EJA

Eur J Anaesthesiol 2017; **34:**748–754

ORIGINAL ARTICLE

Echocardiography and passive leg raising in the postoperative period

A prospective observational study

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BACKGROUND Signs of hypovolaemia are frequent in the postoperative period, but not all patients need or respond to fluid administration. An increase in cardiac output (CO) after passive leg raising (PLR) has been demonstrated to be useful as a volume response predictor in non-surgical, spontaneously breathing patients.

OBJECTIVE The objective of this study was to evaluate the accuracy of transthoracic echocardiography after PLR to predict fluid responsiveness in post-surgical patients.

DESIGN A prospective observational study.

SETTING A tertiary hospital between January and July 2015.

PATIENTS Fifty-one spontaneously breathing postoperative patients with suspected hypovolaemia (arterial hypotension, oliguria, tachycardia or delayed capillary refill) were considered for the study.

INTERVENTION Demographic and personal data were collected, as well as heart rate variations, mean arterial pressure during PLR and after administering 500 ml of Ringer's lactate solution. CO was measured by transthoracic echocardiography.

MAIN OUTCOME MEASURES The primary outcome was measurement of CO before and after PLR and Ringer's lactate administration.

RESULTS Forty-one patients were included in the study (six patients were excluded because of a poor echocardiographic window and four because of misalignment of the Doppler and outflow tract of the left ventricle). Twenty-two patients (54%) were considered responders to fluid therapy, with an increase of stroke volume greater than or equal to 15% after 500 ml lactated Ringer's infusion. The highest area under the curve was found for an increase in CO (0.91 ± 0.05; 95% confidence interval 0.78 to 0.97). An increase in CO greater than 11% after the PLR manoeuvre predicts a volume response with 68% sensitivity and 100% specificity.

CONCLUSION This is the first study showing that measurement of CO after PLR can predict volume response in spontaneously breathing postoperative patients.

Published online 15 July 2017

Introduction

Perioperative fluid management influences patients' outcomes. It is a controversial and challenging issue in the anaesthesia setting. Both overhydration and conservative fluid therapy can lead to perioperative complications. The problem facing anaesthesiologists is that we do not know the volume needed by the patient. Goal-directed therapy sheds light on this debate, demonstrating that its use reduces postoperative complications and hospital stay.[1–3]

In the immediate postoperative period, signs of low cardiac output (CO) such as oliguria, tachycardia or arterial hypotension are frequent. However, these signs could be secondary to other factors. There could also be patients with real hypovolaemia but their ventricles operate at the non-responder point of the Frank–Starling curve. Is it possible to know if our patient will respond to fluids without administration, avoiding the negative consequences of excessive volume? A simple, non-invasive

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DOI:10.1097/EJA.0000000000000679

manoeuvre to predict response to volume in postoperative patients is passive leg raising (PLR). Raising the patient from a semi-recumbent position to a position with the head at 0° and the legs raised to a 45° angle, it is possible to increase venous return, mobilising blood from the lower limbs and splanchnic territory to the central compartment.

PLR has been demonstrated to produce changes in preload, increasing stroke volume (SV) significantly in patients who meet at the responder part of the ventricular function curve of Frank–Starling. Likewise, this is considered a reversible filling volume test as its effect on SV disappears after the patient returns to the supine position.[4] This manoeuvre simulates a pre-load increase, distinguishing responders to fluid therapy from non-responders.

Studies validating PLR have been carried out mostly on critical, non-surgical patients and less frequently in a postoperative population.[5,6] PLR has been evaluated with both invasive and non-invasive CO methods, measuring aortic flow or pulse pressure in non-surgical patients with spontaneous breathing.[7,8] However, no studies have validated this manoeuvre with measurement of CO using echocardiography in a surgical population. Post-surgical patients have traditionally been studied independently from medical patients. This is because surgical trauma as well as anaesthetic technique is associated with alterations in the release of hormones and cytokines. This causes a haemodynamic response to stress that is different from medical patients and, moreover, with different behaviours among surgical patients. This explains why in studies referring to haemodynamic monitoring, the surgical population should be studied separately. The objective of this study was to evaluate the ability of echocardiographic measurement of changes in CO during a PLR manoeuvre to identify those spontaneously breathing post-surgical patients who will respond to volume expansion.

