CRITICAL CARE

COMMENTARY

Pitfalls in haemodynamic monitoring based on the arterial pressure waveform

Luigi Camporota and Richard Beale*

See related research of Eleftheriadis *et al.*, http://ccforum.com/content/13/6/R179

Abstract

The accuracy of the arterial pressure-based cardiac output FloTrac-Vigileo system remains unacceptably low during haemodynamic instability. Data show that the measurement of cardiac output (CO) is strongly influenced by changes in factors that affect arterial blood pressure (ABP) – for example, vascular tone and compliance and the arterial site – independently of true changes in CO. Although in theory the autocalibration algorithm of FloTrac-Vigileo should adjust for those changes, the model undercompensates (or overcompensates) for prominent increases (or decreases) in vascular tone and compliance, making the system largely dependent on changes in ABP. These limitations make FloTrac-Vigileo accurate in stable haemodynamic conditions only, and until more robust algorithms and further validation studies become available, we should be aware that during haemodynamic instability or in extreme conditions of vasodilation or vasoconstriction, the measured CO may diverge from an independent bolus indicator dilution measurement, particularly if a peripheral artery is used. In these conditions, we advocate the use of transpulmonary indicator dilution via a femoral artery.

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 (σ_{AP}) around the mean arterial pressure (MAP). σ_{AP} 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

*Correspondence: richard.beale@gstt.nhs.uk
Department of Adult Critical Care - Guy's and St Thomas' NHS Foundation Trust, St Thomas' Hospital, 1st Floor East Wing - Lambeth Palace Road, London, SE1 7EH, UK

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 vasoconstriction). 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 intravascular 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 vasoconstriction 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

σ_{AP} = 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
RB and LC declare that they have no personal competing interests. The Department has received research support from Philips (Amsterdam, The Netherlands), LiDCO (Cambridge, UK), Applied Physiology (Sydney, Australia), Covidien (Dublin, Ireland), and Oxford Biosignals (Carmel, IN, USA).

