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The Use of an Extension Line in Epidural Anesthesia

To the Editor:—Two options that exist for epidural injection are through the needle or through the catheter. Those who prefer the needle as an injection port usually attach the syringe to the needle, then introduce incremental doses of local anesthetic with or without removal of the syringe.

As a modification of the traditional "injection by needle" technique, we place an extension line with a "T" (Abbott® 4616) between the Touhy needle and the syringe (fig. 1). The connector requires only 0.33 ml for priming and has little effect on the typical sensation during the injection, while the built-in clamp prevents back-flow.

In addition, the absence of a Luer®-type twist lock between the needle and the extension line makes the connection simple, further reducing the risk of inadvertent reposition of the needle.

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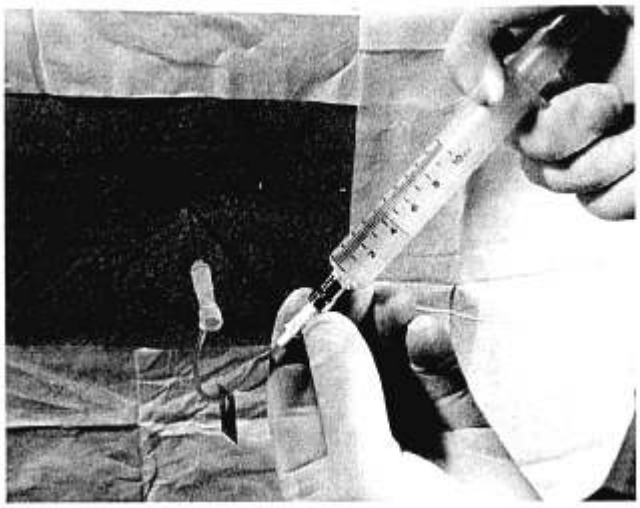

FIG 1. Illustration of extension line for epidural anesthesia.

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Tracheo-bronchial Angles in Infants and Children

To the Editor:—The article by Kubota *et al.*[1] documents the fact that there is indeed a difference in the angles subtended by the left and right mainstem bronchi at the carina in infants and children. They have, hopefully, settled a long-standing controversy.

However, they quote Brown and Fisk[2] (not Fish) as stating that the bevel of the endotracheal tube usually lies to the right on insertion. The fact is that the bevel of the tube faces to the left following insertion, as stated by Brown and Fisk, and the tip of the tube therefore lies to the right of the midline of the trachea. This is the reason for the fact that the tube invariably enters the right mainstem bronchus. The normal bronchial angles are of no real significance. In fact, if one wishes deliberately to advance the tube into the left bronchus, it can be done by rotating the tube through 180° before advancing it beyond the carina.

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65:237, 1986

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. Russell[2] and Vaghadia[3] 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