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Can changes in arterial pressure be used to detect changes in cardiac index during fluid challenge in patients with septic shock?

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Abstract *Purpose:* Response to fluid challenge is often defined as an increase in cardiac index (CI) of more than 10–15%. However, in clinical practice CI values are often not available. We evaluated whether changes in mean arterial pressure (MAP) correlate with changes in CI after fluid challenge in patients with septic shock. *Methods:* This was an observational study in which we reviewed prospectively collected data from 51 septic shock patients in whom complete hemodynamic measurements had been obtained before and after a fluid challenge with 1,000 ml crystalloid (Hartman's solution) or 500 ml colloid (hydroxyethyl starch 6%). CI was measured using thermodilution. Patients were divided into two groups (responders and non-responders) according to their change in CI (responders: %CI >10%) after the fluid challenge. Statistical analysis was performed using a two-way analysis of variance test followed by a Student's *t* test with adjustment for multiple comparisons. Pearson's correlation and receiver operating characteristic curve analysis were also used. *Results:* Mean patient age was 67 ± 17 years and mean Sequential Organ Failure Assessment (SOFA) upon admittance to the intensive care unit was 10 ± 3. In the 25 responders, MAP increased from 69 ± 9 to 77 ± 9 mmHg, pulse pressure (PP) increased from 59 ± 15 to 67 ± 16, and CI increased from 2.8 ± 0.8 to 3.4 ± 0.9 L/min/m^2 (all $p < 0.001$). There were no significant correlations between the changes in MAP, PP, and CI. *Conclusions:* Changes in MAP do not reliably track changes in CI after fluid challenge in patients with septic shock and, consequently, should be interpreted carefully when evaluating the response to fluid challenge in such patients.

Keywords Sepsis · Vascular resistance · Intensive care unit

Introduction

Fluid resuscitation is essential for the restoration and maintenance of adequate intravascular volume and organ perfusion. Fluid challenge, in the context of a provocative test in which an amount of fluid is administered over a defined interval under close monitoring, has been proposed as a useful tool to evaluate the response to fluids in patients with suspected hypovolemia [1]. This technique, originally introduced many years ago [2], is still considered to be part of standard practice in the evaluation and management of septic patients [3, 4]. In addition, even if fluid responsiveness can be predicted by various techniques [5], it is still important to evaluate whether the patient actually

responds to fluids, especially when the variable used to predict fluid response is close to the cut-off value.

The percentage change in cardiac index (CI) following fluid administration has been used to discriminate responders and non-responders to fluid challenge [6, 7]. However, in routine clinical practice, CI is often not measured because invasive methods (i.e., thermodilution technique) are not indicated [8], and less invasive techniques, such as echocardiography and pulse contour methods, are not always available. Consequently, other hemodynamic variables are frequently used to define responders to fluid challenge, with a satisfactory mean arterial pressure (MAP) often a key target, especially in hemodynamically unstable patients [9]. However, whether MAP, compared to CI, can effectively guide fluid administration and be used to discriminate fluid responders and non-responders has not yet been demonstrated. Particularly in patients with septic shock, significant changes in flow may lead to minimal changes in blood pressure because of the low vascular resistance. The aim of this study was, therefore, to assess whether changes in MAP correlated with changes in CI after fluid challenge in patients with septic shock.

Methods

In this observational study, we reviewed prospectively collected data from patients who had been admitted to our 34-bed university hospital mixed medical–surgical ICU between January 2006 and December 2009. Approval was obtained from the Ethics Committee of Erasme Hospital, and informed consent was waived due to the purely observational nature of the study. All adult patients with septic shock as defined by standard criteria [10] who had received a fluid challenge during their stay in the intensive care unit (ICU) and had been monitored with an arterial catheter and a pulmonary artery catheter (Swan Ganz catheter, Edwards Lifesciences, Irvine, CA) and who had complete hemodynamic profiles recorded in our computerized database were included in the study. Patients with acute coronary syndromes or a history of cardiac disease were not included. The CI was determined by the continuous thermodilution technique (Vigilance, Edwards Lifesciences). Mixed venous O_2 saturation (SvO_2) was measured continuously using a co-oximeter.

