Monitoring Arterial Blood Pressure and Cardiac Output using Central or Peripheral Arterial Pressure Waveforms

J. Smith, L. Camporota, and R. Beale

Introduction

Arterial blood pressure and cardiac output are the two most important and frequently measured hemodynamic parameters in critically ill patients as they provide indirect information on global tissue perfusion and oxygen delivery, and can guide fluid management and vasoactive drug use [1, 2]. Inaccurate measurement of these parameters, both in the intensive care unit (ICU) and the operating room (OR), can lead to misdiagnosis and inappropriate treatment, potentially impacting on patient morbidity and mortality. In the ICU, arterial blood pressure is commonly measured invasively via a peripheral artery (e.g., radial) or less frequently via a central artery (e.g., femoral). However, because the arterial blood pressure is not constant throughout the arterial tree – as a consequence of changes in hydrostatic pressure, arterial stiffness, and pressure wave reflection that are dependent on individual characteristics (e.g., age, height, gender), disease state (e.g., sepsis), and the administration of vasoactive drugs – the site of arterial blood pressure measurement may not faithfully reflect organ perfusion pressure.

Cardiac output is routinely measured using a variety of methods [3–5], but increasingly popular are those that use an indicator dilution technique to calibrate a continuous cardiac output measurement which is based on the analysis of the arterial pressure waveform obtained through a radial or a femoral arterial catheter [6–11]. Several different commercially available systems give a continuous cardiac output value based on an arterial waveform, but these differ considerably from one another in the way they relate changes in arterial blood pressure to changes in stroke volume [1, 2]. One approach is to calculate continuous cardiac output from the analysis of the area under the systolic portion of the arterial waveform, which is therefore pressure waveform morphology dependent (pulse-contour method) (i.e., PiCCO, PiCCO*plus* [Pulsion, Munich, Germany]) [12]; another approach is to calculate stroke volume from the entire arterial waveform (not just the systolic area), which is therefore not pressure morphology based (i.e., not a pulse contour method, PulseCO; LiDCO, Cambridge, UK) [13–15]. This latter system uses autocorrelation (a time-based system), rather than Fourier transform (frequency based system) to calculate the net power of the nominal stroke volume (to be converted in actual stroke volume after multiplying by a calibration factor), with the theoretical advantage of being less influenced by the timing of the reflected wave and the degree of damping of the arterial waveform. Other commercially available systems require no initial calibration with bolus indicator dilution technique ('uncalibrated systems') and give information on the changes in continuous cardiac output over time (FloTrac™, Vigileo™; Edwards Lifesciences, Irvine, CA, USA, Pressure Recording Analyt-

ical Method – PRAM, Vytech Health, Padova, Italy and LiDCO*rapid*, LiDCO, Cambridge, UK). Since all the methods that calculate continuous cardiac output rely on the analysis of the arterial waveform, it seems clear that obtaining an accurate arterial blood pressure waveform is crucial not only for the appropriate titration of vasopressors, but also for an accurate estimation and correct interpretation of continuous cardiac output measurement.

The translation of arterial pressure into stroke volume and then into cardiac output is mainly influenced by the following factors: 1) damping and resonance of the system; 2) the non-linearity of the relationship between the change in arterial pressure and the change in stroke volume in the arterial system (i.e., compliance of the system), which mandates a compliance correction to linearize a pressure signal into a arterial volume change; 3) the presence, size and timing of the reflected waves with effects that vary depending on the distance of the measurement site from the heart and on the vascular compliance. Therefore, the site where an arterial waveform is recorded (central versus peripheral) will determine the absolute value of blood pressure and the shape of the arterial pulse, and ultimately the dose of vasopressors and the estimation of cardiac output. In this chapter, we discuss the effects of the site of measurement of arterial blood pressure on continuous cardiac output values and arterial pressure which, if not considered in clinical practice, may lead to important misuse of fluids and vasoactive drugs.

