confuse the clinical picture unless the associated electrocardiographic changes and other evidence of systemic gas embolism are recognized.

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Analgesic Nephropathy, A Form of Chronic Renal Disease

To the Editor: — I read with great interest the case report by Sivarajan concerning a 38-yr-old who underwent lumbar discectomy and subsequently developed acute tubular necrosis (ANESTHESIOLOGY, 1997; 86:1390–2). The authors imply that preoperative intake of nonsteroidal antiinflammatory drugs (NSAIDs) was the culprit in this otherwise healthy patient's perioperative renal dysfunction.

However, the authors have failed to notice the patients admitted intake of acetaminophencodeine (300 mg–30 mg) tablets every 6 h as another possibility. A form of chronic renal disease called *analgesic nephropathy* can occur in patients who ingest large quantities of acetaminophen and prostaglandin inhibitors. It is believed the minimal requirements for the development of renal damage are 2–3 kg of acetaminophen taken over a 3-yr period.

Pathologically, the acetaminophen injures cells by covalent binding and oxidative damage. The NSAIDs may then potentiate the effect of the acetaminophen by inhibiting the vasodilatory effects of prostaglandin, thus predisposing the papilla to ischemia. Thus, the papillary necrosis may be a result of the combination of the direct toxic effects of acetaminophen and of the ischemic injury to tubular cells and vessels.[1]

Luckily for this particular patient, after appropriate treatment and cessation of the offending drugs, his blood urea nitrogen and serum creatinine levels returned to normal. However, if his renal failure actually was a result of analgesic nephropathy, it is believed that he is at increased risk for the development of transitional papillary carcinoma of the renal pelvis.[2]

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In Reply: — We were aware of analgesic nephropathy occurring in patients ingesting large quantities of acetaminophen alone or in combination with NSAIDs. The first reference by Clive and Stoff in our case report has a detailed description of analgesic nephropathy. Analgesic nephropathy is a *chronic* disease that causes tubulointerstitial damage and dysfunction that presents as leukocyturia, hematuria, and inability to concentrate urine.[1] As mentioned in the case report, our patient exhibited none of these, and result of preoperative urinalysis was normal. Our patient suffered acute renal failure, and postoperative urinalysis was characteristic of acute tubular necrosis from which full recovery took place. Therefore, we did not consider analgesic nephropathy as a possibility.

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The Practice of Using Sevoflurane Inhalation Induction for Emergency Cesarean Section and a Parturient with No Intravenous Access

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To the Editor: — Schaut *et al.*[1] described a successful mask inhalation induction for emergency cesarean section in a patient without intravenous access. The authors acknowledged the limitations associated with two other anesthetic options for this challenging scenario: local infiltration by the obstetrician and intramuscular rapid sequence induction. A third alternative in this situation is an awake intubation under topical anesthesia followed by inhalation induction. Although this method requires some time and patient cooperation, securing the airway before induction of anesthesia affords an element of safety lacking in a mask inhalation induction.[2] Less optimal, alternative approaches include a single shot spinal anesthetic (in the case of a prolapsed umbilical cord, performed with the patient in the lateral decubitus position) with simultaneous intramuscular ephedrine, or perseverance in securing peripheral or central venous access before induction. Although all of the previously mentioned approaches have significant limitations, one should at least acknowledge that a variety of options exist.

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To the Editor: — We read with interest the case report by Dr Schaut *et al.*, on sevoflurane inhalational induction for emergency cesarean section in a parturient with no intravenous access (ANESTHESIOLOGY 1997; 86:1392–4). The authors did not address two important concerns in their case report: implications of commencement of surgery in absence of intravenous access, and medicolegal issues.

Cesarean section is not a benign procedure and can be associated with life-threatening complications such as uterine atony hemorrhage, hypotension, amniotic fluid embolism, air embolism, disseminated intravascular coagulation, uterine tears, and so on. These could lead to disastrous consequences in absence of an intravenous access. Baby delivery before intravenous access, therefore, may impose additional risks to risks already associated with mask anesthesia. Although the relative incidence of each of these complications is low, the combined risk of mask anesthesia and surgery without an intravenous access should not be disregarded. This raises an important question of whether the obstetricians were justified to put intense pressure on the anesthesiologists as described by authors. There appeared to be no time to discuss the additional risks involved with a technique deviating from standard of care with the patient or the husband.

During cesarean section, the risk-to-benefit ratio implies risk to the mother *versus* benefit to the baby. The primary responsibility of the anesthesiologist and the obstetrician is directed toward the safety of the mother. We congratulate the authors for producing a successful outcome. One favorable outcome, however, does not imply that the anesthetic approach is justified, safe, or advisable. If this mother had suffered some sort of catastrophe resulting from lack of intravenous access, it would be hard to defend the technique as performed here. Publication of this case report should not imply advocacy of this technique in similar circumstances, nor