Correspondence

References


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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

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lost during transit through the output line. The patient input temperature would then approach ambient temperature.

In this context it is worth emphasizing that the use of disposable blood warming devices at low flow rates is of no benefit in maintaining temperature homeostasis. Measurements of input temperatures in 23 patients receiving blood via a disposable blood warming device showed that patient input temperatures were 0–2°C above the operating room temperatures. In all cases the rate of blood transfusion was less than 30 ml·min⁻¹. This suggests a wasteful use of expensive blood warming equipment. Vaghadia has already shown that when the rate of blood transfusion is less than 25 ml·min⁻¹ (approximately equivalent to 1 unit of whole blood given over 18 min), there is no advantage in using a disposable warming device. In fact, higher patient input temperatures can be achieved by simply immersing the iv tubing in a warming bath.

Hopefully, anesthesiologists will take into account the anticipated flow rate before resorting to the use of warming devices. If the flow rate is less than 30 ml·min⁻¹ and temperature homeostasis is important (e.g., in infants), consideration should be given to insulating the iv lines to minimize heat loss.

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Do Not Discontinue Antiarrhythmic Drugs Preoperatively

To the Editor.—Drs. Weiskopf and Stead have recently presented a current and interesting discussion of polymorphous ventricular tachycardia during coronary artery bypass surgery. However, an ambiguity in phrasing appeared in the text that may needlessly delay surgery in some patients with a prolonged QT interval. They state: "In patients on class I antidysrhythmic agents, a prolongation of the QT interval can occur, thereby increasing the susceptibility of the patient to Torsade des pointes. Decreasing the dose of the drug or discontinuing the drug entirely until the QT interval returns to normal is the appropriate treatment." Clearly the two references cited for this statement refer to patients who have a history of Torsade, not to all patients receiving drugs that prolong the QT interval as a therapeutic side effect. This recently came to our attention when we encountered a diabetic patient scheduled for elective surgery. He had a history of uncomplicated silent myocardial infarction; subsequent administration of quinidine for frequent premature ventricular beats (PVBs) had resulted in prolongation of the QT interval compared with previous ECGs (from 0.43 to 0.47 s). Surgery was unnecessarily postponed until an appropriate consult could be obtained, with the recommendation that quinidine therapy be continued throughout the perioperative period with no increased risk of Torsade de novo.

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