CORRESPONDENCE

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