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ORIGINAL RESEARCH

Evaluation of cardiac output variations with the peripheral pulse pressure to mean arterial pressure ratio

Audrey Tantot[1,2] · Anais Caillard[1,2] · Arthur Le Gall[1,2,3,4] · Joaquim Mateo[1] · Sandrine Millasseau[5] · Alexandre Mebazaa[1,2] · Etienne Gayat[1,2] · Fabrice Vallée[1,2,3,4]

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Abstract
Cardiac output (CO) optimisation during surgery reduces post-operative morbidity. Various methods based on pulse pressure analysis have been developed to overcome difficulties to measure accurate CO variations in standard anaesthetic settings. Several of these methods include, among other parameters, the ratio of pulse pressure to mean arterial pressure (PP/MAP). The aim of this study was to evaluate whether the ratio of radial pulse pressure to mean arterial pressure (ΔPPrad/MAP) could track CO variations (ΔCO) induced by various therapeutic interventions such as fluid infusions and vasopressors boluses [phenylephrine (PE), norepinephrine (NA) or ephedrine (EP)] in the operating room. Trans-oesophageal Doppler signal and pressure waveforms were recorded in patients undergoing neurosurgery. CO and PPrad/MAP were recorded before and after fluid challenges, PE, NA and EP bolus infusions as medically required during their anaesthesia. One hundred and three patients (mean age: 52 ± 12 years old, 38 men) have been included with a total of 636 sets of measurement. During fluids challenges (n = 188), a positive correlation was found between ΔPPrad/MAP and ΔCO (r = 0.22, p = 0.003). After PE (n = 256) and NA (n = 121) boluses, ΔPPrad/MAP positively tracked ΔCO (r = 0.53 and 0.41 respectively, p < 0.001). By contrast, there was no relation between ΔPPrad/MAP and ΔCO after EP boluses (r = 0.10, p = 0.39). ΔPPrad/MAP tracked ΔCO variations during PE and NA vasopressor challenges. However, after positive fluid challenge or EP boluses, ΔPPrad/MAP was not as performant to track ΔCO which could make the use of this ratio difficult in current clinical practice.

Keywords Cardiac output · Pressure pulse analysis · Vasopressor challenges · Pulse pressure · Mean arterial pressure · PP/MAP

1 Introduction

Cardiac output (CO) monitoring is a leading component of hemodynamic assessment in operating theatre and his optimisation during surgery is associated with a significant decrease in mortality of high-risk surgical patients [1–3].

✉ Audrey Tantot
audrey.tantot@wanadoo.fr

1 Department of Anesthesiology and Critical Care, Hôpitaux Universitaires Saint-Louis-Lariboisière, Assistance Publique - Hôpitaux de Paris, Université Paris Diderot, 2 rue Ambroise-Paris, 75475 Paris Cedex 10, France

2 Inserm, UMRS-942, Paris Diderot University, Paris, France

3 MΞDISIM, Inria Paris-Saclay, Palaiseau, France

4 LMS, Ecole Polytechnique, Palaiseau, France

5 Pulse Wave Consulting, Saint Leu La Foret, France

The two most frequently used CO monitoring in operating theatres are oesophageal Doppler (DCO) and Pulse Pressure Analysis (PPA). DCO is accurate but requires access to the oesophagus that might be not possible in some oral or neurological surgery settings and also relies on frequent manual adjustments to obtain optimal signal.

The existence of Pulse pressure analysis (PPA) technology has existed for many years now, but the introduction of this method for estimating CO has been greatly developed over the last decade in clinical practice. This method is based on the estimation of CO from arterial pressure waveform using the three-element Windkessel model: Pulse pressure (PP) variations are related to the interactions between stroke volume (SV) and arterial properties [4]. Therefore, for reliable CO assessment, this method requires accurate estimation of arterial load. However, a complete and precise description of arterial load remains particularly complex in current routine clinical practice [5, 6]. If individual arterial

load is estimated by external or internal calibration on different PPA devices, recent studies provided evidences that vasopressor therapy could induce changes in arterial load and largely affect the reliability of these PPA-based CO estimation [7–10]. Monge et al. have indeed shown that vasopressor infusion induced changes of arterial elastance (E_a) and total arterial compliance (C) which particularly affected the agreement between estimated CO from PPA (PPCO) and oesophageal Doppler (DCO) [10].