For the fluid challenge, crystalloids (Hartman's solution; Baxter, Lessines, Belgium) or synthetic colloids [hydroxyethyl starch (HES) 6%; Voluven, Fresenius, Bad Homburg, Germany) were administered at a rate of 1,000 ml/30 min or 500 ml/30 min, respectively. The choice of type of fluid and the decision to stop the fluid challenge was made by the treating physician according to predetermined safety limits for each patient. No change in catecholamine administration or any other therapeutic intervention was allowed during the fluid challenge.

Demographics, type of fluid used for the fluid challenge test, type of patient (surgical or medical), and clinical data concerning treatments (mechanical ventilation, inotropic agents) were collected for each patient. Arterial pressures, central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), heart rate (HR), CI, systemic vascular resistance (SVR), pulse pressure (PP), and SvO_2 values were recorded at baseline and at the end of the fluid challenge. Relative changes in CI (%CI) and MAP (%MAP) were calculated, and fluid responders were defined as those patients who showed a $>10\%$ increase in CI.

Statistical analysis

Statistical analysis was performed with SPSS software (SPSS, Chicago, IL). A two-way analysis of variance test followed by a Student's t test with adjustment for multiple comparisons were used for continuous variables. Pearson's correlation was applied. Receiver operating characteristic (ROC) curve analysis was used to assess the effectiveness of MAP, systolic and diastolic arterial pressure, and PP as indicators of fluid responsiveness. Statistical significance was defined as $p < 0.05$.

Results

Fifty-one patients met our entry criteria, and their demographic characteristics are shown in Table 1. Patients had a mean age (±standard deviation) of 67 ± 17 years and a mean Sequential Organ Failure Assessment (SOFA) score on admittance to the ICU of 10 ± 3. Twenty-five of the patients (49%) responded to the fluid challenge according to the predetermined criterion (%CI >10%). There were no significant differences between the groups in terms of types of fluid administered, with crystalloid being the most commonly used fluid.

The hemodynamic data of responders and non-responders, both before and after fluid challenge, are presented in Table 2 [and in Electronic Supplementary Material (ESM) Tables 1 and 2 according to type of fluid]. CVP and PAOP increased in both groups, CI, SvO_2, PP, and MAP increased significantly only in the responders, and HR and SVR decreased significantly only in the responders. Among the group of responders, patients with a low baseline MAP (<70 mmHg) had a greater increase in MAP than patients with a high initial MAP; non-responders did not show this difference in MAP response to fluid (Table 3).

Table 1 Demographic characteristics of the patients

Demographic characteristics	Responders	Non-responders
Number of patients	25	26
Age (years)[a]	69 ± 15	66 ± 19
Male (*n*)	17	15
Mechanical ventilation (*n*)	23	26
Type of patient (*n*)		
Medical	13	18
Surgical	12	8
Types of inotrope, *n* (%)		
Norepinephrine	15 (60)	18 (69)
Dobutamine	14 (56)	11 (42)
Dopamine	14 (56)	8 (30)
Type of fluid, *n* (%)		
Hartman's solution	14 (56)	15 (58)
HES solution	11 (44)	11 (42)
SOFA score[a]	11 ± 3	10 ± 3
Mortality, *n* (%)	13 (52)	11 (42%)

HES hydroxyethyl starch, *SOFA* Sequential Organ Failure Assessment

[a] Mean ± standard deviation (SD)

