higher than LVMEP. The mean bias was thus lower than the 5 mm Hg threshold recommended by the Association for the Advancement of Medical Instrumentation, which is consistent with interchangeability of the two measures.[10] Finally, when systolic arterial pressure is obtained from a radial or femoral arterial catheter in ICU patients, preliminary unpublished data from our laboratory confirm the superiority of CPO_{SAP} over CPO_{MAP} to estimate CPO at bedside. The potential interest of CPO_{SAP} for haemodynamic phenotyping and risk stratification deserves further clinical studies.

Authors' contributions

Study conception/design: MJ, J-LT, DC
Patient recruitment: MJ, DC
Data collection: MJ, DC
Data analysis/interpretation: all authors
Drafting of report: MJ, DC
Writing/approval of final version: all authors
MJ is the guarantor of the content of the article, including the data and analysis.

Declarations of interest

The authors declare that they have no conflicts of interest.

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doi: 10.1016/j.bja.2020.06.010
Advance Access Publication Date: 6 June 2020

Large underestimation of arterial pressure after vasodilator medication overdose

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Keywords: arterial pressure; critical care; femoral-to-radial arterial pressure gradient; norepinephrine; poisoning; vasodilator; vasoplegia

Editor—The femoral-to-radial arterial pressure gradient results in an underestimation of invasive arterial pressure at the radial site compared with the aortic site (usually obtained by femoral arterial line). This phenomenon can lead to inappropriate therapeutic management with excessive vasopressor infusions when based on the radial arterial line pressure. We report a case of femoral-to-radial arterial pressure gradient in a critically ill paediatric patient

self-poisoned with a vasodilator. Her legal representatives signed informed consent for this publication.

A 13-yr-old girl without any underlying medical condition was admitted to the ICU for self-drug poisoning following a family conflict. She weighed 50 kg (BMI of 18 kg m^{-2}), and she orally ingested ramipril 200 mg and amlodipine 150 mg.

Upon admission to the ICU 7 h after ingestion, a 4 cm line was inserted into the right radial artery (SELDICATH® 3F; PRODIMED, Neuilly-en-Thelle, France), which revealed an arterial pressure of 94/45 mm Hg (mean: 60 mm Hg) with a norepinephrine infusion rate of 3.5 μg kg^{-1} min^{-1}. Transthoracic echocardiography revealed a hyperkinetic ventricle with cardiac output up to 5.6 L min^{-1} m^{-2}. Serum lactate was 2.8 mM, and $Scvo_2$ was 75%. Physical examination revealed no mottling, with normal capillary refill time. Electrocardiogram revealed incomplete right bundle branch block with no other abnormalities. She had a normal level of consciousness, no respiratory failure, and Stage 1 acute kidney injury. We administered multiple rescue therapies for refractory vasoplegic syndrome (terlipressin, methylene blue, hydrocortisone, and ascorbic acid) in addition to standard antidote therapy with euglycaemic insulin and calcium salts. Despite these therapies, norepinephrine infusion was increased up to 4.7 μg kg^{-1} min^{-1} to maintain mean arterial pressure >60 mm Hg.

At 12 h after ICU admission (19 h after ingestion), a 16 cm line was inserted in the right femoral artery (PiCCO Catheter 4F; PULSION Medical Systems, Feldkirchen, Germany). The monitor processed both pressure signals (IntelliVue MP70; Philips, Amsterdam, Netherlands) and revealed a femoral-to-radial arterial pressure gradient of 31 mm Hg for mean arterial pressure. Based on femoral mean arterial pressure, norepinephrine infusion was reduced from >4 to <0.3 μg kg^{-1} min^{-1} 3 h after femoral arterial line insertion. A significant femoral-to-radial arterial pressure gradient persisted during the first 6 h after femoral arterial line insertion. Figure 1 shows norepinephrine doses and invasive arterial pressures obtained with both radial and femoral arterial lines during the first day of ICU admission. Norepinephrine was weaned at Day 3, and the patient recovered and was discharged at Day 6 to the paediatric psychiatric ward.