Methods

Ethical approval for this study (Ethical Committee no. NAC 140155) was provided by the Ethical Committee NAC of Hospital Universitario de Gran Canaria Dr Negrín, Las Palmas de Gran Canaria, Spain (Chairperson Prof P. Lara) on 1 August 2014. We carried out a prospective observational study in an 18-bed post-anaesthesia care unit (PACU) between January and July 2015. After obtaining their informed consent, patients in whom low CO was suspected in the immediate postoperative period (general, vascular or orthopaedic surgery) were included. The following clinical signs were selected: arterial hypotension, systolic arterial pressure below 90 mmHg, mean arterial pressure (MAP) less than 70 mmHg or blood pressure (BP) decreased by more than 40 mmHg compared with usual BP; oliguria, urine output less than 0.5 ml kg^{-1} h^{-1} for more than 2 consecutive hours; tachycardia, heart rate (HR) higher than 100 bpm; delayed capillary refill, longer than 3 s.

Patients with aortic valvulopathy (known or discovered during the study) and mitral valvulopathy (mitral insufficiency greater than grade II or mitral stenosis) were not included, as well as patients who had undergone intracranial surgery, patients with any contra-indication to PLR manoeuvres (hip replacement or deep venous thrombosis), clinical signs of haemorrhage and patients with severe pain [visual analogue scale (VAS) > 3)]. We excluded patients with unsatisfactory cardiac echogenicity, such as poor echocardiographic window or misalignment between the Doppler and the outflow tract of the left ventricle (LV).

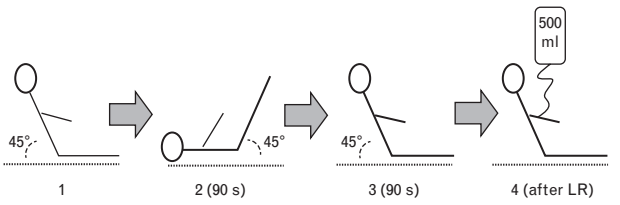
Data collected at the beginning were age, sex , American Society of Anesthesiologists' physical status, weight and height, to calculate BSA. The presence of arterial hypertension, diabetes mellitus, coronary arterial disease, chronic obstructive pulmonary disease and atrial fibrillation were recorded, as were the types of anaesthesia and surgery, postoperative analgesia, Ramsay sedation scale and VAS. The presence or absence of motor block in the lower limbs was also registered.

During the study, the following variables were collected: haemodynamic parameters, HR and MAP; echocardiographic parameters, LV outflow tract diameter (LVOTd), maximum velocity originated in the LV outflow tract (LVOTv) and velocity-time integral of the outflow tract (VTiOT). These measurements were made at four different times (Fig. 1).

We considered 'Responders' to be patients in whom SV increased by 15% or more after volume expansion with 500 ml of lactated Ringer's solution (B. Braun Medical SA, Barcelona, Spain).

The echocardiographic examination was performed by the same trained operator using a transthoracic ultrasound device, MyLab Five (Esaote, Maastricht, Netherlands).

Fig. 1



Graphic description of the study protocol and positions in which measurements were performed: (1) Baseline measurement in the supine position with chest raised to 45°. (2) The bed was then raised to elevate the patient's legs to 45°. After 90 s, the second measurement was taken. (3) The patient was then returned to the basal position for 90 s, when measurements were recorded. (4) Finally, 500 ml of lactated Ringer's solution (B. Braun Medical SA, Barcelona, Spain) was administered quickly, and the final measurements were done. LR, lactated Ringer's solution.

LVOTd was measured using the parasternal long axis view 1 cm below the insertion of the aortic annulus. Assuming that the LV outflow tract area (LVOTa) was spherical, the LVOTa was calculated as follows: LVOTa = 0.78 × LVOTd^2. As the diameter was assumed to remain constant during the study, it was measured only once at the baseline position.

LVOTv was measured using the apical five-chamber view, with the pulsed-wave Doppler aligned to the LV outflow tract, and the sample volume fixed 1 cm below the aortic annulus, and the VTiOT was calculated. SV was measured as follows: SV = LVOTa × VTiOT. Then, CO was determined as follows: CO = SV × HR. These values were averaged over four measurements at the end-expiratory period. In patients suffering from atrial fibrillation, these values were averaged over more than eight measurements when no significant changes in the ventricular rate were noticed.

