

# Femoral–radial arterial pressure gradients in critically ill patients

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Invasive arterial pressure monitoring is fundamental to intensive care practice, but debate persists about the ideal monitoring site. Ideally, one wishes to measure central (aortic) pressure, as this best reflects perfusion of vital organs. In practice, the radial site is most often used, with the assumption that this peripheral pressure is an accurate surrogate for central pressure.<sup>1</sup> This premise may be flawed in the abnormal haemodynamic state of critical illness.

The arterial pressure waveform undergoes characteristic morphological changes as it travels from central to peripheral vessels. These complex, dynamic changes involve reflection and summation of pressure waves, modulated by factors such as stroke volume, heart rate and arterial elastance.<sup>2–6</sup> In health, distal pulse amplification results in higher systolic arterial pressures at the periphery, while diastolic and mean arterial pressures (MAP) are relatively preserved. Compared with arterial pressure waveforms measured in the central arteries (eg, aorta or femoral artery), those at the periphery (eg, radial artery) characteristically have steeper upstrokes, higher systolic peaks, a later appearing diastolic notch, more prominent diastolic waves and lower end-diastolic pressure.<sup>2–6</sup> Yet, under normal physiological conditions, only a negligible decrement in MAP occurs.<sup>6</sup>

In this context, the radial and femoral sites appear conveniently interchangeable for measurement of MAP. In intensive care practice, the radial site is most commonly used but, during aberrant haemodynamic conditions, gradients in MAP may develop from the central to the peripheral arterial tree. These gradients have been repeatedly demonstrated in cardiac surgical patients undergoing cardiopulmonary bypass, although the mechanisms responsible are poorly understood.<sup>7–11</sup>

The phenomenon has not been well documented in heterogeneous critically ill populations. Dorman et al noted large discrepancies in MAP measured at the radial and femoral sites in patients receiving high-dose vasopressors,<sup>12,13</sup> but this has not been a consistent finding.<sup>14</sup> Additionally, the specific clinical and demographic factors that predict or engender such a difference have not been identified. Doubt remains as to the equivalence of central and peripheral monitoring of arterial pressure in the critically ill. If measurement at the periphery leads to underesti-

## ABSTRACT

**Objective:** To investigate the presence and determinants of femoral–radial gradients in mean arterial pressure (MAP) in a critically ill population.

**Design:** Prospective observational study.

**Setting and participants:** Critically ill patients who were undergoing simultaneous monitoring of arterial pressure by radial arterial catheterisation and transpulmonary thermodilution (via femoral arterial access) in a Level 3, mixed medical–surgical intensive care unit, December 2007 to May 2008.

**Main outcome measures:** Level of agreement between simultaneous measurements of MAP via the femoral and radial arteries, determined by Bland–Altman analysis; haemodynamic and demographic factors associated with a MAP gradient, assessed by multiple linear regression.

**Results:** 131 observations were made in 24 patients. Mean age of patients was 56 (SD, 18) years, and mean APACHE II score was 27 (SD, 8). Overall mean bias between radial and femoral MAP measurements was 4.27 mmHg (limits of agreement, –3.41 to 11.94 mmHg). Fifteen patients (62%) had maximum MAP gradients >5 mmHg, and seven of these (29% of the total) had maximum gradients >10 mmHg. The largest discrepancy in MAP was 18 mmHg in a patient with septic shock resistant to high-dose catecholamine infusion. Regression analysis failed to identify any statistically significant associations between patient factors and MAP gradient.

**Conclusion:** A systematic difference in MAP measured at the radial and femoral sites was demonstrated. In some critically ill patients, the femoral artery may be the preferred site for systemic arterial pressure monitoring.

Crit Care Resusc 2009; 11: 34–38

mation of central pressure, then excess vasoactive medications or fluid therapy may be used.

This study aimed to determine the difference between radial (peripheral) and femoral (central) arterial pressures measured simultaneously in a group of critically ill patients. A secondary goal was to identify haemodynamic or demographic factors associated with these gradients.

## Methods

The study protocol was approved by the Royal Adelaide Hospital Research Ethics Committee. The requirement for informed consent was waived by the committee, as insertion of arterial catheters was part of patient clinical care.

We studied consecutive patients from a mixed surgical–medical, tertiary referral ICU who required monitoring with a radial arterial line as well as transpulmonary thermodilution via femoral arterial access between December 2007 and May 2008. In this ICU, the default site for invasive arterial pressure monitoring is the radial artery. Patients requiring advanced haemodynamic monitoring typically undergo either pulmonary or femoral arterial catheterisation to allow transpulmonary thermodilution, with the femoral route increasingly used.

