taught me a lot. Because of her expertise I began using PEEP during most general anesthetics. I also began using recruitment maneuvers; intermittent manual sigh breaths, to reopen atelactatic alveoli during many types of procedures. This study doesn’t tell us that we don’t ever need to use recruitment maneuvers, but it does show us why we should be using PEEP unless there is a good reason not to.
Atelectasis is the beginning of many a postoperative complication. Based upon this evidence and more I urge you to make PEEP a regular practice during general anesthesia. We used to use PEEP only when we thought there was a good reason to use it. We know better now. Now we should use PEEP unless there is a good reason not to.

Michael A. Fiedler, PhD, CRNA

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