Statistical analysis

Statistical analysis was performed using R Core Team 2014 version (R Foundation for Statistical Computing, Vienna, Austria) and MedCalc version 9.2.1.0 software (MedCalc Software, Mariakerke, Belgium).

Results of qualitative variables are expressed as frequency and percentage. Quantitative variables are expressed as mean ± SD. Normality was tested by using the Shapiro–Wilk test. Variables were compared using a Student *t* test for continuous variables and a χ^2 test for frequencies. Variables were compared between responders and non-responders before PLR and after volume expansion using the non-parametric Mann–Whitney *U* test. Quantitative variables with more than two categories were compared using the Kruskal–Wallis test. Receiver-operating characteristic curves were constructed to evaluate the ability of the changes of each variable during PLR to predict the fluid responsiveness after volume expansion. The area under the curve (AUC) was calculated for all parameters and expressed as AUC ± SD. SD. Sensitivity, specificity, negative and positive predictive values, negative and positive likelihood ratio and rate of correct classification were calculated to choose the cut-off values. Probability (*P*) values of less than 0.05 were considered statistically significant.

Results

Fifty-one patients were considered for the study. Six patients were excluded because of a poor echocardiographic window, and four due to misalignment between the Doppler and the outflow tract of the LV. Consequently, 41 patients were included in the study. Table 1 summarises the characteristics of the patients as well as their distribution between responders and non-responders.

The reason for inclusion was oliguria in half of the patients (51.2%) followed by arterial hypotension (26.8%), hypotension and tachycardia (9.8%), tachycardia (4.9%) and oliguria and tachycardia (2.4%). Only two patients (4.9%) required vasoactive support with norepinephrine, but no modification of its dose was required during the study. Twenty patients (48.8%) had been submitted to general surgery, 16 (39.0%) to orthopaedic surgery and only five (12.2%) to vascular surgery. The Ramsay score was II in all patients except one. VAS scores reported by patients were 1 in 34.2%, 2 in 48.8% and 3 in 17%.

Twenty-two patients (54%) were considered to be fluid responders, with an increase of SV at least 15% after 500 ml of lactated Ringer's solution. The remaining 46% were considered 'Non-Responders'. No statistical significant differences were found in clinical characteristics between these groups (Table 1).

When analysing baseline data (haemodynamic and echocardiographic measurements), no statistically significant differences were found between 'Responders' and 'Non-Responders' except in MAP, which was higher in the 'Responders' (Table 2). Registered data during the different times of the study are shown in Table 3.

Table 1 Characteristics of the study population

	All patients (41)	Responders (22)	Non-responders (19)	*P* value
Age (years)	41 ± 65.5	64.1 ± 15.2	67.2 ± 13.9	0.51
Sex (male/female) (%)	63.4/36.6	68.1/31.9	57.9/42.1	0.53
BSA (m^2)	1.81 ± 0.16	1.80 ± 0.2	1.82 ± 0.11	0.71
ASA physical status (1/2/3/4) (%)	4.9/51.2/41.5/2.4	4.5/41.0/50.0/4.5	5.3/63.2/31.5/0	0.15
Co-morbidities				
Arterial hypertension (%)	63.4	68.2	57.9	0.53
Diabetes mellitus (%)	24.4	36.4	10.5	0.08
Chronic obstructive pulmonary disease (%)	4.9	0	10.5	0.21
Atrial fibrillation (%)	7.3	13.6	5.3	0.61
Anaesthesia				
General (%)	61.0	59.1	63.1	0.69
Regional (%)	36.6	40.9	31.6	
Combined (%)	2.4	0	5.3	
Epidural/intravenous analgesia (%)	2.4/97.6	4.5/95.5	0/100	1
Motor block (yes/no) (%)	19.5/80.5	18.2/81.8	21.1/78.9	1

Data are expressed as mean ± SD or percentage. ASA, American Society of Anesthesiologists'.

Table 2 Baseline data in responders and non-responders

	Responders	Non-responders	*P* value
HR (bpm)	77.4 ± 21.8	70.9 ± 17.2	0.32
MAP (mmHg)	87.1 ± 17.4	75.4 ± 15.4	0.03
LVOTv (cm s^{-1})	1.0 ± 0.2	1.0 ± 0.2	0.54
VTiOT index (cm)	11.0 ± 2.8	12.0 ± 2.9	0.26
SV index (ml m^{-2})	32.0 ± 9.2	33.9 ± 8.9	0.5
CO index (l min^{-1} m^{-2})	2.35 ± 0.55	2.30 ± 0.45	0.37

Data are expressed as mean ± SD. CO, cardiac output; HR, heart rate; LVOTv, maximum velocity originated in left ventricular outflow tract; MAP, mean arterial pressure; SV, stroke volume; VTiOT, velocity-time integral of the outflow tract.