Wave Contour in the Central and Peripheral Arteries

At each systole, the heart ejects a volume of blood into the aorta (stroke volume) and generates a forward pressure wave which then travels along the arterial tree. During this transit, the mean arterial pressure (MAP) progressively falls by 1 – 3 mmHg between the ascending aorta and brachial or radial artery, but the systolic and pulse pressure progressively increase and at the extremities the systolic blood pressure (SBP) can be twice the SBP in the ascending aorta [16], with absolute differences (radial-aortic) of up to 40 mmHg [17]. The contour of the pressure wave is modified as it travels from the central arteries to the peripheral vessels. This is accepted as being a consequence of the duration of systole, MAP, vasomotor tone, pulse wave velocity and 'pressure augmentation' by wave reflection and resonance arising from reflection and re-reflection of the pulse between the upper and lower part of the body. In the proximal aorta, the arrival of the reflected wave is in early diastole and if the velocity of the waveform increases, the reflected wave will arrive earlier in systole with the effect of increasing the systolic pressure. The major sites of wave reflection in the circulatory system are the points of impedance discontinuity, such as arterial branching and arterial-arteriolar junctions and particularly high-resistance arterioles where a pulse waveform entering the aorta is exposed to a sudden impedance change, resulting in a large increment in resistance and producing reflected pulse waveforms [18]. Morphologically, the amplified peripheral pressure wave has a shorter interval between the initial systolic peak and diastolic wave suggesting 'resonance' in a shorter system [19] and is displayed as a sharp narrow arterial wave, which usually contains the summation of two systolic peaks: One represents the 'forward pressure' wave generated by the heart, and the second, a superimposed wave, is the 'backward pressure' wave reflected from the peripheries [18]. The contribution of the reflected wave to the measured SBP occurs earlier in the periphery, where the SBP may be up to 35 mmHg higher than the central aortic

pressure [17]. This distal pulse amplification by wave reflection is always present when peripheral vascular resistance is high [20] and is most marked in young adults, in whom the amplitude of the radial pulse pressure may be 50 % greater than that in the ascending aorta, but is reduced during a Valsalva maneuver, hypotension, hypovolemia, and in the presence of vasodilatation. The latter is particularly well documented after administration of nitrates, when a significant reduction in aortic systolic pressure may not be appreciated by recording brachial or radial artery pressure (reviewed in [16]). In the contrary situation, during intense vasoconstriction, an increased wave reflection in the peripheries can lead to an underestimation of central aortic pressure if the SBP is measured in the radial artery [19]. Drug-induced alteration in central blood pressure and wave contours may be explained on the basis of change in arterial caliber, arterial stiffness and wave reflection.

A similar mechanism can occur also in the absence of drugs, in any clinical condition that leads to vasodilatation, such as in septic shock where radial systolic pressure may grossly overestimate by up to 20 mmHg the pressure in the central arteries as a result of a decreased wave reflection from the lower body which contributes to the reduced SBP in the central arteries but does not influence SBP in distal peripheral arteries to the same degree [19, 21]. The appreciation of this phenomenon is important because clinicians target a blood pressure value as though this was constantly the same, under all conditions and in all arteries. Even more therapeutically important is the situation that occurs during intense vasoconstriction (i.e., high doses of vasopressors or after cardiac surgery). While the arterial pressure is normally amplified in the radial artery and, therefore, is higher than the central arterial pressure, under conditions of excess vasoconstriction (typically high dose vasopressor use and relative hypovolemia) the radial pressure may underestimate central systolic and organ perfusion pressure, to the extent that it can grossly misguide the requirement of fluid and vasoactive agents (reviewed in [19]). The effect of vasoconstriction (e.g., during norepinephrine use) on the pressure waveform or flow wave contours is much less the more centrally the measurements are made (even if this can reduce mean flow), and this is explained by the fact that in the femoral artery the arteriolar tone is already high, and so the reflection coefficient (the ratio between the reflected wave and the incident wave in the frequency domain) already generated from this point in the circulation can be increased only marginally by intense vasoconstriction [19].