Nevertheless, some recent studies pointed out that CO variations (ΔCO) estimated by an algorithm first described by Liljestrand Zander in 1928, had a greater agreement with DCO during arterial load changes induced by vasopressors than from PPA [11, 12]. Interestingly, this algorithm contains the ratio of pulse pressure to mean arterial pressure (PP/MAP). Pulse pressure, the pulsatile component of the pressure waveform is often used as an estimation of arterial stiffness while MAP, the steady component is related to vascular tone. The introduction of MAP in the formula was suggested to overcome the nonlinearity of the arterial system, as PP value depend on both stroke volume (SV) but also MAP level. Thus, the ratio PP/MAP might be related to the global arterial loads of the system and could provide an acceptable estimation of CO during acute changes induced by vasopressors. The aim of this study was hence to evaluate whether PPrad/MAP variations (ΔPPrad/MAP) could track CO variations (ΔCO) induced by therapeutic interventions such as fluid infusions or vasopressors boluses [phenylephrine (PE), norepinephrine (NA) or ephedrine (EP)] in the operating room.

2 Materials and methods

2.1 Patients

A prospective, observational study was conducted from November 2011 to July 2012 in the Department of Anaesthesiology at Lariboisiere University Hospital (Paris, France). Oral informed consent was obtained for each patient and the investigation approved by the institutional research ethics committee (Comité d'Ethique de la Société de Réanimation de Langue Française No.11–356). Consecutive neurosurgical patients involving hemodynamic monitoring by invasive blood pressure and DCO were enrolled in this study. Neurosurgical patients with reduced cerebral compliance and/or potential bleeding in whom haemodynamic monitoring, including invasive blood pressure, DCO, and therapeutic challenges represented the usual care in our institution were included in the study. Underage (< 18 years), pregnant patients and patients with chronic cardiac arrhythmia or any contraindication to the use of DCO were excluded.

2.2 Study protocol

General anaesthesia was induced and maintained with propofol and remifentanil target controlled infusion. All patients were intubated and mechanically ventilated with a tidal volume of 7 ml kg^{-1}, positive end expiratory pressure of 5 cm H_2O, at a frequency of 12–16 breaths per minute to keep the end tidal CO_2 between 35 and 38 mmHg. After induction of anaesthesia, DCP probe (I_2P, Deltex Medical, UK) was inserted in the oesophagus and a radial arterial line (SAC 00520 20GA, Arrow International, Reading, PA, UA) in the left arm.

For all patients, baseline measurement of MAP was taken from the cuff monitor at steady state before the induction of general anaesthesia and was considered as the reference value. Following our standard of care procedures, a decrease of 20% from this reference MAP led to a therapeutic intervention. The physician in charge could choose between volume expansion or vasopressor infusion. In case of clinical hypovolemia defined by a significant decrease in CO associated with surgical haemorrhage, a volume expansion was realized before any vasopressor infusion (250 ml of saline in 10 min). Positive fluid challenge was defined by an increase in CO of at least 10% after volume expansion.

If MAP was not restored after volume expansion, or in case of general anaesthesia-induced arterial hypotension, a bolus of vasoconstrictor was administered according to the choice of the attending physician. Three different vasopressors were available: bolus of 50 mcg of phenylephrine (PE), 5 mcg of norepinephrine (NA) or 9 mg of ephedrine (EPH). Repeated boluses could be delivered to achieve the MAP goal. However, in case of multiple boluses in a short interval, we analysed only the first bolus if the delay between the first and the second bolus was less than 5 min to try to eliminate the confounding factors such as synergism between the drugs and repetitive boluses.