The femoral-to-radial arterial pressure gradient has been described mainly in cardiac surgical patients with multiple risk factors, including pre-existing hypertension, in high-risk patients undergoing long and complex cardiac surgery, and in shorter patients.[1] It has also been described in critically ill patients with sepsis receiving vasopressors and in patients undergoing orthotopic liver transplantation.[2,3] The mechanisms are still poorly understood, and some authors suggest radial arterial diameter reduction induced by vasopressors as a possible mechanism.[4]

This report is the first description of a poisoned patient with a significant femoral-to-radial arterial pressure gradient, which led to inappropriate management before femoral arterial line insertion. We administered multiple rescue medications with potential adverse effects, such as terlipressin and methylene blue, in addition to massive doses of norepinephrine, which may have been prevented by earlier insertion of a femoral arterial line. Although radial arterial line dysfunction could explain the difference, the radial arterial waveform was considered valid during the study period with a dicrotic notch present. Superimposition of the two signals at the end of the first ICU day suggests the absence of radial arterial line dysfunction (Fig. 1). Moreover, we performed multiple pressure transducer calibrations during the study period to exclude baseline drift.

Femoral-to-radial arterial pressure gradient is probably common in the critically ill population. Femoral arterial line insertion should be considered promptly to prevent inappropriate therapeutic management in cases where a high dose of vasopressor is required.

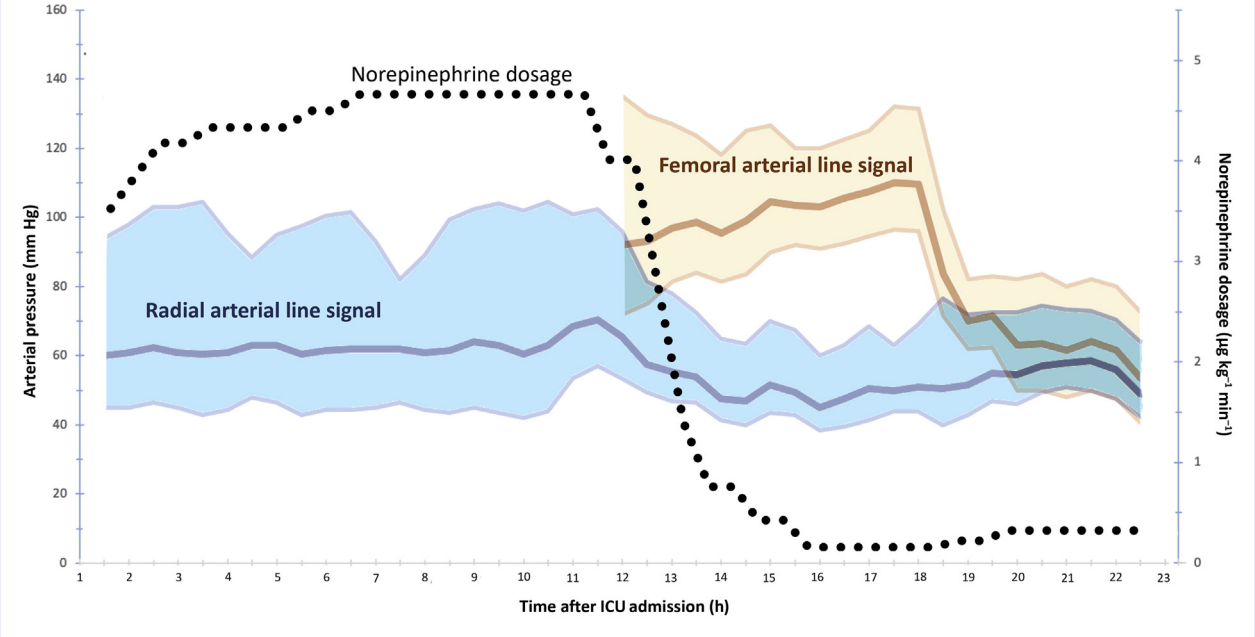

Fig 1. Arterial pressure signals obtained with both lines and norepinephrine dosage during the first ICU day.

