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HEMODYNAMIC CHANGES IN CLINICAL ORTHOTOPIC LIVER TRANSPLANTATION

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SERIAL CHANGES were measured in the cardiohemodynamics of 7 patients undergoing orthotopic liver transplantation, with the specific objective of determining the effects of simultaneously cross-clamping the portal vein and inferior vena cava before, during, and after the anhepatic phase.

METHODS

Cardiac output (with cardiogreen dye dilution), heart rate, stroke volume, mean arterial pressure, and central venous pressure (SVC) were measured: 1) following induction of anesthesia while the abdomen was being opened, 2) during mobilization of the recipient's liver, 3) shortly following cross-clamping the inferior vena cava and portal vein (early anhepatic phase), 4) just prior to revascularization of the donor liver (late anhepatic phase), 5) one hr later, and, 6) several hours following transplantation with the recipient awake. Each patient suffered from cirrhosis, portal hypertension, and extensive venous collaterals. There were two children (ages 3½ and 3½ years) with biliary atresia; 3 adolescents (ages 11, 15½ and 16 years) with chronic aggressive hepatitis, Wilson's disease and chronic aggressive hepatitis, respectively; and 2 adults (ages 47 and 47 years) with Laennec's cirrhosis.

RESULTS

Intermittent decreases in cardiac index, stroke volume, and arterial blood pressure were observed before the anhepatic phase apparently because of displacement of the native liver and partial

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interruption of the venous return. These changes became pronounced during the ensuing 52 to 116 min of the anhepatic phase (average 71 min) with the cross-clamping of the portal vein and inferior vena cava. The average maximum decrements were 47% ± 15 (SD)% of cardiac index 45% ± 26 (SD)% of stroke volume, and 19% ± 13 (SD)% of mean arterial blood pressure. The peripheral vascular resistance index increased by 95% ± 78 (SD)% whereas the heart rate increased by only 7% ± 11 (SD)%. During the late anhepatic phase, the alterations in hemodynamics were less noticeable than at the beginning, probably due to an intentional overtransfusion of blood in anticipation of blood loss incurred during revascularization of the new organ. The central venous pressure (SVC) did not change significantly during the operation. The transfusion of blood was insufficient in one severely ill child (3½ years). The control cardiac index was only 1.3 L/min/M² which fell to 0.7 L/min/M² during the anhepatic phase. After revascularization of the new liver in this patient, cardiac arrest occurred which was immediately reversed by external cardiac massage, adequate blood replacement, and administration of inotropic drugs. The alterations of hemodynamics noted during mobilization of the recipient's liver and during the anhepatic phase reverted to control levels shortly after revascularization of the new organ in this patient as well as in the other 6.

SUMMARY

In spite of pre-existing hepatic disease, portal hypertension, and extensive venous collaterals, significant cardiohemodynamic depression occurred during the anhepatic phase of liver replacement. With simultaneous cross-clamping of the portal vein and inferior vena cava, there was a moderate reduction in arterial blood pressure and a minimal increase in the pulse rate, but the cardiac index and stroke index fell sharply along with a substantial increase in peripheral vascular resistance. These changes were promptly reversed with revascularization of the homograft. A highly significant pooling with consequent reduction of venous return during cross-clamping of the great veins is offered as an explanation. The acute insult appeared to be well tolerated as previously reported (1), but the anhepatic period is potentially dangerous unless appropriate monitoring and therapy are provided at this critical intra-operative time.

REFERENCE

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