2.3 Hemodynamic monitoring

Hemodynamic variables were continuously recorded using an invasive arterial radial line, (SAC 00520 20GA, Arrow International, Reading, PA, UA) and an oesophageal Doppler probe inserted after the induction of the general anaesthesia and both connected to the Combi Q® monitor (Deltex Medical, UK). After radial insertion, the radial line was zeroed to atmospheric pressure and the arterial pressure waveform was carefully checked using a fast-flush test to ensure optimal invasive radial waveform quality. Before and after the end of volume expansion or at the highest arterial pressure after vasopressor injection, a set of hemodynamic measurements was recorded with Combi Q® monitor.

Radial systolic pressure (SAP_{rad}), diastolic pressure (DAP_{rad}), mean arterial pressure (MAP_{rad}) and radial pulse pressure (PP_{rad} = SAP−DAP) were recorded. The PP_{rad} and the ratio PP_{rad}/MAP were manually calculated and averaged from three consecutive heart beats.

Heart rate (HR), stroke volume (SV) and cardiac output (CO) was measured from the DCO after adjusting the position of the probe in order to obtain the highest quality of velocity complexes.

2.4 Statistical analysis

Data are expressed as mean ± standard deviation or as median and interquartile range (IQR), as appropriate. Comparisons between values recorded before and after therapeutic interventions (volume expansion or vasopressor bolus injection) were tested by using a paired Wilcoxon analysis. Changes before and after therapeutic intervention were expressed as % changes from baseline. As several therapeutic interventions were conducted per patients, data were averaged and weighted depending on the number of measurements performed. Meta regression results were expressed as slope and 95% confidence interval.

Vasopressor subgroups were then defined as PE, NA and EP subgroups. Haemodynamic parameters were compared between subgroups by using a Mann Whitney U test.

The ability to track ΔCO changes with ΔPP_{rad} and ΔPP_{rad}/PAM variations was analysed by using the Spearman correlation for non-parametric data and completed by a concordance analysis. Percentage of the concordance was defined using the percentage of weighted data set in which the direction of change was in agreement in four-quadrant plots. We arbitrary chose 5% for defining the exclusion area in concordance analysis as previously described [13].

A p value < 0.01 was considered significant. Statistical analysis was performed using GraphPad Prism 6.0™ software (GraphPad Software Inc., San Diego, CA, USA).

3 Results

One hundred and three patients were included in this study. Patient's characteristics are summarized in Table 1.

In total, 636 sets of measurements were performed, including 448 vasopressor bolus injections and 188 volume expansions.

Table 1 Characteristics of patients

All patients (n = 103)	
Male n (%)	38 (37)
Age (years)	52 (40–64)
BMI (kg/m^2)	25 (21–28)
ASA n (%)	
1	25 (24)
2	65 (63)
3	13 (13)
Comorbidities	
Arterial hypertension n (%)	27 (26)
Mellitus diabetes n (%)	4 (4)
Coronaropathy n (%)	4 (4)
Renal dysfunction n (%)	0 (0)
COPD n (%)	9 (9)
Procedure	
Intra cranial surgery n (%)	75 (73)
Extra cranial surgery n (%)	28 (27)
Duration of surgery (min)	330 (240–440)
Duration of anesthesia (min)	400 (309–510)
Sets of measurements	n = 636
Vasopressors (n = 448)	
Phenylephrine	256 (57)
Norepinephrine	121 (27)
Ephedrine	71 (16)
Fluid challenges n = 188	
NaCl 0.9%	188

3.1 Correlations of ΔCO after therapeutic interventions with changes in PP_{rad} and PP_{rad}/MAP ratio

3.1.1 All interventions

A total of 636 sets of measurements were performed. Therapeutic interventions led to an increase in MAP (from 70 to 81 mmHg, p < 0.001). In the same time, CO and SV decreased (from 4.9 to 4.2 l min^{-1}, and from 73 to 66 ml, respectively, p < 0.001). ΔPP_{rad}/MAP followed both ΔCO and ΔSV and decreased from 0.64 to 0.62 (p < 0.0001). PP_{rad} varied in the opposite direction and increased from 45 to 50 mmHg (p < 0.001) (Table 2a).

