Commentary

Searching for an ideal hemodynamic marker to predict short-term outcome in cardiogenic shock

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See related research by Torgersen *et al*., http://ccforum.com/content/13/5/R157

Abstract

Cardiogenic shock is a lethal condition. Physicians are searching for hemodynamic markers which could help risk-stratification of patients in this picture. Torgersen and coworkers present an hourly time integral of the cardiac power index and cardiac index drops to predict outcomes in the setting of cardiogenic shock. Continuous monitoring of hemodynamic markers may have a role in prediction of outcomes.

In the previous issue of *Critical Care*, Torgersen and coworkers present data about careful evaluation of hemodynamic monitoring of patients with cardiogenic shock (CS) in the intensive care unit as continuous variables during the initial 24-hour period [1]. Hemodynamic parameters influencing short-term mortality were identified. The authors stated that hourly time integrals of the cardiac index (CI) (cardiac output per body surface area) and the cardiac power index (CPI) (cardiac power output per body surface area) were the most important hemodynamic variables designating mortality as continuous parameters. In addition, instead of intermittent measures of the CI and the CPI, the authors analyzed the area under a given level divided into hourly intervals, named hourly time integrals. These results have the potential to provide a paradigm shift in the setting of CS.

Two take-home messages can be extracted from the study of Torgersen and coworkers [1]. First is the emphasis on the importance of continuous monitorization in patients with CS, and the second message is providing data for the importance of the interpretation of the CPI as a continuous variable in an acute setting.

CS is traditionally defined as a state of severe tissue hypoperfusion secondary to cardiac dysfunction. It carries high mortality risk, around 50% in the acute term [2]. Therefore, it is vital to identify signals driving the CS prognosis. For decades, hemodynamic monitoring has been desired for patients with acute pathologies with the aim of guiding therapy and risk-stratification [3]. Many parameters derived from hemodynamics – such as the cardiac output, systemic blood pressure, systemic vascular resistance, stroke volume, and pulmonary capillary wedge pressure – have so far been investigated to designate prognosis with unequivocal results [4,5]. Among these hemodynamic parameters, the cardiac power output and the CPI serve as interesting markers showing cardiovascular coupling at one glance, in contrast to other parameters that provide information about either the cardiac system or the vascular system [5].

Drops in the CPI were described by Torgersen and colleagues to predict outcome [1]. A critically low CPI might be a result of unresponsiveness to therapeutic interventions rather than a causative factor for death. One should mention that although the hourly time integrals of drops in the CI and CPI predicted short-term mortality, this might not mean that the CI and the CPI are targets for treatment. Indeed, in many cases, patients die despite high CI levels. Intra-aortic balloon pump (IABP) use is known to increase cardiac power output, and hence the CPI [6]. Adjustment should have been performed for the use of an IABP (37.8% of patients). Adjustment for gender was also needed, as the cardiac power output is also lower in females [7]. In a very recent meta-analysis, it was shown that a percutaneous left ventricular assist device yielded higher CI and higher mean arterial pressure compared with the IABP. This could be translated as a higher CPI with the left ventricular assist device compared with that under IABP use. The 30-day mortality was similar in both interventions, however, despite

CI = cardiac index; CPI = cardiac power index; CS = cardiogenic shock; IABP = intra-aortic balloon pump.

better early hemodynamic status with the left ventricular assist device [8]. A better CI or CPI may therefore not always translate into better outcomes. Torgersen and coworkers brought us some novel thresholds, however, which should be tested in multicenter trials. Eventually, these thresholds can serve as targets.

The present paper emphasizes the continuous effort that should be made to risk-stratify patients with CS. Hemodynamic variables are important to consider in the setting of CS, particularly those parameters integrating generation of cardiac energy with spreading of blood flow, such as the CPI. Since it was shown that almost one-half of nonsurvivors of CS die with a normal CI, this finding changed the paradigm of CS recently from being only a cardiac problem into a disease of the entire circulatory system [9,10]. Data tell us the importance of systemic inflammatory response upon release of inflammatory mediators and neurohormones yielding alterations in tissue microvasculature, which may result in multiorgan dysfunction syndrome in CS [11]. We therefore agree that hemodynamic signals combining cardiac function with tissue perfusion such as the CPI may be optimal markers of outcome. On the contrary, it might be as important to consider any invasive hemodynamic parameter as a continuous (and not intermittent) marker, as presented by Torgersen and coworkers, instead of considering them per piece.

Competing interests

The authors declare that they have no competing interests.

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