Published: 5 March 2010

References

1. Eleftheriadis S, Galatoudis Z, Didilis V, Bougioukas I, Schon J, Heinze H, Berger KU, Heringlake M: **Variations in arterial blood pressure are associated with parallel changes in FlowTrac/Vigileo(R)-derived cardiac output measurements: a prospective comparison study.** *Crit Care* 2009, **13:**R179.
2. Biancofiore G, Critchley LA, Lee A, Bindi L, Bisà M, Esposito M, Meacci L, Mozzo R, DeSimone P, Urbani L, Filipponi F: **Evaluation of an uncalibrated arterial pulse contour cardiac output monitoring system in cirrhotic patients undergoing liver surgery.** *Br J Anaesth* 2009, **102:**47-54.
3. Biais M, Nouette-Gaulain K, Cottenceau V, Vallet A, Cochard JF, Revel P, Sztark F: **Cardiac output measurement in patients undergoing liver transplantation: pulmonary artery catheter versus uncalibrated arterial pressure waveform analysis.** *Anesth Analg* 2008, **106:**1480-1486, table of contents.
4. Sakka SG, Kozieras J, Thuemer O, van Hout N: **Measurement of cardiac output: a comparison between transpulmonary thermodilution and uncalibrated pulse contour analysis.** *Br J Anaesth* 2007, **99:**337-342.
5. Compton FD, Zukunft B, Hoffmann C, Zidek W, Schaefer JH: **Performance of a minimally invasive uncalibrated cardiac output monitoring system (Flotrac/Vigileo) in haemodynamically unstable patients.** *Br J Anaesth* 2008, **100:**451-456.
6. Mayer J, Boldt J, Schollhorn T, Rohm KD, Mengistu AM, Suttner S: **Semi-invasive monitoring of cardiac output by a new device using arterial pressure waveform analysis: a comparison with intermittent pulmonary artery thermodilution in patients undergoing cardiac surgery.** *Br J Anaesth* 2007, **98:**176-182.
7. Mayer J, Boldt J, Poland R, Peterson A, Manecke GR Jr.: **Continuous arterial pressure waveform-based cardiac output using the FloTrac/Vigileo: a review and meta-analysis.** *J Cardiothorac Vasc Anesth* 2009, **23:**401-406.
8. Chauhan S, Saxena N, Mehrotra S, Rao BH, Sahu M: **Femoral artery pressures are more reliable than radial artery pressures on initiation of cardiopulmonary bypass.** *J Cardiothorac Vasc Anesth* 2000, **14:**274-276.
9. Manecke GR Jr., Parimucha M, Stratmann G, Wilson WC, Roth DM, Auger WR, Kerr KM, Jamieson SW, Kapelanski DP, Mitchell MM: **Deep hypothermic circulatory arrest and the femoral-to-radial arterial pressure gradient.** *J Cardiothorac Vasc Anesth* 2004, **18:**175-179.
10. Rivers EP, Lozon J, Enriquez E, Havstad SV, Martin GB, Lewandowski CA, Goetting MG, Rosenberg JA, Paradis NA, Nowak RM: **Simultaneous radial, femoral, and aortic arterial pressures during human cardiopulmonary resuscitation.** *Crit Care Med* 1993, **21:**878-883.
11. Dorman T, Breslow MJ, Lipsett PA, Rosenberg JM, Balser JR, Almog Y, Rosenfeld BA: **Radial artery pressure monitoring underestimates central arterial pressure during vasopressor therapy in critically ill surgical patients.** *Crit Care Med* 1998, **26:**1646-1649.
12. Arnal D, Garutti I, Perez-Pena J, Olmedilla L, Tzenkov IG: **Radial to femoral arterial blood pressure differences during liver transplantation.** *Anaesthesia* 2005, **60:**766-771.
13. Nichols WW, O'Rourke MF: *McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles*. 5th edition. London: Hodder Arnold; 2005.
14. Hofer CK, Button D, Weibel L, Genoni M, Zollinger A: **Uncalibrated radial and femoral arterial pressure waveform analysis for continuous cardiac output measurement: an evaluation in cardiac surgery patients.** *J Cardiothorac Vasc Anesth* 2009 Aug 21. [Epub ahead of print].
15. Schramm S, Albrecht E, Frascarolo P, Chassot PG, Spahn DR: **Validity of an arterial pressure waveform analysis device: does the puncture site play a role in the agreement with intermittent pulmonary catheter thermodilution measurements?** *J Cardiothorac Vasc Anesth* 2009 Aug 21. [Epub ahead of print].

doi:10.1186/cc8845
Cite this article as: Camporota L, Beale R: **Pitfalls in haemodynamic monitoring based on the arterial pressure waveform.** *Critical Care* 2010, **14:**124.

CRITICAL CARE

COMMENTARY

Pulse pressure analysis: to make a long story short

Maurizio Cecconi and Andrew Rhodes*

See related research by Monnet *et al.*, http://ccforum.com/content/14/3/R109

Abstract

Pulse pressure analysis algorithms are commonly used to measure cardiac output and to allow for the rational titration of therapy in critically ill patients. The ability of these algorithms to accurately track changes in stroke volume (and cardiac output) is thus very important. Most of the currently available algorithms can provide robust data so long as there is no fundamental change in the vasomotor tone (arterial compliance or impedance). If the tone changes significantly, for instance with vasodilatation or vasoconstriction, then the data become less robust. For this reason, unless there is a mechanism for compensating for changes in vasomotor tone, these algorithms are best used only over short time periods in order to get the most accurate and precise data on changes in cardiac output.

Many authors have discussed the importance of measuring cardiac output and then titrating therapy according to these measurements in patients in the operating theatre [1,2] and intensive care environments [3]. Indeed, in some circumstances these measurements have led to changes in therapy that, in themselves, have been associated with improvements in outcomes [3]. The 'art' or 'science' of measuring this variable is therefore rightly given significant airplay in the ongoing literature of our specialty [4].

There are nowadays many devices available that purport to measure cardiac output. These include methodologies based on indicator dilution or thermodilution, Doppler principles, the Fick technique and also pulse pressure analysis. The pulse pressure analysis techniques have become increasingly popular due to the rising number of companies now marketing these devices [4]. It is incumbent on us as practicing clinicians to understand the similarities and differences between these devices so that we can ensure that we use techniques that we can rely upon to be accurate and precise in the clinical environment and also then integrate with therapies that are beneficial to our patients.

