Proposition pertaining to the PhD thesis

THE RENAL MICROCIRCULATION
AS A TARGET FOR THE TREATMENT OF ACUTE KIDNEY INJURY
IN MODELS OF CRITICAL ILLNESS

1. Renal oxygenation is mainly regulated by systemic and renal autoregulatory mechanisms for water reabsorption, electrolytes balance, macromolecules transport and urine production (this thesis).

2. Ischemia, hyperoxia, local and systemic inflammation, and oxygen radicals which mediates renal injury, these appears to be the main reasons for acute kidney injury (this thesis).

3. Acute kidney injury may be solved by microcirculatory-targeted therapies including immune modulation, antioxidants, adequate intravascular volume expansion with proper fluids, peripheral vasodilators and oxygen carriers (this thesis).

4. The resuscitation fluids regardless of types or composition, as well as blood may have some detrimental effect on the microcirculation and ultimately organ oxygenation (this thesis).

5. Acetate-buffered balanced fluids show superior buffering effects when compared with Ringer's lactate or saline and it may be the most efficient bicarbonate precursor regardless of liver function (this thesis).

6. In addition to systemic variables, convection and diffusion distance is preeminently effective in sufficient microcirculatory function and oxygenation (Ince C; Curr. Opin. Crit. Care, 2014).

7. Effective therapies that expected to resolve AKI will have to control inflammation and restore homeostasis between oxygen, nitric oxide, and reactive oxygen species (Ince C; Nephron. Clin. Pract., 2014).

8. The ultimate culprit of AKI leading to renal failure is the dysfunction of its microcirculation (Guerci P; Best Pract. Res. Clin. Anaesthesiol., 2017).


10. Therapeutic resolution of persistent heterogeneous microcirculatory alterations is expected to improve outcomes in critically ill patients (Ince C; J. Appl. Physiol., 2016).

11. Science is the only true guide in life (Mustafa Kemal Ataturk).

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