Early Hemodynamic Changes are Associated with Mortality from Atrio Ventricular Node Arrhythmias Following Hemorrhage in a Swine Model

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Background: Life threatening hemorrhage is the leading cause of mortality on the battlefield. During a study on the effects of remote ischemic preconditioning in pigs subjected to controlled moderate hemorrhage, we noticed that all pigs that died prior to the end of the experiment developed Mobitz type 1 atrial ventricular (AV) node block or a junctional rhythm prior to death. Mobitz-1 or junctional rhythm did not occur in surviving pigs. Hemodynamic parameters between groups were analyzed further.

Methods: Hemorrhagic shock was induced in 16 pigs by removing 50 mL aliquots from a femoral arterial line to achieve a total of 35% reduction in blood volume. The animals were observed for 7 hours (or until death) following bleeding. Measured hemodynamic parameters included: mean arterial blood pressure (MAP), cardiac output, mixed venous oxygen saturation (SvO₂) and heart rate. Blood samples were taken hourly for complete blood count, selected biochemistry, and blood gasses. No other interventions were performed. Oxygen delivery (DO₂), oxygen consumption (VO₂) and oxygen extraction ratio (ER) were calculated. Cardiovascular and laboratory parameters were compared between pigs with and those without arrhythmias.

Results: Five animals had arrhythmias and died, and 10 had no arrhythmias and survived. One was excluded due to suspected malignant hyperpyrexia. During the first 2 hours after achieving 35% blood loss, unlike the pigs that survived, those that died did not have compensatory increase in their cardiac output (CO) (p=0.008), oxygen delivery (p=0.016), or MAP (p=0.013) (Figure 1). Moreover, SvO₂ remained low (p=0.012). Changes in heart rate, VO₂ and ER were not statistically different between groups. There was a corresponding increase in serum lactate (p=0.075) and decrease in pH (p=0.012) by the second hour of observation (Figure 2). There was no difference in other measured values. These reported differences continued until the animals expired.

Figure 1: Cardiac Output (CO), Heart Rate (HR) and Mean Arterial Pressure (MAP) vs. Time

CO = Cardiac Output, L min⁻¹; MAP = Mean Arterial Pressure, mmHg; HR = Heart Rate, Beats Min⁻¹; Time - Minutes

Fig 2: Lactate, Potassium and pH over time

Lactate, mmol L⁻¹; Potassium mmol L⁻¹; Time - Hours

Conclusions: These results suggest that inadequate early compensatory physiological may be predictive of poor outcome after moderate hemorrhage. To the best of our knowledge this is the first time in a large animal model that maladaptive responses across a range of cardiovascular parameters have been reported to begin immediately after hemorrhage.