morphine sulfate 2 mg iv and 40% oxygen by mask, which resulted in a Pa_{O_2} of 70 mmHg. She received an additional 20 mg furosemide and was admitted to the intensive care unit. Her pulmonary edema gradually cleared over 36 h. Upon discharge her chest radiograph showed complete clearing of the pulmonary edema, and her Pa_{O_2} on room air was 98 mmHg.

In the two cases reported by Lee and Downes[1] and the cases detailed by Jenkins[2] and Cozanitis *et al.*[3] laryngospasm occurred during emergence from anesthesia, and development of pulmonary edema was immediate. The case reported by Jackson *et al.*[4] occurred following inability to accomplish intubation during anesthesia induction, and signs of pulmonary edema developed within 20 min.

The case reported by Oswalt *et al.*[5] also occurred secondary to airway obstruction during anesthesia induction, necessitating an emergency tracheostomy. Signs of pulmonary edema were not manifested for 2 h, but this diagnosis was clouded by subsequent development of lobar pneumococal pneumonia.

The mechanism for the development of postlaryngospasm pulmonary edema is said to be the large subatmospheric transpulmonary pressure gradients caused by attempts to ventilate against a closed glottis and/or hypoxia. These have been discussed in the previous case reports.

This case illustrates some important additional points. Laryngospasm can occur at any time during general anesthesia, not just during induction or emergence. Although this patient appeared well anesthetized for the pelvic examination, her depth of anesthesia was not appropriate for the strong stimulus engendered by cervical dilatation.

Although the patient subsequently was intubated for 20 min, there was no immediate development of pink frothy secretions and the lungs were clear to auscultation. Her clinical status did not deteriorate until 85 min after arrival in the recovery room. Many patients undergoing short procedures with general anesthesia are discharged to home after 60–90 min in the recovery room. Because of the previous reports of pulmonary edema following laryngospasm, we had planned to monitor this patient's postoperative respiratory status for several hours, and this vigilance proved to be necessary.

A third point concerns her hospital course. She was given intravenous diuretics and supplemental oxygen, but reintubation was deemed unnecessary. Improvement was steady, with return of her cardiopulmonary parameters to preanesthetic values during the next 36 h. The "lesion" at the pulmonary alveolar–capillary interface needs time to resolve.

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Anesthesiology
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Narcotic Analgesia—Ceiling Effect

To the Editor:—The letter from Eisele and Steffey[1] on the ceiling effect of narcotic analgesia is timely but difficult to resolve in the absence of any model for measurement of a very high intensity of analgesia in the human. The only apt relevant "experiment" is the use of narcotic analgesics as anesthetics for major surgery.

With partial agonist opioids, investigation is easier, as the ceiling effect is lower, at a level of analgesia appropriate for treatment of severe postoperative pain. We believe that we have evidence for a ceiling effect for analgesia using nalbuphine, at a dose comparable with the ceiling effect for respiratory depression, in patients given access to doses up to 200 mg/h "on-demand."[2] This evidence is reinforced by our experience with the

use of nalbuphine as analgesic component of minor and major surgery.*

However, the situation in the case of partial agonist opioids is complicated by what we take to be kappa-receptor-mediated effects, sedation and amnesia. In the case of pentazocine and nalbuphine, these effects readily are seen to be still increasing as the dosage is increased beyond that causing maximum mu-receptor-mediated respiratory depression (and analgesia?). We have provided anaesthesia in unconscious, spontaneously breathing patients for surgery such as vaginal repair or herniotomy, using pentazocine 3 mg/kg (not to be recommended). Many of our patients who used very large doses of nalbuphine, attempting to treat severe postoperative pain, were unsuccessful, judged by the linear analogue pain scores recorded but spent most of their time sleeping and had no subsequent memory of the period.

It seems obvious that kappa-mediated effects do not necessarily have the same dose–response relationship as mu-mediated effects, and this makes the assessment of analgesia even more complicated. Martin[3] suggests that kappa effects in dogs include "spinal analgesia" along with "sedation and anaesthesia." Such analgesia is not readily apparent in humans, but does kappa-induced narcosis count as analgesia? The question also arises as to the contribution that kappa-induced "anesthesia" could make to volatile anaesthetic anaesthesia. MAC is a measurement of only one element of such anesthesia; perhaps we must develop other methods of assessing narcotic or other elements.

So far as narcotic analgesic agonists are concerned, there is also evidence to support the importance of what may be kappa-receptor-mediated effects. Most anesthetists who have used opiate "anesthesia" are aware that patients so treated may be completely analgesic (surely a ceiling effect) but aware and that far greater doses of opiate may be required to ensure unconsciousness, presumably through a less-potent kappa-receptor effect.

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High Oxygen Saturation Does Not Always Indicate Arterial Placement of Catheter during Internal Jugular Venous Cannulation

To the Editor:—Catheterization of an internal jugular vein may be complicated by arterial puncture. Such an occurrence may be recognized by observing the force and color of blood flow. More reliable information can be obtained by connecting the catheter directly to a transducer and displaying the typical venous waveform and pressure. Measurement of oxygen content of blood obtained from the catheter should confirm the site of placement.[1] Failure to recognize this event and use of the Seldinger technique may lead to the placement of a large bore cannula in the artery with potentially lethal consequences.[2] We experienced an instance of abnormally high oxygen saturation, compatible with arterial blood, in a sample obtained from a central venous catheter. A 30-year-old man with end-stage renal disease secondary to insulin-dependent diabetes mellitus underwent kidney transplantation. After induction of anesthesia, the right internal jugular vein first was located with a 23-Ga exploring needle and then catheterized with an 18-Ga catheter. A syringe connected to a 20-inch extension tube was attached to the catheter, and a column of blood was freely aspirated. On disconnection of the syringe, the tube behaved as a manometer, and free fall of the column of blood was noted. This indicated venous placement of the catheter. The 18-Ga catheter then was exchanged using the Seldinger technique for an 8.5-Ga sheath. A triple-lumen catheter was advanced to a distance of 20 cm through the sheath. During the