3.1.2 During fluid administration

One hundred and eighty-eight fluid challenges were administered. Sixty-six (35%) of those fluid challenges induced an increase in CO of at least 10% and were hence classified as positive fluid challenge. Overall, during fluid administration, MAP increased from 73 to 75 mmHg (p = 0.07) while CO increased from 4.9 to 5.2 l min^{-1},

Table 2 Variations of haemodynamic parameters (median [interquartile range]) after all therapeutic intervention and fluid challenge (a) and after vasopressor bolus injections (b)

(a)

	All therapeutic interventions n = 636		Fluid challenge n = 188		Positive fluid challenge n = 66	
	Baseline	Bolus	Baseline	Bolus	Baseline	Bolus
MAP (mmHg)	70 (65–76)	81 (74–89)**	73 (68–80)	75 (68–81)	73 (69–80)	73 (67–80)
HR (bt/min)	69 (60–78)	65 (57–74)**	65 (58–73)	64 (59–73)	62 (54–74)	64 (57–74)*
CO (l/min)	4.9 (4–6)	4.2 (3.4–5.5)	4.9 (3.5–6)	5.2 (3.7–6.2)**	3.9 (2.8–5.3)	4.8 (3.7–6.4)**
SV (ml)	73 (59–87)	66 (53–81)**	73 (57–88)	78 (61–90)**	62 (51–78)	72 (61–90)**
PPrad (mmHg)	45 (39–51)	50 (43–58)**	48 (42–54)	49 (43–55)**	44 (41–49)	47 (42–52)
PPrad/MAP	0.64 (0.54–0.74)	0.62 (0.53–0.72)**	0.64 (0.55–0.73)	0.65 (0.57–0.75)**	0.61 (0.52–0.68)	0.65 (0.57–0.70)**

(b)

	All vasopressors n = 448		Phenylephrine n = 256		Norepinephrine n = 121		Ephedrine n = 71	
	Baseline	Bolus	Baseline	Bolus	Baseline	Bolus	Baseline	Bolus
MAP (mmHg)	69 (64–73)	83 (78–90)**	69 (64–76)	84 (61–92)**	69 (64–73)	82 (77–88)**	67 (62–77)	82 (73–88)**
HR (bt/min)	70 (61–80)	65 (56–75)**	68 (59–75)	61 (54–69)**	72 (64–80)	67 (59–76)**	72 (62–81)	74 (64–85)**
CO (l/min)	4.9 (4.2–6)	4 (3.3–5.1)**	4.8(3.9–5.9)	3.8 (3–4.6)**	4.8 (4.2–6)	3.9 (3.2–4.5)**	5.5 (4.5–7.1)	5.8 (4.8–7.6)
SV (ml)	74 (61–86)	63 (51–77)**	73 (60–86)	62 (52–74)**	73 (57–84)	59 (48–75)**	82 (7–96)	84 (67–99)
PPrad (mmHg)	44 (38–50)	51 (43–59)**	45 (39–52)	51 (44–59)**	43 (36–48)	49 (42–55)**	44 (39–50)	56 (49–66)**
PPrad/MAP	0.63 (0.54–0.74)	0.61 (0.51–0.70)**	0.63 (0.55–0.75)	0.61 (0.52–0.70)**	0.62 (0.49–0.72)	0.59 (0.50–0.67)**	0.66 (0.55–0.77)	0.71 (0.57–0.78)**

*p < 0.01; **p < 0.001 compared to baseline

p < 0.001 (Table 2a). A small positive correlation was found between ΔCO and ΔPP_{rad}/MAP (Fig. 1, left panel, r = 0.22 (0.07–0.35), p = 0.003). By contrast, no correlation was observed between ΔPP_{rad} and ΔCO. Those results were similar when considering only positive fluid challenge (Table 2a).

