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International Journal of Anesthesiology & Research (IJAR)
ISSN 2332-2780

Can Partial Oxygen Pressure Of Urine Be An Indicator For Tissue Perfusion? Review'

Review Article

Melis TOSUN*

Aksaray Eskil Devlet Hastanesi, Department of Anesthesiology and Reanimation, Aksaray- 68100, Turkey.

Abstract

The main purpose of advanced monitorisation is to detect hypoperfusion before any irreversible damage. However, none of the advanced monitorisation procedures focusing only on the hemodynamics and blood gas parameters are sufficient to estimate tissue perfusion adequately. As low urine partial oxygen pressure (PuO_2) is an indicator for medullary hypoperfusion, PuO_2 measurement is one of the promising markers for early detection of hypoperfusion. Based on this hypothesis, we had designed a study to evaluate the use of PuO_2 together with routine systemic tissue perfusion parameters in patients undergoing open-heart surgery with extracorporeal circulation. Fifty patients undergoing elective coronary artery bypass graft surgery had included. In addition to the routine hemodynamic monitorization; cardiac output (CO), Pv-aCO_2, blood, and urine gas analysis were performed at 180th, 360th, and 540th minutes postoperatively, and creatinine was measured repetitively postoperatively. None of the patients developed any hemodynamic event related to hypoperfusion or acute renal injury. No correlation between hemodynamic and blood gas parameters with PuO_2 could be shown in this study. In conclusion, further studies on patients with hypoperfusion and transient renal ischemia may correctly resolve the correlation.

Undetected hypoperfusion, improper delivery of oxygen, or impaired oxygen consumption may lead to irreversible organ damage or even death. The main purpose of advanced monitorisation is to detect hypoperfusion before any irreversible damage. However, none of the advanced monitorisation procedures focusing only on the hemodynamics and blood gas parameters are sufficient to estimate tissue perfusion adequately [1]. Limited information obtained by the routine measurements has led us to look for new parameters to predict hypoperfusion.

As cardiac surgery with extracorporeal circulation (ECC) is a high-risk surgery and the end-organ damage secondary to impaired microcirculation is one of the main determinants of postoperative mortality, ensuring the adequacy of the microcirculation should be the main focus. However, the impaired cardiac pressure-volume relationship of these patients makes the routine evaluation of tissue perfusion more difficult.

The renal medullary oxygenation is determined by four parameters; medullary blood flow (MBF), medullary oxygen consumption rate ($\dot{V}O_2$,M), hemoglobin (Hb) concentration, and renal perfusion pressure, and renal medulla is highly sensitive and one of the first damaged tissues by hypoperfusion [2-4]. It has shown that urine partial oxygen pressure (PuO_2) is altered by renal arterial flow, hence renal medullary oxygen pressure, and PuO_2 sampled from the collecting tubules is a marker for medullary oxygenation [5].

As low PuO_2 is an indicator for medullary hypoperfusion, PuO_2 measurement is one of the promising markers for early detection of hypoperfusion. Based on this hypothesis, we had designed a study to evaluate the use of PuO_2 together with routine systemic tissue perfusion parameters in patients undergoing open-heart surgery with ECC [6].

Fifty patients undergoing elective coronary artery bypass graft surgery had included. In addition to the routine hemodynamic monitorization; cardiac output (CO), Pv-aCO_2, blood, and urine gas analysis were performed at 180th (T0), 360th (T1), and 540th (T_2) minutes postoperatively. Urine was collected through a silicone urine catheter inside the bladder. CO measurement was performed by using a finger cuff method. Through the ECC, hematocrit was kept at 23%-30%. The pump flow rate was maintained >2 L m^{-1} during ECC, mean arterial blood pressure (ABP) was kept between 50-80 mmHg. Moderate hypothermia (32°C) was applied to all patients. To the evaluation of renal functions, serum

***Corresponding Author**:
Melis TOSUN,
Aksaray Eskil Devlet Hastanesi, Department of Anesthesiology and Reanimation, Aksaray- 68100, Turkey.
Tel: +90-536-6694002
E-mail: melistosun@gmail.com

Received: November 20, 2021
Accepted: January 22, 2022
Published: February 07, 2022

Citation: Melis TOSUN. Can Partial Oxygen Pressure Of Urine Be An Indicator For Tissue Perfusion? Review'. *Int J Anesth Res.* 2022;10(1):673-675.

