SPINAL CORD HEMODYNAMICS DURING DELIBERATE HYPOTENSION

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Introduction: Deliberate hypotensive anesthesia is often used to minimize blood loss and provide a clear surgical field during spinal surgery. A potentially increased risk of neurological injury due to a diminished spinal cord blood flow with its resultant hypoxia has been suggested as a contraindication to the use of the hypotensive technique. Data speaking to this point are lacking. Therefore this study was undertaken to measure blood flow and metabolic rate of the canine spinal cord under the halothane - sodium nitroprusside (NPS) technique.

Methods: Regional cord (Cervical, T_1-T_4, T_5-T_{11}, T_{12}-T_{13},L-S) blood flow was measured with radioactive microspheres (15±3μ) in twelve halothane anesthetized (mask induced) mongrel dogs (26.9±1.3 kg) under control mean arterial pressure (MAP) of 88 mmHg (C), after 1 hour at 60 mmHg MAP using NPS (H_{60}) after 30 minutes at 50 mmHg MAP using NPS (H_{50}) and after 1 to 1½ hours following reestablishment of normotension (R). Pulmonary capillary wedge pressure was maintained at a mean value of 14.4±1.0 (SEM) before and after the hypotensive episodes. Mean delivered halothane was between 1.1% and 1.3% throughout. Body temperature was held constant for the entire series of interventions. Arterial PCO_2 was maintained at 37.4±1.6 torr, pH at 7.35±0.01 and oxygen saturation at greater than 95% throughout by appropriate ventilator manipulations, bicarbonate infusions or adjustment of inspired O_2 concentration. Mixed venous blood gases were measured as was venous blood assummed to be spinal column drainage. This spinal venous blood was drawn through a catheter inserted through the azygous vein to the T_6-T_7 level and advanced deep towards the spinal cord at the level of an intercostal vein. Appropriate arterial-mixed venous O_2 content differences ($C_{(A-V)}O_2$) were derived and whole body and an indication of T_5-T_{11} cord metabolic rates (VO_2) were calculated. Heart rate (HR) was counted from an ECG trace. Flow probes were placed around the T_5 and T_{10} intercostal arteries between the spinal column and the sympathetic chain in order to ascertain if a correlation existed between intercostal flow and actual spinal cord flow. Cardiac output (Q) was measured in triplicate by thermodilution using 3 ml iced D5W with injection beginning at the start of the expiratory phase of the ventilator cycle. Left ventricular wall (LV) and cerebral cortical grey (CG) and white (CW) matter were used as "external" comparisons for the microsphere flow data. Controlled ventilation was used through the complete protocol. A one-way analysis of variance was used for data analysis.

Results: The hemodynamic and oxygen consumption data are shown in Table I. To be noted are the increased assummed cord $C_{(A-V)}O_2$ at H_{60} and H_{50} and the increased assummed cord VO_2 under H_{50}. Flow data are shown in Table II. No changes were apparent in any of the tissues studied under any condition. There was no relationship between microsphere determined T_5-T_{11} cord flow and T_5 and T_{10} flow probe measurements as determined by a least squares linear regression analysis (r=0.1249, P=0.42).

Discussion: The data clearly show that there is only a minimal decrease in blood flow to any segment of the cord under NPS hypotension. There was a significant trend (Wilcoxon Signed rank test) for a diminished flow in the upper areas of the cord under H_{60} but actual flow remained unchanged. The external comparison values on LV, CG and CW are within the range of published values as are those for the spinal cord using other techniques. This lends credence to our data. Moreover, under H_{60} and H_{50}, there appeared to be an increased O_2 extraction by the cord which more than compensates for any diminished flow. The lowest value for assummed cord venous O_2 content was 6.9 vol% indicating that even under our worst conditions further O_2 extraction was still possible. It was hypothesized at the onset of this study that a correlation would exist between intercostal flow and cord blood flow thus allowing human cord flow studies under the hypotensive technique during spinal surgery. Such a correlation was not found thereby removing one clinical extension of this study. In light of these data it seems unlikely that deliberate NPS hypotension contributes to a diminished cord blood flow or to hypoxia.

Table I. Hemodynamic parameters under four levels of mean arterial blood pressure.

	C	H_{60}	H_{50}	R
MAP	87.8±3.5	59.8±0.4*#	50.5±0.6*#	78.7±3.1*#x
HR	148±5	162±8	176±9*	150±8x
Q	3.14±0.39	2.21±0.22	2.23±0.32	2.80±0.32
T_5-T_{11} $C_{(A-V)}O_2$	3.95±0.71	7.33±0.88*	7.93±1.11*	5.25±0.93
T_5-$T_{11}VO_2$	0.61±0.11	0.97±0.15	1.18±0.12*	0.85±0.12
Body VO_2	4.68±0.45	5.35±0.44	4.90±0.39	4.84±0.44

*P<0.05 from C, #P<0.05 from H_{60}, xP<0.05 from H_{50}

Table II. Blood flows (ml/100 gm - min) to segments of the spinal cord and to comparison tissues.

	C	H_{60}	H_{50}	R
Cervical	19.5±3.3	14.3±1.3	20.1±2.5	20.0±4.5
T_1-T_4	17.2±2.6	12.0±1.6	17.7±3.2	20.9±4.9
T_5-T_{11}	17.2±2.9	12.4±1.3	16.4±2.3	19.6±4.6
T_{12}-T_{13}	16.8±2.2	13.1±1.3	15.1±1.9	18.9±4.2
LS	20.8±3.6	18.7±2.0	20.7±3.3	23.6±5.7
LV	77.5±6.9	65.4±3.9	88.0±17.3	72.9±10.9
CG(n=4)	59.4±12.8	68.0±17.7	69.9±19.9	42.5±12.3
CW(n=3)	30.9±5.5	34.2±5.3	38.4±4.9	23.8±6.3