3.1.3 During vasopressor administration

During vasopressor bolus injections (n = 448), MAP increased from 69 (64–73) to 83 (78–90) mmHg, p < 0.001 (Table 2b) while CO decreased from 4.9 l min^{-1} (4.2–6) to 4 l min^{-1} (3.3–5) (p < 0.001) (Table 2b). PP_{rad} varied in the opposing direction than ΔCO in 84% of the cases and increased from 44 (38–50) mmHg to 51 (43–59) mmHg (p < 0.001, Table 2b).

By contrast, ΔPP_{rad}/MAP followed ΔCO with a decrease from 0.63 (0.54–0.74) to 0.61 (0.51–0.70) (p < 0.001, Fig. 1, right panel). ΔPP_{rad}/MAP were positively correlated to ΔCO (r = 0.53, p < 0.001) with 69% of concordance. An association of PP_{rad}/MAP percentage variation after CO changes was found: a 1% ΔCO led to a 0.5% ΔPP_{rad}/MAP (Fig. 1).

3.2 Impact of the type of vasopressor: phenylephrine, norepinephrine and ephedrine

Hemodynamic variables analysis was assessed in the three subgroups of vasopressors: PE (n = 256), NA (n = 121), and EP (n = 71) (Table 2b).

All vasopressor bolus induced an increase in MAP [16 (10–22) mmHg, 12 (9–16) mmHg and 14 (11–19) mmHg for PE, NA and EP subgroups respectively, p < 0,001].

PE and NA infusions induced a reduction in CO (ΔCO = − 1.14 (− 1.67 to − 0.70) l min^{-1} and − 0.7 (− 1.15 to 0.45) l min^{-1}, p < 0.001) while EP led to an increase in CO (ΔCO = + 0.2 (− 0.3 to + 0.72) l min^{-1}). PP_{rad}/MAP followed the same trend, with a reduction of − 0.04 (− 0.09 to 0.02) and − 0.01 (− 0.06 to 0.02), after PE and NA respectively (p < 0.001) and an increase after EP [+ 0.02 (− 0.012 to 0.064), p < 0,001]. Significant correlations were observed between ΔCO and ΔPP_{rad}/MAP during PE (r = 0.53, p < 0.001) and NA (r = 0.41, p < 0.001) infusions (Fig. 2a, b). In these two subgroups, PP_{rad}/MAP variations predicted CO changes with a concordance percentage of almost 80%. However, no significant correlation was observed between ΔPP_{rad}/MAP and ΔCO after EP (r = 0.10, p = 0.39, Fig. 2c).

