TITLE: EFFECTS OF INFERIOR VENA CAVA (IVC) CLAMPING ON RENAL PERFUSION PRESSURE DURING PEDIATRIC LIVER TRANSPLANTATION

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During liver transplantation IVC clamping may result in decreased renal perfusion, limiting the ability of the kidney to eliminate the hyperosmolar, acid, Ca^{2+}, and K^+ loads from massive transfusion.

In 27 pediatric liver recipients (13 males, 14 females, 17 ± 2 kg [mean ± SEM], 4 ± 1 year, .6 ± .05 m^2 BSA, extrahepatic biliary atresia 12, alpha$_1$-antitrypsin deficiency 6, others 9) anesthetized, monitored and transplanted as previously reported renal perfusion pressure (RPP, mmHg) inferior vena cava pressure (IVCP, mm/Hg), mean aortic pressure (MAP, mmHg), pulmonary artery wedge pressure (PAWP, mmHg), urine output (UO, ml/kg/hr) and hemodynamic profiles, were measured during surgical Phases I (dissection), II (anhepatic) and III (reperfusion).

Phase I (before IVC clamping) RPP, IVCP, MAP, PAWP, cardiac index (CI, L/min/m2 BSA) and systemic vascular resistance index (SVRI, dynes cm-5 sec/m2 BSA) were 69 ± 3, 16 ± 1, 85 ± 3, 15 ± 1, 9 ± 1 and 775 ± 66, respectively. Following IVC clamping (Phase II), RPP and CI significantly fell from Phase I values by 17 ± 5% ($p < .01$), and 24 ± 5% ($p < .01$), respectively. There were no significant changes in MAP or PAWP, but IVCP and SVRI rose by 126 ± 18% ($p < .001$) and 58 ± 15% ($p < .01$) respectively. At midanhepatic phase, RPP, IVCP, MAP, PAWP, CI and SVRI values stabilized at 61 ± 3, 35 ± 2, 95 ± 3 13 ± 1, 7 ± 1 and 1191 ± 116, respectively. Following IVC unclamping (Phase III) RPP, IVCP, PAWP, MAP, CI and SVRI were 70 ± 3, 20 ± 2, 15 ± 1, 88 ± 3, 8 ± 1 and 942 ± 89 respectively, values which were not different from before clamping with the exception of IVCP which was significantly higher ($p < .001$). UO during Phases I, II and III, which was forced with dopamine (2 mcg/kg/min), mannitol (150 mg/Kg) and furosemide (0.1 mg/kg/h), was 16 ± 2, 21 ± 4 and 28 ± 7, respectively. UO rates were not predicted by RPP, its MAP, IVCP or their percent changes during any of the surgical phases.

In contrast to adults in whom there exists a linear dependance between the fall in UO rate and RPP during Phase II , IVC clamping and unclamping in pediatric liver recipients, without veno venous bypass support, had little effect on UO rates and RPP, thus, not affecting fluid excretion by the kidney.

References
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TITLE: DISPOSITION AND RESPIRATORY EFFECTS OF INTRATHECAL MORPHINE IN CHILDREN

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The disposition and respiratory effects of intrathecal morphine have not been defined in children. Therefore we correlated spinal fluid (CSF) and serum (S) levels of morphine with the ventilatory response to CO_2 during 18 hours after intrathecal morphine administration.

Six children (5 males, 1 female) with ages of 0.3 -15 yrs (mean 6.2 yrs) underwent repair of craniofacial defects. All patients had normal cardiorespiratory and CNS function. No premedication was given and anesthesia was induced and maintained with halothane, N_2O, O_2, and pancuronium. Morphine 0.02 mg/kg via intrathecal catheter was given 3 hrs prior to the end of surgery. Neuromuscular blockade was reversed with neostigmine and atropine at the end of surgery and the patients remained intubated for the next 18 hrs. Analysis of morphine levels and CO_2 response occurred 6, 12, and 18 hrs after morphine administration. Minute ventilatory response (V_E) to end tidal CO_2 ($P_{ET}CO_2$) was determined by rebreathing a mixture of 95% O_2 and 5% CO_2 from a closed circuit in which a pneumotachograph and capnograph were placed in line.

S morphine levels (radioimmunoassay) remained undetectable at 6-18 hrs (Table). Compared to the 6 hr value, CSF morphine level fell to 14% (12 hr) and 9% (18 hr) respectively. The slope of the CO_2 response curve ($V_E/P_{ET}CO_2$) was depressed by 25-30% for up to 18 hrs after morphine administration compared to the preoperative slope obtained in 2 patients. The intercept of the CO_2 response curve defined as $P_{ET}CO_2$ at zero V_E did not change between 6 and 18 hrs after morphine administration.

We conclude that respiratory depression persists in children for up to 18 hrs after intrathecal morphine despite a marked decline in CSF morphine levels. Persistent respiratory depression despite falling CSF morphine levels may be due to binding of morphine to brain receptors.

TABLE

	PRE+	6h	12h	18h
$V_E/P_{ET}CO2$ (ml/kg/mmHg)	33 43	19.5±3.7	23.8±2.2	24.5±4.0
intercept(mmHg)	43 22	39±2	36±4	32±2
S MS**(ng/ml)		0	0	0
CSF MS (ng/ml)		3229±770	446±186*	287±197*

*p>0.05 compared to 6h value (ANOVA).
+preop values obtained in 2 patients.
**MS-morphine sulfate