

End-expiratory occlusion manoeuvre does not accurately predict fluid responsiveness in the operating theatre

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Editor's key points

- Fluid responsiveness can be predicted by interactions between circulatory and respiratory function during mechanical ventilation.
- The utility of changes in stroke volume or end-tidal carbon dioxide produced by the end-expiratory exclusion manoeuvre to predict fluid responsiveness was studied in surgical subjects.
- Neither index was capable of predicting fluid responsiveness in healthy subjects, in contrast to data from critically ill subjects.

Background. The objective of this study was to determine whether assessment of stroke volume (SV) and measurement of exhaled end-tidal carbon dioxide (E'_{CO_2}) during an end-expiratory occlusion (EEO) test can predict fluid responsiveness in the operating theatre.

Methods. Forty-two subjects monitored by oesophageal Doppler who required i.v. fluids during surgery were studied. Haemodynamic variables [heart rate, non-invasive arterial pressure, SV, cardiac output (CO), respiratory variation of SV (ΔrespSV), variation of SV during EEO, and E'_{CO_2}] were measured at baseline, during EEO (ΔE_{EEO}), and after fluid expansion. Responders were defined by an increase in SV over 15% after infusion of 500 ml of crystalloid solution.

Results. Of the 42 subjects, 28 (67%) responded to fluid infusion. A cut-off of $>2.3\%$ ΔSV_{EEO} predicted fluid responsiveness with an area under the receiver-operating characteristic (AUC) curve of 0.78 [95% confidence interval (95% CI): 0.63–0.89, $P=0.003$]. The AUC of ΔrespSV was 0.89 (95% CI: 0.76–0.97, $P<0.001$). With an AUC of 0.68 (95% CI: 0.51–0.81, $P=0.07$), $\Delta E'_{CO_{2EEO}}$ was poorly predictive of fluid responsiveness.

Conclusions. ΔSV_{EEO} and $\Delta E'_{CO_2}$ were unable to accurately predict fluid responsiveness during surgery.

Keywords: anaesthesia; cardiac output monitoring; end-expiratory occlusion; end-tidal carbon dioxide; intraoperative monitoring

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Fluid administration is frequently used to treat hypovolaemia in order to enhance cardiac function by increasing preload. Several studies have demonstrated that fluid responsiveness can be predicted by using indices based on interactions between respiratory and circulatory function under positive pressure mechanical ventilation, such as respiratory variation of stroke volume (ΔrespSV).^{1–4} However, these respiratory-derived indices are reliable predictors only under strict conditions.

Recently, Monnet and colleagues^{5,6} developed a functional test to predict fluid responsiveness: the end-expiratory occlusion (EEO) test. They demonstrated that an increase in pulse pressure or cardiac output (CO) during EEO accurately predicted further increase in CO with fluid expansion in critically ill patients. By abolishing the inspiratory increase in intrathoracic pressure, EEO increases venous return and CO that could act as a volume challenge for detecting preload responsiveness.⁵ The EEO test can be used even in patients not fully adapted to mechanical ventilation and ventilated with low tidal volume, in the presence of cardiac arrhythmias, or both.^{5,6} Such situations are frequent during anaesthesia and can limit the use of dynamic indices at bedside.⁷

During general anaesthesia, patients are monitored by measuring exhaled CO_2 (E'_{CO_2}).⁸ Over short time periods and assuming a constant metabolic status, a qualitative relationship has been demonstrated between E'_{CO_2} and CO.^{9–12} Changes in E'_{CO_2} might reflect changes in CO.^{9–13} The primary objective of this study was to assess the ability of the variation of SV measured by oesophageal Doppler monitoring (ODM) during an EEO test to predict fluid responsiveness. The secondary objective was to assess the ability of E'_{CO_2} changes during an EEO test to predict fluid responsiveness.

Methods

Subjects

After Institutional Review Board (IRB) for human subjects approval, we conducted a prospective, observational study at Amiens University Hospital. Subjects aged >18 yr and monitored by ODM in whom the anaesthetist decided to infuse i.v. fluids were included. Indications for volume expansion were: optimization of CO, arterial hypotension, or haemorrhage. Subjects with right ventricular failure, contraindications to ODM

probe insertion, drug administration during the study period, or laparoscopic surgery were excluded.

Anaesthesia

Each subject was monitored by a three-lead electrocardiogram, pulse oximetry, and non-invasive arterial pressure measurement. Balanced general anaesthesia with tracheal intubation and mechanical ventilation was used. Induction was performed with propofol or etomidate and sufentanil according to the anaesthetist's preference. Maintenance was achieved with desflurane or sevoflurane and sufentanil. Tracheal intubation was facilitated with i.v. cisatracurium (0.15 mg kg^{-1}). Ventilation was performed in a volume-controlled mode with a tidal volume set to $7\text{--}9 \text{ ml kg}^{-1}$ of ideal body weight, and ventilatory frequency adjusted to maintain \dot{V}_{CO_2} at $30\text{--}35 \text{ cm H}_2\text{O}$ ($2.9\text{--}3.4 \text{ kPa}$); PEEP of $3\text{--}5 \text{ cm H}_2\text{O}$ ($0.29\text{--}0.49 \text{ kPa}$) was applied.

