

# Preoxygenation and Prevention of Desaturation During Emergency Airway Management

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Patients requiring emergency airway management are at great risk of hypoxemic hypoxia because of primary lung pathology, high metabolic demands, anemia, insufficient respiratory drive, and inability to protect their airway against aspiration. Tracheal intubation is often required before the complete information needed to assess the risk of periprocedural hypoxia is acquired, such as an arterial blood gas level, hemoglobin value, or even a chest radiograph. This article reviews preoxygenation and peri-intubation oxygenation techniques to minimize the risk of critical hypoxia and introduces a risk-stratification approach to emergency tracheal intubation. Techniques reviewed include positioning, preoxygenation and denitrogenation, positive end expiratory pressure devices, and passive apneic oxygenation. [Ann Emerg Med. 2012;59:165-175.]

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## INTRODUCTION

Maintaining hemoglobin saturation during airway management is critical to patient safety. Desaturation to below 70% puts patients at risk for dysrhythmia, hemodynamic decompensation, hypoxic brain injury, and death.<sup>1,2</sup> The challenge for emergency physicians is to secure a tracheal tube rapidly without critical hypoxia or aspiration. In patients without pulmonary pathology, adequate hemoglobin, or low metabolic demands and an initial pulse oximetry reading of 100% on room air, there is a low risk of desaturation after adequate preoxygenation. Conversely, in a septic patient with multilobar pneumonia who is already hypoxemic (oxygen saturation  $\leq 90\%$ ) despite 100% oxygen at high flow, there is an immediate risk of critical tissue hypoxia during tracheal intubation.

This article reviews preoxygenation and peri-intubation oxygenation techniques to minimize the risk of hypoxemia during emergency tracheal intubation of adult patients. It introduces a risk-stratification approach based on initial pulse oximetry level in response to oxygen administration and provides recommendations about specific techniques based on periprocedural risk. Techniques reviewed include positioning, preoxygenation and denitrogenation, use of positive pressure devices to increase mean airway pressure, and passive apneic oxygenation during tracheal intubation efforts.

## WHAT IS THE RATIONALE FOR PROVIDING PREOXYGENATION BEFORE TRACHEAL INTUBATION?

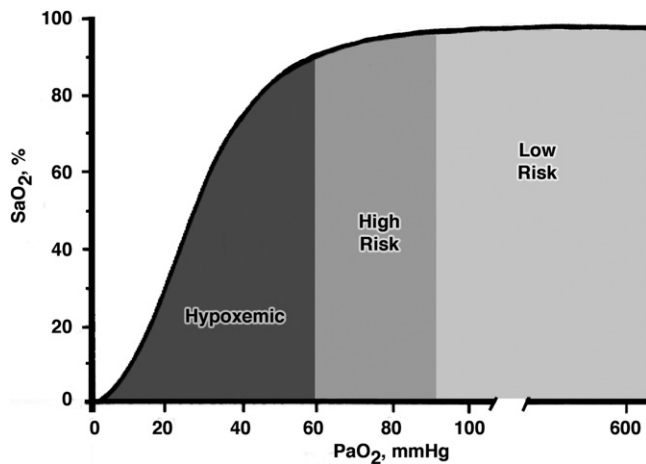
Preoxygenation allows a safety buffer during periods of hypoventilation and apnea. It extends the duration of safe

apnea, defined as the time until a patient reaches a saturation level of 88% to 90%, to allow for placement of a definitive airway. When patients desaturate below this level, their status is on the steep portion of the oxyhemoglobin dissociation curve and can decrease to critical levels of oxygen saturation ( $<70\%$ ) within moments (Figure 1).<sup>3</sup>

The standard anesthesia induction of elective operative patients is performed by administering a sedative, providing manual ventilations, administering a muscle relaxant, and then continuing manual ventilations until placement of a definitive airway. Preoxygenation is not mandatory in these patients because ventilation is continued throughout the induction period and because they have normal physiology and low metabolic needs.

For operative patients with high aspiration risk caused by bowel pathology, body habitus, or critical illness, anesthesiologists developed rapid sequence induction. As originally conceived, this technique is the simultaneous administration of the sedative and paralytic with no ventilation while waiting for the paralytic to take effect, unless needed to prevent hypoxemia. This induction method has been adapted to the emergency department (ED), where all patients requiring airway management are assumed to be at risk for aspiration; our default technique is a rapid sequence tracheal intubation.

In a patient breathing room air before rapid sequence tracheal intubation ( $\text{PaO}_2 \approx 90$  to 100 mm Hg), desaturation will occur in the 45 to 60 seconds between sedative/paralytic administration and airway placement. In the 1950s, anesthesiologists realized that the safest way to perform rapid sequence tracheal intubation would be by filling the patient's alveoli with a high fraction of inspired oxygen ( $\text{FiO}_2$ ) before the



**Figure 1.** Oxyhemoglobin dissociation curve demonstrates the  $\text{SpO}_2$  from various levels of  $\text{PaO}_2$ . Risk categories are overlaid on the curve. Patients near an  $\text{SpO}_2$  of 90% are at risk for precipitous desaturation, as demonstrated by the shape of the curve.

procedure.<sup>4</sup> Studies by Heller and Watson<sup>5</sup> and Heller et al<sup>6</sup> show markedly increased time to desaturation if the patients received preoxygenation with 100% oxygen rather than room air before tracheal intubation.<sup>5,6</sup>

For preoxygenation in the ED, we have 3 goals: (1) to bring the patient's saturation as close to 100% as possible; (2) to denitrogenate the residual capacity of the lungs (maximizing oxygen storage in the lungs); and (3) to denitrogenate and maximally oxygenate the bloodstream.<sup>7</sup> The first 2 goals are imperative; denitrogenating and oxygenating the blood adds little to the duration of safe apnea<sup>8</sup> because oxygen is poorly soluble in blood, and the bloodstream is a comparatively small oxygen reservoir compared with the lungs (5% versus 95%).

Recommendation: Preoxygenation extends the duration of safe apnea and is recommended for every ED tracheal intubation.

### WHAT IS THE BEST SOURCE OF HIGH $\text{FiO}_2$ FOR PREOXYGENATION?

The duration of safe apnea times in most of the preoxygenation literature is predicated on anesthesia circuits that are capable of delivering 90% to 100%  $\text{FiO}_2$  when used with a well-fitting mask. However, the usual source of oxygen during ED preoxygenation is a facemask with an oxygen reservoir. This device is erroneously referred to as the nonrebreather mask despite an absence of 1-way valves covering all of its ports. True nonrebreather masks set at 15 L/minute for patients with normal ventilatory patterns are capable of delivering near 90%  $\text{FiO}_2$ ,<sup>9</sup> but these devices are rarely available in EDs. Standardly available nonrebreather masks at flow rates of 15 L/minute deliver only 60% to 70%  $\text{FiO}_2$ , do not provide complete denitrogenation, and accordingly do not maximize the duration of safe apnea.<sup>9</sup>

Standardly available nonrebreather masks can deliver  $\text{FiO}_2$  greater than or equal to 90% by increasing the flow rate to 30 to 60 L/minute.<sup>10</sup> Such flow rates may be achievable on most flow regulators in EDs by continuing to open the valve, though there will be no calibrated markings beyond 15 L/minute.

Some ED providers use the self-inflating bag-valve-mask device to provide preoxygenation. Bag-valve-mask devices lacking 1-way inhalation and exhalation ports will deliver only close to room air  $\text{FiO}_2$  when not actively assisting ventilations.<sup>11,12</sup> Even with ideal 1-way valves, the devices will deliver oxygen only in 2 circumstances: the patient generates enough inspiratory force to open the valve or the practitioner squeezes the bag. In both circumstances, to obtain any  $\text{FiO}_2$  above that of room air, a tight seal must be achieved with the mask, which usually requires a 2-handed technique.<sup>13</sup> A bag-valve-mask device hovering above the patient's face provides only ambient  $\text{FiO}_2$ .

