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.

Eugene Y. Cheng, M.D.
Harvey Woehlck, M.D.
Department of Anesthesiology
Medical College of Wisconsin
Froedtert Memorial Lutheran Hospital
9200 West Wisconsin Avenue
Milwaukee, Wisconsin 53226

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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.

Marie Csete Prager, M.D.
Department of Anesthesiology
University of California, Los Angeles
School of Medicine
1083 Le Conte Avenue
Los Angeles, California 90024-1778

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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

cauda equina syndrome have been reported associated with the use of small-bore catheters, but only one case since 1984 associated with a larger catheter. However, do we have any information on the frequency with which continuous spinal anesthesia has been used since the advent of smaller catheters, compared with the previous era when the idea of making an 18-G or larger dural puncture made continuous spinal anesthesia a less attractive anesthetic alternative? Do we know the relative frequency of use of small-bore *versus* larger-bore catheters since microcatheters became freely available? Might not the surge in awareness of the risk of cauda equina syndrome with the technique since 1991 have produced an increase in reporting of a complication that may often have escaped the attention of anesthesiologists in the past?

I am concerned that the anesthesia community might be distracted by the focus on microcatheters and their withdrawal by the FDA, from the wider and perhaps more relevant question of safe *versus* unsafe ways to administer a continuous spinal anesthetic through a catheter of any size. The current response of regulatory bodies both in the United States and in Australia has done little to prevent the administration of hyperbaric local anesthetic solutions through larger-bore catheters (such as the 20-G epidural catheters that have been used intrathecally for years) by practitioners who are not well informed about the important issues that Rigler *et al.* and Rigler and Drasner have raised.[1,2]

Dr. Philip Peyton
Department of Anaesthesia
Austin Hospital
Heidelberg, Victoria
3084 Australia

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In Reply:—We appreciate Peyton's comments. We share his concern that the current focus on microcatheters and their withdrawal by the Food and Drug Administration not distract us from considerations more fundamental to the safe intrathecal administration of local anesthetic.

As his letter suggests, recent regulatory decisions have not (nor could they have) eliminated the risk of injury—to avoid injury requires understanding the factors that contribute to neurotoxicity and informed clinical decisions. A small diameter may, in fact, adversely affect distribution; however, maldistribution can occur with catheters of any size. As Peyton notes, in our model, administration of anesthetic through any of the sacrally directed catheters resulted in high "subarachnoid" concentrations; clinically, maldistribution has been documented with catheters as large as 3.5 French.[1] Even if the relative incidence of neurotoxic injury were to differ according to catheter size, risk remains—indeed, one of the 12 cases occurred with a large-bore catheter. And, as Peyton points out, recent regulatory decisions do not prevent the administration of hyperbaric local anesthetic through large-bore catheters.

Moreover, maldistribution occurs not only with catheters of any size, but also with any spinal technique. Maldistribution is perhaps the most common cause for a failed single-injection spinal anesthetic; because repeat injection to overcome failure has the potential to produce the same restricted distribution, if maldistribution is the etiology for failure, there is risk of neurotoxicity.[2]

The events of the last 3 yr also have identified other issues relevant to the safe practice of spinal anesthesia. For example, there is a substantial body of evidence to suggest that neurotoxicity is concentration-dependent.[3,4] Consequently, we have suggested that anesthetics be administered at their lowest effective concentration.[5] Thus, the use of 5% lidocaine for either continuous spinal anesthesia or single-injection spinal anesthesia should be reconsidered since this concentration far exceeds what is needed for adequate blockade. Similarly, consideration should be given to administering anesthetics at the lowest effective tonicity, since tonicity may be an important factor in neurotoxicity.[6,7]

Kenneth Drasner, M.D.
Associate Professor of Anesthesia
Department of Anesthesia
University of California, San Francisco
San Francisco, California 94143-0648

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