An increase in CO greater than 11% during the PLR test distinguished responders from non-responders with a sensitivity of 68.2% and a specificity of 100% (Table 4, Fig. 2). Similarly, an increase in VTiOT higher than 11% during PLR was able to predict an increase in SV at least 15% after volume administration, with a sensitivity of 81.8% and a specificity of 89.5%.

The highest AUC was found for an increase in CO [0.91 ± 0.05; 95% confidence interval (CI) 0.78 to 0.97] and an increase in VTiOT (0.89 ± 0.05; 95% CI 0.75 to 0.96) (Fig. 3). No haemodynamic or echocardiographic data measured in the basal position were able to predict a positive response to volume infusion (Fig. 4).

Discussion

The current study shows that CO measured by transthoracic echocardiography along with a reversible and well-tolerated PLR manoeuvre can predict a positive response to volume infusion with high sensitivity and specificity in spontaneously breathing post-surgical patients with suspected central hypovolaemia.

Traditionally, volaemic status has been evaluated using MAP, HR, capillary refill and diuresis.[9] Now, it is known that MAP and HR cannot be used reliably to measure changes in central blood volume. These two variables do not change with the initial volume loss because the mobilisation of blood from the splanchnic circulation to the core compartment retards the appearance of hypotension and tachycardia.[10] In our study, these variables had too low a predictive value to foresee the volume response.

Other invasive static measurements have been used to evaluate volaemia, such as central venous pressure or pulmonary capillary wedge pressure. It has been demonstrated that these parameters are bad indicators of volaemia and are not useful as predictors of an adequate response to fluid therapy.[11,12] However, the use of haemodynamic variables derived from lung–heart interactions and respiratory variations in SV have been demonstrated to be useful.[7,8] Nevertheless, this monitoring is not always applicable in spontaneously breathing patients in the PACU.[13] Moreover, its indiscriminate use would be economically unsustainable.

As an alternative to situations in which parameters that depend on the lung–heart interaction cannot be used, CO variation after PLR has been studied as a predictor of fluid response.[4,14] The validation of PLR as a virtual filling manoeuvre has been tested with invasive and non-invasive techniques. Among the invasive techniques, there is aortic blood flow by Doppler oesophageal probe,[8] thermodilution[7] or AUC of the systolic component of invasive arterial pressure.[15] Among the non-invasive methods that validate PLR, there is transthoracic echocardiography[5,6] and transcutaneous Doppler.[16] A large number of the studies that used echocardiography included patients on mechanical ventilation.[17] However, Maizel *et al.*,[6] in a prospective study with 34 critical non-intubated patients, demonstrated that an increase of more than 12% in CO or SV during PLR predicted that a patient was a responder to volume. Lamia *et al.*[5] evaluated 24 critical patients who were breathing spontaneously, but the majority were receiving mechanical ventilation. They concluded that PLR induced an increase of 15% or more in SV after volume expansion with a sensitivity of 77% and a specificity of 100%[5]. Nevertheless, in both studies, the majority of the patients included had not undergone surgery, so these results cannot be extrapolated directly to surgical populations. Post-surgical patients behave differently

Table 3 Evolution of haemodynamic parameters during the study (baseline, 90 s after passive leg raising and after fluid infusion)

	Baseline measurements		PLR	After fluid infusion
HR (bpm)	74.4 ± 19.8		74.2 ± 20.1	72.7 ± 19.3
MAP (mmHg)	81.7 ± 17.3		83.6 ± 14.6	85.1 ± 16.5
		Variation	3.0 ± 7.9	
LVOTv (cm s^{-1})	1.0 ± 0.2		1.1 ± 0.2	1.1 ± 0.2
		Variation	5.6 ± 11.2	
VTiOT index (cm)	11.5 ± 2.9		13.0 ± 3.2	13.2 ± 3.2
		Variation	11.8 ± 10.6	
SV index (ml m^{-2})	32.9 ± 9.0		37.1 ± 10.5	37.7 ± 10.2
		Variation	11.8 ± 10.6	
CO index (l min^{-1} m^{-2})	2.33 ± 0.50		2.62 ± 0.61	2.63 ± 0.65
		Variation	11.46 ± 10.21	

Values are expressed as mean ± SD. CO, cardiac output; HR, heart rate; LVOTv, maximum velocity originated in left ventricular outflow tract; MAP, mean arterial pressure; PLR, passive leg raising; SV, stroke volume; VTiOT, velocity-time integral of the outflow tract.

