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TITLE: CHANGES IN SOMATOSENSORY EVOKED RESPONSES AND CEREBRAL BLOOD FLOW FOLLOWING INDUCED HYPOTENSION

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Introduction. Previous reports have shown that changes in somatosensory evoked responses (SEP) can be correlated to changes in cerebral perfusion pressure and consequently to cerebral blood flow[1-3]. Induced hypotension has been shown to affect brain function and in its clinical application is often carried to the limits of the autoregulatory capacity of the brain. It appears none the less, that reduction of the mean arterial pressure to 50 mmHg are compensated by the brain's autoregulatory mechanism resulting in no long term alteration in brain activity. Branston and his colleagues have shown that significant changes in evoked potentials occurred only after cerebral blood flow falls below 16 ml/min/100 gm of tissue. The possibility arises that induced hypotension which drives arterial pressure to the limits of autoregulation may transiently reduce cerebral blood flow below this critical limit.

Method. To evaluate these changes, nineteen mongrel dogs weighing 15-20 kg were anesthetized with 25 mg/kg sodium pentobarbital administered intravenously. The animals were intubated and ventilated so as to maintain blood gases within normal ranges. Arterial and venous pressures as well as lead 2 of the ECG were monitored throughout the procedure. A craniotomy was performed over the right temproparietal area and the dura incised and reflected. A recording electrode was placed on the pial surface at the level of the somatosensory cortex. Reference and ground electrodes were placed subcutaneously anteriorly and posteriorly to the cranial defect. Arterial pressure was lowered to 50 mmHg by the administration of an IV infusion of Trimetaphan (500 mg/1000ml 5% dextrose and water). The median nerve was stimulated by constant current pulses at a rate of 1/sec. at an amplitude twice that necessary to elicit a motor response. One hundred and twenty eight sweeps were averaged having a duration of 100 msec each, immediately after and at 15 min. intervals following hypotension. Cerebral blood flow was quantified using 15 micron microspheres, I_{125}, Ce_{141}, Sr_{85}, Sc_{46}, injected into the left ventricle prior to hypotension and at 15 and 60 min. after the induction of hypotension.

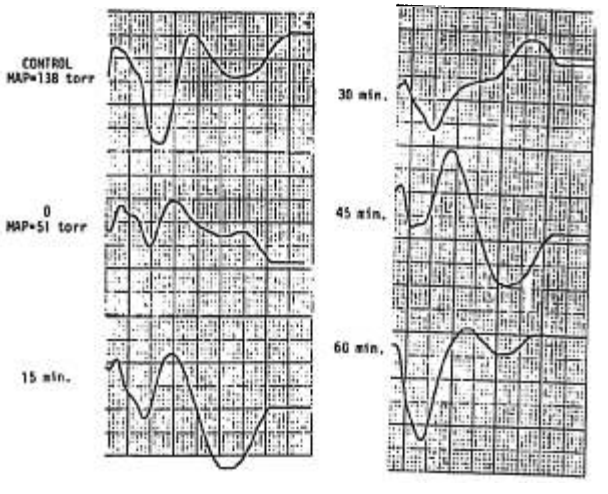
Results. A reduction in MAP from 138 to 51 torr (Table 1, Fig. 1) resulted in an immediate reduction in SEP amplitude to 58% of normal. This reduction was concommitant with a decrease in CBF to 47% of control. Within 15 min. the SEP and CBF had recovered to 70% and 85% of control values respectively. At 30 and 45 minutes, no further improvement in SEP's were noted. At 60 min., however, the SEP's were not statistically different when compared to control values and CBF had recovered to 94% of normal.

Discussion. Our data clearly indicates that transient alteration of SEP does occur following drug induced hypotension. This reduction is directly correlated to changes in cerebral blood flow. The autoregulatory mechanism also appears to re-establish perfusion in two phases. The initial drop in cerebral perfusion is followed by a rapid compensation re-establishing flow to about 85% within the first 15 minutes. This phase is followed by a slower reperfusion rate lasting as long as 45 min. thus returning the brain to equilibrium. It appears that the SEP may provide a useful monitor for cerebral perfusion under conditions where the brain is placed at risk during neurosurgical procedures. Supported in part by NIH Grant # NS 15316-02.

Table 1

	CONTROL	0	15	30	45	60
MAP mmHg	138±16	51±4	52±3	50±4	51±4	53±2
SEP % of control	100	58.1	70.1	63.5	61.2	84.9
p value	----	.004	.010	.020	.003	.620
CBF ml/min/100g	30.6±3.4	14.5±2.6	26.1±2.5	----	----	28.8±3.3
p value	----	.001	.020	----	----	.200

Figure 1



Effect of Induced Hypotension on SEP Amplitude

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