Recommendation: Standard reservoir facemasks with the flow rate of oxygen set as high as possible are the recommended source of high  $\text{FiO}_2$  for preoxygenation in the ED.

### FOR WHAT PERIOD OF TIME SHOULD THE PATIENT RECEIVE PREOXYGENATION?

Ideally, patients should continue to receive preoxygenation until they denitrogenate the functional residual capacity of their lungs sufficiently to achieve greater than 90% end-tidal oxygen level.<sup>14</sup> Although the mass spectrometers in many EDs allow the measurement of end-tidal oxygen levels, in practice this is rarely performed. Instead, expediency often demands an empiric timing of preoxygenation.

Three minutes' worth of tidal-volume breathing (the patient's normal respiratory pattern) with a high  $\text{FiO}_2$  source is an acceptable duration of preoxygenation for most patients.<sup>15,16</sup> This tidal-volume breathing approach can be augmented by asking the patient to exhale fully before the 3-minute period.<sup>17,18</sup>

Cooperative patients can be asked to take 8 vital-capacity breaths (maximal exhalation followed by maximal inhalation).<sup>16,19,20</sup> This method generally can reduce the preoxygenation time to approximately 60 seconds.<sup>16</sup> Unfortunately, many ill ED patients cannot take vital-capacity breaths.

The above times are predicated on a source of  $\text{FiO}_2$  greater than or equal to 90% and a tightly fitting mask that prevents entrainment of room air.<sup>21</sup>

Recommendation: Patients with an adequate respiratory drive should receive preoxygenation for 3 minutes or take 8 breaths, with maximal inhalation and exhalation.

### CAN INCREASING MEAN AIRWAY PRESSURE AUGMENT PREOXYGENATION?

Mean airway pressure may be increased during preoxygenation through the use of techniques such as

**Table 1.** Evidence for increased mean airway pressure as a preoxygenation technique.

| Study                         | Patients                                                    | Intervention                                                                                                                | Comparator                               | Outcome                                                                                                                                                                                          |
|-------------------------------|-------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------|------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Delay et al <sup>25</sup>     | RCT of 28 obese, operative patients                         | Noninvasive ventilation                                                                                                     | Spontaneous ventilation at zero pressure | The patients in the noninvasive positive-pressure ventilation group achieved faster and more complete denitrogenation than the standard group, as measured by an exhaled oxygen level >90%.      |
| Futier et al <sup>26</sup>    | RCT of 66 obese, operative patients                         | Two treatment groups: noninvasive ventilation or noninvasive ventilation with post-tracheal intubation recruitment maneuver | Spontaneous ventilation at zero pressure | At the end of preoxygenation, PaO <sub>2</sub> was higher in the NPPV and NPPV+RM groups compared with the spont vent group and remained higher after TI and the onset of mechanical ventilation |
| Cressey et al <sup>27</sup>   | RCT of 20 morbidly obese women undergoing bariatric surgery | CPAP preoxygenation                                                                                                         | Spontaneous ventilation at zero pressure | Shown a 40-s increase in time to desaturation through the use of noninvasive positive pressure. Nonsignificant primary outcome.                                                                  |
| Gander et al <sup>28</sup>    | RCT of 30 morbidly obese operative patients                 | CPAP preoxygenation                                                                                                         | Spontaneous ventilation at zero pressure | The time until reaching a saturation of 90% after apnea was extended by a minute in the CPAP group                                                                                               |
| Herriger et al <sup>29</sup>  | RCT of 40 ASA I–II operative patients                       | CPAP preoxygenation                                                                                                         | Spontaneous ventilation at zero pressure | Application of positive airway pressure during induction of anesthesia in adults prolongs the nonhypoxic apnea duration by >2 min                                                                |
| Antonelli et al <sup>30</sup> | RCT of 26 hypoxemic ICU patients requiring bronchoscopy     | Noninvasive ventilation                                                                                                     | Spontaneous ventilation at zero pressure | The PaO <sub>2</sub> /FiO <sub>2</sub> ratio improved in the noninvasive positive-pressure ventilation group and worsened in the high-FiO <sub>2</sub> -alone group                              |

RCT, Randomized controlled trial; NPPV, noninvasive positive-pressure ventilation; RM, recruitment maneuver; CPAP, continuous positive airway pressure; ASA, American Society of Anesthesiologists; TI, tracheal intubation.

noninvasive positive-pressure ventilation. If patients have not achieved a saturation greater than 93% to 95% before tracheal intubation, they have a higher likelihood of desaturation during their apneic and tracheal intubation periods.<sup>2,16,18,22,23</sup> If patients do not achieve this saturation level after 3 minutes of tidal-volume breathing with a high FiO<sub>2</sub> source, it is likely that they are exhibiting shunt physiology; any further augmentation of FiO<sub>2</sub> will be unhelpful.<sup>24</sup> Shunt physiology refers to alveoli that are perfused but not ventilated because of conditions such as pulmonary edema or pneumonia, in which the alveoli are filled with fluid or collapsed. The net effect is a physiologic right-to-left shunt; blood coming from the pulmonary arteries is returned to the pulmonary veins without oxygenation. In the short term, shunt physiology can be partially overcome by augmenting mean airway pressure, thereby improving the effectiveness of preoxygenation and extending the safe apnea time.

Evidence for the use of increased mean airway pressure as a preoxygenation technique can be found in Table 1. In a study

particularly relevant to the ED, Baillard et al<sup>23</sup> examined hypoxemic critically ill patients requiring tracheal intubation in the ICU. At the end of the preoxygenation period, the noninvasive positive-pressure ventilation group had a 98% mean SpO<sub>2</sub>, whereas the standard group had one of 93%. In the noninvasive positive-pressure ventilation group, 6 of 26 patients were unable to improve their hypoxemic saturations with high FiO<sub>2</sub> until they received positive pressure. During the tracheal intubation procedure, the standard group's levels decreased to saturations of 81% compared with 93% in the noninvasive positive-pressure ventilation group. Twelve of the control group and 2 of the noninvasive positive-pressure ventilation group had saturation levels below 80%.

No negative cardiovascular effects<sup>27,31</sup> or appreciable gastric distention was observed in noninvasive positive pressure preoxygenation studies.<sup>25</sup> The latter result was likely due to the low pressures used in these studies; gastric distention and resulting aspiration is unlikely at pressures below 25 cm H<sub>2</sub>O.<sup>32–35</sup> The data on the absence of any cardiovascular side



**Figure 2.** A disposable PEEP valve. This inexpensive item is a strain gauge capable of PEEP settings from 5 to 20 cm H<sub>2</sub>O. When placed on the exhalation port of a bag-valve-mask device (inset), it allows the device to provide PEEP/CPAP when the patient is spontaneously breathing and during assisted ventilations. If combined with a nasal cannula set to 15 L/minute, it will provide CPAP even without ventilations. The generation of positive pressure is predicated on a tight mask seal.

effects may not be applicable to volume-depleted critically ill patients.<sup>36</sup>

The benefits of noninvasive positive-pressure ventilation for preoxygenation in the ED should primarily be observed in patients who cannot achieve acceptable saturations with high FiO<sub>2</sub> alone.<sup>31</sup> If tracheal intubation is attempted with saturations below 90%, these patients can critically desaturate in seconds.<sup>37</sup> Positive pressure may be applied with standalone disposable continuous positive airway pressure (CPAP) masks<sup>38</sup>; with CPAP masks connected to noninvasive machines or standard ventilators; or by using a positive end-expiratory pressure (PEEP) valve on a standard bag mask (Figure 2). Even in departments without immediate CPAP systems, single-use PEEP valves are inexpensive and require minimal modification of practice.

