Letters

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Methemoglobinemia and Pulse Oximetry

In reviewing the article "Methemoglobinemia: Sudden Dyspnea and Oxygen Desaturation After Esophagoduodenoscopy," in the August 2004 issue of RESPIRATORY CARE,[1] I found the article suggested that, during the esophagoduodenoscopy procedure the patient's blood oxygen saturation (measured via pulse oximetry) began to drop rapidly, and a saturation of 54% registered on the pulse oximeter while the patient was breathing 100% oxygen via non-rebreathing mask. The arterial blood gas analysis subsequently showed a P_{aO_2} of 117 mm Hg. It is my understanding that a pulse oximeter is capable of reading only "functional" hemoglobin, that is, only the hemoglobin bound with or capable of binding with oxygen.

If the P_{aO_2} was 117 mm Hg, the oxyhemoglobin was 22.2%, and the methemoglobin was 77.4%, the oximeter should have been reading about 98%, since the maximum functional hemoglobin was 22.6% (100% –77.4% methemoglobin) and the actual oxyhemoglobin was 22.2%.

The article gives the impression that a pulse oximeter can detect dysfunctional oxyhemoglobin states, when in fact the oximeter will often mislead practitioners about a patient's true oxygen content when dysfunctional hemoglobin (ie, carboxyhemoglobin or methemoglobin) is present. Why was there a discrepancy with the pulse oximetry readings initially?

Tom Richardson RRT
Southern Patient Care
Alabaster, Alabama

REFERENCE

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The authors reply:

A pulse oximeter functions by emitting light at 2 wavelengths: 650 nm and 805 nm. It measures the light absorbed as a proportion between those 2 wavelengths. Because various forms of hemoglobin (eg, oxyhemoglobin and deoxyhemoglobin) absorb light at different wavelengths, the pulse oximeter reports an estimated percentage of oxyhemoglobin. However, the pulse oximeter cannot detect other forms of hemoglobin, such as carboxyhemoglobin or methemoglobin. In general, a methemoglobin percentage $> 10\%$ will cause an unreliable oximeter reading that is often lower than expected. Therefore, pulse oximetry should be used only as a screen. Co-oximetry should be used instead to monitor response to treatment.

Ellen Lunenfeld MD
Gregory C Kane MD
Department of Medicine
Jefferson Medical College
Philadelphia, Pennsylvania

Dynamic Hyperinflation, Intrinsic Positive End-Expiratory Pressure, and Respiratory Rate

In the November 2004 issue of RESPIRATORY CARE, I read with great interest the article "Determinants of Dynamic Hyperinflation in a Bench Model," by Drs Thorevska and Manthous.[1] The recognition and management of dynamic hyperinflation and intrinsic positive end-expiratory pressure are very important to all respiratory care clinicians who work with mechanically ventilated patients, and Thorevska and Manthous's work helps to illustrate the related factors. I would like to offer one clarification, related to Thorevska and Manthous's statement that their results differed with those of Tuxen and Lane,[2] and Williams et al.[3] Thorevska and Manthous found that dynamic hyperinflation increased with a decrease in tidal volume (from 1.0 L to 0.6 L) with constant minute ventilation and duty cycle (ratio of inspiratory time to total respiratory-cycle time). They mentioned that this may be due to increased elastic recoil with higher tidal volume, which generates a higher expiratory flow rate, which is correct. But there is another explanation that would account for these findings, as illustrated by the following calculations.

If one delivers a constant minute ventilation with different tidal volumes, different respiratory rates will be required. If a constant duty cycle of 0.25 and a constant minute ventilation of 15 L/min are assumed, it is possible to calculate several variables (Table 1).

From the calculations in Table 1 it is clear that using a larger tidal volume to provide a constant minute ventilation with a constant duty cycle will allow the use of a lower respiratory rate and substantially longer expiratory time. If the mechanical factors of compliance and resistance in the test lung remain unchanged, a longer expiratory time coupled with the higher expiratory flow rate generated by the larger tidal volume will result in improved lung emptying and, thus, reduced dynamic hyperin-

Table 1. Respiratory Variables

Calculated Variables	V_T 0.6 L	V_T 1.0 L
Respiratory rate (breaths/min) required to maintain minute ventilation at 15 L/min	25	15
T_{tot} (s)	2.4	4
T_I/T_{tot}	0.25	0.25
T_I/T_E	1:3	1:3
T_I (s)	0.6	1
T_E (s)	1.8	3

V_T = tidal volume
T_{tot} = total respiratory-cycle time
T_I = inspiratory time
T_E = expiratory time

flation. Specific emphasis on allowing an adequate expiratory time, as dictated by the resistance and compliance of the lung, is critical to properly manage intrinsic positive end-expiratory pressure and dynamic hyperinflation.

