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.
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 LiDCOplus 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 PiCCOplus versus the intermittent thermococulation 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 PiCCOplus 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, Soppott 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. Pease 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, 34:998-304.
6. Jansen JR, Versprille A: Improvement of cardiac output estimation by the thermococulation 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, Renzo 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 vasodilatation 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 vasodilatation on cardiac output measured by PulseCO. J Clin Monit Comput 2007, 21:335-339.
15. Mayer J, Boldt J, Pollard 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, Osram D, Riedl 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 Monroe JC: Arterial pressure changes during the Valsalva maneuver to predict fluid responsiveness in spontaneously breathing patients. Intensive Care Med 2009, 35:77-84.

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