Figure 1: Urine pO_2 and PaO_2 levels through the periods.

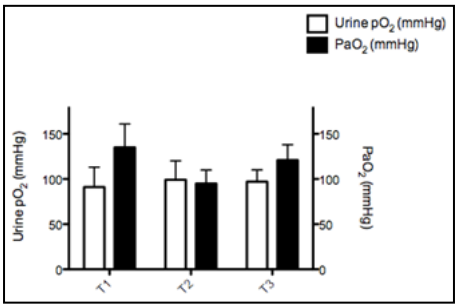


Figure 2. Urine pO_2 and lactate concentration through the periods.

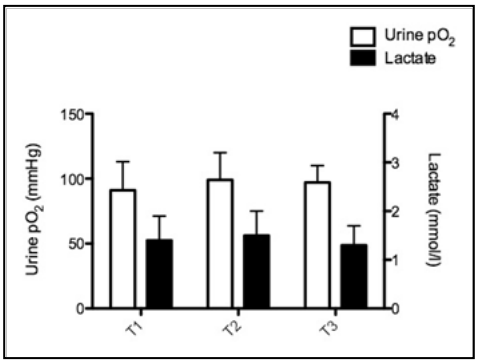
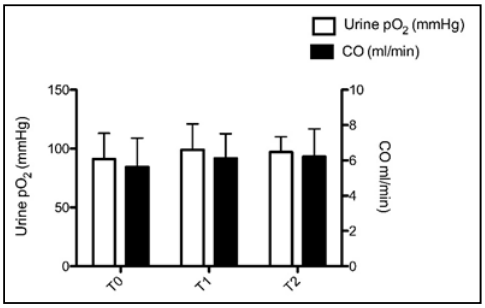


Figure 3. Urine pO_2 and cardiac output concentration through the periods.



creatinine was measured perioperatively.

The patients were hemodynamically stable throughout the surgery and post-extubation period in terms of heart rate, mean arterial pressure, and CO ($p>0.05$). Additionally, blood gas analyses and electrolyte concentrations were also found within the normal limits ($p>0.05$).

Postoperative PuO2 was measured as 91±22, 99±22, and 97±13 mm Hg, respectively, and was no significant difference at any time point. Moreover, there were no significant correlations between PuO_2 and other tissue oxygenation parameters ($p>0.05$) (Figures 1-3).

Serum creatinine was measured preoperatively and postoperative first and 5th days as 0.92±0.5, 0.93±0.5, and 0.85±0.5 mg dL−1, respectively, and none of the patients developed acute renal injury (ARI).

The limited information obtained from routine monitoring methods has encouraged us to look for new monitoring methods. The new technique should be non-invasive, easy to perform, safe, low in price, and capable of detecting the early stages of hypoperfusion. In this concerns, we investigated the correlation of PuO_2 with advanced monitoring parameters in a patient group at risk for hypoperfusion.

Although there are animal studies in the literature showing a correlation between cardiac output and mean arterial pressure with PuO_2[7], no correlation between hemodynamic and blood gas parameters with PuO_2 could be shown in this study.

As we mentioned in the original article, our study had some limitations. First, we compared PuO_2 of bladder urine samples instead of renal medullary or pelvic urine. Although bladder urine has been shown to reflect renal medullary oxygenation [8], the PuO_2 that might have been pooled in the bladder may vary.

Although Xing et al.[9] studied PuO_2 measurements for early diagnosis of AKI in septic patients and found a cut-off value for renal injury to find the normal ranges of renal medullary, renal pelvic, and bladder PuO_2 need more studies in larger patient groups. Also, animal studies may help us to find the factors affecting PuO_2 while urine passing through the urinary tract.

In our opinion, since the COs of the patients were within the normal limits, and none of the patients developed an ischemic condition or AKI, the present study might have been unable to determine any correlation. Further studies on patients with transient renal ischemia may correctly resolve the correlation. Additionally, up-to-date AKI markers should be measured in the upcoming studies to detect renal damage that cannot be detected by

creatinine monitoring.

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