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The Risks of Central Neuraxial Anesthesia

To the Editor: — Ho *et al.*[1] report on the successful use of combined spinal and epidural anesthesia for the management of labor and delivery in a patient with idiopathic hypertrophic subaortic stenosis (IHSS).

Idiopathic hypertrophic subaortic stenosis, or hypertrophic obstructive cardiomyopathy (HOCM) as it also is known, is a cardiomyopathy characterized by asymmetric septal hypertrophy, and dynamic left ventricular outflow tract (LVOT) obstruction, which worsens with hypovolemia, increased left ventricular contractility, and vascular dilation.[2] The diagnosis is confirmed with two-dimensional echocardiography, and the LVOT gradient is quantified by Doppler echocardiography. Provocative testing with inhaled amyl nitrate is used to accentuate the gradient.

In the current case, the diagnosis of IHSS was made several years before pregnancy, but we are given no details regarding its severity; specifically, no mention is made of a "provoked" gradient at diagnosis. Without this information, the reader has no way of knowing what the severity of the condition was and therefore no way of knowing the risks of sympathetic blockade with neuraxial anesthesia. For the patient with the potential for severe LVOT obstruction, the risks of central neuraxial anesthesia are profound and should never be underestimated.

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In Reply: — We thank Dr. Oxorn for his interest in our report. During pregnancy, our patient's mild limitation of exercise tolerance remained essentially unchanged, and echocardiography showed a left ventricular outflow tract gradient of 15 mmHg. Although the response to amyl *nitrite* may provide some measure of severity, its use is not universal, it is not commonly used in our institution, and there are few data to support its safe use in pregnancy. We concur with Dr. Oxorn that hypovolemia, in-

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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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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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