Laryngospasm-induced Pulmonary Edema

To the Editor:

The case report by Jackson et al (Chest 1980; 78:819-21) presents interesting lessons on the complications which may befall a patient undergoing "routine" elective anesthesia and surgery. The data presented, however, raise questions about the accuracy of the clinical observations and diagnostic conclusions of the authors.

Ventilation of their patient was impossible following the intravenous administration of 250 mg of thiopental sodium and 100 mg of succinylcholine. The inability to ventilate this patient of "unusual body habits (sic) (obesity, short neck)" was interpreted as "laryngospasm" and was treated with intravenous aminoephphrine and epinephrine. Even more remarkable than their unconventional therapy for laryngospasm was their statement that the patient remained "alert and awake during the event." Although laryngospasm is a known complication of the induction of anesthesia with a number of agents (including thiopental sodium), it has not, to my knowledge, been reported following the administration of succinylcholine, a muscle relaxant which is in fact the indicated therapy for the treatment of severe laryngospasm associated with cyanosis. To assert that the patient remained conscious during the episode suggests either that the intravenous line through which the thiopental sodium and succinylcholine were administered was not functioning or that the clinical observations of the authors are seriously in error: persons receiving the stated dosages of thiopental sodium and succinylcholine are neither alert nor awake.

It is distressing that the authors implicitly recommend aminophylaine and epinephrine to treat laryngospasm. A computerized review of the medical literature of the past 15 years yielded no papers supporting this practice. On the contrary, the initial therapy recommended by every major textbook of anesthesia is to attempt to administer oxygen with gentle positive airway pressure by mask. If that fails and the patient experiences cyanosis, hypotension, or arrhythmia, one administers succinylcholine to induce paralysis of the muscles of adduction of the vocal cords.

"Within three minutes" of the episode of airway obstruction, an analysis of arterial blood gas levels revealed acute respiratory acidosis with an arterial PaCO₂ = 91 mm Hg. Clinical studies of apnea in human volunteers have established that acute airway obstruction of the type described must persist for eight to 15 minutes to produce a 50 mm Hg rise of PaCO₂. It would appear that much more time was spent re-establishing the airway than was appreciated.

This length of time increases the likelihood that unrecognized aspiration of gastric contents occurred. It is well known that relaxation of the cricopharyngeus muscle by neuromuscular blocking drugs such as succinylcholine may result in inapparent aspiration of regurgitated gastric content, especially if repeated attempts at intubation occur.

The authors of this article were, unfortunately, unaware of previous reports of pulmonary edema following well-documented laryngospasm. The mechanism proposed by Jackson et al is that previously suggested in three earlier reports. If pulmonary edema occurred as a result of laryngospasm in the patient presented, then vigorous inspiratory efforts should have been clinically apparent during the "laryngospasm." Since this was not stated, one must assume that was not the case. In the absence of vigorous inspiratory efforts to decrease intrapleural pressure enough to favor transudation of fluid from the pulmonary capillaries into the interstitial space, there is no support for the authors' contention that this patient had laryngospasm-induced pulmonary edema.

The information presented by the authors is internally inconsistent and does not support the diagnosis of laryngospasm-induced pulmonary edema. It would appear very much more likely that the obesity and short neck of the patient contributed to unrecognized upper airway obstruction by the tongue and soft tissues of the pharynx following induction of paralysis with succinylcholine, and that repeated attempts at intubation resulted in unrecognized aspiration.

This case report emphasizes once more the already well-known importance of special precautions (eg, awake laryngoscopy with topical anesthesia, intubation over fiberoptic bronchoscope, etc) to prevent serious complications for patients who on physical examination reveal signs that they may be difficult to intubate or ventilate by mask.

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REFERENCES
7 Oswalt CE, Gates GA, Holmstrom FM. Pulmonary edema as a complication of acute airway obstruction. JAMA 1971; 238:1833-35

To the Editor:

In spite of the questions Dr. Poulton raises with respect to the "accuracy of the clinical observations and diagnostic conclusions," we continue to believe that laryngospasm was the main etiologic factor to induce pulmonary edema in this patient for the following reasons:
1. The event immediately followed two separate unsuccessful attempts to advance the endotracheal tube through the larynx. While attempting to gain access to the epiglottis area with the aid of the laryngoscope blade, the tongue and soft tissues were lifted and placed to the left of the mouth, thereby eliminating these structures as potential factors of airway obstruction.

2. Ventilation with a face mask was attempted with the neck in hyperextension and with mandibular support, thus minimizing the possibility that tongue and soft tissues would interfere with airway patency.