Jeffrey M Anderson MA RRT
Department of Respiratory Care
Boise State University
Boise, Idaho

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The authors reply:

We agree.

Natalya Y Thorevska MD
Division of Pulmonary and
Critical Care Medicine
Loyola University
Maywood, Illinois

Constantine A Manthous MD
Division of Pulmonary and
Critical Care
Bridgeport Hospital
Yale University School of Medicine
Bridgeport, Connecticut

Expiratory Rib-Cage Compression, Airway Suctioning, and Atelectasis

Unoki et al[1] recently reported, in RESPIRATORY CARE, their evaluation of the effects of rib-cage compression with and without airway suctioning on P_{aO_2}, P_{aCO_2}, respiratory-system compliance, and mucus clearance in 28 rabbits with atelectasis induced by tracheal infusion of artificial mucus. The rabbits were intubated, paralyzed, mechanically ventilated, tracheostomized, and randomized to one of 4 groups: (1) control, (2) suctioning only, (3) rib-cage compression only, and (4) rib-cage compression plus suctioning.

In the group that received rib-cage compression, P_{aO_2}, P_{aCO_2}, and respiratory-system compliance were significantly worse than the groups that did not receive rib-cage compressions. Endotracheal suctioning with and without rib-cage compression did not improve P_{aO_2}, P_{aCO_2}, respiratory-system compliance, or mucus clearance. There were no significant differences in the weight of aspirated mucus between the groups.

Unoki et al are to be commended for evaluating the effects of a commonly used chest physical therapy procedure with an animal model. However, their conclusions are seriously flawed, because of problems with the study design and inappropriate interpretation of the study results.

All the animals were anesthetized and paralyzed during the study, and were mechanically ventilated initially in a volume-controlled mode. Following the induced atelectasis, the animals were then switched over to a pressure-controlled mode, for reasons unexplained.

First, the pharmacologic paralysis would have ablated the ability to stimulate a cough reflex during endotracheal airway suctioning, which obviously would have reduced the effectiveness of airway suctioning to generate expiratory flow and assist mucus clearance. The use of chest wall compressions to improve expiratory flow and move the airway secretions from the peripheral airways to the more central airways is evidenced by the significant reductions in dynamic lung/thorax compliance in the groups that received compressions. As the rabbits were ventilated in a pressure-controlled mode, the significant reductions in dynamic lung/thorax compliance obviously explains the resultant deterioration in oxygenation and hypercarbia. With the rabbits paralyzed, the central airway secretions could not be cleared, because of the absence of cough reflex, which explains the deterioration in ventilation variables. This must be further explored and underlies their seeming lack of understanding of the effects of these chest physical therapy procedures on these outcome measures.

Recent work by Main et al[2] with pediatric ventilated patients supports this reasoning. Main et al demonstrated that these chest physical therapy procedures can improve airways resistance, which was not measured by Unoki et al. As Unoki et al did not monitor expiratory flow rate or airway resistance,[1] we are unsure as to the effectiveness of the interventions in their animal model.

In summary then, Unoki et al used a pressure-cycled mode, so the significant reductions in dynamic airway compliance support the deduction that the chest wall compressions in fact enhanced the movement of airway secretions to more central airways, resulting in the deterioration in ventilation and oxygenation.

George Ntoumenopoulos PhD, BAppSc, BSC, Grad Dip Clin Epid
St Thomas' Hospital
London, United Kingdom

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The authors reply:

We thank Dr Ntoumenopoulos for his interest in our article[1] and stimulating comments. We would like to address the issues raised by his insights. First, we emphasize that our study was carried out under experimental conditions, using an induced-atelectasis model, so we should be cautious about extrapolating the results to humans, as we mentioned in our article. Second, rib-cage compression has been advocated as an effective technique to treat atelectasis in mechanically ventilated patients, despite little clinical or experimental evidence. Therefore, to elucidate the effects of rib-cage compression under well-controlled and consistent circumstances, we chose an animal study.

Dr Ntoumenopoulos wondered why we chose pressure-controlled ventilation following the induced atelectasis. There are