Being aware of these potential differences between central and peripheral arterial waveforms is important clinically for two main reasons: First, it is the central pressure and not the radial pressure that more directly determines organ perfusion; second, the degree to which radial pressure is variably affected by the pressure amplification that occurs in the peripheries means that it does not always accurately reflect the central pressure, which is usually the intended target of any therapeutic interventions aimed at achieving a particular blood pressure target. These principles emphasize the importance of monitoring central arterial pressure when perfusion pressure and cardiac afterload have to be determined precisely, as in shock states, during high doses of vasoactive drugs, or in the presence of an intra-aortic balloon pump (IABP) [19].

Agreement between Central and Peripheral Blood Pressure in Specific Clinical Situations

Invasive blood pressure measurement is performed through a catheter placed most commonly in the radial artery as it is easy to cannulate and has a low risk of complications. The femoral artery is one of several alternatives [22, 23]. Although the agreement between pressure obtained from a peripheral and central artery has been evaluated by many authors, the degree to which these two measures are interchangeable clinically is still a matter of debate. Some studies have shown a good correlation between the site measurements. Mignini et al. [24], in a recent trial in 55 medical and surgical patients requiring high dose of vasopressors (dopamine = 10 μg/kg/min or epinephrine or norepinephrine = 0.1 μg /kg/min) or low dose of vasopressor, compared simultaneous measurements of arterial pressure in peripheral and central arteries. The study showed no significant difference between the femoral and the radial artery in SBP (135 ± 31 vs 126 ± 30 mmHg), diastolic blood pressure (DBP, 63 ± 14 vs 62 ± 13 mmHg) or MAP (85 ± 17 vs 82 ± 17 mmHg). No difference was found either between the group on high doses of vasoactive drugs versus the group on low doses with a bias ± precision in the blood pressure of 3 ± 4 mmHg and 3 ± 4 mmHg, respectively, showing that the radial and femoral measurement of the blood pressure agreed well regardless of the dose of vasoactive drug used, and the authors suggested that these two measurements are interchangeable. Similarly, Yazigi et al. [25] found that the radial arterial pressure seemed an accurate measure of the central arterial pressure as there was no statistically significant difference between peripheral and central arterial pressures either before, during or after controlled hypotension by vasodilatation in 10 healthy patients undergoing surgery. However, discrepancies between central to peripheral blood pressures have been reported to occur in a number of clinical circumstances such as after cardiopulmonary bypass (CPB) [26–28], during deep hypothermic circulatory arrest [29], cardiopulmonary resuscitation (CPR) [30], isofluorane anesthesia [31], in patients with sepsis treated with high dose vasoconstrictors [32], and in patients during reperfusion post-liver transplant [33].

Kanazawa et al. [26], in patients after CPB, showed the presence of a pressure gradient between central and peripheral sites and changes in the pulse wave velocity (PWV). Of the 12 patients, seven had a pressure gradient and a difference of 27 ± 11 mmHg in the SBP between the aorta and the radial artery and the PWV gradually decreased from the central to peripheral artery. The occurrence of a pressure gradient after CPB was due to a decrease in arterial elasticity from the aorta to the radial artery. Similar blood pressure differences have been described by Baba et al. who reported that 38/75 patients undergoing CPB displayed a gradient (femoral-radial) in MAP > 5 mmHg and radial artery constriction could be responsible for the pressure gradient [27] and Chauhan et al. who showed that femoral artery perfusion pressures were higher and more reliable during the initial part of CPB [28]. Similarly, Gravlee et al. [34] showed that a clinically important (> 10 mmHg) underestimation of systolic aortic pressures occurred in 52 % of radial artery catheters and radial artery MAP underestimated aortic MAP by > 5 mmHg in 61 % of the patients two minutes after CPB. In a different setting, Manecke et al. [29] showed that 76 % of the patients receiving CPB with profound hypothermia and circulatory arrest exhibited a mean arterial gradient of at least 10 mmHg either during or after CPB, with femoral readings being higher. Clinically significant gradients were noted throughout the CPB period and the post-CPB period in these patients. In the 54

