Insufficient Centralization During Hemorrhagic Shock Precedes Occult Hypoperfusion After Resuscitation in a Porcine Polytrauma Model

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Purpose: Occult hypoperfusion (OH) describes the absence of sufficient microcirculation despite normal vital signs. It can be quantified via persistent lactatemia and is associated with complications and a reduced outcome. Our study aims to identify potential causes for OH after hemorrhagic shock (HS) in a porcine polytrauma model.

Methods: 25 male pigs (Swiss Landrace) weighing 50 ± 5 kg underwent general anesthesia for 6 hours. They received a combined injury of blunt chest trauma with a lung contusion, a grade II (AAST [American Association for the Surgery of Trauma]) liver laceration, controlled HS (mean arterial pressure 30 ± 5 mm Hg for 60 minutes), and femoral shaft fracture. After 60 minutes, fractures received intramedullary nailing and polytraumatized animals were resuscitated with crystalloid fluids. Microcirculation was evaluated using near-infrared spectroscopy on the M. vastus lateralis of the fractured and uninjured site as well as on liver, stomach, and colon. Lactate was measured via blood gas analysis. All measurements were taken at baseline, during HS at 15/30/45/60 minutes, and after resuscitation at 2,4, and 6 hours. OH was defined as persistent lactatemia (>2 mmol/L) at 6 hours.

Results: Lactate normalized in 18 animals (72%) after resuscitation (control). Four animals (16%) were diagnosed with OH. Three animals (12%) died during the observation period. Vital parameters (heart rate, respiration rate, SaO2, and shock index) did not differ significantly between groups. Lactate was significantly higher in OH at 3 and 6 hours ($P = 0.039$ and $P = 0.001$). Significantly higher local blood flow was noted during HS on the uninjured M. vastus lateralis at 30, 45, and 60 min ($P = 0.012$, $P = 0.039$, and $P = 0.008$) in OH compared to controls. Oxygen delivery at that site was significantly higher at 45 and 60 min ($P = 0.044$, and $P = 0.004$), but lower at 4 hours ($P = 0.039$).

Conclusion: Our data suggest that failure to centralize during hemorrhagic shock can lead to reduced local microcirculation and persistent lactatemia after resuscitation and OH. Further research is warranted to better understand hemodynamic decompensation and recompensation during HS. Our results support the validity of lactate clearance as a marker of sufficient resuscitation.