There was no significant correlation between the %CI and %MAP ($r^2 = 0.07$) for all patients or when just the responders were considered ($r^2 = 0.06$) (Fig. 1). This result did not change when the absolute changes in CI and MAP were evaluated (Fig. 2). Changes in MAP could not identify responders to fluid challenge [%MAP: area under the concentration–time curve (AUC) 0.624, 95% CI 0.480–0.767, $p = 0.09$; absolute change in MAP: AUC 0.381, 95% CI 0.235–0.524, $p = 0.12$) (ESM Figs. 1 and 2). There was no significant correlation between the %CI ($r^2 = 0.02$) and the relative change in stroke volume (%SV, $r^2 = 0.07$) or between the %SV and the relative change in PP (%PP) (Figs. 3, 4). Similarly the %PP could not differentiate CI responders and non-responders (AUC 0.618, 95% CI 0.474–0.761, $p = 0.113$) (ESM Table 3). Changes in systolic or diastolic arterial pressures could not predict changes in CI (areas under the ROC curves shown in ESM Table 3). Finally, the results were similar when we used a cut-off for CI change of >15% to define fluid responsiveness (ESM Table 4).

Discussion

Our study shows the lack of correlation between changes in arterial pressure and CI after fluid challenge in patients with septic shock. If the SVR remains stable, changes in arterial pressure should parallel changes in CI. Experimentally, when the inferior vena cava is transiently occluded, rapid changes in CI correlate linearly with the %MAP [11]. In our study, even though there was a significant increase in MAP only in those patients with an increase in their CI (responders), the increases in MAP and CI were not correlated.

In contrast to our findings, a recent study by Monnet et al. reported a statistically significant correlation between %MAP and %PP and percentage cardiac output (%CO) after fluid challenge ($r = 0.52$, $p < 0.001$ and $r = 0.56$, $p < 0.001$, respectively) [12]. However, 15% of the patient population of that study suffered shock from causes other than sepsis, whereas we included only patients with septic shock; consequently, the two patient populations differ slightly. The method of measuring the CO was also different, and it is known that changes in CO may vary according to the measurement technique used [13]. Moreover, the correlation between changes in arterial pressure and %CO observed by Monnet and colleagues was very weak. In addition, similar to our study, %MAP, %SAP, and %DAP were neither sensitive nor specific indicators of fluid responsiveness, while %PP had a specificity of only 85% and even lower sensitivity.

Changes in MAP may be affected by changes in vascular tone. In our study, there was a significant decrease in SVR in the responders, indicating a relaxation of vasopressor mechanisms. In a previous study by Monnet et al. [14], the diameter of the descending aorta increased significantly (>15%) in responders to fluid challenge, suggesting a tension effect on the baroreceptors located in the wall of the aortic arch. As septic shock may be associated with downregulation of the sympathetic system [15], arterial baroreceptors may be relatively more sensitive to an abrupt increase in arterial blood pressure, and their effect on vascular tone may be more marked. Interestingly, we found a greater increase in MAP in the responders to fluid challenge who had lower initial values (<70 mmHg) than in those with higher initial values despite the same change in CI (Table 3). This observation is important when CI is measured with devices requiring an indicator dilution CO measurement to calibrate the system, as re-calibration after the fluid challenge is then necessary [16, 17]. Additionally, the changes in MAP may be more valuable as an indicator of response to fluid challenge in patients with hypotension than in patients with normal or high MAP.

The capacity of the vascular system to increase MAP may also affect the response to fluid challenge [18]. In a recent study by Monge Garcia et al., the dynamic arterial elastance measured by the ratio of PP variation/SV variation prior to fluid challenge was found to be a highly sensitive and specific index to predict increases in MAP of >15% [19]. We did not measure SV variation and therefore were unable to calculate this index. However, the changes in vascular resistance that we observed during fluid challenge may make this index less efficient. Indeed, Monge Garcia et al. [19] did not find any changes in SVR before and after fluid challenge and, therefore, there was a strong correlation between changes in PP and changes in SV during the fluid challenge ($r^2 = 0.79$); in contrast, we observed no such correlation in our study (Fig. 4). This discrepancy between these two studies may again be

Table 2 Hemodynamic parameters in responders and non-responders before and after fluid challenge

