

	D		S-CHF		D + S-CHF		1 Y-D	
	HBP	NBP	HBP	NBP	HBP	NBP	HBP	NBP
%Plac	8.2	5.8	5.8	2.3	14.1	7.9	19.6	16.2
%Zof	5.8	5.0	2.5	2.0	8.4	7.0	16.5	11.0
Risk Red %	32	minus 16	58	13	45	13	20	36

The treatment with zofenopri resulted in a greater benefit in HBP (*2p < 0.05, *2p < 0.01)

These data suggest that the benefit of early ACEI in patients with AMI can be enhanced in the hypertensive population.

1135-152 Effects of Enalapril on Tissue Factor and Plasminogen Activator Inhibitor in Patients With Acute Myocardial Infarction

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Background: Administration of angiotensin converting enzyme (ACE) inhibitors in patients with myocardial infarction is reported to reduce the incidence of acute coronary syndromes.

Methods: In a randomized, double-blind, placebo-controlled study beginning 4 weeks after myocardial infarction, 11 patients received placebo (P group) and another 11 received enalapril therapy (E group) for 4 weeks. Blood samplings were performed before the start and on the 3rd, 7th, and 28th days after the start of administration from all patients. We measured ACE activity, tissue factor (TF) antigen levels and plasminogen activator inhibitor (PAI) activity.

Results: There were no differences in the ACE activity (15.3 ± 1.7 and 14.5 ± 1.2 IU/l), TF antigen levels (230 ± 19 and 236 ± 21 pg/ml) and PAI activity (11.1 ± 0.8 and 10.6 ± 1.2 IU/ml) between the P and E groups. The ACE activity (8.1 ± 1.2), TF antigen levels (169 ± 13) and PAI activity (6.6 ± 1.3) decreased on the 28th day in the E group. However, three variables were unchanged (15.2 ± 2.0, 247 ± 19, 11.5 ± 1.1) during the study period in patients received placebo.

Conclusions: Enalapril therapy reduced the TF antigen levels and PAI activity in patients with myocardial infarction. The improvements in the coagulation and the fibrinolytic system may be associated with the reduction in the risk of coronary thrombosis seen with the use of ACE inhibitor.

1135-153 AT₁-Receptorblocker Versus Angiotensin Converting Enzyme Inhibitor in Acute Myocardial Infarction? Role of Dosage and Bradykinin on Remodeling, Hemodynamics and Mortality in the Rat Model

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Although ACE inhibitors may reduce mortality in subacute myocardial infarction (MI) they could be harmful during intense neurohumoral activation in acute MI. We investigated the contribution of angiotensin-II (All) synthesis inhibition, bradykinin (BK) breakdown, and dosage to the effects of ACE inhibitors. Three hours after coronary ligation or sham operation, rats received the AT₁-receptorblocker Candesartan (Can, 4 mg/kg/d, n = 52), high or low dose Quinapril (Qui-hi, 50 mg/kg/d, n = 56; Qui-lo, 6 mg/kg/d, n = 52), BK-B₂ receptor antagonist Hoe 140 (H, 500 µg/kg/d, n = 76), H + Qui-hi (n = 74), or Placebo (P, n = 51). After 7 days, mortality was analyzed. In surviving rats, left ventricular systolic (LVSP, mm Hg) and end-diastolic pressures (LVEDP, mm Hg) were measured by Millar catheter and left ventricular (LV) volume (Vol, ml/kg) was derived from passive pressure-volume relations. After MI, mortality with Can (13%), H (13%), H + Qui-hi (15%) and Qui-lo (19%) was similar as with P (18%, p = n. s.) but was higher with Qui-hi (38%, p < 0.05). In sham, mortality was zero with various treatments. LVSP, LVEDP and Vol were reduced to similar levels with Qui-hi and Can but remained unchanged with Qui-lo, H and H + Qui-hi treatments. Table shows data of large MI (≥30% LV circumference):

	P	Qui-hi	Qui-lo	Can	H	H + Qui-hi
LVSP	104 ± 3	87 ± 6	104 ± 4	85 ± 4*	111 ± 3 ¹⁵	104 ± 3 ¹⁵
LVEDP	15 ± 1	10 ± 2	12 ± 2	10 ± 1	15 ± 1	15 ± 1 ¹⁵
Vol	1.88 ± 0.03	1.74 ± 0.10	2.07 ± 0.10 ⁵	1.62 ± 0.07	1.87 ± 0.06	1.90 ± 0.08

(Mean ± SEM; *p < 0.05 vs. P, †p < 0.05 vs. Qui-hi, ⁵p < 0.05 vs. Can)

Thus, the AT₁-receptorblocker is safe in acute MI and prevents early remodeling, whereas the ACE inhibitor is only effective at high dose which increases mortality in this model.

