

## Commentary

# Why measure cardiac output?

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### Abstract

Although cardiac output is a primary determinant of global O<sub>2</sub> transport there are no absolute values that reflect circulatory adequacy, though very low values are of negative prognostic use. There is no agreement as to what constitutes a clinically relevant change in cardiac output. A recent clinical trial suggests that early goal-directed therapy aimed at increasing cardiac output improves survival. Thus, in some patients, measurement of cardiac output is indicated as an aid to prognosis, diagnosis and to monitor the adequacy of therapy. Gonzalez *et al.* compared PAC thermodilution cardiac output with indirect Fick measures of cardiac output. They found that at lower cardiac outputs (<5 l/min) the agreement between the two techniques is good, whereas at higher flows increased differences exist between the two measures. As discussed in this commentary, this study did not address the three potential questions related to PAC monitoring of cardiac output. These questions are: can the PAC cardiac output data be used to monitor cardiac output? Do technical and physiological constraints limit the accuracy of PAC cardiac output? And; are PAC cardiac output measurement errors due to respiratory variation in pulmonary blood flow? Ways of answering each question are given.

**Keywords** clinical trial, circulatory shock, hemodynamic monitoring

Cardiac output is a primary determinant of global oxygen transport from the heart to the body. Also, because the major function of the cardiovascular system is to supply sufficient amounts of oxygen to meet the metabolic demands of the tissues, it appears reasonable to measure cardiac output in the assessment of cardiovascular insufficiency. Regrettably, numerous studies have shown that neither absolute values for cardiac output nor its change in response to therapy reflect the adequacy of local blood flow or outcome from critical illness [1,2]. Clearly, one may have a cardiac output within the normal range (e.g. 2.5 l/min per m<sup>2</sup>) and still be in circulatory shock if metabolic demand is increased or blood flow distribution is deranged. Treating septic shock patients with the goal of augmenting cardiac output to high levels (i.e. >3.5 l/min per m<sup>2</sup>) does not improve survival rates [3–5] and may actually increase mortality [6]. Thus, why measure cardiac output?

Clearly, a very low cardiac output is detrimental. Critically ill patients who are unable to sustain a cardiac index in excess of 2 l/min per m<sup>2</sup>, despite aggressive therapy, have a very

high mortality rate [4]. In many of these patients the cause of the low cardiac output is inadequate cardiac filling, which is responsive to fluid resuscitation. However, in patients with combined cardiac and respiratory disease, it is often difficult to assess the adequacy of resuscitation without measurement of cardiac output. Furthermore, a recent single center study of septic patients treated in an emergency department [7] documented that rapid early resuscitation with a goal of re-establishing adequate oxygen delivery resulted in a markedly reduced mortality and duration of hospital stay. Thus, at least in some patients, measurement of cardiac output is indicated as an aid to prognosis and diagnosis, and to monitor the adequacy of therapy.

If it is useful to measure cardiac output, then it is also important that its measurement be accurate enough to identify clinically relevant changes. There is no agreement as to what constitutes a clinically relevant change in cardiac output. Operationally, changes in cardiac output of 15% or more are usually taken to reflect a relevant change, because

such changes are at the limit of accuracy of present day measuring techniques and are above the degree of normal variance seen in an otherwise stable person. Furthermore, measurements of cardiac output need to be taken repetitively if they are to be useful in the management of the hemodynamically unstable patient. At the present time the clinician has available an increasing number of potential measuring devices that all purport to measure cardiac output. These include indicator dilution techniques, with or without the use of a pulmonary artery catheter (PAC); arterial pulse contour techniques; aortic pulsed Doppler, both of the ascending and descending aortas; and classical indirect measures of cardiac output using arteriovenous gas content differences and expired gas measures via the Fick equation. Each technique has its own strengths and limitations. Realistically, bolus thermodilution measures of pulmonary blood flow using a balloon floatation PAC is the most common technique available and is the method most often used to measure cardiac output in clinical reports.