patients studied, the SBP gradient was 32 ± 19 mmHg after CPB and the MAP gradient was 6.3 ± 4.9 mmHg. The duration of clinically significant SBP (> 10 mmHg) and MAP (> 5 mmHg) gradients in the postoperative period were 5.2 ± 5.7 hours and 5.8 ± 7.2 hours, respectively, making the recommendation for the use of central arterial pressure monitoring for intraoperative and postoperative care [29]. Similarly, Arnal et al. [33] in 72 patients undergoing liver transplantation found that femoral SBP was significantly higher than radial SBP only during liver reperfusion (92 ± 22 vs 76 ± 22 mmHg, $p < 0.01$); the DBP and MAP did not differ in two sites. In 27 of 72 patients who required vasopressors for hemodynamic instability, there was a statistically significant difference between femoral and radial systolic arterial pressure during the reperfusion period. Taken together, these findings suggest that this is a real and important phenomenon, but only occurs in some patients in some circumstances, and so may be underestimated if evaluated solely by the use of statistics applied to a whole study population.

A systematic discrepancy between radial and central blood pressure measurement has also been demonstrated in an important paper by Dorman et al. [32] on 14 post-operative patients with septic shock requiring high doses of norepinephrine – a situation highly relevant to current critical care practice. In these patients, a consistent underestimation of peripheral blood pressure was observed in the radial measurement. Femoral artery systolic pressures were significantly higher than radial artery systolic pressures (143 ± 8.9 vs 86 ± 4.5 mmHg), but, what is more pertinent is that the MAPs were also higher in the femoral artery than in the radial artery (81 ± 2.5 vs 66 ± 2.2 mmHg). The difference between the two sites was large enough to allow a reduction in vasopressor support in 11 (79 %) of 14 patients (85.6 ± 25.3 to 57.2 ± 16.4 μg /min, $p < 0.05$), even discontinuing it in two patients. These data strongly suggest that in hemodynamically unstable patients requiring large amounts of vasoactive drugs, monitoring arterial pressure from the femoral site seems preferable, as the radial site may significantly underestimate not only SBP but also MAP, with important repercussions on fluid and vasoactive drug use.

Influence of the Site of Blood Pressure Measurement on Continuous Cardiac Output Estimation

The increasingly routine use of continuous cardiac output monitoring derived from the arterial pressure wave (pulse contour analysis), in place of the more invasive pulmonary artery catheter (PAC) [35, 36], makes obtaining an accurate arterial waveform and the understanding of the factors influencing the shape of the waveform essential for the correct clinical interpretation of the cardiac output [37]. Studies comparing continuous cardiac output measurements derived from the peripheral and central artery have shown that the quality of the pressure waveform obtained from the radial artery is accurate also for pulse contour analysis. de Wilde et al. [38] compared femoral and radial artery pressure measurement signals as inputs for the PiCCO system in 14 patients following cardiac surgery. The study showed a high level of agreement between cardiac output in the femoral artery and cardiac output in the radial artery (bias -0.01, SD [0.31] l/min), which suggests the interchangeability of radial and femoral arterial pressure signals for continuous cardiac output monitoring. Similarly, Orme et al. [39] and Wouters et al. [40] studied the accuracy of using the brachial artery to measure cardiac output. The values obtained from the brachial artery agreed with the values obtained from the PAC (bias 0.38, SD 0.77 l/min and bias

