

Questioning Diuretic Use in Acute Negative-pressure Pulmonary Edema

To the Editor:

In their case study of negative-pressure pulmonary edema (NPPE), Krodel *et al.*¹ repeat the oft-cited idea that diuretics should be included in the therapies for this condition. They refer to using furosemide in the postanesthesia care unit for NPPE, which occurred as a result of laryngospasm on emergence. The authors do acknowledge that diuretics are not universally recommended for NPPE, noting that they “are often administered, but their use is controversial and may even be unnecessary.” However, we are surprised that expert opinion continues to afford even a qualified role to diuretics. To our knowledge, there has never been any evidence for doing so. Beyond the knee-jerk association between pulmonary edema and loop diuretic administration, we cannot imagine why NPPE should routinely or even occasionally be managed with diuretics. Indeed, the careful elucidation of pathophysiologic features in this review should demonstrate that neither intravascular nor total body volume is increased in those with NPPE; these volumes, in contrast, may be significantly decreased. The sudden shift of fluid into the pulmonary interstitium has little in common with other scenarios in which diuresis is helpful in reducing excess total body water. In those with NPPE, diuretic administration may be unnecessary and harmful, particularly in patients who are older and less able to compensate for hypovolemia than the 25-yr-old otherwise healthy man who is described. Indeed, anecdotal experience at our institution has shown that furosemide administration to patients with NPPE can result in hypovolemic shock requiring fluid resuscitation.

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Acute Postoperative Negative-pressure Pulmonary Edema

To the Editor:

We read with interest the case scenario regarding acute postoperative negative-pressure pulmonary edema (NPPE).¹ The

authors elegantly discussed the diagnosis, differential diagnosis, epidemiological features, pathogenesis, and clinical management of NPPE. We are concerned that anesthetic management may have inadvertently contributed to the cause of this complication. The patient described was given opioid doses equivalent to 27.5 mg iv morphine (0.25 mg fentanyl = 25 mg + 0.5 mg hydromorphone = 2.5 mg)² and a nondepolarizing muscle relaxant. The fact that the patient (with a normal airway) developed laryngospasm after extubation suggests that the patient was not ready for extubation. In addition, it is possible that reduced pharyngeal muscle tone due to residual neuromuscular blockade resulted in upper airway obstruction.^{3,4} A patient with a “train-of-four” ratio of 0.9 or greater may still develop postoperative hypoxemia⁵ and may require the administration of reversal drugs.

The initial difficulty in mask ventilation after extubation implies that the inspiratory stridor had progressed to a ball-valve obstruction.⁶ Applying positive airway pressure under these circumstances may actually worsen ball-valve closure.⁶ Inflation of the pharynx distends the piriform fossae, pressing the aryepiglottic folds more firmly against each other and reinforcing the closure.⁶

We suggest that the complication presented could have been prevented by delaying extubation.

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