In critically ill patients with very high degrees of shunting, apneic oxygenation (discussed below) alone is unlikely to be helpful.<sup>39</sup> If patients require CPAP during their preoxygenation period, it may benefit them to have the device left on until the moment of tracheal intubation. Whatever predisposed them to experience shunting will recur during the apneic period if they remain at zero airway pressure.<sup>40</sup> PEEP also prevents absorption atelectasis caused by breathing high FiO<sub>2</sub> gas level, increasing the efficacy of apneic oxygenation.<sup>41</sup>

**Recommendation:** CPAP masks, noninvasive positive-pressure ventilation, or PEEP valves on a bag-valve-mask device

should be considered for preoxygenation and ventilation during the onset phase of muscle relaxation in patients who cannot achieve saturations greater than 93% to 95% with high FiO<sub>2</sub>.

## IN WHAT POSITION SHOULD THE PATIENT RECEIVE PREOXYGENATION?

Supine positioning is not ideal to achieve optimal preoxygenation. When one is placed flat, it is more difficult to take full breaths and more of the posterior lung becomes prone to atelectatic collapse,<sup>3</sup> which reduces the reservoir of oxygen contained within the lungs and therefore reduces safe apnea time.

Lane et al<sup>42</sup> performed a randomized controlled trial of patients preoxygenated in a 20-degree head-up position versus a control group that was left supine. After 3 minutes of preoxygenation, the patients received sedation and muscle relaxation and were then allowed to decrease their saturation from 100% to 95%. The head-up group took 386 seconds to reach this saturation versus 283 seconds in the control group.<sup>42</sup> Recently, Ramkumar et al<sup>43</sup> confirmed these results, with the 20-degree head-up group taking 452 seconds to desaturate versus 364 seconds for the supine group.

Altermatt et al<sup>44</sup> examined specifically preoxygenation in obese patients (body mass index >35). This randomized controlled trial compared safe apnea times in intubated patients who received preoxygenation in a sitting position compared with those preoxygenated while lying flat. After rapid sequence induction of anesthesia, the trachea was intubated and the patient was left apneic and disconnected from the anesthesia circuit until SpO<sub>2</sub> level decreased from 100% to 90%. Using the time taken for desaturation to 90% as the outcome, the patients preoxygenated in a sitting position took 214 seconds to desaturate versus 162 seconds for patients preoxygenated while lying flat.<sup>44</sup> Dixon et al<sup>45</sup> showed similar benefits in a third randomized controlled trial in patients with body mass index greater than 40 who were placed in the 25-degree head-up position.

Reverse Trendelenburg position (head of stretcher 30 degrees higher than the foot) also improves preoxygenation and may be useful in patients who cannot bend at the waist or shoulders, ie, those immobilized for possible spinal injury.<sup>46</sup>

An additional benefit of head elevation is better laryngeal exposure during direct laryngoscopy.<sup>47</sup>

**Recommendation:** Patients should receive preoxygenation in a head-elevated position whenever possible. For patients immobilized for possible spinal injury, reverse Trendelenburg position can be used.

## HOW LONG WILL IT TAKE FOR THE PATIENT TO DESATURATE AFTER PREOXYGENATION?

Although breathing at a high FiO<sub>2</sub> level will slightly increase the bloodstream stores of oxygen, the primary benefit of preoxygenation is the creation of a reservoir of oxygen in the alveoli. When a patient is breathing room air,

450 mL of oxygen is present in the lungs; this amount increases to 3,000 mL when a patient breathes 100% oxygen for a sufficient time to replace the alveolar nitrogen. A patient breathing room air will have a total oxygen reservoir in the lungs and bloodstream of approximately 1.0 to 1.5 L, whereas an optimally preoxygenated patient will have 3.5 to 4.0 L.<sup>3</sup> Oxygen consumption during apnea is approximately 250 mL/minute (3 mL/kg per minute); in healthy patients, the duration of safe apnea on room air is approximately 1 minute compared with approximately 8 minutes when breathing at a high  $\text{FiO}_2$  level.<sup>3</sup>

Benumof et al<sup>48</sup> used physiologic modeling to calculate the time to desaturation less than 90% after the administration of succinylcholine for various patient groups after apnea. The times were 8 minutes, 5 minutes, and 2.7 minutes for healthy adults, moderately ill adults, and obese adults, respectively.

The desaturation curves of Benumof et al<sup>48</sup> have been extensively reproduced in the emergency medicine literature, but the extended times in these models are predicated on factors that are very different from those present in many emergency tracheal intubations. The curves assume complete denitrogenation and alveolar concentrations of oxygen at 80%. The model by Benumof et al<sup>48</sup> assumes preoxygenation with a device capable of generating approximately 90%  $\text{FiO}_2$  and optimal time for preoxygenation. It also ignores pulse oximeter lag time. In sick patients, especially those with poor cardiac output, the readings on a finger-probe pulse oximeter may lag behind the actual central arterial circulation by 30 to 60 seconds.<sup>49</sup> The times depicted by the desaturation curves are not applicable to critically ill ED patients or those with poor cardiac output.<sup>50-52</sup>

The effect of shunting, increased metabolic demand, anemia, volume depletion, and decreased cardiac output are synergistic in dramatically reducing oxygen storage in the lungs and shortening safe apnea in critically ill patients. As calculated by Farmery and Roe,<sup>51</sup> desaturation to 85% may be as short as 23 seconds in critically ill patients versus 502 seconds in a healthy adult.<sup>51</sup>

Mort<sup>52</sup> studied ICU patients with blood gas measurements before and after preoxygenation, using bag-valve-mask ventilation. Average  $\text{PaO}_2$  was only 67 mm Hg at baseline and increased to a mean of only 104 mm Hg after 4 minutes of bag-valve-mask ventilation. In less than 20% of patients did preoxygenation increase the  $\text{PaO}_2$  by 50 mm Hg. The author's conclusion was that preoxygenation is only marginally effective in critically ill patients. It is important for clinicians to appreciate that  $\text{PaO}_2$  values in this range are on the steep section of the pulse oximetry curve (Figure 1).

**Recommendation:** Given the unique variables involved in each ED tracheal intubation, it is impossible to predict the exact duration of safe apnea in a patient. Patients with high saturation levels on room air or after oxygen administration are at lower risk and may maintain adequate oxygen

saturation as long as 8 minutes. Critically ill patients and those with values just above the steep edge of the desaturation curve are at high risk of hypoxemia with prolonged tracheal intubation efforts and may desaturate immediately.

## CAN APNEIC OXYGENATION EXTEND THE DURATION OF SAFE APNEA?

Alveoli will continue to take up oxygen even without diaphragmatic movements or lung expansion. In an apneic patient, approximately 250 mL/minute of oxygen will move from the alveoli into the bloodstream. Conversely, only 8 to 20 mL/minute of carbon dioxide moves into the alveoli during apnea, with the remainder being buffered in the bloodstream.<sup>53</sup> The difference in oxygen and carbon dioxide movement across the alveolar membrane is due to the significant differences in gas solubility in the blood, as well as the affinity of hemoglobin for oxygen. This causes the net pressure in the alveoli to become slightly subatmospheric, generating a mass flow of gas from pharynx to alveoli. This phenomenon, called apneic oxygenation, permits maintenance of oxygenation without spontaneous or administered ventilations. Under optimal circumstances, a  $\text{PaO}_2$  can be maintained at greater than 100 mm Hg for up to 100 minutes without a single breath, although the lack of ventilation will eventually cause marked hypercapnia and significant acidosis.<sup>54</sup>

Apneic oxygenation is not a new concept; it has been described in the medical literature for more than a century, with names such as apneic diffusion oxygenation, diffusion respiration, and mass flow ventilation.<sup>55,56</sup> A summary of human evidence for apneic oxygenation is available as a supplement to this article (Table E1, available online at <http://www.annemergmed.com>).

