TITLE: EFFECTS OF THREE MODES OF ABDOMINAL COMPRESSION ON VITAL ORGAN BLOOD FLOW IN A PIGLET CPR MODEL

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Augmentation of cerebral blood flow (CBF) during CPR by phasic abdominal compression (AC) has been described in adult CPR models. Our study was designed 1) to evaluate effects of various AC modes in an infant CPR model, and 2) to identify potential mechanisms of augmentation by different timing of AC in relation to chest compressions (CC).

Anesthetized piglets (5.0±0.1 kg) were catheterized for measurement of aortic, right atrial, sagittal sinus pressures and regional organ blood flows (radiolabelled microspheres). CPR was started immediately after fibrillation and injection of epinephrine (10 μg/kg bolus + 4 μg/kg/min i.v.), with a CC rate of 100/min, a CC/ventilation rate of 4:1 and a CC duty cycle (DC) of 40%, using a Thumper$^{R}$. Piglets were randomized into 5 groups: Controls (CON; n=6) underwent CC-CPR only. Group VEST (n=7) were snuggly fitted with an unpressurized abdominal vest. Group BIND (n=6) had this vest inflated permanently to a pressure of 100 mmHg. Group SEQ (n=7) had the vest sequentially and phasically inflated to 100 mmHg for 30% of the cycle immediately prior to CC. In group SIM (n=5) AC and CC occurred simultaneously over 40% of the cycle.

Neither CBF nor myocardial (MBF) blood flow generated by AC-CPR in groups BIND, SEQ and SIM differed significantly from each other or from controls (groups CON and VEST) (Table; ANOVA for repeated measures). Cerebral and myocardial perfusion pressures were not different among groups. The deterioration of perfusion pressures and flows over time could not be modified by addition of phasic or continuous AC modes.

We conclude that, in this infant model of CPR, AC has no significant benefit to cerebral or myocardial perfusion. Enhanced diastolic cardiac filling, prevention of splanchnic venous pooling or increases in systemic vascular resistance were either not achieved in this model or were not effective in augmenting flows. (Supported by NIH NS20020 and NS01293).

| CBF: | | | | | (mean ± SE in ml/min/100g) |
|---|---|---|---|---|---|
| Group | Pre-Arrest | 10 | 20 | 35 | 50 min CPR |
| CON | 30±4 | 13±5 | 11±3 | 8±3 | 6±4 |
| VEST | 38±8 | 7±2 | 7±3 | 4±3 | 4±2 |
| BIND | 32±3 | 13±3 | 10±4 | 7±3 | 6±3 |
| SEQ | 31±4 | 13±2 | 6±2 | 3±2 | 2±1 |
| SIM | 30±2 | 12±3 | 12±4 | 6±4 | 5±3 |
| MBF: | | | | | |
| CON | 143±12 | 33±11 | 21±9 | 13±8 | 9±7 |
| VEST | 133±30 | 18±5 | 17±7 | 6±3 | 4±3 |
| BIND | 148±23 | 43±12 | 30±15 | 16±9 | 13±10 |
| SEQ | 109±15 | 27±6 | 11±8 | 10±8 | 8±6 |
| SIM | 127±25 | 21±9 | 27±18 | 23±21 | 17±16 |

## A301

TITLE: FAILURE OF A 21-AMINOSTEROID (U74006F) TO IMPROVE POSTISCHEMIC MYOCARDIAL FUNCTION OF ISOLATED RAT HEART

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Postischemic myocardial injury results from the ischemic injury plus multifactorial secondary factors associated with reoxygenation-reperfusion injury, which most likely leads to lipid peroxidation and membrane dissolution (1). We sought to determine whether a new compound U74006F (Upjohn, Kalamazoo, MI), a 21-aminosteroid (Lazaroid; LAZ) with purported lipid peroxidation inhibition activity would ameliorate postischemic myocardial injury of isolated perfused rat hearts. 30 male Sprague-Dawley rats (300-350g) were lightly anesthetized with ether and their hearts excised, chilled, and mounted on a Langendorff preparation. Hearts were allowed to equilibrate with Krebs-Henseleit buffer (KHB) and membrane oxygenation (pH 7.4, $PO_2$ >400, $PCO_2$±40), for 15 min. Baseline parameters of peak left ventricular systolic pressure (PLVSP), developed pressure (DP), end-diastolic pressure (EDP), (+) dp/dt, (-) dp/dt, EKG (via bipolar ventricular epicardial leads), and coronary flow (CF), were taken. Hearts were then exposed to 25 min of global normothermic (37°C) ischemia followed by 30 min of reperfusion with 1.5 mg/ml of LAZ vs. no treatment (KHB). At the end of 30 min of reperfusion myocardial parameters were again measured in both groups. Results indicate that LAZ at 1.5 mg/ml provided no resuscitative action when compared with controls (Table). We conclude that LAZ at the concentration studied does not have myocardial resuscitative capability after global myocardial ischemia of 25 min in this model.

Table
Myocardial Function Parameters

| Postischemic variable at 30 min | Control (n=10) | LAZ (n=11) | |
|---|---|---|---|
| PLVSP | *82.7±14.8 | 84.0±12.0 | (NS) |
| DP | 16.9± 4.0 | 24.4± 4.9 | (NS) |
| EDP | 65.8±12.8 | 59.6± 9.8 | (NS) |
| (+) dp/dt | 229.2±56.6 | 301.9±69 | (NS) |
| (-) dp/dt | 166.7±42.8 | 200 ±55.6 | (NS) |

*Variable±SE.
NS=not significant.

References

1) Circulation, 74; 2:215, 1986.