TITLE: EFFECT OF HYPERTONIC SALINE RESUSCITATION ON MYOCARDIAL PERFORMANCE IN A HEMORRHAGIC SHOCK MODEL: ASSESSMENT BY PRESSURE-VOLUME LOOPS

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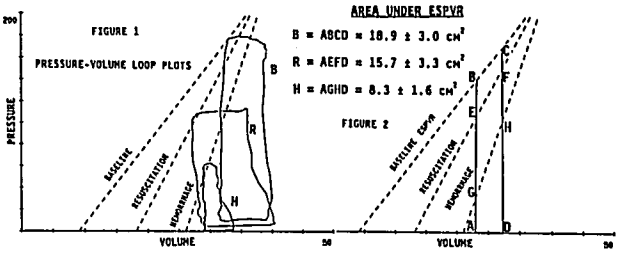
Introduction. Hypertonic saline solution (HSS) has been established as a resuscitation regimen in burns and other forms of shock. After HSS, total blood volume remains below baseline, however cardiac output remains above baseline. These observations suggest that HSS may have a direct effect on myocardial performance. This study was designed to evaluate the effect of 7.5% HSS on myocardial performance by assessment of left ventricular end-systolic pressure-volume relationships (ESPVR).

Methods. Six mongrel dogs were anesthetized and instrumented with the following: femoral artery and vein catheters, swan-ganz in pulmonary artery, micro-tipped Millar pressure catheter and Webster volume-conductance catheter in left ventricle, and inferior vena cava occluder. ESPVR generated during baseline, after hemorrhage to MAP of 50 for 60 minutes, following resuscitation with 4 ml/kg HSS. Resuscitation data collected at 5, 15 and 30 minutes. Statistical significance with ANOVA.

Results. Figures 1&2 show P-V loops and ESPVR respectively.

	BASELINE	HEM	RES$_5$	RES$_{15}$	RES$_{30}$
HR	163 ±14	169 ±13	179 ±9	184 ±10	179 ±12
MAP	150 ±13	51 ±2*	123 ±17+	107 ±10*+	97 ±14*+
SV	20.8 ±2.4	8.4 ±1.6*	18.1 ±2.6+	15.7 ±2.4*+	17.2 ±4.5
CO	3.38 ±.49	1.36 ±.16*	3.31 ±.52+	2.97 ±.51+	2.94 ±.60+
SVR	3569 ±256	2989 ±333	3665 ±410	3191 ±372	2654 ±382*
Hct	48 ±2	43 ±2	34 ±1*+		
Na	152 ±1	151 ±3	166 ±4*+		
ESPVR (area)	18.9 ±3.0	8.3 ±1.6*			15.7 ±3.3

Values = mean ±SE. P<0.05, * vs Baseline; + vs Hemorrhage.



Conclusion. Hemodynamic indices were restored to control levels following HSS resuscitation. The ESPVR has been shown to be a load-independent index of myocardial contractility.1 However, areas beneath the ESPVR more accurately describe contractility because volume intercepts are normalized.[2] Using the area-ESPVR index our study suggests that HSS restores myocardial performance from the hemorrhaged state.

1. Sunagawa K, Circ Res, 50:727, 1982.
2. Crottogini A, Circulation, 76:1115, 1987.

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Title; Myocardial intracellular Ca++ "overload" is the cause of arrhythmia during hypokalemia.

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Introduction: Some studies have shown that hypokalemia leads to increased myocardial arrhythmia, others have failed to see an increase. Previous studies in our laboratory, however, have clearly shown that myocardial arrhythmia is significantly increased. Specifically, our studies with rats fed low potassium (K) diets show that at least two weeks of hypokalemia are necessary before increased myocardial irritability is achieved. We noted that at the point of increased myocardial irritability heart K was only reduced by a modest 15%. We concluded that other ionic changes were occurring which might contribute to the appearance of arrhythmia. It was the purpose of this study, therefore, to measure changes in myocardial K, sodium (Na), and calcium (Ca).

Methods Laboratory rats were placed on K deficient diets. After 5, 10, 15 and 20 days of diet, groups of rats were sacrificed and the heart removed for K, Na, Ca analysis. Anesthesia was maintained with 1.5% halothane. Epinephrine was injected in order to induce arrhythmia. In the rat, epinephrine arrhythmias tend to occur in an uninterrupted chain, the duration of which is proportionate to the dosage of epinephrine.

Results/Discussion Data from this study confirm our earlier observation that heart K remains normal in rats fed low K diets up to 15 days despite an immediate 50% decline in blood K. At 15 days, heart K declines only 15% while the duration of arrhythmia increases suggesting a possible relationship between heart K and arrhythmia. However, our new findings show that both heart Ca and Na increase by day 15. These ionic movements are in agreement with the literature. For example, in our study, as heart K fell, heart Na accumulated in exchange. Since it has been shown that intracellular Na depresses cellular Ca efflux, the accumulation of Na in our study probably caused the accumulation of intracellular Ca. Increases in intracellular Ca are of concern. Studies have shown that any pathology which causes intracellular Ca "overload" also causes serious arrhythmia. We conclude that arrhythmia associated with chronic hypokalemia may be initiated by myocardium Ca "overload" rather than modest K depletion.

Day	Heart K	Heart Ca	Heart Na	Arrhythmia Duration (Sec.)
0	102±6	3.8±0.2	11±3	52±6
10	98±5	4.0±0.1	15±2	57±5
15	95±5*	4.3±0.2*	21±3*	60±5
20	86±4*	4.6±0.3*	26±3*	127±14*

* Significantly changed from Day 0, p<0.05; mean ± SE. K and Na = mmol/kg wet wt; Ca = μmol.