Neurocritical care physicians and neurologists are familiar with the use of apneic oxygenation to prevent desaturation while performing brain death examinations.<sup>63-65</sup> Apneic oxygenation has also been used to continue oxygenation during bronchoscopies and otolaryngeal procedures.<sup>66-68</sup>

Two studies merit specific mention because they extend the technique to situations applicable to emergency tracheal intubation. In a randomized controlled trial of anesthesia patients, Taha et al<sup>69</sup> demonstrated no desaturation during the course of 6 minutes in patients receiving 5 L/minute of oxygen through a nasal catheter. Conversely, the control group desaturated to the study cutoff of 95% in an average of 3.65 minutes. Ramachandran et al<sup>70</sup> performed a randomized controlled trial of obese patients requiring tracheal intubation for elective surgery. The apneic oxygenation group received 5 L/minute of oxygen through nasal cannulas during their apneic period. The apneic oxygenation group had significant prolongation of the time spent with a  $\text{SpO}_2$  greater than or equal to 95% (5.29 versus 3.49 minutes), a significant increase in patients with  $\text{SpO}_2$  greater than or equal to 95% at the 6-minute mark (8 patients versus 1 patient), and significantly higher minimum  $\text{SpO}_2$  (94.3% versus 87.7%).

Apneic oxygenation as described above will allow continued oxygenation but will have no significant effect on carbon dioxide levels. Although extracorporeal carbon dioxide removal, high oscillatory flow, or acid buffers can allow apneic ventilation, as well as oxygenation, these therapies are outside the scope of current ED practice.

To provide apneic oxygenation during ED tracheal intubations, the nasal cannula is the device of choice. Nasal cannulas provide limited  $\text{FiO}_2$  to a spontaneously breathing patient,<sup>71</sup> but the decreased oxygen demands of the apneic state will allow this device to fill the pharynx with a high level of  $\text{FiO}_2$  gas.<sup>70</sup> By increasing the flow rate to 15 L/minute, near 100%  $\text{FiO}_2$  can be obtained. Although providing high flow rates with a conventional, nonhumidified nasal cannula can be uncomfortable because of its desiccating effect on the nasopharynx, after the patient has been sedated it should cause no deleterious effects for the short interval of airway management. Tailor-made high-flow nasal cannulas are also available that will humidify the oxygen, allowing flow rates up to 40 L/minute.<sup>72</sup>

The patients' mouths being open did not negatively affect the  $\text{FiO}_2$  provided.<sup>73</sup> If any potential obstruction caused by redundant nasal tissue is discovered, nasal trumpets will allow a direct conduit from the nasal cannula to the oropharynx.

Although facemasks and nonrebreather masks can provide high  $\text{FiO}_2$  levels to spontaneously breathing patients (depending on flow rates), they provide minimal oxygen to apneic patients,<sup>74</sup> likely because of the 2 exhalation valves venting the fresh gas flow. Bag-valve-mask devices provide no oxygen to apneic patients unless manual ventilations are delivered. Even with manual ventilations, a continuous flow of high-level  $\text{FiO}_2$  will not be available with this device.

Conventional ventilators or noninvasive ventilation machines do not supply a continuous flow of gas when placed on conventional or noninvasive settings. The minimal negative inspiratory pressures generated during apneic oxygenation are insufficient to trigger gas delivery with these machines. Although the high-flow CPAP setups found in many ICUs can provide both positive pressure and high  $\text{FiO}_2$  levels for apneic oxygenation, they are not commonly available in EDs.

An additional benefit to the use of nasal cannula devices is that they can be left on during the tracheal intubation attempts. This has been described with an acronym, NO DESAT (nasal oxygen during efforts securing a tube)<sup>75</sup>; it allows the continued benefits of apneic oxygenation while tracheal intubation techniques are performed. The nasal cannula can be placed under a facemask (or bag-valve-mask device) during preoxygenation, and then it remains on, administering oxygen through the nose throughout oral tracheal intubation with direct or video laryngoscopy.

**Recommendation:** Apneic oxygenation can extend the duration of safe apnea when used after the administration of sedatives and muscle relaxants. A nasal cannula set at 15

L/minute is the most readily available and effective means of providing apneic oxygenation during ED tracheal intubations.

## WHEN AND HOW SHOULD WE PROVIDE MANUAL VENTILATIONS DURING THE APNEIC PERIOD?

Practitioners should not initiate laryngoscopy before full muscle relaxation to maximize laryngeal exposure and to avoid triggering the patient's gag reflex and active vomiting just before apnea. Ventilation provides 2 potential benefits during the onset phase of muscle relaxation: ventilation and increased oxygenation through alveolar distention and reduction in shunting.

The first benefit is minimal in most clinical scenarios. On average,  $\text{PaCO}_2$  increases 8 to 16 mm Hg in the first minute of apnea and then approximately 3 mm Hg/minute subsequently.<sup>53</sup> It is rare that this degree of  $\text{PaCO}_2$  increase and pH decrease will be clinically significant. An exception is a profound metabolic acidosis, such as severe salicylate toxicity, in which patients compensate for the acidosis through hyperpnea and tachypnea. Aggressive ventilation is needed for such patients because cardiovascular collapse with cessation of self-ventilation has been reported.<sup>76</sup> A second exception is in situations of increased intracranial pressure, in which the carbon dioxide increase can lead to cerebral vasodilation.

The second benefit, increased oxygenation, is crucial; patients starting with a pulse oximetry reading of less than or equal to 90% will not tolerate apnea for 60 seconds. Ventilation during the onset phase of muscle relaxation can create alveolar distention and lengthen the duration of safe apnea during tracheal intubation efforts, assuming it is conducted carefully. Bag-valve-mask device inspiratory pressures greater than 25 cm  $\text{H}_2\text{O}$  can overwhelm the esophageal sphincter and put the patient at risk for regurgitation and aspiration.<sup>34,35</sup> When providers are inexperienced or under stress, this pressure is easily exceeded.<sup>77</sup> If ventilations during apnea are required, it may be preferable to use an automated device such as a handheld or conventional ventilator with built-in inspiratory pressure limits and PEEP.<sup>78,79</sup>

If a bag-valve-mask device is used during the onset of muscular relaxation, a PEEP valve will provide sustained alveolar distention. Ventilations should be delivered slowly (during 1 to 2 seconds), involve a low volume (6 to 7 mL/kg), and be administered at as low a rate as tolerable for the clinical circumstances (6 to 8 breaths/min).<sup>80</sup> Although not clinically proven, there may be a benefit to head elevation in reducing the risk of passive regurgitation in such patients, in addition to the significant physiologic benefits of oxygenation in a head-elevated position.

A significant risk of positive-pressure ventilation in the critically ill patient involves decreased venous return and hypotension. This is especially significant in low flow states from any cause (hypotension), volume depletion, acute respiratory distress syndrome, and obstructive airway disease (with attendant risks of intrinsic PEEP). Overventilation in such

patients may precipitate hemodynamic collapse, and clinicians must be mindful of rate, volume, and speed of ventilation in these situations. Relative hypoventilation and resultant permissive hypercapnia may be required to avoid hemodynamic collapse.<sup>81</sup>

**Recommendation:** The risk/benefit of active ventilation during the onset phase of muscle relaxants must be carefully assessed in each patient. In patients at low risk for desaturation (>95% saturation), manual ventilation is not necessary. In patients at higher risk (91% to 95% saturation), a risk-benefit assessment should include an estimation of desaturation risk and the presence of pulmonary pathology. In hypoxemic patients, low-pressure, low-volume, low-rate ventilations will be required.

