

variable. Another difference between our studies was the cohort groups tested. Our study volunteers were ages 60–84 years, compared to 50–74 years in the Neset study. Our volunteers were also retired health care professionals. Additionally, it is not known if there was a difference, independent of age, in baseline physical fitness between cohorts.

Most importantly, we do not state or mean to imply that older rescuers should not be taught or encouraged to perform bystander CPR. We agree with Neset and colleagues that bystander CPR is a key element in the chain of survival for victims of cardiac arrest. The issue of fatigue and adequacy of chest compressions is an important subject that deserves further clarification. This is especially important when considering data linking greater chest compression depth with improved survival, as well as AHA's recent increase in recommended chest compression depth (5). When only one rescuer is present, the lone rescuer must do his/her best in a stressful situation. However, in many circumstances there are other bystanders available who may be able to take over chest compressions when the first rescuer fatigues. Studies on the effects of fatigue may lead to recommendations on the appropriate time period bystanders should perform CPR before asking to change rescuers. We recommend that if more than one rescuer is available, the person giving chest compressions should rotate every 1–2 min until medics arrive.

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<http://dx.doi.org/10.1016/j.jemermed.2012.11.086>

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□ RE: PREOXYGENATION, REOXYGENATION, AND DELAYED SEQUENCE INTUBATION IN THE EMERGENCY DEPARTMENT

□ To the Editor:

We read with interest the recent article in *The Journal of Emergency Medicine* titled “Preoxygenation, Reoxygenation, and Delayed Sequence Intubation in the Emergency Department,” and feel it warrants some comment (1).

As anesthesiologists, our main concern is that the authors seem to have missed the main physiological background and rationale for performing preoxygenation. Namely, to denitrogenate the functional residual capacity (FRC) of the lungs with near 100% O₂ to provide a reservoir of oxygen that will continue to oxygenate the blood during the apneic period after induction of anesthesia.

Our impression that they have not fully understood or explained the physiological basis of this procedure is further reinforced later in the discussion where the assertion seems to be made that preoxygenation of a patient with SpO₂ on the steep portion of the oxy-hemoglobin dissociation curve, where the hypoxia is a result of shunt, is futile unless recruitment of alveoli occurs. This, they suggest, will be best done through the application of non-invasive intermittent ventilation (NIV) before attempting rapid sequence induction.

We would contest this due to the following:

- a) SpO₂ is not a measure of preoxygenation. The best measure of this in clinical practice is the comparison of end-tidal O₂ with inspired O₂, ideally achieving >90% E_TO₂. Even in the presence of shunt or reduced lung volume, the increased alveolar partial pressure of oxygen will continue to oxygenate the blood after induction, albeit not for as long as in a well patient without shunt. Therefore, it is still a useful maneuver, and although the patient will remain on the “steep portion of the curve,” the patient will be held at this point until the FRC oxygen reserve is exhausted.
- b) If physiological shunt (as compared to true anatomical shunt) is the main source of hypoxia as a result of atelectasis, then intubation is undoubtedly superior to NIV. Intubation allows transmission of higher inflation pressures (>20 mm Hg, but ideally <30 cm H₂O) without the risk of gastric insufflation or aspiration. In this case, intubation and mechanical ventilation is the therapy of choice.
- c) In our experience, patients who are combative due to hypoxia or hypercapnia often do not tolerate or comply with NIV and it would be inappropriate to use it as a preoxygenation strategy.
- d) The use of intravenous ketamine in what could be approaching an anesthetic dose at 1.5 mg/kg

may not be as problem-free as described by the authors.

- e) The use of bag-valve-mask techniques to provide continuous positive airway pressure/spontaneous positive end expiratory pressure is widely practiced by anaesthetists in the United Kingdom.

Further to these concerns, we do not entirely understand the statement regarding minute ventilation in the re-oxygenation section. The authors state that to provide high levels of oxygenation, all that is required at an FiO_2 of 0.5 is a minute volume of 500 mL/min. This would provide the minimum quoted oxygen requirement for basal metabolic function at rest, but will not provide "high levels of oxygenation," and oxygen requirement in a critically ill patient is likely to exceed this figure.

The article suggests that using nasopharyngeal airways or oropharyngeal airways (NPAs/OPAs) are advanced techniques best performed by experts and may be dangerous in the hands of novices. In our opinion, the performance of a rapid sequence induction and intubation, especially in a critically unwell patient, is an advanced technique and should be performed only by experienced personnel. The recent National Audit Project 4 performed by the Royal College of Anaesthetists highlighted how dangerous intubation in the Emergency Department can be (2). NPAs/OPAs are simple airway adjuncts that should be familiar to most health care providers working in the acute setting.

We do, however agree entirely with the comments made regarding the importance of maintenance of a patent airway post induction to allow for apneic oxygenation. The importance of reoxygenation in the face of dropping saturations, rather than persisting with attempts at intubation, also cannot be overstated.

In summary, in rapid sequence induction, the time from induction to establishing a definitive airway should be as short as possible due to a potential risk to the airway or likely precipitous fall in oxygenation. Preoxygenation is an integral part of this by providing a supply of oxygen that will maintain oxygenation during apnea. To delay the definitive treatment of patients in respiratory distress does not strike us as best practice.

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<http://dx.doi.org/10.1016/j.jemermed.2012.01.071>

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RE: PREOXYGENATION, REOXYGENATION, AND DELAYED SEQUENCE INTUBATION IN THE EMERGENCY DEPARTMENT

To the Editor:

I thank Drs. Gill and Edmondson for an opportunity to expand on some of the points in my manuscript (1). That previous sentence was the first in the response I began to draft when I received the letter to the editor printed above. I then realized that this form of communication was outdated; I would write a response, perhaps addressing the questions raised or perhaps missing their intent entirely. No actual communication or fostering of more refined ideas would be achieved. Instead, I searched for contact information for Dr. Gill and e-mailed him to ask if he'd like to have an actual conversation about the things he had written. Further, I got his permission to record the conversation and make it freely available. So although I will respond briefly to each of the points of the original letter below, I believe more understanding may be gained by listening to the podcast of the conversation available here: [<http://emcrit.org/preox-response/>].

The first point we discussed was the pulse oximetry reading as a measure of preoxygenation. Although maximal denitrogenation is indeed a goal during preoxygenation, in a critically ill patient it is necessary, but insufficient. As mentioned in the original paper, denitrogenation can be achieved with eight vital capacity breaths or 3 min of tidal volume breathing while on a high FiO_2 source. But if the patient's oxygen saturation is still low after being placed on high FiO_2 , the patient is exhibiting physiological shunt. In fact, the work by Davis et al. demonstrated that the pre-rapid sequence intubation (RSI) oxygen saturation is the best predictor of the rapidity of decline during the intubation with a curve that nearly mirrors the oxyhemoglobin dissociation curve (2). This is physiologically intuitive, because as the arterial saturation diminishes, progressively lower-saturated venous blood will be sent through the shunt areas of the lung. The larger the shunt fraction, the worse will be this rapid cyclical decline of arterial oxygen saturation.