If we step back and look carefully at how these tools are used, then we would purport that there are two different scenarios that could be discussed. The first scenario is where a snapshot of the circulatory status is required. This needs an accurate and precise measurement in order to provide useful information [5-7]. The second scenario is where clinical interventions are titrated against changes in cardiac output - for instance, with a passive leg raise [8,9] or volume challenge [2]. In this scenario it is less relevant that we have an accurate and precise measurement, although it is more important that we can track the changes in the underlying signal reliably [10]. On the whole, the pulse pressure analysis techniques for estimating cardiac output are better placed at helping us with this second scenario than the first. In order to have an accurate and precise measurement, the relationship between arterial pressure and central impedance needs to be clarified and this usually means having to make an independent measurement as impedance is notoriously difficult to measure. Most companies therefore market these devices combined with another method of measuring cardiac output to calibrate the pulse pressure algorithm at baseline for this problem - commonly with either transpulmonary thermodilution or lithium (indicator) dilution techniques.

On a beat to beat basis pulse pressure provides a very good surrogate of changes in stroke volume. As the time interval lengthens, however, this relationship becomes less robust as the vascular tone will change, thereby adversely influencing this signal. The same holds true for the measurement of changes in stroke volume and/or cardiac output from pulse pressure tracking techniques. Over time many of the competing influences on the systemic vasculature will alter - level of preload, compliance, arterial resistance, and so on. This makes the assumption that changes in the arterial pressure signal directly relate to changes in flow less robust. On a beat to beat basis many of the marketed technologies will provide reliable information. Unfortunately, these tools are rarely used over a beat to beat basis and are more commonly used

*Correspondence: andyr@sgul.ac.uk
Department of General Intensive Care, St George's Healthcare NHS Trust, London, SW17 0QT, UK

over a period of time that may be 30 minutes or perhaps over an hour. If we look at the variety of methodologies used for giving a fluid challenge we can see this all too vividly. Many authors give the fluid over a 30 to 60 minute time window [11]. After 60 minutes it is quite possible that the vascular tone has changed significantly, thereby raising the question as to whether the change in flow estimated from the pressure signal is real or artefactual.

In order to understand this problem a number of authors have investigated these techniques under changing circulatory conditions. In an elegant study, Marquez and colleagues [12] demonstrated that the LiDCO*plus* algorithm, when compared against aortic flow probes, was able to track changes in stroke volume in response to a venous occlusion, although there tended to be an underestimation at higher values. Yamashita and colleagues [13,14] assessed how the precision of the algorithms was maintained under therapeutic vasodilatation with prostaglandin E1 during cardiac surgery. They tested the LiDCO™*plus* and the pulse contour method of the PiCCO*plus* versus the intermittent thermodilution of the pulmonary artery catheter. These studies suggested that after significant haemodynamic change (vasodilatation), the algorithms may underestimate the cardiac output and therefore not give a reliable estimate in the change of the signal. More recently, Monnet and colleagues [1] assessed how the PiCCO*plus* and the Vigileo (v1.10) handle vasoconstriction induced by infusion of norepinephrine. They concluded that the Vigileo algorithm was less able to track the changes in cardiac index during these situations. A further important consideration from all of these studies is that each algorithm, or algorithm update, will behave differently and will require independent validation. This can be seen in the meta-analysis published by Mayer and colleagues [15] looking at the new and older versions of the Vigileo algorithms where dramatically differing levels of accuracy and precision were seen.

It seems clear that if these devices are to be used to be able to track changes in cardiac output induced by changes in preload, then much care must be taken to ensure that in addition there are no major influences from altered vascular tone. The only way of ensuring this is to make the time interval between measurements short - perhaps minutes rather than hours. If we want to assess the circulation over longer time intervals, then a measurement independent of pulse pressure analysis needs to be included to compensate for these changes in vascular tone. When designing methodologies for assessing the response to a passive leg raise [8], an end expiratory occlusion [16], a Valsava manoeuvre [17] or a fluid challenge [2] this message needs to be understood. Perform the intervention quickly and the monitor should be able to track the change reliably and the correct interpretation should be made.