The principal inclusion criterion for the study was the use of simultaneous monitoring with radial arterial catheterisation and transpulmonary thermodilution via femoral arterial access, as directed by the treating clinician. Typical indications for monitoring with transpulmonary thermodilution are characterisation and management of shock; respiratory failure, including acute respiratory distress syndrome; and multiple organ dysfunction syndrome with any cause. No invasive monitoring lines were placed solely for the purpose of the study. Exclusion criteria for the study included patient age < 18 years; use of intra-aortic balloon counterpulsation; severe peripheral vascular disease or critical ischaemia of the lower limbs; and arrhythmia resulting in wide fluctuations in beat-to-beat blood pressure (atrial fibrillation, and frequent atrial or ventricular ectopy).

At patient enrolment, we collected demographic and clinical data on age, sex, height, weight, APACHE II score, admission diagnosis, and history of diabetes, hypertension or smoking. At the time of blood pressure measurements, we also recorded data on parameters that potentially influenced generation of a central–peripheral pressure gradient, including heart rate, use and dose of vasopressor and inotropic therapy, mode of mechanical ventilation and body temperature.

### Blood pressure measurements

Blood pressure was measured at the radial artery using a 20-gauge short Teflon catheter (Arrow International, Penn, USA), connected to a pressure transducer and 30 cm of non-compliant extension tubing with a continuous flush device via a three-way stopcock (Transpac, Abbott, Ill, USA). The pressure transducer system was connected to a bedside monitor (Solar 8000, GE Marquette, Milwaukee, USA) to display the arterial waveform and systolic, diastolic and mean arterial pressures. Non-invasive (oscillometric) measurement of brachial blood pressure was performed bilaterally to ensure equivalence of blood pressure in the upper

**Table 1. Characteristics of the study population**

Characteristic	No. of patients*
No. of patients studied	24
Mean age in years (SD)	56 (18)
Sex	
Men	15 (63%)
Women	9 (37%)
Mean APACHE II score (SD)	27 (8)
ICU mortality	6 (25%)
30-day mortality	10 (42%)
Admission diagnosis	
Sepsis	15 (62%)
Cardiogenic shock	5 (21%)
Other <sup>†</sup>	4 (17%)
Mechanical ventilation	23 (96%)
Vasoactive medication	24 (100%)

\* Continuous data are presented as mean (SD).

<sup>†</sup> Other diagnoses were acute pancreatitis (2), haemorrhagic shock from upper gastrointestinal bleeding (1), and acute respiratory distress syndrome after lung transplantation (1).

**Table 2. Use and dose of vasoactive medications**

Medication	No. of patients who received medication (n = 24)	Mean dose (SD)
Noradrenaline	19 (79%)	0.26 (0.21) µg/kg/min
Adrenaline	5 (21%)	0.11 (0.18) µg/kg/min
Dobutamine	2 (8%)	5.7 (3) µg/kg/min
Vasopressin	1 (4%)	0.02 (0.02) U/min

limbs. Patients with a significant discrepancy were excluded from the trial.

Blood pressure was measured at the femoral artery using a 20 cm 5F thermistor-tipped catheter (PiCCO catheter PV2015L20) connected to the PiCCO<sup>plus</sup> System using the proprietary pressure transducer and 150 cm extension tubing (PiCCO Monitoring Kit PV8115, Pulsion Medical Systems, Munich, Germany). A slave connection was made to the bedside monitor, so that radial and femoral arterial pressures were reproduced and displayed simultaneously on the same monitor.

Blood pressure measurements at each site were taken as the value averaged by the bedside monitor over a 1-minute period. Before recording, all pressure lines were zeroed to atmospheric pressure and referenced to the mid-axillary line with the patient in the supine position. All pressure measurements were referenced to the phlebostatic axis. The adequacy of the frequency response and damping coefficient

lients was assessed using the rapid flush test.<sup>15-17</sup> Blood pressure measurements were recorded whenever transpulmonary thermodilution was performed, as part of routine care, to determine cardiac output and derive volumetric parameters. Data collection ceased when either the radial or the femoral catheter was removed at the discretion of the treating clinician.