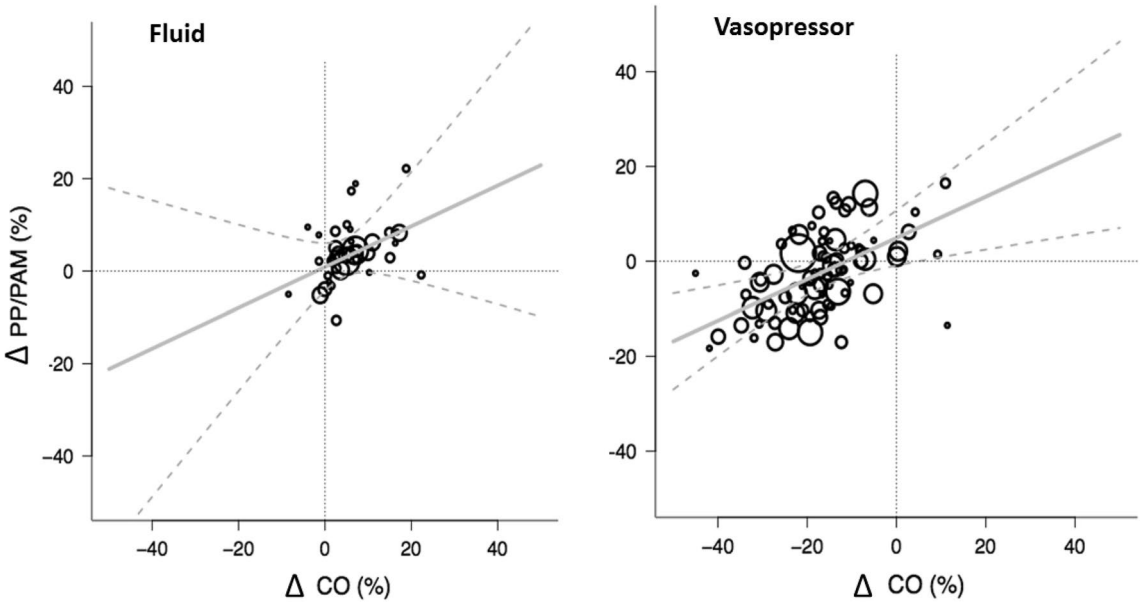

Fig. 1 Correlations between PPrad/MAP with CO changes after fluid and vasopressor bolus injection. Weighted data are shown on correlation graphs, the size of points is related with the number of measurements per patient

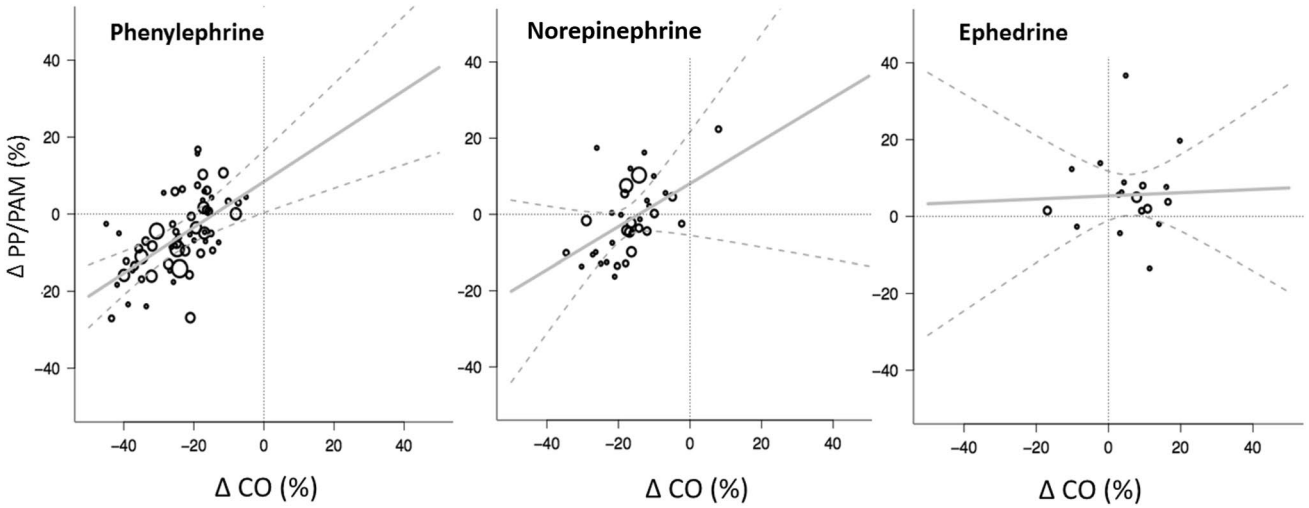

Fig. 2 Correlations between PPrad/MAP changes with CO changes after bolus injections of Phenylphrine (left), Norepinephrine (middle) and Ephedrine (right). Weighted data are shown on correlation graphs, the size of points is related with the number of measurements per patient

After PE and NA vasopressors, a 1% CO variation led to a change of 0.5% in PP_{rad}/MAP.

4 Discussion

Our study shows that: (1) variations in PP_{rad}/MAP ratio correlated with CO variations after PE and NA boluses but was not as performant after EP bolus or fluid challenge; (2) variations in ΔPP_{rad}/MAP performed better than variations in PP_{rad} alone.

PPA is an attractive method to monitor changes in CO during surgery and anaesthesia. The Windkessel model describes the pressure waveform diastolic decay in terms of compliance and vascular resistance and hence afterload. However many PPA algorithms including algorithms based on the Windkessel model have difficulties to track CO during vasopressor administration. Meng et al. showed that while PPA could track changes after tilt-test, it was not reliable

after PE boluses with many false positive and true negative [14]. Caillard et al. confirmed this finding and identified the Liljestrand-Zander model as the best model to track change during vasopressors challenges [12]. This model takes into consideration vessel compliance (also estimated with PP) and continuous pressures variations (MAP). For this reason, we looked to the variation of the PP_{rad}/MAP ratio during fluid challenges and vasopressors challenges.

