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. 2011;xx:xxx.]

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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.[1,2] 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 ≤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).[3]

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 (PaO_2 ≈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 (FiO_2) before the

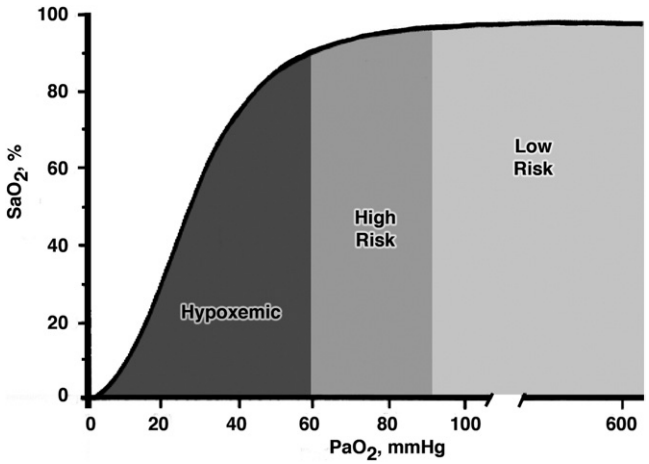

Figure 1. Oxyhemoglobin dissociation curve demonstrates the SpO_2 from various levels of PaO_2. Risk categories are overlaid on the curve. Patients near an SpO_2 of 90% are at risk for precipitous desaturation, as demonstrated by the shape of the curve.

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

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.[7] The first 2 goals are imperative; denitrogenating and oxygenating the blood adds little to the duration of safe apnea[8] 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 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% 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% FiO_2,[9] but these devices are rarely available in EDs. Standardly available nonrebreather masks at flow rates of 15 L/minute deliver only 60% to 70% FiO_2, do not provide complete denitrogenation, and accordingly do not maximize the duration of safe apnea.[9]

Standardly available nonrebreather masks can deliver FiO_2 greater than or equal to 90% by increasing the flow rate to 30 to 60 L/minute.[10] 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 FiO_2 when not actively assisting ventilations.[11,12] 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 FiO_2 above that of room air, a tight seal must be achieved with the mask, which usually requires a 2-handed technique.[13] A bag-valve-mask device hovering above the patient's face provides only ambient FiO_2.

Recommendation: Standard reservoir facemasks with the flow rate of oxygen set as high as possible are the recommended source of high 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.[14] 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 FiO_2 source is an acceptable duration of preoxygenation for most patients.[15,16] This tidal-volume breathing approach can be augmented by asking the patient to exhale fully before the 3-minute period.[17,18]

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

The above times are predicated on a source of FiO_2 greater than or equal to 90% and a tightly fitting mask that prevents entrainment of room air.[21]

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[25]	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[26]	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_2 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[27]	RCT of 20 morbidly obese women undergoing bariatric surgery	CPAP preoxygenation	Spontaneous ventilation at zero pressure	Showed a 40-s increase in time to desaturation through the use of noninvasive positive pressure. Nonsignificant primary outcome.
Gander et al[28]	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[29]	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[30]	RCT of 26 hypoxemic ICU patients requiring bronchoscopy	Noninvasive ventilation	Spontaneous ventilation at zero pressure	The PaO_2/FiO_2 ratio improved in the noninvasive positive-pressure ventilation group and worsened in the high-FiO_2-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.[2,16,18,22,23] If patients do not achieve this saturation level after 3 minutes of tidal-volume breathing with a high FiO_2 source, it is likely that they are exhibiting shunt physiology; any further augmentation of FiO_2 will be unhelpful.[24] 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[23] 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_2, 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_2 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[27,31] or appreciable gastric distention was observed in noninvasive positive pressure preoxygenation studies.[25] 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_2O.[32-35] The data on the absence of any cardiovascular side

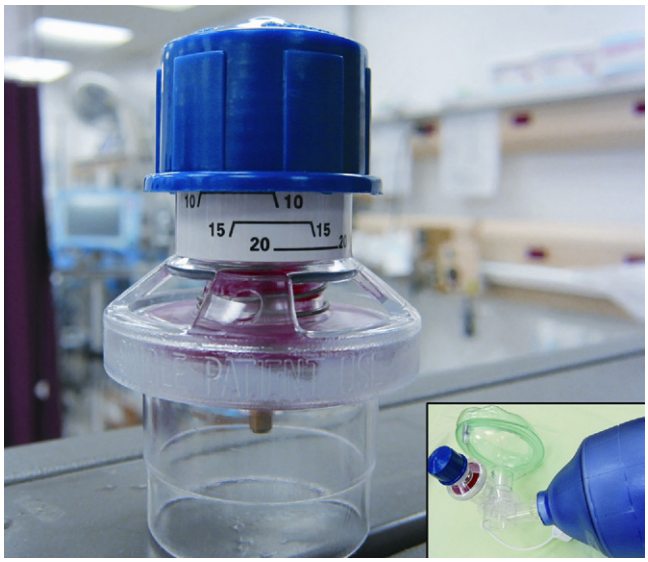

Figure 2. A disposable PEEP valve. This inexpensive item is a strain gauge capable of PEEP settings from 5 to 20 cm H_2O. When placed on the exhalation port of a bag-valve-mask device (inset), it allows the device to provide PEEP/CPAP when the patient is spontaneously breathing and during assisted ventilations. 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.[36]

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_2 alone.[31] If tracheal intubation is attempted with saturations below 90%, these patients can critically desaturate in seconds.[37] Positive pressure may be applied with standalone disposable continuous positive airway pressure (CPAP) masks[38]; 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.[39] 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.[40] PEEP also prevents absorption atelectasis caused by breathing high FiO_2 gas level, increasing the efficacy of apneic oxygenation.[41]

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_2.

