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CORRESPONDENCE

Improved estimation of cardiac power output by including pulsatile power

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Editor—The essential function of the heart is the conversion of chemical to mechanical energy to eject blood into the arterial system, and cardiac power is a major aspect of cardiovascular function.[1,2] Cardiac power output (CPO) is the hydraulic energy delivered by the left ventricle (LV) to the systemic circulation per unit time. Cardiac power output is a strong independent predictor of adverse outcomes in heart disease, heart failure, and cardiogenic shock, and it can be included in algorithms for critical care management of these patients.[3–7] CPO can be estimated as the product of the left ventricular mean ejection pressure (LVMEP) times cardiac output (CO) (CPO=LVMEP×CO).[2] In most cases, it is not possible to measure LVMEP precisely, and MAP is most commonly used as a surrogate of LVMEP in the absence of outflow tract obstruction (CPO_{MAP}=MAP×CO).[3] CPO_{MAP} reflects the amount of power that would be required to produce steady non-pulsatile flow.[2] On average, pulsatile power is relatively small (10–15% of CPO),[2] and is thus generally neglected. However, in patients with stiff arteries, this pulsatile component might be significant.[1,2] The aim of our study was to improve the calculation of CPO by taking into account both its steady and pulsatile components. Because systolic aortic pressure (SAP) reflects the combined influences of the steady and pulsatile components of the aortic load,[1,2] we hypothesised that SAP is more closely related to LVMEP than MAP, and that a new equation based on SAP will improve estimation of CPO by taking into account pulsatile load.

We conducted a *post hoc* analysis of our high-fidelity aortic pressure database (n=139).[8] All investigations had been approved by our institution, and informed consent was obtained for all patients.[8] The adult patients prospectively enrolled were free of aortic stenosis or LV outflow tract obstruction. LVMEP was not available in 25 patients, leaving 114 patients in the final analysis (27 with normal cardiac function and coronary angiograms, 46 with hypertension, 17 with grafted heart, 10 with idiopathic dilated cardiomyopathy, and 14 with miscellaneous cardiac diseases). Amongst them, 26 were women and 88 were men, with age ranging from 20 to 74 yr. Stroke volume was calculated from monoplane angiograms (area–length method) or by pulmonary artery thermodilution. Time-averaged MAP, SAP, diastolic aortic pressure (DAP), and pulse pressure (PP=SAP–DAP) were automatically calculated. LVMEP was automatically calculated as the area under the systolic part of the aortic pressure waveform divided by LV ejection time. The pulsatile load was quantified by the PP/MAP ratio. The study population was divided into two. In the first 57 patients (test sample), we documented the relationship between LVMEP and the different aortic pressures (SAP, DAP, and MAP). We found that LVMEP was most strongly related to SAP, and the LVMEP *vs* SAP linear relationship was very close to a proportional relationship, with 0.87 proportionality constant. In the remaining 57 patients (validation sample), we aimed to validate a new CPO equation: CPO=0.87×SAP×CO product. Baseline characteristics (sex and age) of patients from the test and validation samples were similar. Variables were expressed as mean (standard deviation), and CPO estimates were compared using the Bland–Altman method. Regression analyses were also performed. We used MedCalc 11.6.0 software (Mariakerke, Belgium), and P-values <0.05 were considered statistically significant.

In the first 57 patients (test sample), LVMEP was strongly related to both SAP (r=0.97) and MAP (r=0.83) (each P<0.001). The LVMEP/SAP ratio was 0.87 (0.04). In the remaining 57 patients (validation sample), MAP was 116 (18) mm Hg, LVMEP was 139 (20) mm Hg, and the 0.87×SAP product was 142 (22) mm Hg. The CPO_{MAP} underestimated CPO by 16 (7%) (1.90 [0.47] *vs* 2.27 [0.47] W; Fig. 1a). Our new CPO estimate (CPO_{SAP}=0.87×SAP×CO) resulted in a 2 (5%) overestimation of CPO (2.32 [0.51] *vs* 2.27 [0.47] W; Fig. 1b). Unlike CPO_{MAP}, the difference between CPO_{SAP} and CPO was not influenced by PP/MAP (Fig. 1c and d).

Reliable tools for assessment of cardiac function are crucial for the evaluation of prognosis and treatment efficacy. Here, we validated a new estimate of CPO that takes account of the pulsatile power (CPO_{SAP}=0.87×SAP×CO). Unlike CPO_{MAP}, CPO_{SAP} leads to negligible bias and is not influenced by the pulsatile load imposed on the heart. This may provide an accurate and precise characterisation of the true hydraulic power. As recommended for this kind of study, we used a reasonably large sample (i.e. n>85).[9] The main study limitation was that the clinical significance of CPO_{SAP} for assessing prognosis or risk of patients was not tested, and thus deserves further study. Similarly, our new CPO calculation remains to be tested in postoperative care. Moreover, further studies are needed to document the potential impact of major comorbidities on the accuracy and precision of our new CPO calculation.

