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TITLE: SODIUM BICARBONATE IMPROVES CARDIAC RESUSCITABILITY, 24 HOUR SURVIVAL AND NEUROLOGICAL 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 clinical resuscitation. This study determined whether or not treatment of metabolic acidosis with $NaHCO_3$ altered outcome after 10 minutes of ventricular fibrillation (VF) in an animal model of cardiac arrest. This model included post arrest intensive care so that the role of metabolic acidosis in impeding spontaneous circulation after restarting the heart could be dissected from the role of metabolic acidosis in preventing restoration of spontaneous circulation. Methods: Forty dogs of 10-16 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 halothane/ N2O/O2 with pancuronium 0.1 mg/kg prn after endotracheal intubation. Catheters were placed percutaneously or by cutdown for 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 and expired CO2. After 10 min VF without CPR, resuscitation consisted of CPR with a THUMPER and canine ACLS protocols. 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° was analyzed with Fisher's Exact test and all other parameters with repeated measures analysis of variance (RMANOVA) and the t-test for independent samples. Results: After the first dose of $NaHCO_3$, pHa was 7.26 ± 0.12 (SD) in the $NaHCO_3$ group (N=20) and 7.20 ± 0.14 (NS) in the C group (N=20); PaCO2s were 50.1 ± 9.7 and 36.8 ± 12.2 mmHg p<0.02, and 20 base deficits were 5.5 ± 7.5 and 14.3 ± 4.0 respectively (p<0.005). Restoration of spontaneous circulation was possible in all of the $NaHCO_3$ dogs but only 15 of the C group (p<0.05). RMANOVA revealed no difference between groups in arterial oxygen or CO2 tension, arterial or mixed venous oxygen content, cardiac output, or oxygen delivery or transport. However, the C group had substantially higher inotrope and antidysrhythmic requirements. At 24 h, 19 HCO3 dogs were alive versus 11 in the C group (p<0.01) and NDS were 26 ± 6% in the $NaHCO_3$ group and 65 ± 11 in the C group (p<0.0005). Our results suggest a substantial benefit to the administration of $NaHCO_3$ after 10 min cardiac arrest in dogs.
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TITLE: URAPIDIL INCREASES INTRACRANIAL PRESSURE IN HEAD TRAUMA PATIENTS

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Urapidil has been established as a valuable tool in the treatment of hypertensive crisis. The mode of action as a peripheral alpha1-adrenoceptor antagonist and as an agonist of central serotonin (5-HT1a) receptors leads to a decrease in blood pressure without a significant sympathetic activation (reflex-tachycardia) (1). Intracranial pressure (ICP) seems to be unaltered by urapidil (2).
We report data on ICP elevation following urapidil treatment in 6 head-trauma patients of either sex.

Methods:
All patients had a CTscan of the brain on admission, reveiling signs of cerebral congestion due to traumatic lesions, but not requiring surgical treatment. A GAELTEC transducer was inserted into all patients for ICP-monitoring. The classical treatment for elevated intracranial pressure (hyperventilation to PCO2 at 30 mm Hg, sedation, relaxation) was performed. Supplementary, a continuous infusion of thiopentone (1.5 to 2.5 grams per day) was administered. ICP, mean arterial pressure (MAP) and heart rate (HR) were recorded on line. The cerebral perfusion pressure (CPP) was calculated according to the formula MAP - ICP = CPP. To control episodes of arterial hypertension the patients received a small bolus of urapidil (10 mg) iv. Hypoxia, hypercarbia and insufficient sedation and relaxation could not be accounted for the hypertensive status. Data were evaluated three minutes after intravenous administration of urapidil. Paired t-test was used for statistical analysis. A p-value <0.05 was seen as significant.

Results: (n=6)

	Control	Urapidil 10mg iv (after 3 minutes)
HR (b/min)	84±6	87±11
MAP (mmHg)	98±8	89±7
ICP (mmHg)	27±3	40±6 *
CCP (mmHg)	71±6	49±7 *

HR and MAP showed no change before and after administration of urapidil. ICP increased and CPP decreased significantly (* p<0.05). ICP and CPP returned to control values in a time period of up to ten minutes.

Conclusion:
As the small dose of urapidil left HR and MAP unaltered, it can reasonably be speculated to locate this phenomenon in the cerebrovasculature. Although contradictory to some reports in the literature, similar results have been described (3). It is conceivable that the compromised autoregulatory function of the cerebral vascular tree (cerebrovascular compliance) in a status of elevated ICP together with the alpha1-adrenoceptor antagonism of urapidil play a major role in this case. The small influence of alpha1-adrenoceptors in the cerebral vasculature may - **under these special circumstances** - become of significant importance. Therefore, in contrast to Van Aken's results the recommendation for urapidil in patients with elevated ICP has to be a very cautious one.

1. GROSS, G., et al. Naunyn Schmiedebergs Arch. Pharm. 336:597-601, 1987
2. VAN AKEN, H., et al. Int. Care Med. 12: 106-112, 1986
3. SINGBARTL, G., METZGER, G. Int. Care Med. 16: 272-274, 1990