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Pulmonary Hypertension and Liver Transplantation

To the Editor:—We read with interest the reports by Prager *et al.* and Cheng *et al.* on pulmonary hypertension (PH) in two patients with end-stage liver disease.[1,2] We agree that liver transplantation in patients with PH requires complex management and carries a high perioperative mortality.

At the University of Pittsburgh, we have had experience with seven patients with end-stage liver disease and moderate to severe PH (range of systolic pulmonary arterial [PA] pressure: 55–79 mmHg). All of these patients were challenged with pulmonary vasodilators before or during surgery, including nitroglycerin, prostaglandin E_1, and nifedipine, and none of them had a significant decrease in PA pressure. Five patients underwent liver transplantation; two of these patients died intraoperatively, two died in the early postoperative period (one of these patients was described in a previous report[3]), and one patient survived. One patient died while waiting for a liver transplant, and the last patient continues to await liver transplantation.

In addition to these seven patients, six patients with moderate to severe PH (range of systolic PA pressure 63–110 mmHg) are still under evaluation. However, three of these patients have died during the evaluation period. This high preoperative mortality rate reflects the dismal outcome of PH associated with portal hypertension; the mean survival after diagnosis has been reported to be 15 months, which is worse than in patients with PH alone.[4]

In our experience, patients with significant PH may survive liver transplantation only if they have good right ventricular function, have no other significant disease (such as coronary artery disease), have an expeditious surgical procedure, and receive a graft of excellent quality. Any significant complication (*e.g.*, sepsis, pulmonary infection, poor graft function) will likely result in death.

Despite the high risks, we have had one long-term survivor (20 months as of August 1992) after orthotopic liver transplantation.[5] Intraoperatively, the PA pressure was as high as 100/50 mmHg. Although this patient had an unstable intraoperative and postoperative course, he survived with good liver function. In contrast to the findings of Prager *et al.*,[1] this patient had normalized PA pressures when catheterized 1 yr after surgery. Thus, it appears that PH, on occasion, can be reversed by successful liver transplantation. Reversibility of PH may depend on several factors, as suggested by Prager *et al.* Fixed anatomic changes in the pulmonary vasculature may lead to irreversible PH, and the cause of the liver disease also may play a role.

In conclusion, we believe that PH should not be an absolute contraindication for liver transplantation. However, proper patient selection is extremely important, and even in the most suitable patients, perioperative management remains extremely difficult.

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In Reply:—It is not surprising with the increasing number of liver transplants that more anesthesiologists are encountering patients with liver failure and pulmonary hypertension. The experience of DeWolf *et al.* with their small series of liver transplant patients with pulmonary hypertension reinforces the fact that these patients have a high perioperative mortality rate.

The main reasons for organ transplantation are to improve lifestyle and increase longevity. If these goals are not achieved, the pro-

cedure must be questioned for the affected patient population. We are not advocating that patients with liver failure and pulmonary hypertension be excluded from liver transplantation. However, with the scarcity of donor organs, proper selection of organ recipients is important to ensure optimal patient and organ survival. It is hoped that further reports from physicians such as DeWolf *et al.* involved with liver transplantation will identify predictive factors that will help in selecting patients with liver failure and pulmonary hypertension who will do well after liver transplantation.

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In Reply:—The comments from the Pittsburgh group are appreciated, since their experience dramatically emphasizes the high perioperative mortality of patients with pulmonary hypertension undergoing liver transplantation. In addition, since we reported our case, we have heard from other transplant anesthesia teams who described intraoperative deaths in this particular patient population. I hope that our experience added to Pittsburgh's more extensive experience, as reported in their letter, will aid physicians who care for liver transplant patients.

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Cauda Equina Syndrome and Continuous Spinal Anesthesia

To the Editor:—Recently the Food and Drug Administration (FDA) withdrew manufacturers' marketing approvals for small-bore catheters (under 27 G) for intrathecal use because of a particular reported association of these catheters with the development of cauda equina syndrome.

The current controversy regarding the association of cauda equina syndrome and continuous spinal anesthesia originates from the excellent paper by Rigler *et al.*[1] published in 1991. They described four cases of cauda equina syndrome among a population of several thousand patients undergoing continuous spinal anesthesia. They attributed this to local anesthetic neurotoxicity.

In a subsequent study by Rigler and Drasner,[2] which involved a model of the subarachnoid space, concentrations of lidocaine were little different following introduction of local anesthetic solution *via* either of two types of a 20-G catheter or a 28-G catheter. Most importantly, all three catheters, when inserted so as to lie with their tip in the simulated sacral curve, produced marked pooling when hyperbaric solutions of lidocaine were injected, a phenomenon not seen with isobaric solutions.

The concept of pooling and the production of potentially neurotoxic concentrations of local anesthetic is not new. That pooling might occur with use of hyperbaric solutions of local anesthetic was suggested as long ago as 1937, when Ferguson and Watkins described 12 cases of cauda equina syndrome following single-shot spinal anesthesia with hyperbaric procaine.[3] In 1956, Payne suggested that the continuous technique might pose particular risks for the development of adhesive arachnoiditis (another condition possibly related to tissue toxicity of local anesthetic solutions) by allowing large or repeated doses of the agent to be administered.[4] In 1951, Morch *et al.* inadvertently demonstrated the possibility of pooling and the production of alarming concentrations of hyperbaric lignocaine should an intrathecal catheter pass caudally.[5]

Rigler *et al.*[1] reminded us of these dangers and went on to describe a number of sensible precautions that the anesthesiologist should take to minimize the risk of inadvertently producing neurotoxic concentrations of local anesthetic in the sacral cerebral spinal fluid. Also, they highlighted the common features of their cases that should alert the anesthesiologist to the danger of maldistribution—large or repeated doses of hyperbaric solutions and patchy or failed block. The caliber of the catheter involved was not one of these features, as one of their cases involved a 20-G catheter.

The FDA safety alert states that since December 1989, 11 cases of