## WHAT POSITIONING AND MANEUVERS SHOULD THE PATIENT RECEIVE DURING THE APNEIC PERIOD?

Apneic oxygenation requires a patent airway for oxygen to reach the hypopharynx and be entrained into the trachea; once the patient is sedated and paralyzed, it is imperative to keep the posterior pharyngeal structures and tongue from occluding the passage of gas. Head elevation, chin lift, and jaw thrust will accomplish this in most patients; a jaw thrust alone should be used if there is risk for cervical spine injury. In some patients, a nasal trumpet or oral airway may also be required. Patients with sleep apnea or obesity often need a combination of jaw distraction, lifting of submandibular soft tissue, and nasal or oral airways.

Positioning the patient with their external auditory meatus on the same horizontal plane as their sternal notch maximizes upper airway dimensions and facilitates direct laryngoscopy.<sup>82,83</sup> Head elevation relative to the thorax also permits optimal jaw distraction, and conversely atlanto-occipital extension pivots the base of tongue and epiglottis against the posterior pharynx and promotes obstruction. In all but the thinnest patients, a head-elevated position requires lifting of the head of the bed somewhat, plus padding under the head and upper shoulders. The face plane of the patient should be parallel to the ceiling. For the superobese, this positioning requires a very large ramp. In cervical spine precautions, elevating the head relative to the neck is not possible, but as previously noted the foot of the stretcher should be tilted downward to improve pulmonary function.

Cricoid pressure, considered an essential aspect of rapid sequence tracheal intubation when it was first conceived, has come under increasing scrutiny within anesthesia and emergency medicine.<sup>84,85</sup> Theoretically, the application of firm pressure to the cricoid cartilage compresses the esophagus while keeping the trachea patent, but in practice this is not always the case.<sup>86</sup> Computed tomography and magnetic resonance imaging scanning have shown that cricoid pressure causes lateral displacement of the esophagus in more than 90% of patients and laryngeal/tracheal compression in 80%.<sup>87,88</sup> Numerous ventilation studies have found that cricoid pressure hinders bag-

valve-mask device ventilation, increases peak inspiratory pressure, and reduces tidal volumes.<sup>34,84,89-94</sup> For the same reasons that the airway obstruction induced by cricoid pressure may preclude effective manual ventilation, it may limit the effectiveness of apneic oxygenation as well.

**Recommendation:** Patients should be positioned to maximize upper airway patency before and during the apneic period, using ear-to-sternal notch positioning. Nasal airways may be needed to create a patent upper airway. Once the apneic period begins, the posterior pharyngeal structures should be kept from collapsing backwards by using a jaw thrust. Cricoid pressure may negatively affect apneic oxygenation, but studies examining this question in the setting of modern emergency airway management do not exist to our knowledge.

## DOES THE CHOICE OF PARALYTIC AGENT AFFECT PREOXYGENATION?

The choice of paralytic agent may influence the time to desaturation during airway management. In a study of operative patients, the time to desaturation to 95% was 242 seconds in patients receiving succinylcholine versus 378 seconds in a group given rocuronium.<sup>95</sup> Similarly, in obese patients undergoing surgery, the succinylcholine group desaturated to 92% in 283 seconds versus 329 seconds in the rocuronium group.<sup>96</sup> When used at a dose of greater than or equal to 1.2 mg/kg, rocuronium provides intubating conditions identical to those of succinylcholine.<sup>97</sup>

It is hypothesized that the fasciculations induced by succinylcholine may cause increased oxygen use. Pretreatment medications to prevent fasciculations minimize the difference in desaturation times.<sup>95</sup>

**Recommendation:** In patients at high risk of desaturation, rocuronium may provide a longer duration of safe apnea than succinylcholine.

## RISK STRATIFICATION AND CONCLUSIONS

Patients requiring emergency airway management can be risk stratified into 3 groups, according to pulse oximetry after initial application of high-flow oxygen. The recommended techniques to use for patients in each group are shown in Table 2, and a logistic flow of preoxygenation steps is shown in Figure 3.

Head-elevated positioning is simple and easy to apply in all patients; in the patient immobilized for cervical spine injury, it is beneficial to tilt the foot of the bed downward.

Patients at lowest risk (saturation of 96% to 100%) should be properly positioned and receive preoxygenation; active ventilation is not needed. Passive apneic oxygenation during tracheal intubation efforts may not be necessary in these low-risk patients, but it will extend safe apnea in the event of repeated tracheal intubation attempts.

Patients in the 91% to 95% saturation group after receiving high FiO<sub>2</sub> levels are at high risk of critical desaturation during

**Table 2.** Risk categorization of patients during preoxygenation.\*

| <b>Risk Category, Based on Pulse Oximetry While Receiving High-Flow Oxygen</b> | <b>Preoxygenation Period (3 Minutes)</b>                      | <b>Onset of Muscle Relaxation (≈60 Seconds)</b>                                           | <b>Apneic Period During Tracheal Intubation (Variable Duration, Depending on Airway Difficulty; Ideally &lt;30 Seconds)</b> |
|--------------------------------------------------------------------------------|---------------------------------------------------------------|-------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------|
| Low risk, SpO <sub>2</sub> 96%–100%                                            | Nonrebreather mask with maximal oxygen flow rate              | Nonrebreather mask and nasal oxygen at 15 L/min                                           | Nasal oxygen at 15 L/min                                                                                                    |
| High risk, SpO <sub>2</sub> 91%–95%                                            | Nonrebreather mask or CPAP or bag-valve-mask device with PEEP | Nonrebreather mask, CPAP, or bag-valve-mask device with PEEP and nasal oxygen at 15 L/min | Nasal oxygen at 15 L/min                                                                                                    |
| Hypoxemic, SpO <sub>2</sub> 90% or less                                        | CPAP or bag-valve-mask device with PEEP                       | CPAP or bag-valve-mask device with PEEP and nasal oxygen at 15 L/min                      | Nasal oxygen at 15 L/min                                                                                                    |

\*Risk categories are based on patient's initial response to high-flow oxygen through a tightly fitting nonrebreather mask. Patients who are already hypoxemic exhibit shunt physiology and are prone to rapid desaturation during the peri-intubation. Patients with saturations of 91% to 95% have values close to the precipice of the steep portion of the oxyhemoglobin dissociation curve and should be considered high risk. Patients with saturations greater than or equal to 96% are at low risk for peri-intubation desaturation. Patients in all risk categories should receive preoxygenation in a head-elevated position (or reverse-Trendelenburg if there is a risk of spine injury).

emergency tracheal intubation. Positioning, preoxygenation, and passive oxygenation should be used. For these patients, consideration should be given to using PEEP during preoxygenation and while awaiting muscle relaxation, but the

risks and benefits of these techniques must be assessed case by case.

