Title : EFFECTS OF HYPOXIC HYPOXIA ON SOMATOSENSORY EVOKED POTENTIALS

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Introduction: Somatosensory evoked potentials (SEP) are cortical responses to contra lateral nerve stimulation. The SEP has been used intraoperatively to measure the effects of surgery involving the spinal cord. The SEP has also been shown to be altered in a predictable manner due to tissue compression by retractors during neurosurgical procedures. Studies have shown that the amplitude of the SEP is decreased as cerebral blood flow (CBF) is decreased. This study was undertaken to determine whether alterations in the SEP reflect changes in oxygen utilization of the brain in situations where CBF is not decreased.

Methods: Five mongrel dogs anesthetized with sodium pentobarbital 30mg/kg IV were studied. The animals were intubated and mechanically ventilated. CBF was measured continuously by the venous outflow technique. Electrodes were placed in proximity to the dura in small holes filled with conductive gel. Stimulating electrodes were placed percutaneously in the area of the ulnar-nerve bilaterally. A stimulus rate of 1.9/sec with current 2 times that necessary to elicit a motor response was used. One hundred and twenty eight stimuli were averaged utilizing a Nicolet Med 80. Replications were obtained to insure correlation of the waves with stimulus. During the hypoxic studies, a single side was stimulated and followed throughout the experiment. Simultaneous arterial and cerebral venous blood samples were obtained at various levels of inspired oxygen, during the mid-point of the determiation of the SEP. Cerebral 0_2 consumption ($CMRO_2$) was calculated as the arterial minus cerebral venous 0_2 content times the CBF.

After a stablization control period, inspired oxygen was decreased from room air to 6%, and 4.5% each for a period of approximately 5 minutes. Animals were then returned to room air and sequential blood samples and SEP's were obtained during recovery.

Results: As arterial PO_2 was reduced from control (93± 3.5torr) to 21.4 ± 1.3 then to 15.4 + .11, CBF increased from control (24± 2.2 ml/min) to 250% and 200% of control. Arterial blood pressure increased from control (120± 8.0mmHg) to 152± 13.5 at 6% and decreased to 87 ± .11 at 4.5%. $CMRO_2$ was unchanged from control (1.68 ± .12 ml/0_2/min 0_2) (Table 1) except at the most severe hypoxic level ($Pa0_2$= 15.4± .11) when $CMRO_2$ was reduced to 53% of control. At 4.5% 0_2, a significant decrease in $CMRO_2$ was correlated with an increase in latency and decrease in amplitude of SEP. During the onset of hypoxia, the change in latency of the SEP correlated the change in oxygen uptake with an R value of -.611 (P<.005). The change in amplitude (percentage of control) vs the change in oxygen uptake was also well correlated with an R value of .62 (P<.005).

During recovery from hypoxia, the SEP returned to control at varying rates in individual animals. The latency of the primary wave was well correlated with the return of $CMRO_2$ to normal with an R value of -.577 (P<.005). During the recovery period, a poor correlation was noted between amplitude and oxygen uptake (R=0.10).

Discussion: These data show that SEP's change both in latency and amplitude in a consistent fashion during hypoxic hypoxia and that this change is correlated with oxygen uptake of the brain. No correlation in latency or amplitude was observed with CBF. During the recovery phase, the latency of the primary component is inversely correlated with the ability of the brain to utilize oxygen. The lack of correlation of amplitude and oxygen utilization during the recovery phase differs from that observed during initiation of hypoxia. Inspection of individual values indicates that the lack of correlation is due to an increase in amplitude proceeding a return of oxygen uptake.

These results suggest that the alterations in SEP during hypoxia are related to 0_2 utilization of the brain and may be useful in monitoring cerebral function in situations other than ischemia insults. During the initiation of an insult, both the alteration in latency and amplitude leads to information concerning $CMRO_2$. During recovery from an insult, the alterations in latency, but not amplitude, also appeared to reflect the metabolic status of the brain.

TABLE I

FI02	CBF ml/min	PO_2 mmHg	0_2 Uptake ml/min 0_2	Latency mili/sec	Ampl. µ/vis
Room Air	24.0 ±2.2	93.0 ±3.5	1.68 ±.12	17.8 ±.62	3.21 ±.72
6.0%	60.2 ±6.5	21.4 ±1.3	1.67 ±.28	17.7 ±.57	3.60 ±.90
4.5%	48.0 ±7.8	15.4 ±.11	0.90** ±.11	21.4* ±1.7	0.63* ±.17

Mean ± SEM
* P< .05 Compared to 6.0% 0_2 Value.
** P<.025 Compared to 6.0% 0_2 Value.