Table 4 Accuracy of velocity–time integral of the outflow tract and cardiac output changes after passive leg raising to predict fluid responsiveness

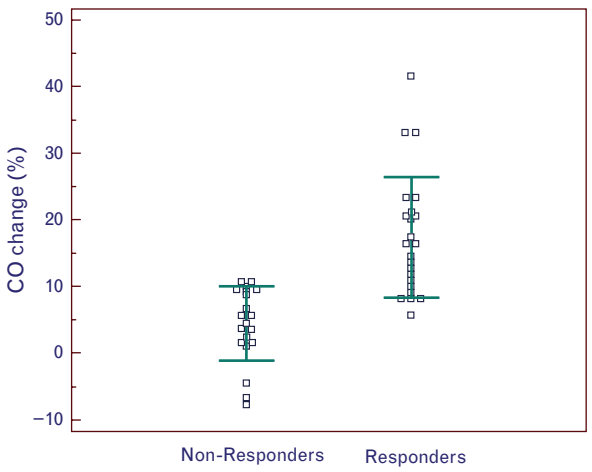
Cut-off point (%)	Sensitivity	Specificity	PPV	NPV	PLR	NLR	Rate of correct classification
VTiOT > 5	95.5	52.6	70.0	90.9	2.0	0.1	75.6
CO > 5	100	47.4	68.8	100	2.1	0	75.6
VTiOT > 8	86.4	63.2	73.1	80.0	2.3	0.2	75.6
CO > 8	95.5	63.2	92.3	75.0	2.6	0.1	80.5
VTiOT > 10	81.8	84.2	85.7	80.0	5.2	0.2	82.9
CO > 10	77.3	84.2	85.0	76.2	4.9	0.3	80.5
VTiOT > 11	81.8	89.5	90.0	81.0	7.8	0.2	85.4
CO > 11	68.2	100	100	73.1	0	0.3	82.9
VTiOT > 12	68.2	89.5	88.2	70.8	6.5	0.4	78.1
CO > 12	63.6	100	100	70.4	0	0.4	80.5

CO, cardiac output; NL, negative likelihood ratio; NPV, Negative predictive value; PL, positive likelihood ratio; PLR, passive leg raising; PPV, positive predictive value; VTiOT, velocity-time integral of the outflow tract.

because they undergo pathophysiological changes inherent to their surgery and the anaesthetic agents. In PACU, they frequently display signs suggestive of hypovolaemia. However, these signs could be due to other factors. For example, the presence of arterial hypotension and tachycardia could be a consequence of vasoplegia induced by the residual effects of anaesthetic agents or by inflammatory response syndrome due to the surgery. A reduction of urinary flow, representing the reason for inclusion in 51.2% of our patients, could be caused by secretion of anti-diuretic hormone secondary to surgery, pain, use of opioids or to the presence of nausea or vomiting after surgery.[18] All of this could explain why only 54% of the patients included who had received volume change were true responders.

Moreover, the response to PLR may be affected by frequent situations that arise in the postoperative period. Adrenergic vasoconstriction due to postoperative pain may reduce the total volume of blood stored in the lower limbs, underestimating the amount of blood displaced during the manoeuvre. For that reason, patients with a VAS higher than 3 were excluded. In contrast, the presence of sympathetic block due to regional analgesia techniques may mask the effect of the PLR test on a patient's volume status.[19] In our study, we showed that this manoeuvre was able to discriminate between responder and non-responder patients, even in patients with motor block.

