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Resuscitation Science Symposium

Session Title: Session III: Best of the Best Oral Abstract Presentations

Abstract 4: Small-Volume 7.5% NaCl Adenocaine/Mg²⁺ Preserves Cardiac Function During Hypotensive Resuscitation in the Pig Following Severe Hemorrhagic Shock

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Background: Cardiac rescue is critical for successful resuscitation. Previously, we reported that ~1 ml/kg 7.5% NaCl with Adenocaine (adenosine and lidocaine) and Mg²⁺ (ALM) successfully resuscitated rats following severe hemorrhagic shock, and that in pigs ALM significantly reduced the volume of Ringers acetate required to raise MAP to a target of 50 mmHg following 74% blood loss. The present study examines the effect of small volume 7.5% NaCl ALM on cardiac function during 60 min hypotensive resuscitation following 90 min shock in the porcine model.

Methods: Pigs (35–40kg) were subjected to pressure-controlled hemorrhage at a mean arterial pressure (MAP) of 35–40mmHg for 90 min (~75% blood loss). Pigs were randomly assigned to receive either 4 ml/kg 7.5% NaCl ALM (ALM n=8) or 4 ml/kg 7.5% NaCl (Control n=8). Cardiac output (CO), stroke volume, left-ventricular pressures and hemodynamics were collected continuously. Significance *p<0.05.

Results: MAP, end-systolic pressure and CO were significantly higher in the ALM pigs at the end of hypotensive resuscitation (Table). The higher CO was due to a higher stroke volume (SV) maintained at a significantly lower heart rate (HR). Paradoxically dP/dtmax tended to be higher in the Control group during the first 45min of hypotensive resuscitation (p=0.066) with no difference at 60min. Systolic ejection times were significantly increased in the ALM group and inversely related to HR. There was no difference in LV end-diastolic pressure between the two groups indicating that preload was similar during hypotensive resuscitation. LV blood flow at 30min resuscitation was comparable in the two groups (Control: 2.86±0.6 vs. ALM: 4.1±1.4 ml/min/g; p=0.7)

Conclusion: Superior cardiac rescue was achieved in ALM by a higher SV and CO, lower HR and longer ejection times vs Controls. Longer isovolumic contraction and ejection times may be due to suppression of SA nodal activity or rebalancing of sympathetic/parasympathetic control.

Time	Cardiac output(l/min)		Stroke volume(ml /beat)	
	Control	ALM	Control	ALM
Baseline	3.8±0.3	4.3±0.2	57±3	61±4
90min bleeding	1.8±0.1	1.7±0.2	9±1	9±1
15min fluid	3.4±0.2	3.7±0.6	17±1	20±2
30min fluid	2.9±0.2	3.3±0.5	15±1	17±2
45min fluid	2.5±0.2	3.2±0.4	12±1	17±1
60min fluid	2.0±0.2	3.1±0.5*	10±1	17±2*
Time	Heart rate (Beats pr. min)		Ejection time (ms)	
	Control	ALM	Control	ALM
Baseline	67±2	68±3	290±5	296±12
90min bleeding	205±8	195±7	88±11	111±10
15min fluid	196±10	180±7	120±8	143±5
30min fluid	204±11	184±8	81±13	127±9
45min fluid	210±8	185±8	74±12	127±10
60min fluid	205±7	183±11*	84±12	129±10*
Time	LV End-systolic pressure (mmHg)		dP/dt _{max} (mmHg/sec)	
	Control	ALM	Control	ALM
Baseline	105±3	114±9	1489±34	1649±85
90min bleeding	53±3	56±3	2784±233	2284±279
15min fluid	71±4	72±4	3796±457	2770±315
30min fluid	69±3	73±4	3912±395	2978±304
45min fluid	62±4	72±4	3471±459	2776±238
60min fluid	49±6	66±5*	2347±317	2552±281

One animal died during hypotensive resuscitation so n=7 in control group during hypotensive resuscitation. Values are Mean±SEM.
Control 7.5% NaCl ALM 7.5% NaCl ALM

Author Disclosures: **A. Granfeldt**: Research Grant; Significant; Partial grant support from Hibernation Therapeutics. **H.L. Letson**: None. **J.A. Hyldebrandt**: None. **E.R. Wang**: None. **P.A. Salcedo**: None. **T.K. Nielsen**: None. **E. Tønnesen**: None. **J. Vinten-Johansen**: Consultant/Advisory Board; Significant; Consultant for Hibernation Therapeutics. **G.P. Dobson**: Consultant/Advisory Board; Significant; Consultant for Hibernation Therapeutics.

Key Words: Resuscitation · Cardioprotective drugs · Adenosine