Cardiac output and volumetric variables were measured using the single-indicator transpulmonary thermodilution technique. Measurements were taken as the mean of three consecutive injections, randomised throughout the respiratory cycle, of 20 mL of cold saline solution (temperature, <math><8^{\circ}\text{C}</math>) via the distal port of a central venous catheter placed in the internal jugular or subclavian vein. Cardiac output was determined using a modified Stewart–Hamilton equation, and the volumetric data by assessment of the mean transit time and exponential downslope time of the thermodilution curve.<sup>18</sup> Global ejection fraction was expressed as the ratio of four times the stroke volume to global end-diastolic volume.<sup>19</sup> Following calibration for individual arterial compliance with transpulmonary thermodilution, pulse contour variables (stroke volume variation) were calculated using the area and shape of the systolic portion of the arterial pressure curve.<sup>20</sup>

### Statistical analysis

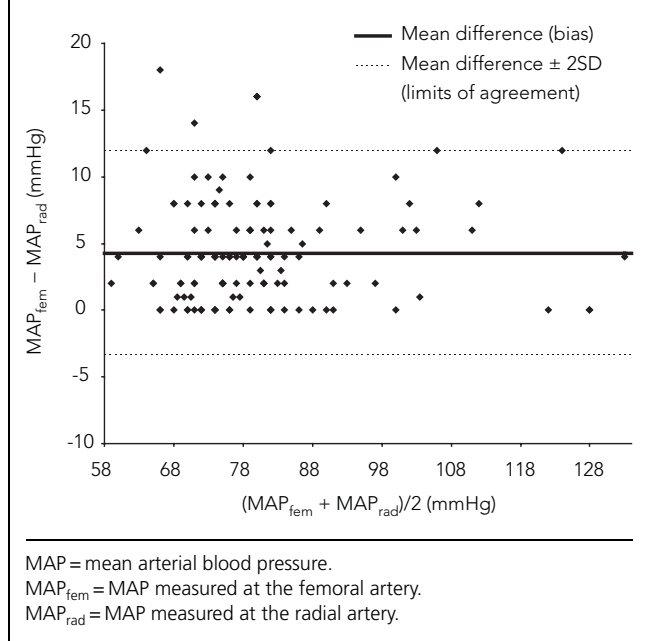
Mean and standard deviation were calculated for continuous data. Bland–Altman analysis<sup>21</sup> was used to compare agreement of radial and femoral measurements of systolic, diastolic and mean arterial pressures. Multiple linear regression was used to determine demographic or haemodynamic factors associated with a MAP gradient. Data were analysed using Microsoft Excel and Minitab 15 software.

### Results

A total of 131 observations were made in 24 patients, with a mean of 5.5 (SD, 3.7) observations per patient. Characteristics of the patients are shown in Table 1. Primary diagnostic categories included septic shock in 15 patients, and cardiogenic shock or admission after cardiac arrest in five patients. All patients were supported with vasoactive medications; the use and dose of specific medications is shown in Table 2.

Agreement between the radial and femoral measurements of MAP is shown in Figure 1. The overall mean bias between radial and femoral MAP measurements was 4.27 mmHg. Although this mean bias is modest, the limits of agreement were large (–3.41 to 11.94 mmHg). Fifteen patients (62%) had maximum MAP gradients >5 mmHg, and seven of these (29% of all patients) had maximum gradients >10 mmHg. In all cases, femoral MAP equalled or

**Figure 1. Bland–Altman plot showing agreement between MAP measurements at the radial and femoral arteries**



was greater than radial MAP. When there was a significant femoral–radial MAP gradient, the femoral (higher) pressure was assumed to be the better indicator of perfusion of vital organs, and the dosage of vasopressor medications was down-titrated. The largest difference in MAP between measurement sites was 18 mmHg in a patient with septic shock resistant to high-dose catecholamine infusion. There were also marked discrepancies in femoral and radial systolic arterial pressure. Mean bias was 8.8 mmHg, with limits of agreement of –21 to 38 mmHg (not shown).

Multiple regression analysis failed to identify any statistically significant associations between a haemodynamic or demographic factor and MAP gradient. Generation of a robust model was not feasible given the small size of this study and limited number of observations.

### Discussion

This study confirms a high prevalence of a systematic bias between MAP measurements at the radial and femoral sites in critically ill patients who require treatment with vasoactive medications. The definition of a clinically significant difference is arbitrary, but a change in MAP of 5 mmHg or more may result in changes to clinical management, such as vasopressor dose or fluid administration. By these criteria, up to 62% of our patients exhibited a clinically significant MAP gradient.