Our findings showed that the agreement between ΔPPrad/MAP and ΔCO depends on the type of vasopressor used. PE and NA mainly act on alpha receptors as a vessel vasoconstrictor. Both PPrad and MAP increased after PE and NA boluses. However there was a reduction in PPrad/MAP ratio and PPrad/MAP ratio accurately tracked CO in 80% of the cases. PP_{rad}/MAP ratio could accurately track CO variations induced by PE and NA vasopressors better than PP_{rad} alone as it also take into account arterial peripheral resistance changes.

Ephedrine (EP) is a sympathomimetic amine that increases norepinephrine activity of beta-adrenergic receptors, leading to more important inotropic changes than effects on peripheral resistance. Therefore, PP_{rad}/MAP ratio could not accurately track CO changes after EP boluses.

Our results are in agreement with some recent studies that pointed out the poor agreement between Windkessel algorithm using PP changes alone and CO variations, as opposed to the ratio PP/MAP derived from the Liljestrand Zander model [11, 12].

Almost 65% of the fluid challenges were negative (= no CO improvement) in our study. On the remaining positive fluid-responders, fluid administration led to a significant discrepancy in the direction of ΔPP_{rad}/MAP and ΔCO variations. Clinical relevance of PP_{rad}/MAP ratio in tracking CO variations during fluid challenge need more investigations [12].

4.1 Limitations of the study

The main limitation of our study resided in the assessment of pulse pressure. Indeed PP was recorded invasively at the radial level and thus it was a measure of peripheral pulse pressure and might not represent aortic pulse pressure. Pulse pressure waveforms analysis are mostly studied from aortic pressure waveforms or central pressure derived from radial pressure waveforms using a generalized transfer function [15]. Using the PPaortic/MAP ratio might have led to different results. Second, our population is highly selected and includes only neurosurgery patients. Our results must be confirmed in other populations such as septic and/or circulatory failure patients. Furthermore, we analysed the whole population of this study without considering age or hypertensive history, which both contribute to high arterial load and increased arterial stiffness [16, 17]. This might influence the response of PPrad/MAP ratio to vasopressors. We used DCO as our reference technique, which does not represent the usual gold standard method and the question of aortic diameter measurement is still a debate [18].. Nevertheless, the accuracy of DCO in measuring CO has been widely demonstrated [6]. Finally, clinical application of PP_{rad}/MAP must be partially offset by the relatively small variations: 1% CO variations induced barely 0,5% PP_{rad}/MAP. This restricted percentage of variation could prove difficult to assess CO changes in current clinical practice unless a fixed multiplying factor is used to provides higher numerical changes. These limitations have to be kept in mind when using the PP_{rad}/MAP to track acute changes in CO induce by vasopressors.

5 Conclusion

Variations of the PPrad/MAP ratio could accurately track CO variations during PE and NA vasopressor challenges and PPrad/MAP ratio performs better than PP_{rad} alone. However, it did not perform so well to track CO changes after EP or fluid challenges. Use of the PPrad/MAP ratio to assess CO changes in current clinical practice might hence be difficult.

Compliance with ethical standards

Conflict of interest Alexandre Mebazaa has received speaker honoraria from Abbott, Novartis, Orion, Roche and Servier and fee as member of advisory board and/or steering committee from Cardiorentis, Adrenomed, MyCartis, ZS Pharma and Critical Diagnostics. Etienne Gayat has received consulting fees from Magnisense, research support from Sphingotec, Deltex Medical and Retia Medical. Fabrice Vallée has received research support from Radiometer, Deltex Medical and Retia Medical. Sandrine Millasseau has received consulting fees from Alam Medical, AtCor Medical, Mesi Medical and Omron.

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