creased contractility, and vasodilation may worsen the condition. However, sympathetic stimulation associated with painful labor also may have detrimental consequences, and therefore adequate analgesia for the first stage and a controlled second stage of labor is important.[1] By using a small dose of intrathecal fentanyl followed by a dilute epidural infusion, we obtained satisfactory analgesia without adverse hemodynamic changes. We anticipated that any changes that were to occur would be gradual and amenable to early intervention. In this respect, the predictive value of an amyl nitrite provocative test is unknown. In this case, our estimation of the risks of traditional methods of central neuraxial anesthesia prompted us to consider our modified approach.

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(Accepted for publication September 4, 1997.)

Anesthesiology
1998; 88:273

Laser Treatment of Endobronchial Lesions

To the Editor:—In her interesting article about complications of endobronchial laser treatment, Dullye *et al.* describe three cases of gas embolization. The underlining pathophysiologic mechanism is similar to gas emboli resulting from barotrauma in diving accidents. Because of high pressure, air is forced to enter into the pulmonary vasculature through ruptured lung tissue. Bubbles in the coronary arteries or the central nervous system can be life-threatening or result in prolonged neurologic deficits. Rapid repressurization according to the US-Navy Dive Table 6A can be a life-saving procedure and is described to improve especially the neurologic outcome even if treatment is delayed up to 30 h.[1] Limiting factors for hyperbaric therapy in critical ill patients are the access to an adequately equipped pressure chamber and concomitant diseases like chronic obstructive pulmonary disease, untreated pneumothorax, or severe seizure disorders, which may be exacerbated when oxygen is breathed under pressure.[2] In my opinion, hyperbaric therapy should be considered as a therapeutic option, even if the gas embolus is not the result of a classical diving accident.

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(Accepted for publication September 9, 1997.)

Anesthesiology
1998; 88:273–4

In Reply:—Dr. Schulz-Stübner brings up an excellent therapeutic option for those patients undergoing laser treatment of endobronchial lesions who suffer a cerebral air embolus. In the three patients we described in our report, only the second patient would fit into this category.[1] The first patient, we believe, suffered a helium gas embolus, delivered from the coaxial gas channel of the laser. We are not aware that there is any evidence that hyperbaric therapy is efficacious in this incidence. The second patient suffered an air and oxygen mixture gas embolus, and certainly hyperbaric therapy should be considered in this situation. The third patient's cerebral gas embolus was, we believe, 100% oxygen, and this rapidly dissolved without any neurologic deficit. Because many institutions do not have hyperbaric facilities readily available and because these patients often are not in the best physical condition to travel, the use of high concentrations of oxygen, when the risk of combustion is limited, is, perhaps, another alternative technique for avoiding a permanent deficit if a cerebral gas embolus occurs.

However, before hyperbaric therapy can be considered, the appropriate diagnosis needs to be made because the differential diagnosis of cerebral metastatic lesion or cerebral vascular accident may

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

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(Accepted for publication September 9, 1997.)

Anesthesiology
1998; 88:274

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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(Accepted for publication September 10, 1997.)

Anesthesiology
1998; 88:274–5

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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