Title: RELATIONSHIP BETWEEN CEREBRAL BLOOD FLOW AND SOMATOSENSORY EVOKED POTENTIALS DURING GRADED INCOMPLETE ISCHEMIA IN GOATS UNDER GENERAL ANESTHESIA

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Introduction: Most experimental studies on the effects of ischemia on CNS electrical activity used temporary occlusion of cerebral arteries to produce total ischemia. To approximate conditions encountered clinically the present study investigated the relationship between global cerebral blood flow (CBF) and somatosensory evoked potentials (SEP) in the time course of graded progressive incomplete ischemia and subsequent recirculation in goats under anesthesia with nitrous oxide and etomidate.

Methods: In 10 male goats weighing 15-32 kg anesthesia was achieved by etomidate (0.5 mg/kg body weight) p. inf. and nitrous oxide in oxygen (FIO_2=0.3, mechanically ventilated). $P_{ET}CO2$ was adjusted to 33-38 mmHg. CBF was measured by a magnetic flow probe placed around the internal maxillary (IM) artery after ligation of the infradental, temporal, occipital and the contralateral IM. The buccinator, ophthalmic and ethmoidal arteries of both sides were artificially thrombosed by injection of 1500 I.E. thrombin into the IM cranial to the ligation so that virtually all of the blood supplying the brain via the rete mirabile was delivered by one IM as the basilar artery of the goat provides only minimal flow to the circle of Willis (1). The femoral vein / artery and sinus sagittalis were cannulated and analog signals of heart rate (HR), arterial blood pressure (AP), central venous pressure (CVP) and intracranial pressure (ICP) were stored on magnetic tape (Racal, 7DS®). Arterial, central venous and Sinus sagittalis blood samples were withdrawn every 15 min for measurement of blood gas tensions. Stimuli were delivered to the exposed median nerve at the paw at a rate of 5/s by constant current pulses (stimulation intensity: 2-fold motor threshold; duration: 0.2 ms, bandpass: 10-2000 Hz; analysis time: 80 ms). SEP recordings were made by platinum needle electrodes in the bone overlying the frontal and both somatosensory cortices, and the 6th cervical vertebra (Cv6) with a frontal reference. After recording of control values CBF was progressively reduced every 30 min (occlusion of the external carotid artery) by 50% until a basal blood flow of 20 $ml.min^{-1}$ was achieved. CBF was calculated per 100 g tissue weight post mortem. Statistical analyses were made using Students's T-test for paired and unpaired data (significance at $p<0.05$). Linear regression and correlation analysis were performed of SEP on CBF for ischemic and recirculation periods.

Results: Arterial blood gases, HR and AP were maintained constant during progressive ischemia and were comparable in all animals. CBF control values were 55-75 $ml.100g^{-1}. min^{-1}$. Graded occlusion of the external carotid artery led to a concomitant decrease in ICP (-40-60% at CBF: 20-30 $ml.100g^{-1}. min^{-1}$). Reperfusion led to a transient hyperemia (10-30 min) in 5 goats with a 20-140% increase of CBF compared to control. SEP consistently recorded were (neg.=N; pos.= P): N7 (mean=7.4, range=7.1-7.8); N10 (10.1, 9.7-10.5); P14 (13.8, 13.5-14.1), N20 (19.7, 18.9-20.7); P28 (26.8-31.9), N44 (44.2, 38.2-52.3). Components P14/N20 and N20/P28 were reduced significantly at a CBF < 40 $ml.100g^{-1}. min^{-1}$. Maximal depression was at a CBF of 20-25 $ml.100g^{-1}. min^{-1}$. Latencies and amplitudes of components < 14 ms didn't change significantly during ischemic and reperfusion periods but latencies of components ≥14 ms were prolonged in correlation to the CBF-reduction (P14:+1.2 ms; N20:+2.1 ms). Latencies and depressed amplitudes did only partially recover with reperfusion for an observation period of 120 min (Fig.). The correlation coefficient between P14/N20 on CBF was r=0.71 during progressive ischemia and r=0.38 for the reperfusion epoch. Correlation of central conduction time (CCT: difference of latencies: P14-N10) on CBF was r=0.76 during ischemia and r=0.51 after reestablishment of CBF.

Discussion: The data indicate that episodes of incomplete ischemia where critical levels for global cerebral blood flow (<20 $ml.100g^{-1}.min^{-1}$) were not attained may lead to prolonged impairment of brain function. The assumption is that prolonged periods of incomplete ischemia deteriorate brain function at a higher level of CBF than seen after transient epochs of complete ischemia (2). The findings of depressed SEP-amplitudes in combination with a sustained prolongation of CCT post CBF-reduction under steady state systemic circulation parameters might give a hint to depressed regional cerebal blood flows (<20 $ml.100g^{-1}.min^{-1}$) in certain areas (white and gray matter (2)) which were not supplied adequately by a global cerebral blood flow above a presumed critical level of 20 ml. $100g^{-1}. min^{-1}$ (ICP: 5-10 mmHg) under etomidate/nitrous oxide anesthesia. Furthermore it can be stated that hyperemic periods after transient incomplete cerebral ischemia are not necessarily beneficial for the restoration of functional capability .

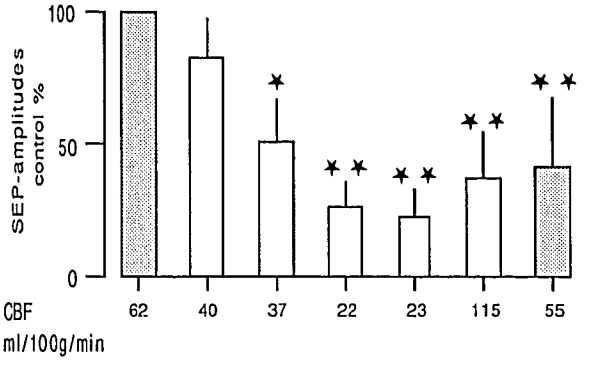

Figure: Relationship between CBF and SEP-amplitude (mean, ± SD) of component P14/N20 during progressively increasing ischemia and reperfusion period in ten goats (Shaded areas indicate difference in SEP-amplitude pre- and postischemic at comparable CBF-values (*p< 0.05;**p<0.01).

References:

(1) Miletich DJ, AD Ivankovic, RF Albrecht, ET Toyooka: Cerebral hemodynamics following internal maxillary artery ligation in the goat. J Appl Physiol 38: 942-945, 1975

(2) Lesnick JE, Michele JJ, Simeone FA, DeFeo S, Welsch FA: Alteration of somatosensory evoked potentials in response to global ischemia. J Neurosurg 60: 490-494, 1984

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