Title: RECOVERY AFTER PROLONGED STANDARD AND "NEW" EXTERNAL CARDIOPULMONARY RESUSCITATION (CPR) IN DOGS.

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Introduction. Standard external CPR (SECPR) produces only 6-30% of normal common carotid artery blood flow (CCABF) (1). This may not sustain brain viability. Various methods have been examined to increase this borderline blood flow (2). "New" external CPR (NECPR), which increases intrathoracic pressure (ITP) fluctuations by lung inflations simultaneous with chest compressions, plus abdominal restraint, increased CCABF in dogs (5) and augmented arterial pressure and common carotid blood flow velocity in humans (6), when compared with SECPR. In this study we compared early recovery following SECPR and NECPR during 30 min in dogs.

Methods. Broad-chested dogs were anesthetized with ketamine and maintained with halothane-N_2O/O_2 50%/50%. Monitored were: $PETCO_2$; T (38°C); MAP; other cardiovascular pressures; EKG; EEG; arterial, venous and sagittal sinus (SS) blood gases, lactate and O_2 content; CSF lact; and pupil size and reactivity. 14 dogs were divided into 2 groups: (1) SECPR; (2) NECPR. Control values were obtained under light halothane anesthesia during steady state. After discontinuance of halothane, vent fibril was induced with 100 V-AC. Circulatory arrest was allowed to persist for 1 min. CPR with one of the above 2 methods was then carried out with a programmable thumper (Michigan Instruments) for 30 min. Standard CPR (n7) was performed with IPPV (TV 10 mg/kg; FIO_2 100%); ext card compr (60/min)-1:5. New CPR (n7) was performed with 40 compr/min (compr:relax time 60:40%), with simultaneous IPPV with each compression (peak airway pres 80-100cmH_2O; TV varied) plus abdominal binding, using a large blood pressure cuff (10 torr intracuff pressure) reaching from xyphoid to symphysis. After 1 min of arrest and 30 min of either SECPR or NECPR, restoration of spont circ was attempted by a standardized sequence of drugs and countershocks. Post-CPR art blood gases and MAP were controlled for 3h. At 6h post-resuscitation, neurologic deficit (ND) (0%= normal; 100%=brain death) was determined. Differences between groups were compared by Student's t-test. $p<0.05$ was considered significant.

Results. NECPR resulted in respiratory alkalemia, superimposed on the anticipated metabolic acidemia. At 30 min of CPR, pHa was significantly higher and $PaCO_2$ lower for NECPR (pHa=7.20±0.1 and $PaCO_2$=17±4 torr) as compared with SECPR (pHa=7.01±0.1 and $PaCO_2$= 39±14 torr). PaO_2 at 30 min of CPR was 284± 66 torr for NECPR as compared with 118±11 torr for standard CPR. SS pH was significantly higher, and SS PCO_2 and PO_2 lower during NECPR (SS PO_2=21±6 torr) as compared with SECPR (SS PO_2=34±9 torr). Both NECPR and SECPR were able to maintain pupillary reactivity and at least minimal EEG activity throughout the 30 min of CPR period. Spontaneous circulation was restored in 5/7 dogs in each group. Final ND scores among survivors were 7.6±15% for NECPR and 4.2±2% for SECPR (NS). CSF lactate after CPR (4-5 μM/ml) was the same in both groups.

Discussion and Conclusions. With this model, "New" External CPR produced (in comparison with Standard External CPR) hyperventilation and reduced cerebral venous PO_2. We had observed previously that increased ITP by NECPR causes increased ICP, decreased CPP and decreased SS PO_2 as compared to SECPR (adjusted for maximal systolic art pres) (2). This negative trend for NECPR may be absent or reversed if compared with less than optimal SECPR (3,4). Abdom. restraint alone increases CCABF, but also increases ICP, decreases CPP and SS PO_2 (and thus may decrease CBF) and can cause liver trauma (1). However the leg garments of pressure suits are useful for auto transfusion or hemostasis during trauma, shock or CPR. For prolonged external CPR in dogs, NECPR provides no advantage over optimal SECPR in terms of cardiac resuscitability, early neurologic recovery and complications.

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