CRITICAL CARE

COMMENTARY

Please don't call me RI anymore; I may not be the one you think I am!

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See related research by Dewitte *et al.*, http://ccforum.com/content/16/5/R165

Abstract

The previous issue of *Critical Care* reports new data on renal resistive index in critically ill patients. Although high renal resistive index may indeed be associated with acute kidney injury, the existence of several determinants of this index, of which renal resistance is only one among many, obscures the usefulness of this index in clinical practice.

Another brick in the wall of knowledge on renal resistive index (RI) was provided by Dewitte and colleagues [1] in the previous issue of *Critical Care*. It is additional evidence that much remains to be done to understand what describes this parameter, as rightly concluded by the authors. Several studies have now accumulated knowledge on RI in the intensive care setting. The overall messages repeatedly emerging from these studies are that renal injury is associated with RI elevation but that considerable overlap between patients with and without acute kidney injury (AKI) precludes the use of this index in daily practice [1-4]. Additional observations have been made with less reproducibility from one study to another as the number of studies increases: elevated RI may differentiate persistent versus transient AKI [1,3], RI increases with lower arterial partial pressure of oxygen (PaO_2) [5], and RI increases with lower mean arterial pressure (MAP) [1,2]. This last point is challenged by the study by Dewitte and colleagues, in which such a relationship was observed only in patients without AKI.

RI data in the critical care setting are reminiscent of the story in the field of nephrology, in which RI is very easy to measure but of little help in practice. Before RI's poor utility in clinical practice and progressive abandonment, initial enthusiastic reports claimed that RI could help to assess the impact of renal urinary tract obstruction, diagnose acute rejection and chronic dysfunction of renal transplant, and predict renal function recovery after renal artery stenosis dilatation [6-8].

We know several reasons why RI failed to hold its promise to predict or accurately diagnose AKI, but we do not know which of the several determinants that impact RI – systemic pulse pressure, MAP, PaO_2, renal vascular resistance, renal vascular compliance, and renal parenchymatous pressure – relate to the pathophysiology of AKI [2,5,6,9,10]. The term 'renal resistive index' is undoubtedly misleading, and the less definite term 'renal vascular index' (RVI) may be better chosen to preclude erroneous interpretation.

Indeed, our understanding of the relationship between RVI and kidney injury is elusive. Several hypotheses may be put forward but all of them can be challenged easily. First, elevated RVI may directly reflect renal damage, especially vascular damage. Indeed, persistent circulatory abnormalities (low renal blood flow) have been observed at the established phase of acute tubular necrosis, and renal vascular damage now emerges as a prominent feature of AKI [11]. However, animal data showed that, at the initiation of septic shock, renal blood flow is maintained or even increased provided that cardiac output is maintained [12]. Renal hemodynamics probably evolves along the different phases of AKI, obscuring what can be inferred by RVI. Second, it has been hypothesized that increased RVI may reveal renal vasoconstriction secondary to inadequate renal perfusion eventually leading to renal ischemia. This led some authors to suppose that RVI may guide hemodynamic therapeutic interventions [13] and notably that lowering RVI may be a resuscitation endpoint. However, the observation by Dewitte and colleagues that the relationship between a parameter of systemic hemodynamics (MAP) and RVI is present only in patients who do not develop AKI may indicate that the use of renal Doppler to optimize renal hemodynamics may be possible only in patients who do not need it. Furthermore, if we make the assumption that RVI actually permits us to evaluate renal vascular resistance, the relationship between RVI and MAP (with lower MAP

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associated with higher RVI) may be difficult to reconcile with the usual concept of physiological regulation of renal circulation. Indeed, although this may be debated, it has been demonstrated that, along a certain range of MAP, renal blood flow remains constant ('autoregulation' phenomenon) [14]; that is, in this range, lowering MAP results in renal vasodilation (unlike other vascular beds); thus, RVI diminution, not augmentation, should be observed. Therefore, RVI does not represent renal vascular resistance, or renal autoregulation is abolished in some or all critically ill patients [14], or patients have been evaluated below the autoregulation MAP range. Nevertheless, the autoregulation phenomenon may participate in the complexity of RVI interpretation.

There is an urgent need to establish pathophysiological models allowing an understanding of what exactly RVI describes. Routine invasive measurements of several hemodynamic parameters may give the critical care field, unlike the nephrology field, the opportunity to refine our understanding of RVI.

Abbreviations

AKI, acute kidney injury; MAP, mean arterial pressure; PaO_2, arterial partial pressure of oxygen; RI, renal resistive index; RVI, renal vascular index.

Competing interests

The author declares that he has no competing interests.

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