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Acute Pulmonary Edema Resulting from Nalbuphine Reversal of Fentanyl-induced Respiratory Depression

To the Editor:—Naloxone, used to antagonize fentanyl, has been reported to cause acute pulmonary edema in young, healthy individuals even when used in conservative doses.^{1,2} Nalbuphine has also been shown to be a clinically effective antagonist of fentanyl.³

Recently, we employed 10 mg of nalbuphine to reverse the respiratory depression of 0.05 mg of fentanyl in an otherwise healthy, 19-yr-old white man undergoing general anesthesia for debridement and arthrodesis of compound fractures of the right hand. Other anesthetic agents included 3 mg of curare, 350 mg of sodium thiopental, 100 mg of succinylcholine for induction and intubation, and isoflurane and nitrous oxide in oxygen for maintenance.

The patient developed mild pulmonary edema in the recovery room about 20 min after receiving nalbuphine. The pulmonary edema, evidenced both by the presence of rales and frothy sputum and radiographically, responded to conservative treatment. Because this patient was completely healthy in every aspect, except his injury, prior to anesthesia and surgery, it appeared to us that the nalbuphine reversal of fentanyl had led to the pulmonary edema.

Pulmonary edema following narcotic reversal by naloxone has been postulated to have a neurogenic basis.^{1,2} Nalbuphine, an agonist-antagonist agent, presumably antagonizes mu-receptor-bound drugs, such as fentanyl, while providing its own analgesia *via* kappa receptors.

That it provides its own analgesia has been thought to eliminate the sympathetic response that may result when a pure antagonist such as naloxone is used. Recently, however, it was shown that patients reversed with nalbuphine can demonstrate a sympathetic response.⁴

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Blood Warming Devices Do Not Guarantee Temperature Homeostasis

To the Editor:—The method for warming intravenous fluids in infants as described by Rosen *et al.*¹ is worth further consideration. Although the method was effective in warming intravenous fluid, as judged by the rise in temperature across the "warmer," its contribution toward causing a "beneficial increase in patient temperature" may

have been negligible. The critical parameter is patient input temperature. Neither this nor the iv flow rate was stated. Russell² and Vaghadia³ have shown that heat loss in the output line is a significant factor in the clinical setting. When the output line is long and the iv flow rate is slow, heat gained from the warming device may be largely