Hemodynamic parameters	Before[a]	After[a]	*p* value	ANOVA[b]
MAP (mmHg)				
Responders	69 ± 9	77 ± 9	<0.01	0.12
Non-responders	71 ± 9	74 ± 9	0.13	
Pulse pressure (mmHg)				
Responders	59 ± 15	67 ± 16	<0.01	0.26
Non-responders	57 ± 17	59 ± 10	0.42	
Heart rate (b/min)				
Responders	103 ± 17	99 ± 16	0.03	0.99
Non-responders	106 ± 21	102 ± 22	0.14	
CVP (mmHg)				
Responders	10 ± 3	13 ± 5	<0.01	0.49
Non-responders	12 ± 4	14 ± 4	<0.01	
PAOP (mmHg)				
Responders	12 ± 3	16 ± 5	<0.01	0.53
Non-responders	14 ± 3	17 ± 6	<0.01	
SvO_2				
Responders	66 ± 8	69 ± 7	0.01	0.19
Non-responders	66 ± 3	66 ± 6	0.67	
Cardiac index (l/min/m^2)				
Responders	2.8 ± 0.8	3.4 ± 0.9	<0.01	0.12
Non-responders	3.1 ± 0.9	3.1 ± 1.0	0.38	
SVR (dynes.s.cm^{-5})				
Responders	1,011 ± 144	865 ± 288	0.01	0.12
Non-responders	902 ± 286	905 ± 326	0.88	

MAP Mean arterial pressure, *CVP* central venous pressure, *PAOP* pulmonary arterial occlusion pressure, *SVR* systemic vascular resistance, *SvO_2* mixed venous oxygen saturation

[a] Values are presented as the mean ± SD

[b] ANOVA (analysis of variance) test for group and time interaction

Table 3 Baseline and relative changes (%) in hemodynamic values in responders and non-responders to the fluid challenge divided into two groups according to their baseline mean arterial pressure

Baseline and relative changes in hemodynamic parameters	Responders			Non responders		
	<70 mmHg	>70 mmHg	*p* value	<70 mmHg	>70 mmHg	*p* value
Number of patients	15	10		12	14	
$MAP_{baseline}$ (mmHg)	63 ± 5	78 ± 7	<0.001	64 ± 4	77 ± 6	<0.001
Cardiac $index_{baseline}$ (l/min/m^2)	2.8 ± 0.8	3.0 ± 0.8	0.234	3.2 ± 1.0	2.9 ± 0.7	0.484
Pulse $pressure_{baseline}$ (mmHg)	56 ± 14	61 ± 14	0.018	50 ± 9	64 ± 18	0.337
$SVR_{baseline}$ (dynes.s.cm^{-5})	871 ± 273	1,093 ± 483	0.24	816 ± 305	948 ± 264	0.125
%MAP	16 ± 7	4 ± 10	0.001	8 ± 11	4 ± 13	0.391
%Cardiac index	21 ± 10	24 ± 11	0.432	2 ± 4	0 ± 8	0.541
%Pulse pressure	20 ± 18	10 ± 17	0.167	14 ± 24	3 ± 16	0.104
%SVR	−7 ± 13	−18 ± 15	0.031	1 ± 12	2 ± 13	0.967
%CVP	32 ± 45	62 ± 127	0.842	20 ± 25	18 ± 16	0.40
%PAOP	33 ± 41	29 ± 35	0.795	19 ± 22	16 ± 31	0.777

Baseline: Just prior to fluid challenge

All values with the exception of number of patients are presented as the mean ± SD

explained by the different types of patient included. Moreover, in our study, the baseline MAP was higher than that in the study by Monge Garcia et al. [19] (70 ± 9 vs. 58 ± 8 mmHg, respectively); we found minimal changes in SVR during fluid challenge in responders among patients with low baseline MAP (<70 mmHg) compared to patients with higher MAPs (Table 3). Hence, the capacity of the vascular system to increase the MAP may change during a fluid challenge. Although there were no significant increases in MAP in the non-responders as a group, some individuals did experience a significant rise in arterial pressure. This increase in the absence of an increase in CO may be the result of a direct effect of the increased blood volume together with a limited increase in vessel diameter.

