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LETTER

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Letter to the Editor: Stroke volume is the key measure of fluid responsiveness

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To the Editor

The authorities in the domain of shock physiology, Vincent and colleagues, have recently offered a concise perspective on intravenous fluid administration [1]. They discuss the concept of the fluid challenge and offer a practical clinical algorithm, but miss an opportunity to underscore that cardiac output (CO) is a complex measure when interpreting the full effect of fluid provision.

Historically, fluid responsiveness (FR) has been defined by change in cardiac output (CO = heart rate x stroke volume) as CO is a key determinant of oxygen delivery [1, 2]. Ignoring issues around measurement precision and defining a true gold standard, we note that relying solely on CO belies the fundamental physiology that *stroke volume* is the closest clinically-available approximation of the cardiac length-tension relationship. This point is highlighted by a hypothetical example.

A patient in early septic shock has a heart rate (HR) of 120 beats per minute (BPM), stroke volume (SV) of 51 mL and, therefore, a CO of 6.1 L per minute (L/min). The patient is given 30 mL/kg of balanced crystalloid; subsequently HR falls to 90 BPM and SV rises to 65 mL resulting in a CO of 5.9 L/min. Was this patient harmed by intravenous fluids? Is this patient 'fluid unresponsive' despite SV rising by over 25%?

An implicit, mathematical assumption of CO is that its augmentation is beneficial independent of the relationship between SV and HR; this may not be universally true. For example, the coronary circulation is perfused preferentially during diastole. Therefore, diminishing HR augments diastolic time and subendocardial perfusion [3]. Accordingly, despite a calculated *fall* in cardiac output, the patient above may have significantly *increased* ventricular oxygen delivery with intravenous fluids, especially if there is co-morbid coronary artery disease and/or ventricular hypertrophy. These principles could partly explain recent data associating diminished diastolic shock index (i.e., HR divided by diastolic blood pressure) with improved outcome [4]. In other words, *low* HR and high diastolic pressure is a good prognosticator in septic shock.

Thus, because CO comprises two variables that may diverge as a normal, adaptive response or following beneficial therapy, relying on it as a lumped index of 'fluid responsiveness' may mislead. Accordingly, clinicians and researchers should rely not merely on CO as it may conceal the effect of fluids on the cardiac length-tension, i.e., Frank-Starling, relationship.

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Authors' response

Stroke volume versus cardiac output during a fluid challenge

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We would like to thank Drs Kenny and Barjaktarevic for their comment about our paper on fluid challenge [1]. The authors are correct to indicate that stroke volume (SV) should be the preferred index for evaluating fluid responsiveness, rather than cardiac output (CO). As indicted in the example provided by Kenny and Barjaktarevic, a patient can respond to fluids by increasing SV and decreasing heart rate, so that CO may not increase. However, if the goal of fluid administration is to increase oxygen delivery (DO_2), this will not be achieved by increasing SV without an increase in CO. A decrease in hemoglobin concentration due to a dilutional effect of a large bolus of fluid may also limit the increase in DO_2.

Measurements of CO rather than SV also have some practical advantages. First, the decrease in heart rate can vary during a fluid challenge. In a study evaluating fluid responsiveness in 491 critically ill patients [5], heart rate decreased minimally in fluid responders and non-responders, and the area under the receiver-operating characteristic curve for changes in heart rate to detect fluid response was only 0.53, i.e., hardly better than flipping a coin. Second, measuring SV is challenging in fluid responders who may have respiratory variations in SV that decrease as fluid is administered [6]. As suggested by Kenny and Barjaktarevic, in some cases, a reduction in heart rate may protect coronary perfusion by prolonging diastole, but prolonging diastole can also decrease diastolic pressure. Increasing vasopressor support may be a better option to increase coronary perfusion [7].

Perhaps, the most important message is that the management of critically ill patients is complex and cannot be based on one variable only. Using SV to assess fluid response is not wrong if one looks at the Frank–Starling relationship, but ultimately the DO_2 is what matters for the tissues.

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