CORRESPONDENCE

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Laryngeal Mask and Pulmonary Edema

To the Editor:—We recently encountered two patients who developed pulmonary edema after insertion of a laryngeal mask.

Two otherwise healthy adult patients scheduled for elective orthopedic surgery were anesthetized with a mixture of nitrous oxide, oxygen, and halothane, while breathing spontaneously.

A laryngeal mask (LM) was inserted under deep anesthesia. In both cases difficulties were encountered in inserting the LM and the patients developed signs of airway obstruction with inspiratory stridor and chest retraction. The LM was successfully reinserted in both patients after the third attempt. Despite relieving the airway obstruction, the hemoglobin oxygen saturation was low (90–92% on a $F_{I_{O_2}}$ of 0.5) and wet rales were heard over both lung fields. An arterial blood sample for blood gas analysis was obtained in both patients. In the first case, there was a mild respiratory acidosis and hypoxemia (pH 7.32, Pa_{CO_2} 47 mmHg, Pa_{O_2} 63 mmHg, Sa_{O_2} 90%, $F_{I_{O_2}}$ 0.4, and spontaneous breathing). In the second case, the blood gas analysis showed severe hypoxemia (pH 7.36, Pa_{CO_2} 35 mmHg, Pa_{O_2} 46 mmHg, Sa_{O_2} 78%, $F_{I_{O_2}}$ 0.4, while breathing spontaneously). In the postanesthesia care unit, the chest x-ray showed frank pulmonary edema, which was promptly and successfully treated with intravenous furosemide and 100% oxygen by mask. There was no fluid overload in either of the cases. The first patient had received 500 ml of balanced Ringer's lactate solution during surgery and 100 ml more prior to the chest x-ray, whereas the second patient had received 560 ml during surgery and 120 ml prior to the x-ray.

Pulmonary edema has been associated with airway obstruction in children and adults.[1,2] The mechanisms causing pulmonary edema induced by airway obstruction are multifactorial[3] and not entirely understood. It is believed that intrathoracic negative pressure created by the inspiratory effort through an obstructed airway will promote an increased venous return to the pulmonary circulation increasing pulmonary capillary hydrostatic pressure. This negative pressure also will decrease pulmonary interstitial hydrostatic pressure, promoting filtration of the fluid from the capillaries into the lung interstitium. Finally, the intense sympathetic discharge triggered by hypoxia will cause increased capillary hydrostatic pressures damaging the alveolocapillary membrane, increasing permeability and further worsening edema.[4]

A 10% incidence of malposition of the LM has been reported.[5] This could lead to upper respiratory airway obstruction. Forceful spontaneous breathing through an obstructed airway could induce pulmonary edema through the mechanisms described above.

We conclude that, apart from laryngospasm and aspiration of stomach content, malposition of the LM can induce frank pulmonary edema due to upper respiratory airway obstruction.

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Preemptive Analgesia or Anoci-Association

To the Editor:—The recent article by Katz *et al.*[1] describing preemptive analgesia is suggestive of an earlier concept proposed by George Crile, Sr. many years ago. Crile introduced the term "anoci-association" as a technique to reduce surgical stress and improve postoperative status. This procedure involved general anesthesia for surgical operations combined with infiltration of local anesthetics to block noxious impulses arising from the surgical wound. In this way, he believed that the patient was protected from the stress of