The criterion for a fluid response in previous publications has consisted of increases in CI of >10% [20, 21] or 15% [22–24] because potential errors in CI measurement are estimated to be around 5–7% [25]. To date,

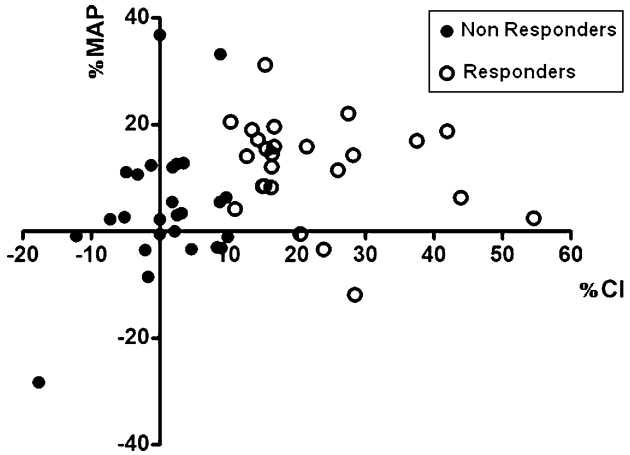

Fig. 1 Correlation between relative changes in cardiac index (*%CI*) and relative changes in mean arterial pressure (*%MAP*) ($r^2 = 0.07$, $p = 0.05$)

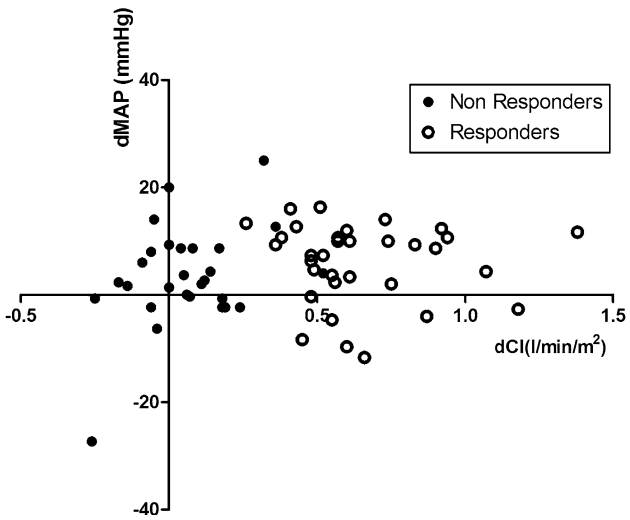

Fig. 2 Correlation between absolute changes in cardiac index (*dCI*) and absolute changes in mean arterial pressure (*dMAP*) ($r^2 = 0.08$, $p = 0.06$)

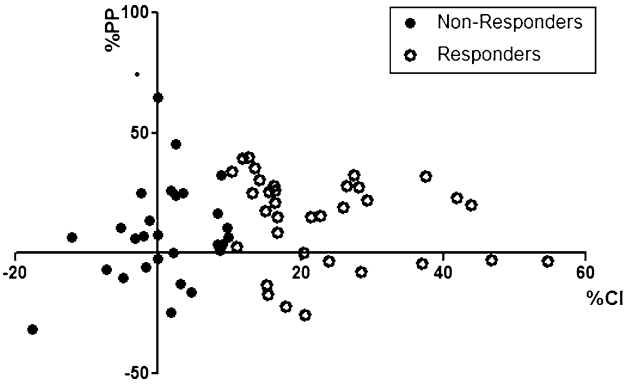

Fig. 3 Correlation between relative changes in cardiac index (*%CI*) and relative changes in pulse pressure (*%PP*) ($r^2 = 0.07, p = 0.155$)