Fig. 2

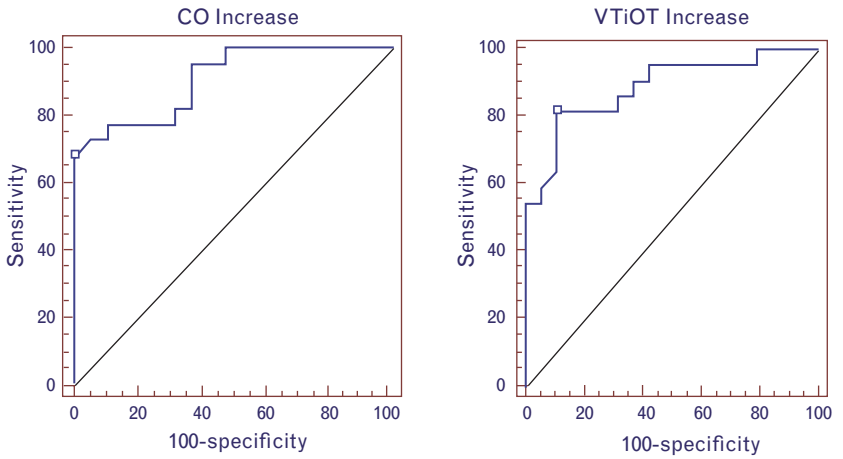


Cardiac output variation in responders and nonresponders. CO, cardiac output.

The CO measurement has already been validated as effective compared with other invasive techniques that use thermodilution.[20] The advantages of using bedside transthoracic echocardiography in the PACU are that it is non-invasive, inexpensive and available 24/7. However, the measurement of CO requires advanced echocardiographic skills and can be complicated because of the patient's body habitus. It is also essential to find the proper alignment of the Doppler axis and the blood flow through the LV outflow tract. This alignment should not be changed during the study, so the observed difference is not merely the result of a variation of the angle.

The main limitation of our investigation is the sample size. Ten patients were excluded because of misalignment of the Doppler axis and the LV outflow tract or because of poor echogenicity. Placing patients in the left lateral decubitus position could have resulted in excluding fewer patients due to a bad echocardiographic window. However, we could not place patients in this position because they had just undergone surgery. It is also difficult to place a patient in left lateral decubitus position at the same time as a PLR manoeuvre is performed. Another limitation is that measurements were performed by a single observer, which made it impossible to take inter-individual variability into account. In relation to the assessment of PLR in patients with sympathetic block, we only recorded whether the patient had motor block at the time of the test, ignoring the persistence of sympathetic block.

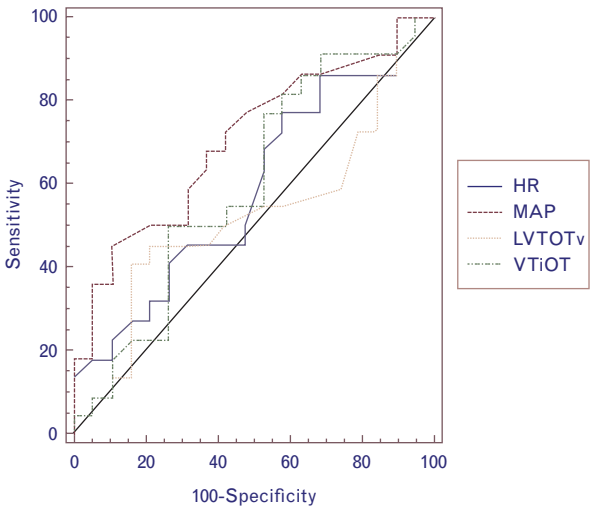
Fig. 3



Receiver-operating characteristic curves showing the ability of passive leg raising-induced increases in cardiac output and velocity-time integral of the outflow tract to discriminate between responders and non-responders.

Despite these limitations, our results show that the prediction of response to volume with echocardiography and PLR is useful in the postoperative period after low-risk surgery. Our study is the first to confirm that in a low-risk surgical population, an increase in CO higher than 11% measured by echocardiography after a PLR manoeuvre predicts that the patient will respond to fluid infusion with high sensitivity and specificity. No haemodynamic or echocardiographic data (HR, MAP, LVOTv and VTiOT) measured in the basal position were able to predict a positive response to volume infusion. The potential benefits of our results on clinical outcomes remain to be clarified.

Fig. 4



Receiver operating characteristic curves showing the lack of ability of measurements in the basal position to discriminate responders from non-responders. HR, heart rate; LVOTv, maximum velocity originated in left ventricular outflow tract; MAP, mean arterial pressure; VTiOT, velocity–time integral of the outflow tract.

Acknowledgements relating to this article

Assistance with the study: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

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