For patients initially hypoxemic with high FiO<sub>2</sub> levels (saturation of 90% or less), aggressive efforts must be made to

### Sequence of Preoxygenation and Prevention of Desaturation

(Assuming 2 oxygen regulators\*)

#### Preoxygenation Period

- Position the patient in a semi-recumbent position (~20°) or in reverse Trendelenburg. Position the patient's head in the ear-to-sternal-notch position using padding if necessary.
- Place a nasal cannula in the patient's nares. Do not hook the nasal cannula to oxygen regulator.
- Place patient on a non-rebreather mask at the maximal flow allowed by the oxygen regulator (at least 15 lpm, but many allow a much greater uncalibrated flow)
- If patient is not saturating > 90%, remove face mask and switch to non-invasive CPAP by using ventilator, non-invasive ventilation machine, commercial CPAP device, or BVM with PEEP valve attached. Titrate between 5-15 cm H<sub>2</sub>O of PEEP to achieve an oxygen saturation > 98%. Consider this step in patients saturating 91-95%.
- Allow patient to breathe at tidal volume for 3 minutes or ask the patient to perform 8 maximal exhalations and inhalations
- Attach a BVM to oxygen regulator and set it to maximal flow (at least 15 lpm). If the patient required CPAP for preoxygenation, attach a PEEP valve to the BVM set at the patient's current CPAP level

#### Apneic Period

- Push sedative and paralytic (preferably rocuronium, if the patient is at risk for rapid desaturation)
- Detach face mask from the oxygen regulator and attach the nasal cannula. Drop the flow rate to 15 lpm.
- Remove the face mask from the patient.
- Perform a jaw thrust to maintain pharyngeal patency.
- If the patient is high risk (required CPAP for preoxygenation), consider leaving on the CPAP during the apneic period or providing 4-6 ventilations with the BVM with a PEEP valve attached. Maintain a two-hand mask seal during the entire apneic period to maintain the CPAP.

#### Intubation Period

- Leave the nasal cannula on throughout the airway management period to maintain apneic oxygenation.

\* If 3 regulators are available, attach reservoir face mask, BVM, and nasal cannula to them. If only one regulator is available, consider using a stand-alone oxygen tank to offer a second source of oxygen.

**Figure 3.** Logistic flow of preoxygenation and prevention of desaturation.

maximize saturation before tracheal intubation. These patients will require PEEP during preoxygenation, ventilation during the onset of phase of muscle relaxants, and passive oxygenation during tracheal intubation.

Videos of the techniques described in this article can be found at <http://emcrit.org/preoxygenation>.

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## REFERENCES

1. Mort TC. The incidence and risk factors for cardiac arrest during emergency tracheal intubation: a justification for incorporating the ASA guidelines in the remote location. *J Clin Anesth*. 2004;16:508-516.
2. Davis DP, Hwang JQ, Dunford JV. Rate of decline in oxygen saturation at various pulse oximetry values with prehospital rapid sequence intubation. *Prehosp Emerg Care*. 2008;12:46-51.
3. Lumb AB. *Nunn's Applied Respiratory Physiology*. 7th ed. Oxford: Churchill Livingstone; 2010:568.
4. Morton HJ, Wylie WD. Anaesthetic deaths due to regurgitation or vomiting. *Anaesthesia*. 1951;6:190-201.
5. Heller ML, Watson TR Jr. Polarographic study of arterial oxygenation during apnea in man. *N Engl J Med*. 1961;264:326-330.
6. Heller ML, Watson TR Jr, Imredy DS. Apneic oxygenation in man: polarographic arterial oxygen tension study. *Anesthesiology*. 1964;25:25-30.
7. Campbell IT, Beatty PC. Monitoring preoxygenation. *Br J Anaesth*. 1994;72:3-4.
8. Baraka A, Ramez Salem M. Preoxygenation. In: Hagberg CA, ed. *Benumof's Airway Management: Principles and Practice*. 2nd ed. Philadelphia, PA: Mosby; 2007:303-318.
9. Vender JS, Szokol JW. Oxygen delivery systems, inhalation therapy, and respiratory therapy. In: Hagberg CA, ed. *Benumof's Airway Management: Principles and Practice*. 2nd ed. Philadelphia, PA: Mosby; 2007:321-345.
10. Earl JW. Delivery of high FiO<sub>2</sub>. Available at: <http://www.rcjournal.com/abstracts/2003/?id=OF-03-257>. Accessed April 11, 2011.
11. Nimmagadda U, Salem MR, Joseph NJ, et al. Efficacy of preoxygenation with tidal volume breathing. Comparison of breathing systems. *Anesthesiology*. 2000;93:693-698.
12. Mills PJ, Baptiste J, Preston J, et al. Manual resuscitators and spontaneous ventilation—an evaluation. *Crit Care Med*. 1991;19:1425-1431.
13. Joffe AM, Hetzel S, Liew EC. *Anesthesiology*. 2010;113:873-879.
14. Bhatia PK, Bhandari SC, Tulsiani KL, et al. End-tidal oxygraphy and safe duration of apnoea in young adults and elderly patients. *Anaesthesia*. 1997;52:175-178.
15. Hamilton WK, Eastwood DW. A study of denitrogenation with some inhalation anesthetic systems. *Anesthesiology*. 1955;16:861-867.
16. Baraka AS, Taha SK, Aouad MT, et al. Preoxygenation: comparison of maximal breathing and tidal volume breathing techniques. *Anesthesiology*. 1999;91:612-616.
17. Baraka AS, Taha SK, El-Khatib MF, et al. Oxygenation using tidal volume breathing after maximal exhalation. *Anesth Analg*. 2003;97:1533-1535.
18. Baraka A, Haroun-Bizri S, Khoury S, et al. Single vital capacity breath for preoxygenation. *Can J Anaesth*. 2000;47:1144-1146.
19. Pandit JJ, Duncan T, Robbins PA. Total oxygen uptake with two maximal breathing techniques and the tidal volume breathing technique: a physiologic study of preoxygenation. *Anesthesiology*. 2003;99:841-846.
20. Rapaport S, Joannes-Boyau O, Bazin R, et al. [Comparison of eight deep breaths and tidal volume breathing preoxygenation techniques in morbid obese patients]. *Ann Fr Anesth Reanim*. 2004;23:1155-1159.
21. Benumof JL. Preoxygenation: best method for both efficacy and efficiency. *Anesthesiology*. 1999;91:603-605.
22. El-Khatib MF, Kanazi G, Baraka AS. Noninvasive bilevel positive airway pressure for preoxygenation of the critically ill morbidly obese patient. *Can J Anaesth*. 2007;54:744-747.
23. Baillard C, Fosse JP, Sebbane M, et al. Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients. *Am J Respir Crit Care Med*. 2006;174:171-177.
24. Nunn JF. *Applied Respiratory Physiology*. 4th ed. Oxford, England: Butterworth-Heinemann; 1995:156-197.
25. Delay JM, Sebbane M, Jung B, et al. The effectiveness of noninvasive positive pressure ventilation to enhance preoxygenation in morbidly obese patients: a randomized controlled study. *Anesth Analg*. 2008;107:1707-1713.
26. Futier E, Constantin JM, Pelosi P, et al. Noninvasive ventilation and alveolar recruitment maneuver improve respiratory function during and after intubation of morbidly obese patients: a randomized controlled study. *Anesthesiology*. 2011;114:1354-1363.
27. Cressey DM, Berthoud MC, Reilly CS. Effectiveness of continuous positive airway pressure to enhance pre-oxygenation in morbidly obese women. *Anaesthesia*. 2001;56:680-684.
28. Gander S, Frascarolo P, Suter M, et al. Positive end-expiratory pressure during induction of general anesthesia increases duration of nonhypoxic apnea in morbidly obese patients. *Anesth Analg*. 2005;100:580-584.
29. Herriger A, Frascarolo P, Spahn DR, et al. The effect of positive airway pressure during pre-oxygenation and induction of anaesthesia upon duration of non-hypoxic apnoea. *Anaesthesia*. 2004;59:243-247.
30. Antonelli M, Conti G, Rocco M, et al. Noninvasive positive-pressure ventilation versus conventional oxygen supplementation in hypoxemic patients undergoing diagnostic bronchoscopy. *Chest*. 2002;121:1149-1154.
31. Jaber S, Jung B, Corne P, et al. An intervention to decrease complications related to endotracheal intubation in the intensive care unit: a prospective, multiple-center study. *Intensive Care Med*. 2010;36:248-255.
32. Vyas H, Milner AD, Hopkin IE. Face mask resuscitation: does it lead to gastric distension? *Arch Dis Child*. 1983;58:373-375.
33. Ho-Tai LM, Devitt JH, Noel AG, et al. Gas leak and gastric insufflation during controlled ventilation: face mask versus laryngeal mask airway. *Can J Anaesth*. 1998;45:206-211.

