In recent years, there has been a trend toward the use, in intensive care units (ICUs) and in operating theatres, of ‘minimally invasive’ haemodynamic monitoring systems for the continuous measurement of cardiac output (CO). In this context, ‘minimally invasive’ has come to mean ‘less invasive than a pulmonary artery catheter’ and is arguably an unhelpful term. Nevertheless, among the available devices, the FloTrac-Vigileo system (FTV) (Edwards Lifesciences LLC, Irvine, CA, USA) does perhaps deserve this epithet as it is designed to run from any arterial line (frequently present in patients in the ICU or undergoing major surgery, at least in Europe) and requires no calibration. This latter capability is a consequence of a sophisticated algorithm that the device employs to analyse the arterial pressure waveform (APW), whether obtained from the radial or the femoral artery, to determine the presumed non-linear proportionality between arterial blood pressure (ABP) and stroke volume (SV) and hence give an estimate of CO. However, despite its simplicity of use, the reliability of this system is uncertain during conditions of haemodynamic instability, when the dose of vasopressors changes rapidly but having an accurate CO is essential to guide appropriate management.

The FloTrac algorithm analyses the statistical distribution of data points of the ABP sampled at 100 Hz and is based on the principle that aortic pulse pressure is proportional to SV, measured as the standard deviation of the arterial pressure (σAP) around the mean arterial pressure (MAP). σAP is then multiplied by a scaling parameter derived by a multivariate polynomial equation that includes the patient’s demographic data, arterial compliance, skewness (symmetry of the waveform) to adjust for vascular tone, and kurtosis (measure of how peaked the APW is) to compensate for the differences in APW due to arterial site.

The fundamental problem with this approach is to be sure that it can identify and accurately represent those situations in which a change in blood pressure (systolic, diastolic, mean and pulse pressures) is associated with a change in SV that is directionally inverse as opposed to directionally similar. In other words, the system should be able to distinguish blood pressure changes due to volume loading manoeuvres, in which the primary intervention is aimed at increasing CO, and so blood pressure will usually change only if this occurs, and in the same direction, although the relative sensitivity of the manner in which the two variables respond can of course be quite different. When the primary change is in arterial resistance, as when a vasopressor is deployed, the situation is
more challenging since the intervention is aimed at generating a blood pressure increase, but the effect upon SV may be in either direction. This is the situation that is most testing for arterial pressure-derived CO algorithms, especially if uncalibrated.

In a previous issue of Critical Care, Eleftheriadis and colleagues [1], who had observed implausible changes in CO when vasopressors were employed in their clinical practice, reported a simple but elegant experiment that shows that, in patients undergoing coronary artery bypass grafting, variations in ABP in response to a stepwise change in noradrenaline lead to parallel changes in CO measured by the second-generation FTV (software version 1.14), which were not present when CO was measured conventionally using a thermodilution pulmonary artery catheter. During these conditions of pharmacologically driven changes in vascular tone, the bias and the limits of agreement of the FTV CO were unacceptably high compared with thermodilution, and furthermore, the divergence in CO obtained by the two methods became greater with each step increase in ABP, demonstrating that (at least in this context) the CO measured by FTV was dependent on MAP.

These findings highlight the fact that arterial pressure-based cardiac output (APCO) methods, particularly when uncalibrated, are still strongly influenced by factors that affect ABP and APW independently of SV and CO. The quality of the APW, the degree of the pressure wave reflection at the arterial site (that is, radial versus femoral), the degree and rapidity of change of vascular tone and compliance, and the geometry of the arterial system can all affect APCO algorithms, making these systems unreliable in patients undergoing rapid changes in ABP due to change in vascular resistance (for example, during pharmacologically induced vasconstriction). So although theoretically the algorithm should compensate for changes in tone and arterial site every 60 seconds in accordance with the model, it seems clear that the autocalibration scaling factor undercompensates for the increase in vascular tone and overcompensates in conditions of low vascular tone, making the system directly proportional to changes in ABP.

In fairness, the second-generation software of FTV has shown improved accuracy and precision in conditions of haemodynamic stability, or during changes in intra-vascular volume in the absence of significant variation in vascular tone, and so may be helpful in guiding volume loading (for example, during ‘early goal-directed therapy’ or pre-operative optimisation for elective surgery). However, unacceptably poor agreement has been shown in studies including patients at extremes of vascular tone and compliance such as cirrhotic patients undergoing liver transplant [2,3], patients with septic shock [4], haemodynamically unstable critically ill patients on large doses of vasopressors [5], and patients undergoing cardiac surgery [6], in which changes in vascular tone and compliance are prominent and the apparent changes in CO are due to the variations in the APW [7].

Another important factor to consider when interpreting CO measured by any APCO system is that the site of ABP measurement (for example, radial versus femoral artery) may significantly affect the APW and therefore CO. Discrepancies between central and peripheral blood pressures have been described in a number of clinical circumstances such as after cardiopulmonary bypass [8], during deep hypothermic circulatory arrest [9], during cardiopulmonary resuscitation [10], in patients with septic shock treated with high-dose vasoconstrictors [11], and in patients during reperfusion after liver transplant [12]. The differences in ABP between different sites may be large and in conditions of intense vasconstriction the radial ABP may underestimate the true aortic ABP, giving a falsely low CO value. It is concerning that in the study by Eleftheriadis and colleagues [1], the large differences in CO between FTV and pulmonary artery catheter were demonstrated despite the fact that the ABP for the FTV was obtained from the femoral artery. Central arteries should be less sensitive to variations in response to vasoactive drugs as the arteriolar tone is already high, and the reflection coefficient (the ratio between the reflected wave and the incident wave in the frequency domain) can be increased only marginally by intense vasoconstriction [13]. Studies looking at the differences in CO when the FTV was connected to a radial or a femoral artery have shown variable results [14,15] but highlight the fact that the impact of the site of the arterial catheter may not be negligible and the algorithm may not be able to compensate for changes in shape and amplitude of the APW in extreme haemodynamic conditions.

In conclusion, autocalibrated systems are useful only when used to monitor changes in SV during fluid challenge in stable conditions but become less accurate with changes in vascular tone and reactivity. Until more robust algorithms and further validation studies in critically ill patients become available, we should be aware that in conditions of haemodynamic instability, uncalibrated ABP CO systems may diverge from independent bolus measurements, particularly if a peripheral artery is used as this may underestimate or overestimate central blood pressure depending on the vascular tone. In these conditions, we advocate the use of systems that are recalibrated frequently using indicator dilution via either the femoral or the pulmonary artery.

**Abbreviations**

\( P_{H} \) = arterial pressure; \( ABP \) = arterial blood pressure; \( APCO \) = arterial pressure-based cardiac output; \( APW \) = arterial pressure waveform; \( CO \) = cardiac output; \( FTV \) = FloTrac®-Vigileo system; \( ICU \) = intensive care unit; \( MAP \) = mean arterial pressure; \( SV \) = stroke volume.
Competing interests
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