Declarations of interest

The authors declare that they have no conflicts of interest.

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doi: 10.1016/j.bja.2020.06.026
Advance Access Publication Date: 9 July 2020

Critical indexed oxygen delivery as a cornerstone of goal-directed perfusion in neonates undergoing cardiac surgery. Comment on Br J Anaesth 2020; 124: 395-402

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Keywords: anaerobic metabolism; cardiac surgery; cardiopulmonary bypass; goal-directed perfusion; lactate; oxygen delivery; paediatric

Editor—We read with great interest the paper by Bojan and colleagues[1] on oxygen delivery during cardiopulmonary bypass (CPB) in neonates. They report a retrospective cohort study on the critical indexed oxygen delivery ($\dot{D}_{O_2}i$) threshold during normothermic CPB in neonates according to serum lactate concentration measured after aortic unclamping (lactOFF). Using lactOFF >2.5 mM to identify anaerobic metabolism, they found that 340 ml min^{-1} m^{-2} is likely to represent the nadir $\dot{D}_{O_2}i$ for maintenance of aerobic metabolism in neonates. A further reduction of 100 ml min^{-1} m^{-2} below the critical $\dot{D}_{O_2}i$ threshold would lead to a 1 mM increment in lactOFF. The results should provide new ideas for optimising perfusion strategy and improving the prognosis in neonatal cardiac surgery with CPB.

Optimal perfusion should maintain microcirculatory and organ function by preserving endothelial function, capillary density, and $\dot{D}_{O_2}$ at the tissue level.[2,3] Thus, $\dot{D}_{O_2}i$ is one of the most important determinants of optimal perfusion during CPB. The minimal safe $\dot{D}_{O_2}i$ during CPB, or critical $\dot{D}_{O_2}i$, is the point when the maximal oxygen extraction is reached, whole-body oxygen consumption ($\dot{V}_{O_2}$) and tissue oxygenation begin to decrease, and anaerobic metabolism and lactic acidosis begin to develop.[3] $\dot{D}_{O_2}i$ is determined by two main variables: haemoglobin concentration and pump flow.[2]

$$\dot{D}O_2i\left(ml\,min^{-1}m^{-2}\right) = pump\ flow\left(L\,min^{-1}\,m^{-2}\right)\Big/ body\ surface\ area\left(m^2\right) \times \Big[1.36 \times haemoglobin\left(g\,L^{-1}\right) \times haemoglobin\ saturation\ (\%) + 0.031 \times partial\ pressure\ of\ arterial\ oxygen\ (mm\ Hg)\Big]$$

Therefore, measuring $\dot{D}_{O_2}i$ should guide the perfusionist in adjusting arterial pump flow according to the haemoglobin concentration and to implement ultrafiltration or red blood cell transfusion.

Maintaining $\dot{D}_{O_2}i$ above the critical value on CPB is vital to improve tissue perfusion and individualise the conduct of bypass to the particular patient, and is the core idea of goal-directed perfusion. This strategy, introduced in recent years, involves aggressive patient management and incorporates continuous monitoring of such oxygen metabolism parameters as $\dot{D}_{O_2}i$, oxygen extraction index, and carbon dioxide production.[4] Goal-directed perfusion could be even more beneficial to early detection of hypoperfusion and anaerobic metabolism during CPB, thus allowing timely and appropriate intervention to ensure adequate or optimal tissue perfusion. Before this report, clinical trials on critical $\dot{D}_{O_2}i$ threshold and goal-directed perfusion approaches focused exclusively on adult cardiac surgery under CPB.[5–10] The landmark goal-directed perfusion studies from Ranucci and colleagues[5,6] showed that a nadir $\dot{D}_{O_2}i$ of 272 ml min^{-1} m^{-2} during CPB was independently associated