The first clinical implication is that CPO_{SAP} could be valuable in patients with stiffened arteries (e.g. older or hypertensive patients). Increases in arterial stiffness are expected to increase CPO because of the increased pulsatile component of cardiac power. This potentially detrimental effect will remain unnoticed if we rely only on the classic CPO_{MAP} estimate (MAP×CO), whilst it can be shown if CPO is estimated using CPO_{SAP}. Whilst it is commonly assumed that CPO_{MAP} reflects cardiac energetics, it may in fact underestimate CPO by up to 30%. Second, the new estimate of LVMEP may help simplify estimation of systolic pressure–time index, one of the main haemodynamic determinants of myocardial oxygen supply/demand ratio.[1,2] The 0.87×SAP product was only 3 mm Hg (2%)

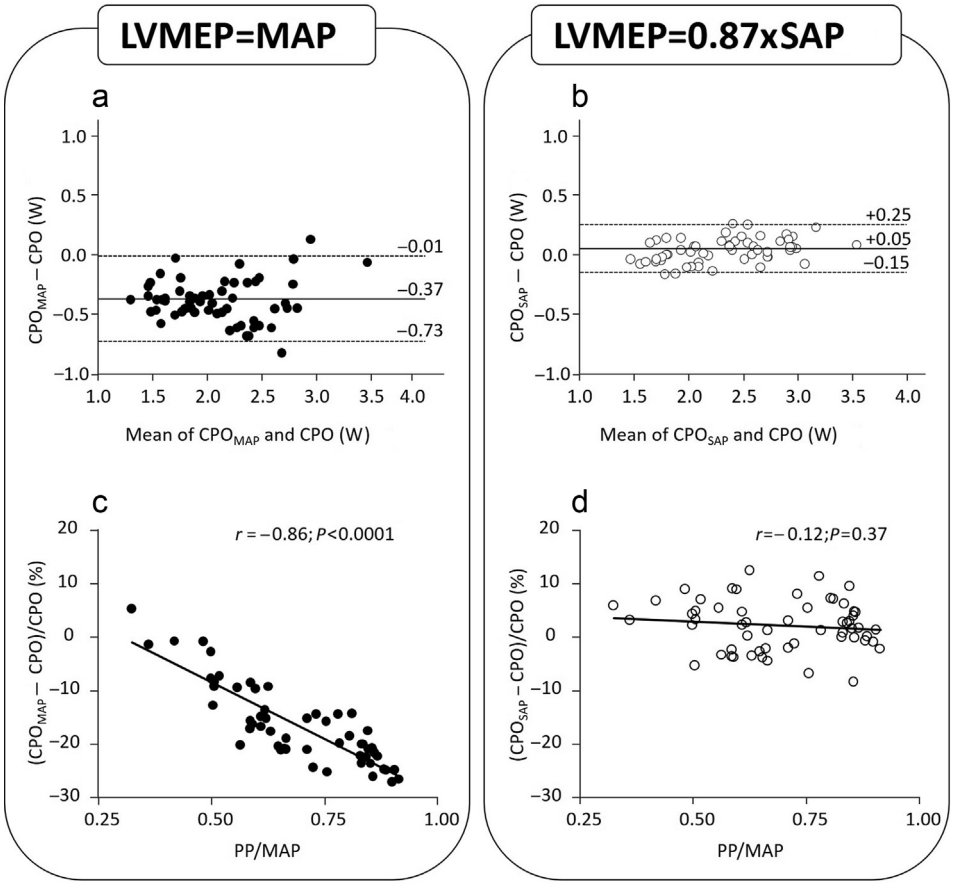

Fig 1. Top: comparison of cardiac power output (CPO=LVMEP×CO) and (a) CPO_{MAP} (MAP×CO) and (b) CPO_{SAP} (0.87×SAP×CO) using the Bland–Altman method in the last 57/114 patients (validation sample). Solid lines: mean bias; dotted lines: limits of agreement (mean [1.96] standard deviation). Bottom: influence of pulsatile load (PP/MAP) (c) on the percentage difference between CPO_{MAP} and CPO (difference [%]=−42.46 PP/MAP+12.82 with 95% confidence interval for the slope [−49.50; −35.66]) and (d) on the percentage difference between CPO_{SAP} and CPO (difference [%]=−3.67 PP/MAP+4.71 with 95% confidence interval for the slope [−11.93; 4.46]) in the last 57/114 patients (validation sample). CO, cardiac output; LVMEP, left ventricular mean ejection pressure; PP, aortic pulse pressure; SAP, systolic aortic pressure.

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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Large underestimation of arterial pressure after vasodilator medication overdose

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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