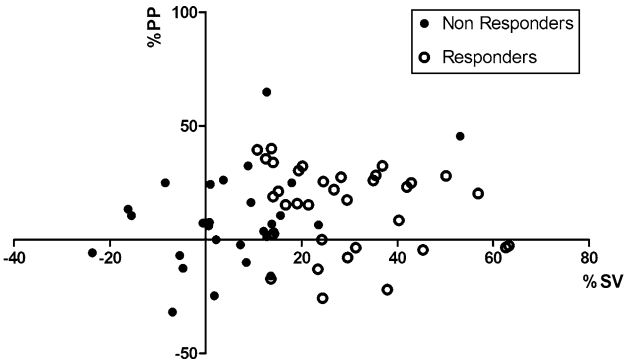

Fig. 4 Correlation between relative changes in stroke volume (*%SV*) and relative changes in pulse pressure (*%PP*) ($r^2 = 0.08$, $p = 0.04$)

no dynamic test has been proven to be predictive of arterial pressure changes during fluid challenge. Although an increase in CI of 10–15% has been defined as the usual cut-off value to quantify fluid responsiveness [26], it is possible that increases in arterial pressure may be associated with smaller increases in CI that are not recognized. We have demonstrated that arterial pressure may increase without a noticeable increase in CI in individual patients. These observations are important because they suggest that some patients in septic shock may still benefit from fluid administration even in the absence of signs of fluid responsiveness [27, 28]. Alternatively, one may consider that the dissociation between changes in MAP and changes in CI may be related to undetected spontaneous changes in the clinical condition of the patient. Indeed, in our study, MAP changed by >20% in several non-responders, and this may have been related to spontaneous changes in vasomotor tone. Whatever the explanation, the results further emphasize that the response to fluids is complex and difficult to detect without CI measurements.

Interestingly, we did not find any correlation between PP and CO changes in our study. It is well known that at rest, PP depends mainly on SV and arterial tone. Thus, SV and PP should uniformly increase or decrease if arterial tone does not change [29]. The arterial pulse wave is a combination of ejection force of the left ventricle to the periphery and a reflection of this force back toward the heart [30]. In patients with severe sepsis and septic shock, this peripheral reflection is decreased due to the significant vasoplegy, causing a decrease of PP in the periphery compared to that of the central arterial compartment [31]. Additionally, the effects of sepsis on peripheral and central vessel compliance are not uniform, and the normal PP amplification phenomenon may be affected [32]. This "peripheral vascular decoupling" may cause a dissociation between the changes in PP seen in the central arterial compartment and those observed in the periphery. Consequently, PP measured in the periphery may not track SV

changes. The aforementioned assumptions may explain why our results do not conform with physiological responses seen in normal subjects. Comparison of our results with those of other similar studies [12, 33] is difficult because we included only patients with septic shock and measured the PP in the radial artery in nearly all patients. However, a strong correlation between PP and CO was not found in any of these other studies.

A potential limitation of our study is that arterial pressure was usually measured from a radial artery, which may not give an entirely reliable measurement. Radial artery pressure has been reported to underestimate central aortic pressure in patients after cardiac surgery [34, 35], possibly due to local changes in resistance during the rewarming process after cardiopulmonary bypass [36]. For patients with septic shock, the data from two studies are conflicting, with one study showing an underestimation [31] of central arterial pressure values when a radial artery was used to measure the blood pressure and the other showing that the radial artery pressure correctly tracked central arterial pressure [37]. However, both studies demonstrated that, in patients treated with vasopressors, the pulse amplification phenomenon disappeared. A further limitation of our study is that we evaluated CO and its changes using a continuous thermodilution method and not by means of a beat-by-beat monitoring system. The continuous thermodilution method, which is routinely used in septic shock patients admitted to our ICU, has a characteristic intrinsically slower response to abrupt changes in CO [38]. Thus, we cannot exclude the possibility that rapid changes in CO may have occurred after the fluid challenge which would not have been detected by our CO monitoring technique.

Conclusions

Changes in arterial pressure are not correlated with changes in CI after fluid challenge in patients with septic shock. Care must be taken when using MAP to interpret the response to fluid challenge in such patients.

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