References


Preoxygenation: Comparison of Maximal Breathing and Tidal Volume Techniques

To the Editor.—Baraka et al.1 recently demonstrated that preoxygenation using eight deep breaths within 60 s (8 DB/60 s) at an oxygen flow of 10 L/min can produce arterial oxygen tension (PaO2) values comparable to those obtained using normal tidal volume breathing (TVB) for 3 min. In addition, they showed that this technique significantly delayed the onset of apnea-induced hemoglobin desaturation.

Before this new method becomes widely accepted, several issues need to be clarified. First, we wonder what role the baseline values for PaO2 played in the delayed hemoglobin desaturation after 8 DB/60 s. For this portion of their study, Baraka et al. used a separate group of subjects, group B, in whom baseline PaO2 values were 407 ± 55 mmHg after 5 min of TVB and 454 ± 45 mmHg after 8 DB/60 s. Both values were higher than those of subjects in group A, in whom 3 min of TVB yielded a PaO2 higher than 392 ± 72 mmHg versus 369 ± 69 mmHg after 8 DB/60 s. It cannot be ruled out that the higher PaO2 values observed in group B after 8 DB/60 s contributed to the delay in hemoglobin desaturation. If subjects from group A were subjected to apnea, the benefit of 8 DB/60 s may not have been evident, or at least may not have been as dramatic.

Second, we think that reporting this technique as eight breaths in 60 s underestimates the number of breaths and the time of preoxygenation. If we understand the protocol correctly, after the eight breaths, a rapid-sequence induction of anesthesia was carried out. During this period, face-mask oxygenation was continued until apnea ensued, a period described as 15 to 30 s during which an additional 2 to 4 deep breaths occurred. Thus, it appears that Baraka et al. actually evaluated the efficacy of 10 to 12 breaths during a 75 to 90 s period rather than eight deep breaths in 60 s. The authors proposed two possible mechanisms for the delayed decrease in hemoglobin saturation during 8 DB/60 s: (1) that the extra 15 to 30 s provided more alveolar oxygenation in patients breathing deeply for 60 s than during TVB; and (2) that the continued deep breathing during this extra time may have opened collapsed airways or lung tissue, with a consequent increase in oxygen store in the functional residual capacity. In his editorial, Benumof6 proposed other explanations, including a leftward shift of oxyhemoglobin dissociation curve secondary to hyperventilation-induced reductions in PaCO2. We propose that by extending the duration of deep breathing beyond 60 s (i.e., to 75-90 s) may have enhanced the potential influence of this factor. A delay in desaturation caused by a leftward shift of the oxyhemoglobin dissociation curve would not necessarily favor improved oxygen transport. Because the authors presented only values for PaO2s, the role of changes in arterial carbon dioxide tension and arterial pH must remain speculative.

Third, Baraka et al. state that using the technique of four deep breaths in 30 s (4DB/30 s), PaO2 values increased exponentially as oxygen flow is increased from 5 to 10 to 20 L/min. Although this description may accurately describe the increase from baseline values, the mean values for PaO2 at 5, 10, and 20 L/min oxygen flow all decrease within the linear, essentially flat portion of the curve. The differences appear minimal, and the authors make no statement concerning the significance of the differences among the values for PaO2, at the three fresh gas flows. Recently, Nimmagadda et al.5 demonstrated that increasing fresh gas flows from 5 to 7 to 10 L/min had no significant effect on end-tidal oxygen or nitrogen during preoxygenation using 4DB/30 s or 2-min TVB techniques in healthy volunteers. Although Nimmagadda et al. did not test 20 L/min, this value is probably not encountered in most circumstances in the operating room. Although interesting and provocative, the study of Baraka et al. is far from conclusive. More studies are required to ascertain if the 8 DB/60 s method actually delays hemoglobin desaturation, and whether this method is more beneficial than the traditional TVB. It is clearly premature to anoint the 8 DB/60 s technique as the method of choice for preoxygenation.

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In Reply.—Drs. Morrison and Videira make the valid point that pediatric patients desaturate faster than adults and that preoxygenation is especially indicated in several types of pediatric patients. In fact, I have previously reported complete apnea-oxyhemoglobin desaturation curves for various humans using computer modeling1 and have showed that a 10-kg pediatric patient will desaturate twice as fast as a 70-kg adult. Rather than offer a convoluted explanation as to how the words "obvious exclusion examples" were chosen, I agree the phrase is too broad and that Dr. Morrison provides examples of two patients who deserve to be preoxygenated.

Dr. Salem et al. are probably correct with their hypothesis that Baraka et al. actually studied 10 to 12 deep breaths in 75 to 90 s rather than eight deep breaths in 60 s. In ongoing studies I have tried to repeat the Baraka et al. methodology. Even with extensive preoxygenation coaching of the patients, I have found that the preoxygenation process is always extended by at least 2 breaths beyond the intended period. In fact, if one wants to study eight deep breaths in 60 s, then the rapid sequence induction must be begun on the inspiratory limb of the sixth breath. I agree with the Salem et al. comment regarding the effect of an extended period of hyperventilation on shifting the oxyhemoglobin curve further to the left.

In summary, I thank Drs. Morrison, Videira, and Salem for their thoughtful insights.

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