Towards an AKI Monitor: Modeling Urinary Oxygen Changes Through the Urinary Tract

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Introduction: Up to 30% of cardiothoracic surgery patients develop acute kidney injury (AKI)[1]. Kidney hypoxia is recognized as an associated risk factor for AKI during surgery[2]. Currently, there is no intraoperative monitor or indicator of AKI or AKI risk. Studies suggest that urinary oxygen tension (PuO_2) may reflect renal oxygenation[3]. We have developed a prototype device which is placed between the urinary catheter and the tubing to the urine collection bag to measure PuO_2 noninvasively. However, one of the major limitations of the device is that the measurement may be affected by oxygen ingress from the tissue in the urinary tract and through the exposed section of the urinary catheter. The aim of this research is to model the change in PuO_2 as urine moves along the urinary tract based on urine flow rate, urine oxygen concentration and other parameters to better predict PuO_2 in the renal pelvis based on the measurement outside the body. Understanding how oxygen concentration changes based on parameters associated with this model will help improve the sensitivity and specificity of the prototype device.

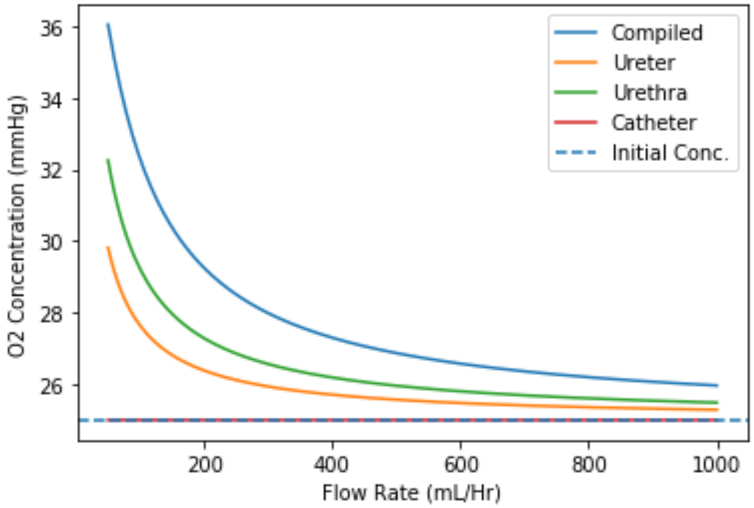
Methods: A model developed by Evans et al. was expanded to fit the prototype device scenario.[4] Urine flow was modeled as a single bolus which traverses the urinary tract. First, the urinary tract was split into three different sections (Ureter, Urethra, and Catheter) and each section was modeled independently. The models were then combined to assess the change in oxygen along the entire urinary tract. The general equation for the individual models is included below, where $C_{outside}$ is the oxygen concentration in the surrounding environment, p_{epith} is the permeability of the material of the flow path, S_{bolus} is the surface area of the bolus, V_{bolus} is the volume of the bolus and $t_{bolus}^{transit}$ is the time it takes for the bolus to traverse the flow pathway. C_{urine}^{in}, the oxygen concentration in the renal pelvis, was set to 25 mmHg to assess the usefulness of the model

$$C_{urine}^{out} = C_{outside} - (C_{outside} - C_{urine}^{in})e^{\left(-\frac{p_{epith}\, S_{bolus}}{V_{bolus}} t_{bolus}^{transit}\right)} \quad (1)$$

Underlying assumptions about model parameters were based on current literature.[5] In addition, the concentration of oxygen in the ureter and urethra tissue was considered equal to the mixed venous oxygen concentration. Also, $t_{bolus}^{transit}$ was estimated based on urinary flow rate. The flow rate was converted to average velocity based on the equation Q = Av, where Q is the volumetric flow rate, A is the cross-sectional area, and v is the average velocity. The bladder was not included in the model as it is assumed a urinary catheter is in place such that urine does not rest in the bladder.

Results: The four different models are shown in figure 1.

Discussion: These results indicate that oxygen ingress occurs primarily through the ureter and urethra. This could be because the oxygen permeability of latex is more than two times smaller than the reported oxygen permeability values of tissue. In addition, these results suggest that the prototype device is within 10 % measurement error when the urine flow rate is greater than 365 mL/hr. This is much larger than the cut off value for diagnosing AKI, which suggests the need for more research to improve and apply the model.



References: **1.** Hobson CE, et. Al, Circulation, 2009; 119; 2444-2453 **2.** Evans RG, et. Al Clin Exp Pharmacol Physiol 40; 106–122; 2013 **3.** Okusa MD, et. Al, Contrib Nephrol 2013; 182; 65-81 **4.** Sgouralis, et. Al Am. J. Physiol. Regul. Inter. Comp Physiol., 311; R532-R544; 2016 **5.** Standring, S. (2016) Elsevier Limited,

Figure 1 - Summary of the four different models that were generated.