Comparison of continuous-wave Doppler ultrasound monitor and echocardiography to assess cardiac output in intensive care patients

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TO THE EDITOR: In their article, Elgendy and colleagues concluded that the Smith–Madigan inotropy index (SMII) was not useful in patients admitted to the intensive care unit, as SMII < 1.1 watts/m² showed only fair correlation and limited sensitivity with low cardiac output. The authors’ definition of low cardiac output specified no threshold value, yet, oddly, a left ventricular ejection fraction (LVEF) < 35% was included.

Low LVEF per se does not indicate low cardiac output; for example, in dilated cardiomyopathy, stroke volume may be normal despite low LVEF. Cardiac output is the product of stroke volume and heart rate, but heart rate data were not presented. High cardiac output with low inotropy is common, as occurs in sepsis, for example, where low afterload results in high cardiac output despite severe myocardial depression. When afterload is low, any old heart can eject a good stroke volume. Likewise, even if inotropy is good, a normal stroke volume cannot be ejected from a poorly-filled ventricle or against a very high afterload. Therefore, preload and afterload are crucial when interpreting LVEF or low cardiac output.

The most common diagnoses in the article were septic and cardiogenic shock, but no details regarding the use of vasoactive therapies were given. Were patients receiving vasopressors, inotropes, nitrates or diuretics?

Inotropy is only one factor in patients with low cardiac output. Stroke volume depends on preload, inotropy and afterload, while cardiac output depends on stroke volume and heart rate; hence, quoting SMII without the other three parameters is meaningless in relation to cardiac output.

SMII measures instantaneous external cardiac power output, which is the best predictor of outcome in left ventricular failure. It is not a measure of current cardiac output, but an indicator of what the heart could produce. If SMII is adequate, then low cardiac output must result from suboptimal heart rate, preload or afterload. Table 1 showed that in patients with low cardiac output, over 70% had low SMII, as opposed to less than 30% of patients with SMII > 1.1 watts/m². When cardiac output was normal or high, 84% of patients had SMII > 1.1 watts/m², while only 16% had low SMII. How many of the 16% of patients had low afterload? The between-group difference for low cardiac output and threshold value of SMII of 1.1 watts/m² was highly significant ($\chi^2 = 33.8; P < 0.000001$).

SMII does not predict a low cardiac output; it explains why stroke volume is low and guides treatment in terms of optimising preload, afterload and inotropes. However, SMII was not compared with the variable on which it exerts its influence: stroke volume. Finally, SMII is not derived from an algorithm, as the authors suggest, but from the standard mathematical equations used in classical hydrodynamics.

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Competing interests
None declared.

References