circulation for normal livers, leading to an impaired flow through this circuit.

In light of the deleterious effects of a hepatic congestion for the portal circulation emphasized by Heinrich and Kao, manipulation of the hepatic arterial flow might be of more limited interest in the reported case. First, the arterial buffer response,[3] which has been confirmed to exist in humans,[4] might have led to a relatively high hepatic arterial flow in this patient with a presumably highly reduced portal venous flow. Second, trying to increase portal inflow to the liver would imply acting on mesenteric blood flow and thus on systemic blood flow,[5] which can be limited in the described clinical situation.

Concerning the atrial fibrillation, we agree on the comment raising the necessity to control the ventricular rate. However, cardioversion was judged dangerous at the acute phase of the decompensation and was considered as a consequence rather than the cause of the right heart failure. Accordingly, normal sinus rhythm spontaneously recovered after the hemodynamic improvement, suggesting that the atrial fibrillation resulted from rather than in hemodynamic dysfunction. Two years after this acute episode, the patient is still in good hemodynamic condition and has been removed from the heart transplantation list.

In conclusion, we believe that the observed beneficial effect of inhaled nitric oxide in this patient was the consequence of a significant decrease in hepatic venous pressure while mean arterial pressure was maintained. For the hospitals that "do not have the proper permission or setup for nitric oxide administration," we could advocate the use of a pharmacologic combination therapy to achieve these goals, although such a combination might be less effective or more difficult to use.

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# Respiratory Arrest after Second Dose of Intrathecal Sufentanil

*To the Editor:*—We describe a case of respiratory arrest after the administration of a second dose of intrathecal sufentanil during labor.

A 34-yr-old gravida 4, para 1, abortion 2 woman was admitted to York Hospital at 8 AM in active labor. She was 5′6″ tall and weighed 156 lb at term. Her medical history was unremarkable. On examination she was noted to have a single fetus in vertex presentation. Fetal heart monitoring by abdominal doppler revealed a reassuring pattern at a rate of 120 beats/min.

At 9 AM, at 5 cm dilatation, she requested labor analgesia for which 12.5 μg intrathecal sufentanil was administered with good result. The procedure was accomplished without difficulty at the L3–L4 interspace *via* a 24-G Sprotte needle. Sufentanil was diluted with cerebrospinal fluid (CSF) to a total volume of 2 ml. Vital signs were recorded every 5 min for the first 15 min after the sufentanil. Pulse and blood pressure remained stable, as did the fetal heart trace.

Four hours after the first dose of sufentanil, the patient reported return of painful contractions. She received another 12.5 μg sufentanil intrathecally *via* the same interspace as before. The second spinal procedure was uneventful, and the patient again reported excellent analgesia. Twenty minutes later, the spouse noted the patient was unresponsive to verbal commands. He immediately called the nurse to the labor room, who noted the patient to be in respiratory arrest.

While awaiting arrival of the emergency team, the nurse administered 100% $O_2$ by a self-inflating device. On arrival, the emergency team noted no spontaneous respirations, pinpoint pupils, pulse 78 beats/min, and blood pressure 118/60 mmHg. She was unresponsive to verbal commands and vigorous shaking. Fetal heart rate (FHR) was 60 beats/min from a baseline of 130. Naloxone (0.4 mg) was given as an intravenous bolus to which the patient responded immediately with prompt awakening. Seven minutes after naloxone, FHR increased to 150–160 beats/min. The rest of her labor course was unremarkable.

At 4:20 PM, the patient underwent a normal spontaneous vaginal delivery of an 8-lb, 1-oz male infant with Apgar scores of 9 and 10 at 1 and 5 min, respectively.

Sufentanil is a highly lipophilic opioid with a strong affinity for the opioid receptors. Its lipophilicity is advantageous in limiting its mean residence time in CSF, thereby minimizing potential side effects, such as delayed respiratory depression.[1,2] However, early respiratory depression is of concern. Recently, a case report appeared that described respiratory depression after a single dose of intrathecal sufentanil in a laboring parturient.[3]

The exact mechanism by which the respiratory arrest occurred in our patient is not clear. According to the study by Hansdottir *et al.*, intrathecal sufentanil has a mean residence time of 0.9 h in CSF but almost 7 h in plasma.[1] They pointed out that, after repeated doses of intrathecal sufentanil, there was a theoretical risk of accumulation of this drug in plasma but not in CSF. This may explain the respiratory arrest seen in our patient. The second dose of sufentanil was given approximately 3 h, 40 min after the first dose, at a time when the plasma concentration of the first dose, insufficient by itself, may have been augmented by the second dose to that above the threshold for respiratory arrest. However, cephalad migration of sufentanil in the CSF, leading to central respiratory depression, cannot be ruled out. D'Angelo *et al.*, from their observation of the cephalad extent of sensory changes resulting from intrathecal sufentanil administered at the lumbar spinal level, cautioned about the potential for respiratory depression.[4]

There are few data regarding the optimal dose of intrathecal sufentanil for labor analgesia, for either the initial bolus or repeat doses. An abstract addressing this issue suggests that there may be no advantage to using doses in excess of 7.5 μg intrathecal sufentanil.* Whether such a dose would reduce the likelihood of early respiratory depression remains to be investigated.

We wish to emphasize that patients receiving intrathecal sufentanil be monitored closely after each dose. As suggested by Hays and Palmer, this should include checking the respiratory rate every 15 min for the first hour after injection and every 30 min for the next 2 h.[3] It may be prudent to note the cephalad spread of sensory changes after each dose. Appropriate resuscitation equipment and personnel must be immediately available. Furthermore, dose-response studies are necessary to establish the optimal dosage schedule for single injection and continuous intrathecal sufentanil for labor analgesia.

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# Sevoflurane, Fluoride Ion, and Renal Toxicity

*To the Editor:*—In a recent editorial regarding a study by Kharasch *et al.*,[1] Brown[2] claims that sevoflurane is "biotransformed in a quantitative fashion similar to enflurane." How "similar" are they? In patients with renal impairment, sevoflurane administration resulted in average serum fluoride concentrations 85% greater than those given enflurane.[3] It has been reported that 8.1% of adult patients given sevoflurane had a serum fluoride concentration greater than 50 μM.[4] What is the corresponding percentage for enflurane? Brown's con-