34. Lawes EG, Campbell I, Mercer D. Inflation pressure, gastric insufflation and rapid sequence induction. *Br J Anaesth*. 1987; 59:315-318.
35. Ruben H, Knudsen EJ, Carugati G. Gastric inflation in relation to airway pressure. *Acta Anaesthesiol Scand*. 1961;5:107-114.
36. Jellinek H, Krafft P, Fitzgerald RD, et al. Right atrial pressure predicts hemodynamic response to apneic positive airway pressure. *Crit Care Med*. 2000;28:672-678.
37. Tanoubi I, Drolet P, Donati F. Optimizing preoxygenation in adults. *Can J Anaesth*. 2009;56:449-466.
38. Leman P, Greene S, Whelan K, et al. Simple lightweight disposable continuous positive airways pressure mask to effectively treat acute pulmonary oedema: randomized controlled trial. *Emerg Med Australas*. 2005;17:224-230.
39. Engstrom J, Hedenstierna G, Larsson A. Pharyngeal oxygen administration increases the time to serious desaturation at intubation in acute lung injury: an experimental study. *Crit Care*. 2010;14:R93.
40. Neumann P, Berglund JE, Fernandez Mondejar E, et al. Dynamics of lung collapse and recruitment during prolonged breathing in porcine lung injury. *J Appl Physiol*. 1998;85:1533-1543.
41. Rothen HU, Sporre B, Engberg G, et al. Prevention of atelectasis during general anaesthesia. *Lancet*. 1995;345:1387-1391.
42. Lane S, Saunders D, Schofield A, et al. A prospective, randomised controlled trial comparing the efficacy of pre-oxygenation in the 20 degrees head-up vs supine position. *Anaesthesia*. 2005;60:1064-1067.
43. Ramkumar V, Umesh G, Philip FA. Preoxygenation with 20° head-up tilt provides longer duration of non-hypoxic apnea than conventional preoxygenation in non-obese healthy adults. *J Anesth*. 2011;25:189-194.
44. Altermatt FR, Munoz HR, Delfino AE, et al. Pre-oxygenation in the obese patient: effects of position on tolerance to apnoea. *Br J Anaesth*. 2005;95:706-709.
45. Dixon BJ, Dixon JB, Carden JR, et al. Preoxygenation is more effective in the 25 degrees head-up position than in the supine position in severely obese patients: a randomized controlled study. *Anesthesiology*. 2005;102:1110-1115; discussion 1115A.
46. Boyce JR, Ness T, Castroman P, et al. A preliminary study of the optimal anesthesia positioning for the morbidly obese patient. *Obes Surg*. 2003;13:4-9.
47. Lee BJ, Kang JM, Kim DO. Laryngeal exposure during laryngoscopy is better in the 25 degrees back-up position than in the supine position. *Br J Anaesth*. 2007;99:581-586.
48. Benumof JL, Dagg R, Benumof R. Critical hemoglobin desaturation will occur before return to an unparalyzed state following 1 mg/kg intravenous succinylcholine. *Anesthesiology*. 1997;87:979-982.
49. Ding ZN, Shibata K, Yamamoto K, et al. Decreased circulation time in the upper limb reduces the lag time of the finger pulse oximeter response. *Can J Anaesth*. 1992;39:87-89.
50. Drummond GB, Park GR. Arterial oxygen saturation before intubation of the trachea. An assessment of oxygenation techniques. *Br J Anaesth*. 1984;56:987-993.
51. Farmery AD, Roe PG. A model to describe the rate of oxyhaemoglobin desaturation during apnoea. *Br J Anaesth*. 1996; 76:284-291.
52. Mort TC. Preoxygenation in critically ill patients requiring emergency tracheal intubation. *Crit Care Med*. 2005;33:2672-2675.
53. Eger EI, Severinghaus JW. The rate of rise of PaCO<sub>2</sub> in the apneic anesthetized patient. *Anesthesiology*. 1961;22:419-425.
54. Nielsen ND, Kjaergaard B, Koefoed-Nielsen J, et al. Apneic oxygenation combined with extracorporeal arteriovenous carbon dioxide removal provides sufficient gas exchange in experimental lung injury. *ASAIO J*. 2008;54:401-405.
55. Hirsch H. *Über künstliche Atmung durch Ventilation der Trachea* [dissertation]. Giessen; 1905.
56. Volhard F. Über künstliche Atmung durch Ventilation der Trachea und eine einfache Vorrichtung zur hytmischen künstlichen Atmung. *Munch med Wochensh*. 1908;209-211.
57. Comroe JH Jr, Dripps RD. Artificial respiration. *JAMA*. 1946;130: 381-383.
58. Enghoff H, Holmdahl MH, Risholm L. Oxygen uptake in human lungs without spontaneous or artificial pulmonary ventilation. *Acta Chir Scand*. 1952;103:293-301.
59. Holmdahl MH. Pulmonary uptake of oxygen, acid-base metabolism, and circulation during prolonged apnoea. *Acta Chir Scand Suppl*. 1956;212:1-128.
60. Frumin MJ, Epstein RM, Cohen G. Apneic oxygenation in man. *Anesthesiology*. 1959;20:789-798.
61. Babinski MF, Sierra OG, Smith RB, et al. Clinical application of continuous flow apneic ventilation. *Acta Anaesthesiol Scand*. 1985;29:750-752.
62. Teller LE, Alexander CM, Frumin MJ, et al. Pharyngeal insufflation of oxygen prevents arterial desaturation during apnea. *Anesthesiology*. 1988;69:980-982.
63. al Jumah M, McLean DR, al Rajeh S, et al. Bulk diffusion apnea test in the diagnosis of brain death. *Crit Care Med*. 1992;20: 1564-1567.
64. Marks SJ, Zisfein J. Apneic oxygenation in apnea tests for brain death. A controlled trial. *Arch Neurol*. 1990;47:1066-1068.
65. Perel A, Berger M, Cotev S. The use of continuous flow of oxygen and PEEP during apnea in the diagnosis of brain death. *Intensive Care Med*. 1983;9:25-27.
66. Barth L. [Therapeutic use of diffusion breathing in bronchoscopy]. *Anaesthesist*. 1954;3:227-229.
67. Payne JP. Apnoeic oxygenation in anaesthetised man. *Acta Anaesthesiol Scand*. 1962;6:129-142.
68. Lee SC. Improvement of gas exchange by apneic oxygenation with nasal prong during fiberoptic intubation in fully relaxed patients. *J Korean Med Sci*. 1998;13:582-586.
69. Taha SK, Siddik-Sayyid SM, El-Khatib MF, et al. Nasopharyngeal oxygen insufflation following pre-oxygenation using the four deep breath technique. *Anaesthesia*. 2006;61:427-430.
70. Ramachandran SK, Cosnowski A, Shanks A, et al. Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration. *J Clin Anesth*. 2010;22:164-168.
71. Kory RC, Bergmann JC, Sweet RD, et al. Comparative evaluation of oxygen therapy techniques. *JAMA*. 1962;179:767-772.
72. Waugh JB, Granger WM. An evaluation of 2 new devices for nasal high-flow gas therapy. *Respir Care*. 2004;49:902-906.
73. Wettstein RB, Shelledy DC, Peters JL. Delivered oxygen concentrations using low-flow and high-flow nasal cannulas. *Respir Care*. 2005;50:604-609.
74. Tiep B, Barnett M. High flow nasal vs high flow mask oxygen delivery: tracheal gas concentrations through a head extension airway model. In: *Respiratory Care 2002 Open Forum 2002*; October 5-8, 2002; Tampa, FL.
75. Levitan RM. NO DESAT! Nasal oxygen during efforts securing a tube. *Emergency Physicians Monthly*. December 9, 2010. Available at: <http://www.epmonthly.com/features/current-features/no-desat/>. Accessed October 22, 2011.
76. Stolbach AI, Hoffman RS, Nelson LS. Mechanical ventilation was associated with acidemia in a case series of salicylate-poisoned patients. *Acad Emerg Med*. 2008;15:866-869.

