

Fig. 1.

and gives mothers-to-be a choice about where to be delivered of a baby and how. Lay publications related to pregnancy, delivery, and confinement and antenatal classes are easily available.

The discussion about whether epidural analgesia for labor increases the risk of cesarean section 1,3,4 is not confined to the United States. We cannot answer the question whether labor epidural increases cesarean section rates in general. The Survey is applied in an unselected population with an observed variability in practice pattern of the obstetricians. This makes it difficult to study one intervention (labor epidural analgesia). We can state, however, that a high epidural analgesia rate does not imply a high cesarean section rate in an obstetric unit. Obviously, the practice patterns described differ from those seen in the United States and in other countries. The data presented do not provide any information about the reasons underlying those differences. However, proper assessment of this comprehensive data base may permit the evaluation of important issues in the future.

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# Acute Bronchospasm Associated with Methylmethacrylate Cement

To the Editor:—We read with interest the case report of acute bronchospasm associated with methylmethacrylate cementing during cranioplasty. The authors proposed that the bronchospasm was directly related to the action of absorbed methylmethacrylate.

We do not believe that they presented credible evidence regarding the cause of bronchospasm. First, they based their proposal on previous reports of reversible small airway obstruction and occupational asthma with methylmethacrylate exposure. <sup>2,3</sup> However, the reports they quoted were with long-term rather than acute exposure to methylmethacrylate.

Second, the authors' exclusion of pulmonary embolism regarding the cause of bronchospasm in their patient is not convincing. Bronchospasm is known to occur as an early manifestation of pulmonary embolism.  $^{4-7}$  Although various mechanisms have been proposed to explain bronchospasm associated with pulmonary embolism, the most plausible mechanism is the decreased local carbon dioxide (CO<sub>2</sub>). Severinghaus *et al.*<sup>6</sup> and Swenson *et al.*<sup>7</sup> showed that with unilateral occlusion of pulmonary artery, the decrease in blood flow shifts the ventilation away from the unperfused areas and local CO<sub>2</sub> decreases. The decreased CO<sub>2</sub> causes direct bronchiolar smooth muscle constriction, which can be prevented by inhalation of 6% CO<sub>2</sub>.  $^{6.7}$ 

Third, the authors excluded pulmonary embolism as the cause of bronchospasm because of the nature of the surgical procedure (cranioplasty) and the supine position of the patient. Although we agree that the incidence of pulmonary embolism during cranioplasty with methylmethacrylate should be very low, as compared to cemented total hip replacement, its occurrence cannot be excluded. Furthermore, pulmonary embolism has been reported in the supine position, although its incidence is lower than in the sitting position. It may occur as long as a negative gradient as small as 5 cm exists between the surgical site and the heart.

Fourth, the rapid resolution of bronchospasm in this patient could be attributed to the size of the emboli and the timely administration of therapeutic maneuvers.

Finally, had air been the cause of pulmonary embolism, the use of mass spectrometry would have indicated the presence of end-tidal nitrogen.

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# Sumatriptan Was Effective in Electroconvulsive Therapy (ECT) Headache

To the Editor:—We would like to draw attention to the benefit of sumatriptan on a 35-yr-old woman with chronic depression who was schedule for a course of electroconvulsive therapy (ECT) during general anesthesia. We were able to find only one reference of this subject. After her initial ECT sessions, the patient complained of severe headache. This appeared just after emergence from the anesthetic and lasted for several hours. After trying different analgesics with only partial or no relief, we decided to try 6 mg sumatriptan administered subcutaneously just after the ECT was performed. The patient had a history of migraine, and she described her ECT headache pain to be similar.

The patient was headache free after the first ECT in which she received sumatriptan. Except for one instance in which she experienced a pressure-tingling sensation, the patient continued to be headache free for the remainder of the final four ECT sessions of a 12-treatment series.

Sumatriptan is a selective 5-hydroxytryptamine (5-HT) receptor agonist<sup>2</sup> used for the acute treatment of migraine and cluster headache. The pain of migraine appears to involve the vessels of the pia and dura mater and the trigeminal nerve, which innervates these vessels. Specifically, activated trigeminovascular axons produce pain and result in the local release of vasoactive neuropeptides, which produce vasodilation.<sup>3</sup> This "neurogenic inflammation" is thought to be the mechanism underlying the pain of migraine. Sumatriptan appears to work *via* specific serotonin receptors

(5-HT 1B and 5-HT 1D) to mediate selective vasoconstriction within the cranial vasculature and to prevent the release of inflammatory mediators from trigeminal nerve terminals. Headache is a well-known side effect of ECT. Although the exact mechanism is not known, it is possible that 5-HT receptors are involved and also that the pain can take the form of migraine. Electroconvulsive therapy acts as well on 5-HT receptors and also on postsynaptic 5-HT receptors.

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