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,[3] which reduces the reservoir of oxygen contained within the lungs and therefore reduces safe apnea time.

Lane et al[42] 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.[42] Recently, Ramkumar et al[43] 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[44] 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_2 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.[44] Dixon et al[45] 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.[46]

An additional benefit of head elevation is better laryngeal exposure during direct laryngoscopy.[47]

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_2 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.[3] 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 FiO_2 level.[3]

Benumof et al[48] 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[48] 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[48] assumes preoxygenation with a device capable of generating approximately 90% 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.[49] The times depicted by the desaturation curves are not applicable to critically ill ED patients or those with poor cardiac output.[50-52]

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,[51] desaturation to 85% may be as short as 23 seconds in critically ill patients versus 502 seconds in a healthy adult.[51]

Mort[52] studied ICU patients with blood gas measurements before and after preoxygenation, using bag-valve-mask ventilation. Average 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 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 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.[53] 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 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.[54]

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.[55,56] 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.[63-65] Apneic oxygenation has also been used to continue oxygenation during bronchoscopies and otolaryngeal procedures.[66-68]

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[69] 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[70] 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 SpO_2 greater than or equal to 95% (5.29 versus 3.49 minutes), a significant increase in patients with SpO_2 greater than or equal to 95% at the 6-minute mark (8 patients versus 1 patient), and significantly higher minimum 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 FiO_2 to a spontaneously breathing patient,[71] but the decreased oxygen demands of the apneic state will allow this device to fill the pharynx with a high level of FiO_2 gas.[70] By increasing the flow rate to 15 L/minute, near 100% 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.[72]

The patients' mouths being open did not negatively affect the FiO_2 provided.[73] 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 FiO_2 levels to spontaneously breathing patients (depending on flow rates), they provide minimal oxygen to apneic patients,[74] 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 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 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)[75]; 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, $PaCO_2$ increases 8 to 16 mm Hg in the first minute of apnea and then approximately 3 mm Hg/minute subsequently.[53] It is rare that this degree of $PaCO_2$ 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.[76] 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 H_2O can overwhelm the esophageal sphincter and put the patient at risk for regurgitation and aspiration.[34,35] When providers are inexperienced or under stress, this pressure is easily exceeded.[77] 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.[78,79]

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).[80] 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.[81]

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.[82,83] 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.[84,85] 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.[86] 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%.[87,88] Numerous ventilation studies have found that cricoid pressure hinders bag-valve-mask device ventilation, increases peak inspiratory pressure, and reduces tidal volumes.[34,84,89-94] 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.[95] Similarly, in obese patients undergoing surgery, the succinylcholine group desaturated to 92% in 283 seconds versus 329 seconds in the rocuronium group.[96] When used at a dose of greater than or equal to 1.2 mg/kg, rocuronium provides intubating conditions identical to those of succinylcholine.[97]

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.[95]

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 (saturations 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_2 levels are at high risk of critical desaturation during

Table 2. Risk categorization of patients during preoxygenation.*

Risk Category, Based on Pulse Oximetry While Receiving High-Flow Oxygen	Preoxygenation Period (3 Minutes)	Onset of Muscle Relaxation (≈60 Seconds)	Apneic Period During Tracheal Intubation (Variable Duration, Depending on Airway Difficulty; Ideally <30 Seconds)
Low risk, SpO_2 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_2 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_2 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_2 levels (saturation of 90% or less), aggressive efforts must be made to

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

Study	Patients	Intervention	Comparator	Outcome
Comroe et al[57]	Obs trial of 2 patients with neurologic injuries	Endotracheal insufflation of 6–11 L/min of oxygen	None	Observation of long duration till SaO_2 <90% in numerous separate experiments
Enghoff et al[58]	Obs study of 7 operative patients	Tubing placed down ET tube connected to 100% FiO_2	None	No decrease in ABG PaO_2 for 5–7 min
Holmdahl[59]	Documentation of separate Obs studies	Endotracheal insufflation of 100% FiO_2	None	Extended time until desaturation in separate studies
Frumin et al[60]	Obs study of 8 operative patients	Intubated, administered 100% FiO_2 and left apneic	None	No desaturation for between 18 and 55 min. Patients had marked hypercapnia and commensurate decreased pH.
Babinski et al[61]	Obs study of 5 operative patients	Two endobronchial catheters placed	None	30 min of apnea without significant desaturation
Teller et al[62]	RCT, blinded, crossover trial of 12 pts	Nasopharyngeal catheters attached to 100% FiO_2 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[69]	RCT of 30 pts	Nasal catheters attached to 5L/min of 100% FiO_2	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[70]	RCT of 30 obese, operative patients	Nasal cannula attached to 5 L/min 100% FiO_2	Room air	The apneic oxygenation group had significant prolongation of SpO_2 ≥95% time (5.29 versus 3.49 min), a significant increase in patients with SpO_2 ≥95% at the 6-min mark (8 patients versus 1 patient), and significantly higher minimum SpO_2 (94.3% versus 87.7%).

Obs, Observational trial.