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 pressure: 55–79 mmHg). All of these patients were challenged with pulmonary vasodilators before or during surgery, including nitroglycerin, prostanludin E1, 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 report3), 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 expedient 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.,6 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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