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Nitric Oxide and the Treatment of Right Heart Failure with Atrial Fibrillation and Rapid Ventricular Response

To the Editor:—Gatecel *et al.* described their experience in the therapeutic application of nitric oxide in the intensive care unit.[1] This case report provides valuable clinical information regarding the hepatic circulation, which is important to liver-transplant anesthesiologists and intensive care specialists.

In the case report, a patient was admitted to the intensive care unit suffering from atrial fibrillation with rapid ventricular response, right heart failure, and liver injury. The patient underwent extensive hemodynamic monitoring and was given supportive measures, but the clinical condition continued to worsen. The patient was given inhaled nitric oxide to reduce the pulmonary arterial pressure, improve the right ventricular function, and decrease the central venous pressure. The hepatic venous oxygen content increased, and hepatic injury reversed. The mechanism is attributed to the increased hepatic perfusion pressure, a term we found to be ambiguous.

We would like to clarify the definition of hepatic perfusion pressure and propose an alternative treatment modality in this clinical scenario to be used in hospitals that do not have the proper permission or setup for nitric oxide administration.

Hepatic perfusion is achieved through two different afferent flows, blood supplied through the hepatic artery and through the portal vein. Hepatic perfusion pressure, the difference between mean arterial pressure and hepatic venous pressure, is mostly mean arterial pressure-dependent because venous pressure is usually low. The portal circulation, which supplies up to 75% of the hepatic blood flow, presents a completely different portal perfusion pressure. The difference between portal venous pressure (normal value 8–10 mmHg) and the hepatic vein is primarily hepatic venous pressure-dependent.[2] An acute 10-mmHg increase of the central venous pressure (transmitted as hepatic venous pressure) can effectively shut down the portal circulation. Conversely, a decrease of 10 mmHg in central venous pressure can reverse the portal circulation stasis. We believe that the patient described benefitted from the increased portal perfusion pressure.

Although the inhalation of nitric oxide apparently assisted in unloading the right heart, in a patient presenting with atrial fibrillation, we propose cardioversion or, when contraindicated, controlling the ventricular response rate as the first-line therapy.[3]

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In Reply:—The authors thank Heinrich and Kao for clarifying the meaning of hepatic perfusion improvement after nitric oxide inhalation in the reported case.[1] We kept this point relatively ambiguous because of space constraints, although we agree this aspect must be clarified. Hepatic arterial flow is proportional to the pressure gradient between mean arterial and hepatic venous pressures, whereas portal venous flow is dependent on the pressure gradient between portal venous and hepatic venous pressures. Considering these mechanical determinants of liver perfusion, we agree with Heinrich and Kao that the patient benefitted from an increased perfusion pressure in the portal circulation. Accordingly, a Starling resistance have been described in the portal circulation,[2] and the high hepatic venous pressure observed before nitric oxide inhalation was much likely to be higher than the critical closing pressure described in the portal venous

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.