0.91, SD 0.41 l/min for the two studies, respectively). Moreover, the pulse contour analysis using a brachial artery catheter was in agreement with pulmonary artery thermodilution, concluding that the brachial artery is a valid alternative to the femoral artery when the femoral approach is not desirable. However, in spite of the reassuring tenor of these studies, since arterial blood pressure and arterial waveform measured at the radial artery can differ significantly from those measured simultaneously in the central arteries particularly during the administration of vasoactive drugs, continuous cardiac output measured at the two sites can vary in a proportion of critically ill patients. In our experience, in patients requiring high doses of vasopressors, femoral arterial pressure is generally higher than radial arterial pressure (**Figs. 1–3**), and

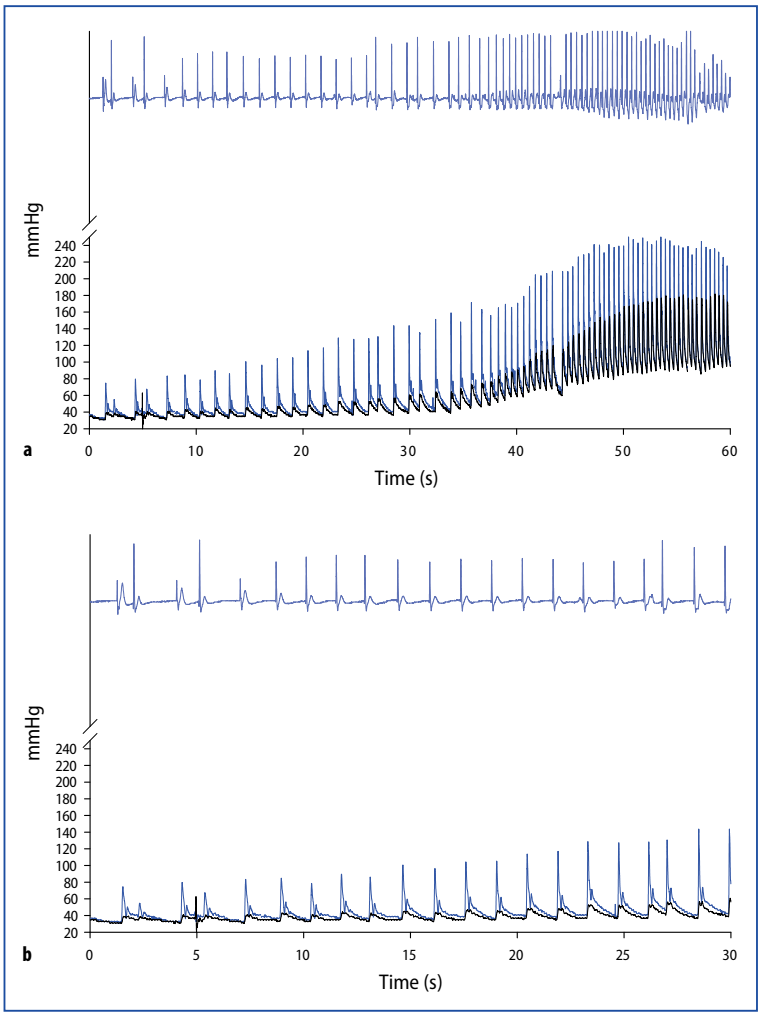


Fig. 1. During extreme hemodynamic conditions, a bolus of epinephrine was administered and the radial (black line) and femoral (blue line) arterial pressures were evaluated. The femoral arterial blood pressure was higher than the radial arterial blood pressure. Panel **a** shows the first 30 seconds of the recording in **b**.

