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

Methods. Breast-chested dogs were anesthetized with ketamine and maintained with halothane-N2O/O2. MAP: PETCO2; T (38°C); MAP; other cardiovascular pressures: EKG, ECG, arterial, venous and sagittal sinus (SS) blood gases, lactate and O2 content; CSF lactate; 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 fribrit 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; FIO2 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 80-100cmH2O; TV varied) plus abdominal binding, using a large blood pressure cuff (at aortic intracuff pressure) reaching from aortop to sympyms. After 1 min of arrest and 30 min of either SECPR or NECPR, restoration of SpO2 was attempted by a standardized sequence of drugs and counter-shocks. Post-CPR art blood gases and MAP were controlled for 6h.

Results. NECPR resulted in respiratory alkaemia, superimposed on the anticipated metabolic acidaemia. At 30 min of CPR, pH was significantly higher and PaCO2 lower for NECPR (pHa=7.20±0.1 and PaCO2=17.4 torr) as compared with SECPR (pHa=7.01±0.1 and PaCO2=39±14 torr). NECPR may be 28466 torr for NECPR as compared with 11±11 torr for standard CPR. SS pH was significantly higher, and SS PCO2 and P02 lower during NECPR (SS P02=21±6 torr) as compared with SECPR (SS P02=34±9 torr). NECPR and SECPR were able to maintain pupillary reactivity and at least minimal EEG activity throughout the 30 min of CPR period. Sudden circulatory arrest was restored in 5/7 dogs in each group. Final ND scores among survivors were 7±1.5 for NECPR and 4.2±2% for SECPR (NS). CSF lactate after CPR (4.5 μM/ml) was the same in both groups.

Conclusion. With this model, "New" External CPR produced (in comparison with Standard External CPR) hyper-ventilation and reduced cerebral venous P02.

We had observed previously that increased ITP by NECPR causes increased CBF, decreased CPP and decreased SS P02 as compared to SECPR (adjusted for maximal systolic art pres) (2,3). Abdom. restraint alone increases CCABF, but also increases ICP, decreases CBF and SS P02 (and thus may decrease CBF) and can cause liver trauma (1). However, the leg garments of pressure suits are useful for auto transfusion and resuscitation 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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References