1136 Technical Refinements in Cardiopulmonary Resuscitation

Tuesday, March 31, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 4:00 p.m.–5:00 p.m.

1136-107 The Impact of Hypertonic Saline Solutions on Myocardial and Cerebral Blood Flow During Cardiopulmonary Resuscitation

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Background: During cardiac arrest hemoconcentration deteriorates rheology of blood, causes sludge phenomena and effectively impairs microcirculation. Considering the success of hypertonic saline solutions (HS) in resuscitation from hemorrhagic shock we assumed that HS after cardiopulmonary resuscitation (CPR) might avoid hemoconcentration, increase blood volume and improve myocardial and cerebral blood flow (MBF, CBF).

Methods: In 30 anaesthetized pigs a 10 minute-normothermic cardiac arrest was induced by ventricular fibrillation. CPR was started with application of epinephrine, open chest cardiac massage and ventilation. The animals randomly received either 7.5% NaCl or normal saline solution. 5 minutes later DC-counter shock was applied to obtain return of spontaneous recirculation (ROSC). MBF and CBF were measured with colored microspheres. Systemic hemodynamics were continuously recorded.

Results: After HS infusion hematocrit was significantly reduced during CPR and within the first 30 minutes after ROSC (43 ± 3 vs. 38 ± 3%), the parameters myocardial (25 ± 12 vs. 36 ± 16 mmHg) and cerebral perfusion pressure (32 ± 14 vs. 59 ± 13 mmHg), cardiac output (0.3 ± 0.1 vs. 0.6 ± 0.2 l min⁻¹), MBF (1.5 ± 0.6 vs. 2.8 ± 1.8 ml g⁻¹ min⁻¹) and CBF (0.5 ± 0.2 vs. 0.9 ± 0.4 ml g⁻¹ min⁻¹) were significantly improved (P < 0.05). The 2 hour survival rate for animal with HC infusion was significantly higher than for controls (P < 0.03).

Conclusions: The improvement of systemic, myocardial and cerebral circulation after HC infusion during CPR after cardiac arrest is correlated with a better outcome.

1136-108 Systemic and Coronary Perfusion During Cardiopulmonary Resuscitation: Comparison of Manual Sternal and Automatic Circumferential Compression by an Automatic Vest

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Background and Purpose: Manual sternal compression, considered as the gold standard for external cardiopulmonary resuscitation (CPR) is of variable efficiency. The automatic pneumatic chest vest (Cardiologic Systems Inc, Hanover, MD) permits fatigue resistant, circumferential chest compression.

Method: Manual and automatic compression were compared in random order in the bovine model (n = 6, 65.3 ± 6.5 kg) with jugulo-carotid cardiopulmonary bypass (CPB) backup. Mean arterial pressure (MAP) was measured through an introducer in the femoral artery and coronary flow velocity (CFV) was recorded with a 0.014 inch intracoronary Doppler wire (Flowwire, Cardiometrics, Mountain View, CA) positioned in the LAD. Animals were reperused with CPB for 7 min between CPR periods (3 min) under various conditions of volemia (extracorporeal blood storage in venous reservoir). Values (MAP & CBV) during CPR are expressed as % of the values registered during CPB under the same volemic conditions.

Results:

Vascular volume litre	MAP (mmHg)	MAP (% of CPB)		CFV (cm/s)	CFV (% of CPB)	
		CPB	manual vest		CPB	manual vest
-2	20 ± 5	72 ± 26	122 ± 41	15 ± 10	130 ± 104	206 ± 186
-1.5	19 ± 8	72 ± 4	134 ± 20	23 ± 5	42 ± 18	100 ± 14
-1	36 ± 14	48 ± 13	96 ± 35	17 ± 8	97 ± 58	137 ± 57
0	55 ± 15	55 ± 10	88 ± 36	23 ± 8	68 ± 36	119 ± 30
+1	47 ± 12	62 ± 16	104 ± 20	25 ± 8	62 ± 26	113 ± 40

Conclusions: Compared to external manual compression, automatic pneumatic vest CPR results in superior, fatigue resistant systemic and coronary hemodynamics in the bovine model.