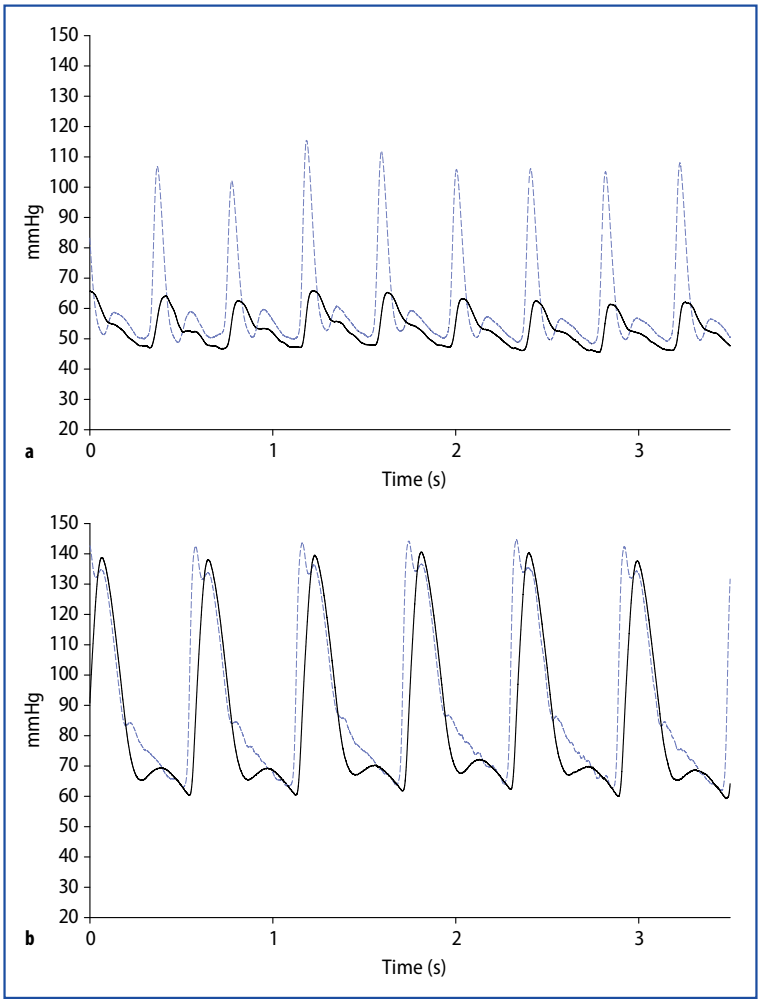


Fig. 2a. A patient with septic shock post bowel resection for fecal peritonitis in hypovolemic state requiring vasopressors (norepinephrine 0.7 μg/kg/min). The discrepancy between radial (solid line) and femoral (dashed line) arterial blood pressure measurement is evident. **b** agreement between the two arterial blood pressure measurements is seen after vasopressor reduction and fluid resuscitation.

this discrepancy can be even more dramatic under extreme hemodynamic conditions (**Fig. 1**). The difference in arterial blood pressure is then reflected in a large difference in the continuous cardiac output value at the two arterial sites (**Fig. 4**) leading to a difference in cardiac output between the two sites of up to 3 l/min [41]. This suggests that radial and femoral artery are not automatically interchangeable sites for cardiac output monitoring anymore than they are for blood pressure monitoring, and there may be important differences in various physiological conditions (e.g., age, aortic compliance) or in association with low flow/cardiac output states, particularly during rapid changes in hemodynamics as a consequence of the use of fluids and high doses of vasoactive agents in shock.

