



Characterisation of a Paediatric Porcine Model of the Microcirculation and Coagulopathy in Traumatic Haemorrhagic Shock

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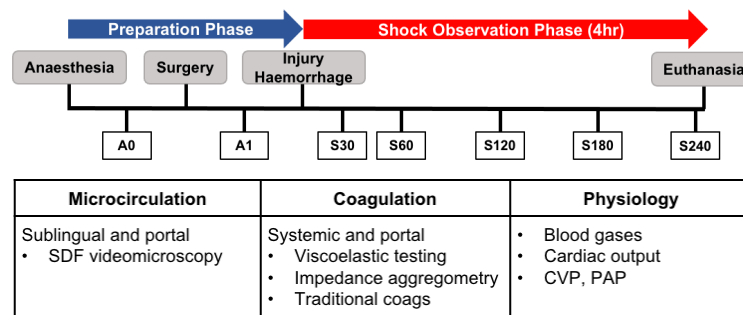
Background and Methods

Injury is a significant contributor to mortality and morbidity

- Traumatic injury complicated by haemorrhagic shock results in microcirculatory dysfunction
- Microcirculatory dysfunction causes tissue hypoperfusion and may contribute to coagulopathy induction
- Temporal profiles of microcirculatory behaviour and coagulation function (in systemic and portal circulations) during traumatic haemorrhagic shock is unclear

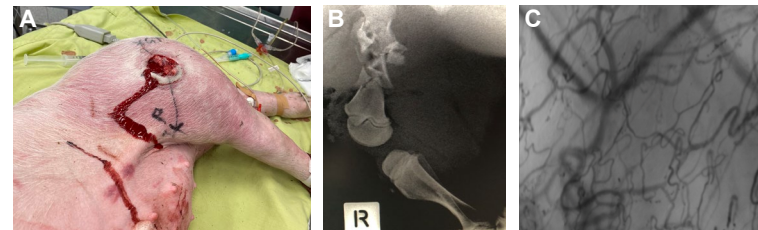
Characterise a paediatric porcine model of traumatic haemorrhagic shock to examine microcirculatory and coagulation function

- Ten female landrace pigs (20kg, 8 weeks)
- Surgery involved splenectomy, portal vein catheterisation, colon exposure (portal microcirculatory imaging)
- Femoral fracture and haemorrhage to 40 mmHg MAP
- Standardised panel of measurements during four hour observation



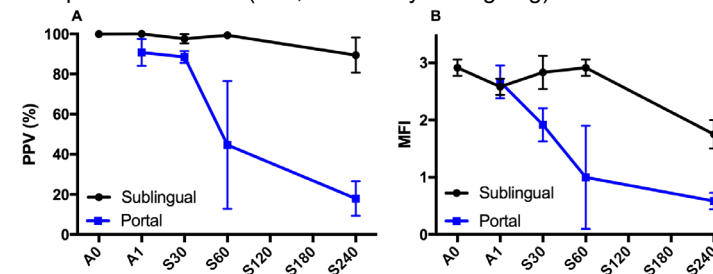
Standardised femoral fracture and videomicroscopy techniques

- Hind limb injury (A) induced using cattle stunner with radiographic fracture confirmation (B)
- Microcirculation imaged at sublingual (C) and mucosal vascular beds



Sublingual and portal microcirculation behaved differently

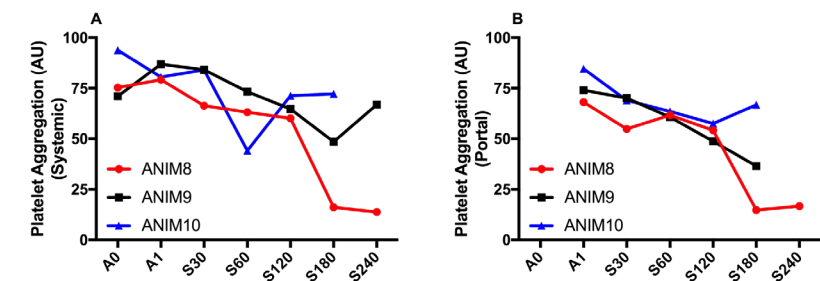
- Reductions in proportion of perfused vessels (PPV) and microvascular flow index (MFI) were greater and occurred earlier in the portal circulation (n=1, data analysis ongoing)



Results

Marked reductions in platelet function during shock

- Reduced aggregation response to ADP agonism
- Similar trend in the systemic (A) and portal (B) circulation



Conclusion

Developed a reproducible paediatric porcine model of trauma and severe haemorrhagic shock

- Facilitates examination of microcirculatory and coagulopathic responses to shock in systemic and portal circulations
- Model will be used to examine responses to variable shock time and resuscitation fluids (plasma and crystalloid)
- Wide reaching applications as a framework for future translational trauma research for mechanistic basis of coagulopathy and potential intervention strategies