Numerous studies over the years have compared PAC thermodilution cardiac output with indirect Fick and aortic flow probe derived measures of cardiac output. In general, they all arrive at the same conclusions. First, because the PAC thermodilution technique measures pulmonary blood flow over a very short time window of 1.5–3 s, the measured cardiac output values are influenced by the phase of the respiratory cycle during which the injection occurs [8]. Second, the manual cold bolus injection technique is subject to systematic bias and intraoperator variability because of errors in ejection technique, and these are independent of the actual accuracy of individual measures of blood flow. Thus, numerous mechanical injection techniques and timed manual injection techniques have been proposed to minimize this problem [9]. Given the above physiologic and technical limitations, it is usually assumed that measures of cardiac output within 15–20% of previous measures reflect no real change in measured flow, but is this assumption correct?

In this issue of *Critical Care*, Gonzalez and coworkers [10] readdress this question. That prospective observational study, conducted in 18 mechanically ventilated critically ill patients, compared agreement in 49 paired measures of cardiac output using the modified Stewart–Hamilton equation and bolus thermodilution from a PAC with the indirect Fick technique using oxygen uptake at the mouth and arteriovenous oxygen content differences. Those investigators reported that at lower cardiac outputs (<5 l/min per m<sup>2</sup>) the agreement between the two techniques is good, whereas at higher flows increased differences exist between the two measures. They concluded that the thermodilution technique is still clinically valuable in assessing cardiac output. Interestingly, their finding of increased differences in paired values at higher cardiac outputs illustrates a systematic bias between the two techniques, rather than a cutoff value below which the two techniques agree.

Regrettably, the study design does not allow sufficient data to make their stated conclusion in a clinically relevant manner. Protocol design becomes a central feature of this type of experiment and can be used to illustrate how such paired cardiac output data can be collected and used to make valuable inferences.

If the question were ‘Can the cardiac output data from the PAC be used to monitor cardiac output?’, then one would need to know not only whether the two measures agree at one point in time but also whether they also track each other as cardiac output varies over time. There are two qualities of a hemodynamic measure that are clinically relevant: first, if no change in cardiac output measure occurs, then the absolute value for cardiac output is accurate and has not changed between the measuring intervals; and second, if a change in cardiac output occurs, then the direction and magnitude are accurately reflected in the measured cardiac output change. Thus, a minimum of two sets of paired observations separated by an intervention presumed to alter cardiac output would need to be performed to address this issue.

If the question were ‘Do technical and physiologic constraints limit the accuracy of PAC thermodilution measures of cardiac output?’, then different injection techniques, injectate temperatures, and respiratory rates would need to be studied, and inclusion of patients with varying degrees of hemodynamic stability would be required. Because any inaccuracy present in the measure of cardiac output by thermodilution reflects not only technical aspects of consistency of injection, catheter temperature, and sensor responsiveness, but also physiologic variables such as ventilation-induced variations in both pulmonary arterial blood flow and tricuspid regurgitation.

If the question were ‘Are measurement errors due to respiratory variation in pulmonary blood flow not seen when measures are made over many respiratory cycles?’, then differences between thermodilution and metabolic estimates of cardiac output could be compared, with great attention paid to partitioning the injections throughout the cardiac cycle rather than timing all injections with a specific point in the ventilatory cycle. Picking a specific point in the respiratory cycle does not allow for a more consistent determination of cardiac output but decreases the accuracy of the absolute value; this is because true blood flow varies over the ventilatory cycle. The advantage of comparing the thermodilution technique with an indirect Fick technique is that the indirect Fick technique measures the average cardiac output over several minutes. Thus, differences between measures will better reflect either direct measurement error or measurement of different actual variations. The indirect Fick method also carries significant bias if lung oxygen consumption is large because the arteriovenous oxygen difference calculation assumes negligible lung oxygen consumption. Thus, if lung oxygen

consumption were increased, as is the case with acute lung injury and acute pneumonitis, then the Fick technique would overestimate true cardiac output.

In the final analysis PAC bolus thermodilution measures of cardiac output, if performed correctly and with inspection of the thermal decay profile to identify poor injection runs, give cardiac output values that may vary by up to 25% at low cardiac outputs (<2 l/min per m<sup>2</sup>) and 20% at higher cardiac outputs, without reliably reflecting actual changes in cardiac output. Although newer and more precise thermodilution techniques exist, they are not readily available. Thus, the use of cardiac output measures as single variables in defining disease and response to therapy require very large differences to reflect actual changes and, outside of simultaneous oxygen consumption measures, often give little insight into the mechanisms responsible for hemodynamic instability and circulatory shock or their response to therapy.

### Competing interests

None declared.

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