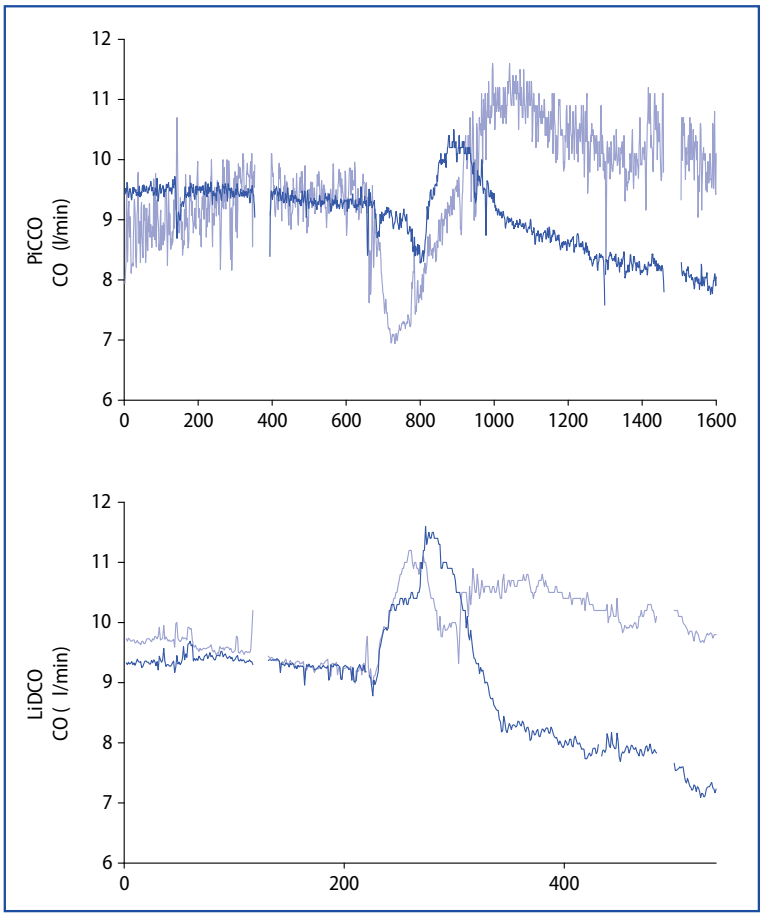
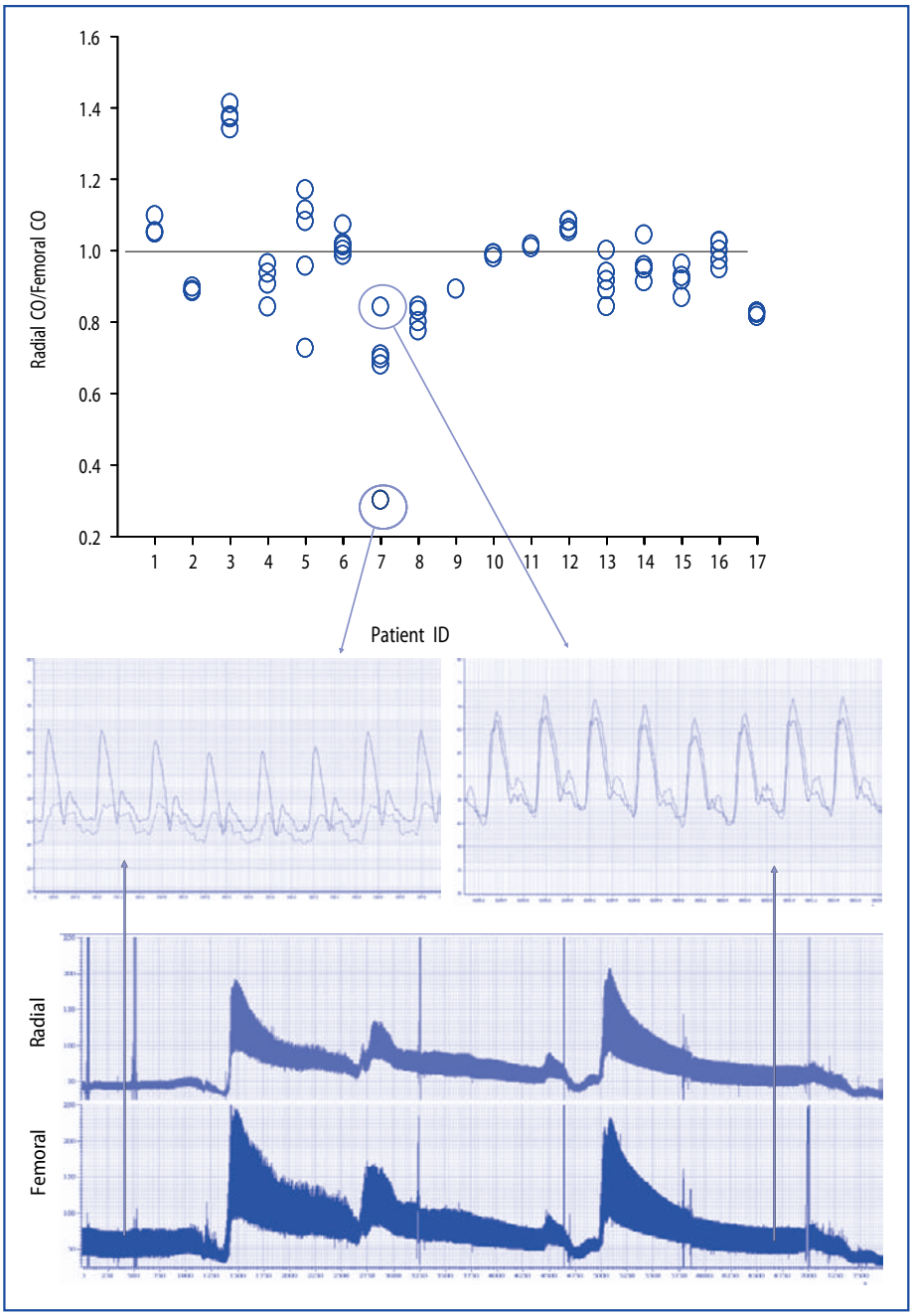