3. Although the epiglottis was identified, but the vocal cords were not visualized, one cannot rule out with certainty that the inability to effectively ventilate the patient was the result of other than laryngospasm, eg, bronchospasm.

4. We are, of course, fully aware of the various steps recommended for the treatment of laryngospasm, including the administration of oxygen under positive airway pressure, and the use of succinylcholine to induce paralysis of the muscles of adduction of the vocal cords. In our patient, the initial treatment of laryngospasm followed precisely these rules and only when the patient failed to respond promptly were atropine and epinephrine administered, since the possibility of a bronchospastic component could not be ignored.

We would like to clarify a statement made by Dr. Poulton regarding analysis of arterial blood gas levels “within three minutes” of the episode of airway obstruction. The blood gases were obtained not three minutes after the onset of respiratory obstruction, but rather three minutes after the administration of the medication, which means that at least 8 to 10 minutes had elapsed from the onset of ventilatory difficulties until arterial blood gases were analyzed.

We also think that further clarification is required regarding our patient’s acuity during the event. She was indeed conscious during this episode and remained conscious for several hours following its onset.

Dr. Poulton’s comments are well taken, though not applicable in this case.

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The Abbreviated Alveolar Air Equation Revisited

To the Editor:

The reports by Raymond1 and Helmholtz2 concerning abbreviation of the alveolar air equation to encourage more general use still result in a rather formidable algebraic expression. The clinician without a handy programmable pocket calculator remains without a truly simplified approach to estimate the P(A-a)O\textsubscript{2}\textsuperscript{(approx)}. Once determined, this altitude-dependent normal sum of the alveolar tensions remains constant and serves as the baseline for a simple subtraction leading directly to the approximation. An uncomplicated calculation of this baseline, as well as an error analysis of the P(A-a)O\textsubscript{2}\textsuperscript{(approx)} follows from analysis of the algebraic representation of Wasserman’s technique:

\[ P(A-a)O_2(\text{approx}) = \text{alveolar sum(baseline)} - \text{arterial sum} = (PaO_2 + PaCO_2) - (PaO_2 + PaCO_2) \]

The first term \((PaO_2 + PaCO_2)\) is the baseline number, \(PaO_2\) is the normal ideal alveolar \(O_2\) tension, and \(PaCO_2\) (equal to \(PaCO_2\)) is the normal alveolar \(CO_2\) tension for any given altitude. \(PaO_2\) is calculated from the alveolar air equation with \(R = 0.8\) using the mean barometric pressure (\(mm\ Hg\)) for the given altitude, \((Pa),\) and \(PaCO_2\). Substituting the altitude-dependent function:

\[ PaCO_2 = 40 - .042(760-Pa) \]

and consolidating terms with the alveolar air equation, the baseline simplifies to:

\[ (PaO_2 + PaCO_2) = .2017 Pa - 11.47 \]

A parabolic fit to the US Standard Atmosphere tables3 allows the baseline to be expressed as a function of altitude:

\[ (PaO_2 + PaCO_2) = 141.8 - 5.495 \times 10^{-4}h + 7.135 \times 10^{-7}h^2 \]

where \(h\) is altitude in feet. Thus, the sea level baseline \((Pa = 760, h = 0)\) calculated from equation (3) or (4) is 142, while the baseline for Denver \((Pa = 628, h = 5280)\) is 115. The \(P(A-a)O_2\ (\text{approx})\) of a patient in Denver breathing room air with arterial gases \(PaO_2\ = 60\) and \(PaCO_2\ = 30\) is easily calculated:

\[ 115 - (60 + 30) = 25 \]

Exact calculation of room air \(P(A-a)O_2\) using the ideal alveolar air equation with \(R = 0.8\) at \(Pa\) reduces to:

\[ P(A-a)O_2(\text{ideal}) = .21 Pa - (PaO_2 + 1.198 PaCO_2) - 9.87 \]

By rearranging and simplifying, the error of the approximation is seen to be:

\[ P(A-a)O_2(\text{approx-ideal}) = .1975 PaCO_2 - 8.295 \times 10^{-3} Pa - 1.596 \]

and is, therefore, a function of both altitude and \(PaCO_2\).

In the figure, the dashed line represents zero error and intersects the \(PaCO_2\) isopleths at the corresponding normal \(PaCO_2\) for given altitude. The figure demonstrates that the sign and magnitude of the error are in the same direction and proportional to the \(PaCO_2\) variation from normal, 1 mm Hg error developing for every 5 mm Hg change in the \(PaCO_2\). Because of the simplicity and minimal error of this method, its use as a bedside approximation of \(P(A-a)O_2\)

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