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Fifty shades of central venous pressure in the cardiorenal syndrome

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Cardio renal syndrome is the result of many hemodynamic, physiological, hormonal, biochemical or structural interactions. The interactions are bidirectional: acute or chronic cardiac failure may induce acute or chronic renal failure.[1] The renal blood flow is kept constant for mean arterial pressure (MAP) between 70 and 130 mmHg.[2] This self-regulation is made possible by two mechanisms. The first is myogenic by the contraction/relaxation of the afferent vessels in reaction to pressure, and the second is the tubule-glomerular feedback, which also regulates the diameter of the afferent arteriole as a function of NaCl concentration in the filtration liquid arriving at the macula densa.[3,4] The sodium concentration is a function of the quantity of blood, which arrives in the afferent arteriole and the glomerulus.[3,4] In pathological situations such as septic shock, the MAP is reduced below 65 mmHg. The collapse of MAP spectacularly reduces the afterload with a cardiac output capable of increasing due to sepsis to values ranging from 10 to 15 L/min.[5] At the same time, fall in MAP decreases renal blood flow following the loss of self-regulation leading to renal failure and so-called "kidney shock".

Previous animal studies have shown that an isolated elevation in central venous pressure (CVP) can impair renal function.[6,7] Mullens et al. studied the impact of CVP measured by a Swan-Ganz catheter on the worsening of renal function (WRF) in patients with advanced decompensated heart failure. Patients who developed WRF had a higher central venous pressure on admission (CVP, 18 ± 7 *vs.* 12 ± 6 mmHg, $P < 0.001$) and after intensive medical therapy (11 ± 8 *vs.* 8 ± 5 mmHg, $P = 0.04$). The development of WRF occurred less frequently in patients who achieved a CVP < 8 mmHg ($P = 0.01$).[8]

In the context of septic shock, Legrand et al. studied 137 cases of septic shock and distinguished two populations: patients developing acute kidney injury (AKI) and those without kidney injury or improving their renal function (no-AKI). In this series, there was no significant difference in MAP pressure, cardiac output and central venous oxygen saturation ($ScVO_2$) between AKI and no-AKI. In contrast, the CVP was higher in the AKI group (11 [8.5–13]) than in the no-AKI group (8.5 [7–11.1], $P = 0.0032$). The CVP value was associated with a risk of developing new or persistent AKI even after adjustment for fluid balance (OR = 1.22 (1.08–1.39), for an increase of 1 mmHg; $P = 0.002$). A linear relationship between CVP and the risk of new or persistent AKI was observed. This article suggests a role for venous congestion in the onset of AKI and challenges the paradigm that high CVP reduces the onset of AKI.[9]

Venous return to the heart and disturb microcirculatory blood flow might be reduced by a high CVP causing tissue congestion and organ failure.[10] CVP is a bedside measure and has long been used to assess preload and response to fluid loading. However, measurement of CVP is not reliable to assess patient's hemodynamic status.[11] An excessive fluid administration may increase CVP and end-diastolic pressure without increasing end-diastolic or stroke volume.[10] But in a cohort of 4,761 critically ill patients with admission CVP measurements, each increase of 1 cm

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H_2O CVP was associated with a 2% increase in the adjusted risk of AKI (95% CI, 1.00–1.03; $P = 0.02$). In this same study, pulmonary edema was not associated with a risk of developing AKI.[12]

In conclusion, the main aim of CVP monitoring should be to ensure a CVP below renal venous pressure (RVP). An increase in CVP induces an increase in RVP that reduces glomerular filtration inducing a feedback in the macula densa with vasodilatation of the afferent arteriole and renin secretion.[4] This increase in "renal afterload" will ultimately lead to a decrease in glomerular filtration and an increase in cardiac afterload via renin and will worsen the cardiorenal syndrome.

Conflict of Interests

The authors declare to have no competing interests.

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