Fig. 3. Continuous cardiac output recording with the PiCCO system and the LiDCO system in radial (pale blue line) and femoral (dark blue line) arteries. The discrepancy between the two sites is evident.

The question therefore arises as to when it is reasonable to assume that the peripheral (radial) pressure measurement is a faithful reflection of the central pressure, either for blood pressure targeting *per se*, or for cardiac output derivation. In our view, the safest option is to use radial pressure if the radial pressure is in the "normal" range, if the pulse wave morphology is well defined, and if peripheral perfusion is clinically good or if a vasopressor is being used at a moderate and/or decreasing dose.

▷

Fig. 4. Top: Cardiac output (CO) ratio between radial and femoral site in 17 ICU patients [40]. The median value of the cardiac output ratio was 0.95 (IQR 0.88 to 1.02), with a high variability among the patients, ranging from 0.3 to 1.41, whereas intra-patient variability was low, with a median CV of 3.26 % (IQR 1.1 to 5.3 %). Bottom: Cardiac output measurement derived from simultaneous radial (pale blue line) and femoral (dark blue line) blood pressures in a single patient. When evaluating the data by individual time point pairs,



the ratio of cardiac outputs between the two sites varied greatly. The difference between the median (range) arterial pressures was 2 mmHg (–3 to 8 mmHg), however, the pulse pressure difference was generally large with a median (range) of 2 mmHg (–26 to 44 mmHg). The difference between the two sites is large enough to be clinically unacceptable without a site-specific recalibration.

In these situations the peripheral blood pressure is assumed to be equivalent to the central pressure. In situations where the radial pressure is low, the pulse wave morphology is 'damped' but technical problems with the arterial catheter or the tubing system cannot be identified (i.e., normal damping and resonance of the system after a 'flush test'), peripheral perfusion is clinically poor and the vasopressor dose is high and increasing, central pressure measurement is preferable and more likely to accurately reflect the 'true' organ perfusion pressure.

Conclusion

There may be a dramatic pressure gradient between femoral and radial arterial pressure in several conditions which are either physiological (e.g., age, aortic compliance) or in association with low flow/cardiac output state, particularly during rapid changes in hemodynamics as a consequence of vasoactive agents in shock. This is especially the case with high doses of vasopressors and hypovolemia. Peripheral pressure waveform may underestimate or overestimate central blood pressure depending on the type of vasoactive drugs (vasoconstrictors or vasodilators) used and the degree of blood flow limitation (cardiac output). It follows that using the peripheral arterial pressure may occasionally lead to false assumptions about the correct therapeutic intervention under these circumstances.

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