



Urinary Oxygen Tension Monitoring As a Measure of Systemic Perfusion

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Introduction

- Up to 45% of cardiothoracic surgery patients develop acute kidney injury (AKI) [1].
- Current diagnostic tools do not have prognostic value. Serum creatinine does not increase until 24-72 hours after the injury [2].
- Renal tissue hypoxia plays a critical role in the initiation of AKI [3]. Urine oxygen tension (PuO_2) reflects renal oxygenation when measured at the renal pelvis [4]. Thus, PuO_2 may be a marker of AKI risk.
- Research is focused on use of PuO_2 as an early indicator of AKI. However, very little is understood about potential interventions to improve renal oxygenation.
- This preliminary analysis analyzed the temporal relationship between PuO_2 and systemic perfusion, as measured by mean arterial pressure.

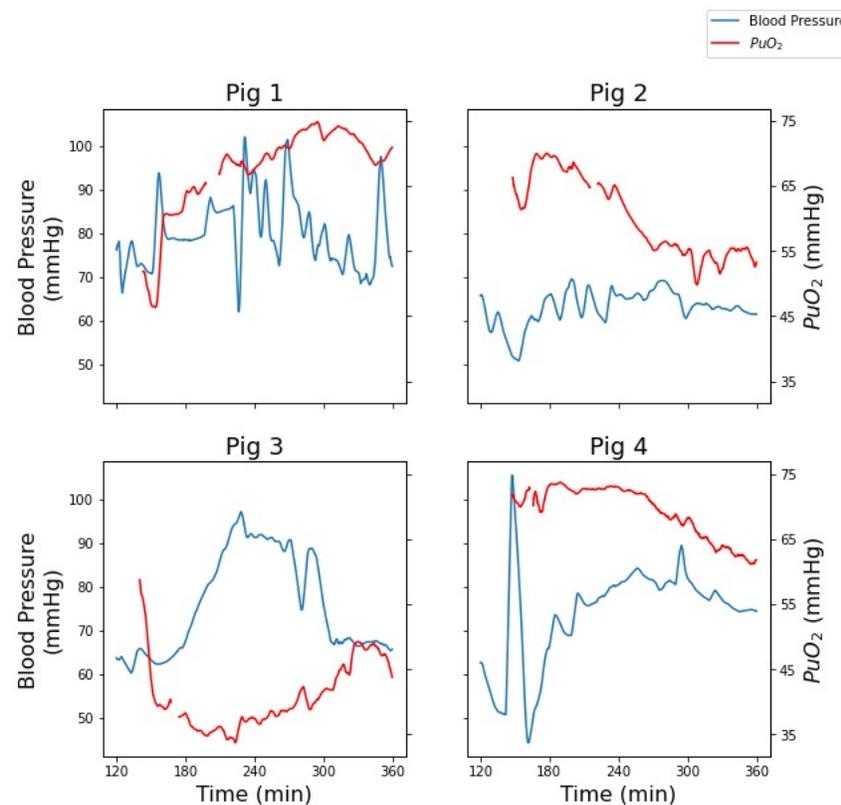
Methods

- Device which measures PuO_2 was placed between catheter and collection bag in 4 pigs.



- At Time 0 hemorrhagic shock was induced by removing an estimated 25% of blood volume over 30 min; subject remained in a hypotensive state for 30 min
- 115 min after experiment started subjects entered critical care phase; subject received combination of crystalloids and norepinephrine to target a MAP > 65 mmHg
- The temporal relationship between PuO_2 and blood pressure was qualitatively assessed

Results



- In pig 2 MAP increases at ~ 150 min and there is a distinct increase in PuO_2 shortly thereafter.
- A similar relationship is seen around ~155 min in pig 1. An increase in MAP followed by an increase in PuO_2 .
- In pig 3, the increase in PuO_2 is gradual compared to the increase in MAP.

Conclusion

- Qualitatively, changes in MAP are reflected by changes in PuO_2 with a slight delay
- Future work will focus on quantifying the relationship and time delay

References

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