Arterial pressure monitoring has fundamental importance in critical care medicine, yet few studies have examined the effect of monitoring site on readings in this population. Our results show that the equivalence of monitoring site for MAP found in healthy people cannot be assumed for all critically ill patients. These results are consistent with previous studies. Although Mignini et al suggested radial and femoral pressure measurements were equivalent, with a small overall MAP difference ( $3 \pm 4$  mmHg), examination of their data reveal that several paired observations were markedly different (up to 15 mmHg).<sup>14</sup> The mean difference in our study was similarly modest, but a substantial proportion of the study population had large central–peripheral gradients. These findings are also consistent with those of the earlier study by Dorman et al,<sup>12</sup> and a more recent study that used methods similar to ours.<sup>22</sup> It thus appears that a proportion of patients receiving vasoactive agents have a clinically important difference in radial and femoral mean arterial pressures.

As central–peripheral pressure gradients are not ubiquitous, it would be useful to identify patients who are at risk, so that arterial pressure could be measured appropriately. Even in the most widely studied demographic of cardiac surgical patients undergoing cardiopulmonary bypass, the factors driving central–peripheral pressure gradients remain ill defined. The best described mechanisms include vasodilation and decreased resistance in the forearm and hand vasculature during rewarming.<sup>4</sup> A similar mechanism may operate in septic shock, in which distal pulse amplification may be mitigated by reduced hand vascular resistance.<sup>13</sup> In our study, we did not characterise regional haemodynamics but only global parameters, such as cardiac output, and derived indices, such as systemic vascular resistance index, the latter bearing little resemblance to in-vivo haemodynamics. The design of our study was inadequate, and the sample size too small, to identify any single factor as a determinant of a central–peripheral pressure gradient. This would require a much larger study population.

Importantly, vasopressor dose was not associated with any difference in MAP measurements at the two sites. This was not surprising as, in a specific individual, vasopressor medications do not have a predictable dose–response relationship, and dosage alone is not a good indicator of the underlying haemodynamic state. Our results complement those of Dorman et al, who showed a high prevalence of MAP gradients, but included only patients receiving very high-dose vasopressors (mean noradrenaline dose,  $85 \mu\text{g}/\text{min}$ ; SD,  $25 \mu\text{g}/\text{min}$ ).

Our study had limitations other than small sample size. Patients were enrolled only if judged by the treating clinician to require monitoring with transpulmonary thermodilution. Thus, the study sample does not represent either the hetero-

geneous critically ill patient population, or the group requiring vasoactive agents. The prevalence of central–peripheral pressure gradients is likely over-represented, as the sickest patients in the ICU were studied. Conversely, during enrolment, several patients monitored with transpulmonary thermodilution were excluded from the study because peripheral perfusion was so poor as to not allow radial artery cannulation. It is likely that the femoral–radial gradients in these patients would have been large.

Additionally, the monitoring equipment, including length of extension tubing and brand of pressure transducer, differed between the two monitoring sites. While these differences undoubtedly influenced the dynamic response of the monitoring system, they were most likely to affect the measurement of systolic and diastolic arterial pressure; they would not be expected to influence MAP significantly. Furthermore, many pairs of radial and femoral MAP measurements were identical over the study period, suggesting that pressure gradients, when observed, were not artefacts.

Lastly, more than half the patients had a primary diagnosis of septic shock. Comparison with patients with a different primary cause for shock (eg, cardiogenic shock) might have helped identify the haemodynamic origins of MAP gradients.

Notwithstanding these limitations, it appears that clinically significant central–peripheral pressure gradients occur in some critically ill patients. Several questions remain. Identifying which demographic or haemodynamic factors contribute to these gradients would be useful in determining when the femoral site is preferable for monitoring. Quantifying the reduction in vasopressor dosage afforded by the finding of an MAP gradient, and determining whether this translates to a meaningful clinical outcome could also be the subject of future research.

An essential caveat to this discussion is that the pressure of interest is the central arterial pressure, as presumably this is the pressure that affects the vital organs. The major physiological effectors of homeostasis, such as regional autoregulation and neurohormonal mechanisms (eg, the renin–angiotensin system), are influenced by this pressure in feedback loops, and not by the pressure sensed in the peripheral circulatory system.

## Conclusions

Clinically significant femoral–radial pressure gradients can occur in critically ill patients receiving vasoactive agents. This study raises several points of interest for the critical care clinician and highlights the limitations in our understanding of the haemodynamics of critical illness. It is important that we develop more sophisticated models of the cardiovascular system,<sup>23</sup> to enable the development of more compre-

hensive and representative monitoring tools and, in turn, therapies to manage the complex haemodynamic perturbations that confront critically ill patients.

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