Conclusions: Evaluation of novel cerebral resuscitation potentials should not be taken from rat data indirectly to patient trials without systematic evaluation of risks and benefits in reproducible outcome models in a large species.

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Mild Cerebral Hypothermia after Cardiac Arrest Mitigates Brain Damage in Dogs

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Objective: To summarize the results of a logical sequence of four cardiac-arrest outcome studies.
Methods: The standardized ventricular fibrillations (VF) 10 or 12.5 min (no-flow) outcome models in dogs were used. In studies #1, #3 and #4, reperfusion was with brief cardiopulmonary bypass (CPB). In study #2, reperfusion was with external CPR. The use of intermittent positive pressure breathing (IPPV) was to 20 h and intensive care with outcome evaluation to 72 or 96 h. Outcome was determined as overall performance, neurologic deficit, and brain histologic damage scores. Mild hypothermia (34°C) was induced from reperfusion to 1–2 h; in study #2, a combination of external cooling methods was started after restoration of spontaneous normotension.
Results: In all four studies, cerebral functional and morphologic outcomes were significantly better in the mild hypothermia groups compared with normothermic concurrent controls. Mild hypothermia was more beneficial than was post-arrest moderate (30°C) or deep hypothermia (15°C), which worsened cardio-vascular variables. When start of cooling was delayed by 15 min after reperfusion, histologic but not functional improvement occurred.
Conclusions: These dog data and others' rat data justify clinical development and evaluation of rapid mild brain cooling methods for use in EMS and hospitals.

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Mild Protective and Resuscitative Cerebral Hypothermia Improves Outcome after Asphyxial Cardiac Arrest in a New Rat Model

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Objective: To explore in cardiac arrest (CA) the ability of mild hypothermia (Hth), tympanic temperature (Tty) 34°C, for protection (cooling before the insult) or resuscitation (cooling after restoration of spontaneous circulation (ROSC), to improve functional and morphologic cerebral outcome in this rat model of eight minutes (min) asphyxiation, to thereby confirm that the model's insult is in the treatable range.
Methods: Twenty-seven rats were randomized into normothermic control Group A (n = 10), resuscitative Hth Group B (n = 9), and protective Hth Group C (n = 8). Cooling was by external means. After eight min asphyxiation (CA 5 min), return of spontaneous circulation (ROSC) was with external CPR, epinephrine intravenous (IV), $NaHCO_3$ IV, and intermittent positive pressure ventilation (IPPV) to one hour (h). Evaluation to 72 h was in terms of neurologic deficit scores (NDS 0–100%), overall performance categories (OPC, 1–5), and whole brain histopathologic damage scores (of at least four coronal slides).
Results: The NDSs were lower (better) in Groups B and C compared with Group A ($p < .05$). Compared with Group A, OPCs were better numerically in group B and significantly better ($p < .05$) in Group C. Total ($p < .05$) and regional (numerical) histologic damage score were lower in Group C and correlated with ND (r = 0.83) and OPC (r = 0.79).
Conclusions: This rat model, with eight-minute asphyxiation (CA 5 min), is suitable for treatment trials as it responds to protective mild hypothermia. Mild resuscitative cerebral hypothermia in rats seems to improve outcome not only in models with incomplete forebrain ischemia (as shown by others previously), but also in a model of total body circulatory arrest.

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Cardiogenic Shock and Multiple-Organ Failure

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Introduction: Multiple-organ failure (MOF) is becoming one of the most important problems during anti-shock therapy of patients suffering from cardiogenic shock. Cardiogenic shock (CGS) was studied and its prognosis was evaluated.
Methods: All patients who were admitted to the emergency center with cardiovascular disease from 1 January 1991 through 31 December 1992 were studied.
Results: The total number of patients studied was 301. Of 301 patients, 30 were found to have cardiogenic shock. They con-

sisted of 17 males (mean age 58.3 ±18.8 years) and 13 females (68.7 ±12.6 years). The basic disease of CGS was predominantly acute myocardial infarction in 20 patients (66.7%), followed by arrhythmia in three patients (18.0%), congestive heart failure in five patients (16.7%), and myocarditis and valvular disease in one patient (3.3%). The involved organs were the lung in 26 (86.7%) patients, liver in 24 patients (80%), and kidneys in 16 patients (53.3%), respectively. Nine patients were found to have the complication of Disseminated Intravascular Coagulation (DIC). The mortality rate was 43%.

Conclusion: The mortality rate is higher in the patient group with severe organ injury. A tendency also was evident, indicating that the larger the number of involved organs, the higher the mortality rate.

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Active Compression-Decompression in CPR

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A new therapeutic principle in cardiopulmonary resuscitation (CPR) recently has been described. Alternate ("active") compression and decompression (ACD) is applied by means of a special device (Ambu CardioPump).[1] The intermittent negative pressure permits four possible improvements of CPR: 1) Increase of "cardiac output" due to suction of blood into the thoracic cage before the next compression closes venous valves; 2) Decrease in central venous pressure facilitates cerebral perfusion (which under conventional CPR nearly may cease); 3) Increased pulse amplitude in aorta implies better coronary perfusion, and thus may facilitate the response to various resuscitation measures; and 4) According to the principles of high-frequency ventilation, small tidal volumes improve alveolar gas exchange.

The first prehospital experiences with this device are presented. The need to deviate from the recommendations concerning compression- and ventilation-rates, suggested by the American Heart Association, soon was realized.[2] These guidelines could not foresee the completely altered cardiopulmonary dynamics obtained with ACD.

Using a compression rate of 40–60/minute (min) and a ventilation rate of 4/min (while maintaining oxygen supply to the tube between positive-pressure ventilations), a good peripheral pulsation, enabling reliable pulse oximetry, was achieved in a preliminary prehospital study. No patients obtained less than 92% oxygen saturation. This study is continuing and its results will be presented.

References

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Active Compression-Decompression Resuscitation: Effect on the Left Ventricular Volume and Transmitral Flow

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Introduction: Recently, cardiopulmonary resuscitation (CPR) incorporating active compression and decompression (ACD) of the chest has been demonstrated to improve hemodynamics in an animal model.

Hypothesis: This study was designed to test the hypothesis that ACD-CPR would increase transmitral flow and end-decompression left ventricular volume (LVV) when compared to standard manual CPR.

Methods: The ACD device was applied mid-sternum in five consecutive patients (3 male, age 44 ±18.5 years) and compared sequentially (in random order) to standard CPR. Both techniques were performed at 80 compressions/minutes, 1.5–2.0 inch compression depth, and a 50% duty cycle. Transesophageal echocardiographic data obtained in each patient during both CPR techniques included: velocity time-integral (VTI) of transmitral pulse-wave doppler recordings and two-dimensional images of left ventricle in long axis. With each CPR technique, planimetry volume measurements of the left ventricle were obtained at end-compression (EC) and end-decompression (ED) and the difference expressed as the stroke volume (SV).

Results:

CPR	EC (ml)	ED (ml)	SV (ml)	VTI
Standard	49.7 ±9.3	69.4 ±10.8	17.6 ±5.2	7.8 ±2.3
ACD	48.6 ±8.5	81.3 ±12.5*	32.6 ±6.8*	15.8 ±4.3*

*$p < .01$

Conclusions: Improved transmitral flow, end-decompression left ventricular volume, and stroke volume are seen with active compression-decompression resuscitation suggesting a biphasic cardiothoracic cycle of flow. Active decompression of the chest is an important adjunct to standard CPR.