Competing interests
MC and AR received lecturing fees and an educational grant from LiDCO. MC received lecturing fees from Edwards.

Published: 12 July 2010

References

1. Monnet XNA, Naudin B, Jabot J, Richard C, Teboul J-L: **Arterial pressure-based cardiac output in septic patients: different accuracy of pulse contour and uncalibrated pressure waveform devices.** *Crit Care* 2010, **14:**R109.
2. Gan TJ, Soppitt A, Maroof M, el-Moalem H, Robertson KM, Moretti E, Dwane P, Glass PSA: **Goal-directed intraoperative fluid administration reduces length of hospital stay after major surgery.** *Anesthesiology* 2002, **97:**820-826.
3. Pearse R, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennett ED: **Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. A randomised, controlled trial [ISRCTN38797445].** *Crit Care* 2005, **9:**R687-693.
4. Hofer CK, Cecconi M, Marx G, della Rocca G: **Minimally invasive haemodynamic monitoring.** *Eur J Anaesthesiol* 2009, **26:**996-1002.
5. Cecconi M, Dawson D, Grounds RM, Rhodes A: **Lithium dilution cardiac output measurement in the critically ill patient: determination of precision of the technique.** *Intensive Care Med* 2008, **35:**498-504.
6. Jansen JR, Versprille A: **Improvement of cardiac output estimation by the thermodilution method during mechanical ventilation.** *Intensive Care Med* 1986, **12:**71-79.
7. Cecconi M, Rhodes A, Poloniecki J, Della Rocca G, Grounds RM: **Bench-to-bedside review: The importance of the precision of the reference technique in method comparison studies - with specific reference to the measurement of cardiac output.** *Crit Care* 2009, **13:**201.
8. Monnet X, Teboul JL: **Passive leg raising.** *Intensive Care Med* 2008, **34:**659-663.
9. Monnet X, Rienzo M, Osman D, Anguel N, Richard C, Pinsky MR, Teboul JL: **Passive leg raising predicts fluid responsiveness in the critically ill.** *Crit Care Med* 2006, **34:**1402-1407.
10. Squara P, Cecconi M, Rhodes A, Singer M, Chiche JD: **Tracking changes in cardiac output: methodological considerations for the validation of monitoring devices.** *Intensive Care Med* 2009, **35:**1801-1808.
11. Michard F, Teboul JL: **Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence.** *Chest* 2002, **121:**2000-2008.
12. Marquez J, McCurry K, Severyn DA, Pinsky MR: **Ability of pulse power, esophageal Doppler, and arterial pulse pressure to estimate rapid changes in stroke volume in humans.** *Crit Care Med* 2008, **36:**3001-3007.
13. Yamashita K, Nishiyama T, Yokoyama T, Abe H, Manabe M: **The effects of vasodilation on cardiac output measured by PiCCO.** *J Cardiothorac Vasc Anesth* 2008, **22:**688-692.
14. Yamashita K, Nishiyama T, Yokoyama T, Abe H, Manabe M: **Effects of vasodilation on cardiac output measured by PulseCO.** *J Clin Monit Comput* 2007, **21:**335-339.
15. Mayer J, Boldt J, Poland R, Peterson A, Manecke GR Jr: **Continuous arterial pressure waveform-based cardiac output using the FloTrac/Vigileo: a review and meta-analysis.** *J Cardiothorac Vasc Anesth* 2009, **23:**401-406.
16. Monnet X, Osman D, Ridel C, Lamia B, Richard C, Teboul JL: **Predicting volume responsiveness by using the end-expiratory occlusion in mechanically ventilated intensive care unit patients.** *Crit Care Med* 2009, **37:**951-956.
17. Monge Garcia MI, Gil Cano A, Diaz Monrove JC: **Arterial pressure changes during the Valsalva maneuver to predict fluid responsiveness in spontaneously breathing patients.** *Intensive Care Med* 2009, **35:**77-84.

doi:10.1186/cc9065
Cite this article as: Cecconi M, Rhodes A: **Pulse pressure analysis: to make a long story short.** *Critical Care* 2010, **14:**175.