TITLE: SODIUM BICARBONATE IMPROVES OUTCOME AFTER TEN MINUTES OF CARDIAC ARREST IN DOGS.

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Introduction: Severe metabolic acidosis may impede or prevent resuscitation from cardiac arrest and adversely influence subsequent neurological outcome. Yet sodium bicarbonate ($NaHCO_3$) has essentially been removed from the armamentarium of resuscitation. The aim of this study is to determine whether or not treatment of metabolic acidosis with $NaHCO_3$ alters outcome after 10 minutes of ventricular fibrillation (VF) in a standardized animal model of cardiac arrest including post arrest intensive care. The latter has been omitted from all previous studies of this topic. Thus, the role of metabolic acidosis in impeding spontaneous circulation after restarting the heart remained undissected from the role of metabolic acidosis in preventing restoration of spontaneous circulation. Methods: Twelve dogs of 10-15 kg body weight with a ratio of AP/lateral diameters of the chest < 1.2 were premedicated with ketamine 10 mg/kg IM. Anesthesia was induced and maintained with halothane/N2O/O2 with pancuronium 0.1 mg/kg prn after endotracheal intubation. Intravascular catheters were placed percutaneously or by cutdown for monitoring of femoral and pulmonary arterial and right atrial pressures and for determining cardiac output and sampling arterial and mixed venous blood gases. The following were also monitored: EKG, urine output, expired CO2, airway pressure, pupillary size and reactivity and EEG. All animals were subjected to 10 min VF without CPR. Resuscitation consisted of CPR with a THUMPER and canine ACLS protocols with and without $NaHCO_3$. In the $NaHCO_3$ group, 1 mEq/kg was given empirically and then base deficit corrected to ≤ 5 mEq. In the control (C) group, no $NaHCO_3$ was administered. All animals were necropsied after the final neurological deficit score (NDS) (0%=normal, 100%=brain dead) determination. Survival for 24^0 was analyzed with Fisher's Exact test and all other parameters with the t-test for independent samples. Results: After the first dose of $NaHCO_3$, pHa was 7.22 ± 0.04 (SD) in the $NaHCO_3$ group (N=6) and 7.09 ± 0.12 ($p<0.01$) in the C group (N=6); base deficits were 9.6 ± 3.1 and 18.4 ± 5.3 respectively ($p<0.001$). Restoration of spontaneous circulation was possible in all of the $NaHCO_3$ dogs but only 4 of the C group (NS). At 24 h, NDS were $26 \pm 4\%$ in the $NaHCO_3$ group and 74 ± 5 in the C group ($p<0.001$). Our results suggest a substantial benefit to the administration of $NaHCO_3$ after 10 min cardiac arrest in dogs.

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TITLE: TREATMENT OF HYPOVOLEMIC SHOCK WITH HYPERTONIC - HYPERONCOTIC SOLUTIONS: EFFECTS ON REGIONAL CEREBRAL BLOOD-FLOW

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In hypovolemic shock hypertonic-hyperoncotic solutions are very efficient to restore blood pressure and cardiac output within a few seconds. The changes on the hemodynamic and circulatory system are well described (1) whereas the effects on cerebral blood flow in particular in cerebral trauma are unknown. We therefore investigated the effect of hypertonic-hyperoncotic solutions on traumatized brain.

Methods: New Zealand rabbits were anesthetized with α-chloralose, tracheotomized and ventilated mechanically. For monitoring of arterial and venous pressure, as well as for blood gas analysis and fluid infusion, catheters were inserted in the abdominal aorta and vena cava inferior. An electromagnetic flow probe was positioned around the pulmonary artery to continuously measure cardiac output. After exposure of the skull an open cranial window was prepared over the left hemisphere for insertion of Pt-electrodes and induction of the cold injury. Regional cerebral blood flow (rCBF) was measured in grey matter at increasing distances from trauma by the H_2-clearance-method. A control phase of 40 min was followed by a focal trauma (freezing lesion) in the occipital part of the brain. 20 min after trauma hemorrhagic shock was induced by withdrawing blood until the mean arterial pressure fell to 40 mmHg. 30 min later the animals were given 4 ml/kg of 7.2 % NaCl & 10 % Dextran 60 i.v. through an ear vein. rCBF was measured during the entire experimental course in 20-min-intervals up to 4 h after trauma.

Results: Cardiac output was 71 ± 14 ml/kg during the control phase and remained constant after trauma. During shock it fell to 17 ± 4 ml/kg; after infusion of NaCl-Dextran it rose to 85 ± 3 ml/kg. After trauma a significant increase in rCBF of $155\ \% \pm 9\ \%$ of control close to the lesion was measured; during shock rCBF was reduced to $115\ \% \pm 18\ \%$, while after infusion of NaCl/Dextran it increased to $152\ \% \pm 8\ \%$. Thereafter rCBF decreased again but reached a second peak 1 hour after NaCl-Dextran-infusion with $150\ \% \pm 15\ \%$ of control; then it returned slowly to baseline levels (Fig.1). In an area distant (13 mm) to the lesion hyperaemia after trauma was more pronounced ($210\ \% \pm 35\ \%$), but there was no initial rise of rCBF after NaCl-Dextran-infusion. The second rCBF-increase 1 h after treatment reached $140\ \% \pm 15\ \%$ of control (Fig.2).

Discussion: In traumatized brain the infusion of a hypertonic-hyperoncotic solution is followed by an initial as well as by a secondary hyperaemia in the adjacent tissue. In contrast distant to the lesion only the secondary increase in cerebral blood flow was found. The initial increase of rCBF in the vicinity of the trauma indicates a transient loss of autoregulation. The second and long-lasting hyperemia may provoke an elevation of the intracranial pressure (ICP) via an increased cerebral blood volume. Therefore ICP-measurements are highly necessary to determine the precise role of NaCl-Dextran in traumatized brain.

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References:
1. Rocha e Silva et al, Crit Care Med 18: 203-207, 1990

Fig. 1

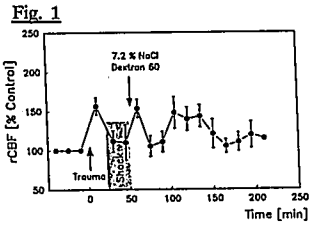
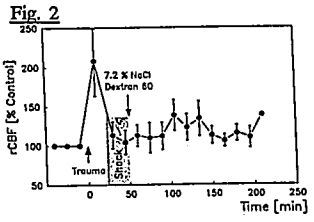


Fig. 2



rCBF close to (Fig. 1) and distant to (Fig. 2) a traumatic brain injury with hemorrhagic shock and subsequent treatment with NaCl-Dextran