77. Aufderheide TP, Lurie KG. Death by hyperventilation: a common and life-threatening problem during cardiopulmonary resuscitation. *Crit Care Med*. 2004;32:S345-351.
78. von Goedecke A, Voelckel WG, Wenzel V, et al. Mechanical versus manual ventilation via a face mask during the induction of anesthesia: a prospective, randomized, crossover study. *Anesth Analg*. 2004;98:260-263.
79. von Goedecke A, Wenzel V, Hormann C, et al. Effects of face mask ventilation in apneic patients with a resuscitation ventilator in comparison with a bag-valve-mask. *J Emerg Med*. 2006;30:63-67.
80. von Goedecke A, Wagner-Berger HG, Stadlbauer KH, et al. Effects of decreasing peak flow rate on stomach inflation during bag-valve-mask ventilation. *Resuscitation*. 2004;63:131-136.
81. Oddo M, Feihl F, Schaller MD, et al. Management of mechanical ventilation in acute severe asthma: practical aspects. *Intensive Care Med*. 2006;32:501-510.
82. Collins JS, Lemmens HJ, Brodsky JB, et al. Laryngoscopy and morbid obesity: a comparison of the "sniff" and "ramped" positions. *Obes Surg*. 2004;14:1171-1175.
83. Levitan RM, Mechem CC, Ochroch EA, et al. Head-elevated laryngoscopy position: improving laryngeal exposure during laryngoscopy by increasing head elevation. *Ann Emerg Med*. 2003;41:322-330.
84. Ellis DY, Harris T, Zideman D. Cricoid pressure in emergency department rapid sequence tracheal intubations: a risk-benefit analysis. *Ann Emerg Med*. 2007;50:653-665.
85. Brimacombe JR, Berry AM. Cricoid pressure. *Can J Anaesth*. 1997;44:414-425.
86. Petito SP, Russell WJ. The prevention of gastric inflation—a neglected benefit of cricoid pressure. *Anaesth Intensive Care*. 1988;16:139-143.
87. Smith KJ, Dobranowski J, Yip G, et al. Cricoid pressure displaces the esophagus: an observational study using magnetic resonance imaging. *Anesthesiology*. 2003;99:60-64.
88. Smith KJ, Ladak S, Choi PT, et al. The cricoid cartilage and the esophagus are not aligned in close to half of adult patients. *Can J Anaesth*. 2002;49:503-507.
89. Allman KG. The effect of cricoid pressure application on airway patency. *J Clin Anesth*. 1995;7:197-199.
90. Hartsilver EL, Vanner RG. Airway obstruction with cricoid pressure. *Anaesthesia*. 2000;55:208-211.
91. Hocking G, Roberts FL, Thew ME. Airway obstruction with cricoid pressure and lateral tilt. *Anaesthesia*. 2001;56:825-828.
92. Saghaei M, Masoodifar M. The pressor response and airway effects of cricoid pressure during induction of general anesthesia. *Anesth Analg*. 2001;93:787-790.
93. Mac GPHJ, Ball DR. The effect of cricoid pressure on the cricoid cartilage and vocal cords: an endoscopic study in anaesthetised patients. *Anaesthesia*. 2000;55:263-268.
94. Palmer JH, Yentis SM. Cricoid pressure application to awake volunteers: discomfort cannot be used to indicate appropriate force. *Can J Anaesth*. 2005;52:114-115.
95. Taha SK, El-Khatib MF, Baraka AS, et al. Effect of suxamethonium vs rocuronium on onset of oxygen desaturation during apnoea following rapid sequence induction. *Anaesthesia*. 2010;65:358-361.
96. Tang L, Li S, Huang S, et al. Desaturation following rapid sequence induction using succinylcholine versus rocuronium in overweight patients. *Acta Anaesthesiol Scand*. 2011;55:203-208.
97. Perry JJ, Lee JS, Sillberg VA, et al. Rocuronium versus succinylcholine for rapid sequence induction intubation. *Cochrane Database Syst Rev*. 2008;(2):CD002788.

**Table E1.** Evidence for apneic oxygenation to extend the duration of safe apnea.

| Study                            | Patients                                         | Intervention                                                          | Comparator                                    | Outcome                                                                                                                                                                                                                                                                                                |
|----------------------------------|--------------------------------------------------|-----------------------------------------------------------------------|-----------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Comroe et al <sup>57</sup>       | Obs trial of 2 patients with neurologic injuries | Endotracheal insufflation of 6–11 L/min of oxygen                     | None                                          | Observation of long duration till SaO <sub>2</sub> <90% in numerous separate experiments                                                                                                                                                                                                               |
| Enghoff et al <sup>58</sup>      | Obs study of 7 operative patients                | Tubing placed down ET tube connected to 100% FiO <sub>2</sub>         | None                                          | No decrease in ABG PaO <sub>2</sub> for 5–7 min                                                                                                                                                                                                                                                        |
| Holmdahl <sup>59</sup>           | Documentation of separate Obs studies            | Endotracheal insufflation of 100% FiO <sub>2</sub>                    | None                                          | Extended time until desaturation in separate studies                                                                                                                                                                                                                                                   |
| Frumin et al <sup>60</sup>       | Obs study of 8 operative patients                | Intubated, administered 100% FiO <sub>2</sub> and left apneic         | None                                          | No desaturation for between 18 and 55 min. Patients had marked hypercapnia and commensurate decreased pH.                                                                                                                                                                                              |
| Babinski et al <sup>61</sup>     | Obs study of 5 operative patients                | Two endobronchial catheters placed                                    | None                                          | 30 min of apnea without significant desaturation                                                                                                                                                                                                                                                       |
| Teller et al <sup>62</sup>       | RCT, blinded, crossover trial of 12 pts          | Nasopharyngeal catheters attached to 100% FiO <sub>2</sub> at 3 L/min | Nasopharyngeal catheters attached to room air | Outcome was a sat of ≤92% or 10 min. None of the patients in the insufflation arm desaturated below 98% during the 10 min.                                                                                                                                                                             |
| Taha et al <sup>69</sup>         | RCT of 30 pts                                    | Nasal catheters attached to 5L/min of 100% FiO <sub>2</sub>           | Room air                                      | No desaturation during the course of the 6-min predetermined stopping point in patients receiving apneic oxygenation. The control group desaturated to the study cutoff of 95% in an average of 3.65 min.                                                                                              |
| Ramachandran et al <sup>70</sup> | RCT of 30 obese, operative patients              | Nasal cannula attached to 5 L/min 100% FiO <sub>2</sub>               | Room air                                      | The apneic oxygenation group had significant prolongation of SpO <sub>2</sub> ≥95% time (5.29 versus 3.49 min), a significant increase in patients with SpO <sub>2</sub> ≥95% at the 6-min mark (8 patients versus 1 patient), and significantly higher minimum SpO <sub>2</sub> (94.3% versus 87.7%). |

Obs, Observational trial.