Measurements

We recorded ventilator settings (tidal volume, plateau pressure, and end-expiratory pressure) at baseline. Exhaled CO_2 was measured by DRAGER® WaterLock2 (Lübeck, Germany). The coefficient of variation of \dot{V}_{CO_2} , precision, and the least significant change (LSC) were calculated as previously described by Monnet and colleagues.¹³ Precision was 2.1% [95% confidence interval (95% CI: 0.99–3.1)] and LSC was 2.9% (95% CI: 2–3.7).

Oesophageal Doppler monitoring

The oesophageal Doppler probe (CardioQ™, Deltex Medical, Gamida, France) was positioned to obtain the optimum signal for descending aorta blood velocity. SV and CO were recorded continuously by the ODM software (beat by beat) from aortic blood flow velocity, and the mean values were calculated over 10 s. Respiratory variation of SV (ΔrespSV) was calculated as previously described.¹⁴ The variation of SV during EEO ($\Delta\text{SV}_{\text{EEO}}$) and the variation of \dot{V}_{CO_2} during EEO ($\Delta\dot{V}_{\text{CO}_{2\text{EEO}}}$) were calculated as the difference between the mean value at baseline and the maximum value reached during the last 5 s of EEO. $\Delta\text{SV}_{\text{EEO}} (\%) = [(\text{SV}_{\text{EEO}} - \text{SV}_{\text{baseline}}) / \text{SV}_{\text{baseline}}] \times 100$. $\Delta\dot{V}_{\text{CO}_{2\text{EEO}}} (\%) = [(\dot{V}_{\text{CO}_{2\text{EEO}}} - \dot{V}_{\text{CO}_{2\text{baseline}}}) / \dot{V}_{\text{CO}_{2\text{baseline}}}] \times 100$. All measurements were analysed off-line using a video sequence of the monitor, and represented the mean of three measurements. The reproducibility of SV measurement was tested before the study: intraobserver and interobserver variability for SV measurements were 0.5 (4)% and 2 (5)%, respectively.

Study protocol

A first set of measurements [heart rate (HR), systolic arterial pressure (SAP), mean arterial pressure (MAP), diastolic arterial pressure (DAP), SV, CO, \dot{V}_{CO_2} , ΔrespSV] was recorded at baseline (Base 1). An EEO test was performed as described by Monnet and colleagues,⁵ by interrupting ventilation at end-expiration for 15 s. A second set of measurements (HR, SV, CO, \dot{V}_{CO_2}) was recorded during the last 5 s of EEO. When all haemodynamic variables returned to baseline, and a third set of measurements (Base 2) (HR, SAP, DAP, SV, CO, \dot{V}_{CO_2} , ΔrespSV) was

recorded. Then, a volume expansion with 500 ml of crystalloid solution (Ringer or Ringer's lactate) over 10 min was performed. A last set of measurements (HR, SAP, DAP, SV, CO, \dot{V}_{CO_2} , ΔrespSV) was recorded immediately after the end of volume expansion (T2).

Statistics

At least 39 subjects would be sufficient to demonstrate that $\Delta\text{SV}_{\text{EEO}}$ can predict an increase of $>15\%$ in SV after fluid expansion with an area under the curve (AUC) >0.75 for a power of 80%, an α of 0.05, and a β of 0.2. The distribution of variables was assessed using the D'Agostino–Pearson test. Data are expressed as mean (SD), or proportion (%), as appropriate. Responders were defined by an increase in SV of $>15\%$ with fluid expansion (between Base 2 and T2). Student's paired *t*-test was used to compare within-group changes in haemodynamic variables. Differences between responders and non-responders were compared by Student's *t*-test. The Pearson rank method tested linear correlations. Statistical evaluation of ΔrespSV , $\Delta\text{SV}_{\text{EEO}}$, and $\Delta\dot{V}_{\text{CO}_{2\text{EEO}}}$ was based on AUC with 95% CI, and likelihood ratio.^{15 16} A receiver-operating characteristic (ROC) curve was established for ΔrespSV measured at baseline, $\Delta\text{SV}_{\text{EEO}}$, and $\Delta\dot{V}_{\text{CO}_{2\text{EEO}}}$. The ROC curves were compared using the DeLong test. Differences with $P < 0.05$ were considered statistically significant. Statistical analysis was performed using IBM® SPSS® Statistics 21 (IBM).

Results

A series of 42 subjects in whom the anaesthetist decided to administer i.v. fluids to expand circulating volume was studied. The mean age was 57 (16), mean height 168 (7) cm, and mean weight 78 (16) kg. Subjects underwent abdominal surgery (colectomy, peritonitis, duodenopancreatectomy, cholecystectomy, cystoprostatectomy, hysterectomy, and ovariectomy, $n=36$), orthopaedic surgery (hip fracture, $n=2$), or vascular surgery (vascular bypass, $n=4$). The mean tidal volume was $8.2 (0.8) \text{ ml kg}^{-1}$, respiratory rate was 14 (2), mean pressure plateau was $19 (4) \text{ cm H}_2\text{O}$, and mean PEEP was $4 (2) \text{ cm H}_2\text{O}$. Twenty-eight of the 42 subjects (67%) showed increased SV by $>15\%$ with volume expansion, and were defined as responders.

Baseline SV and CO were lower and ΔrespSV and $\Delta\text{SV}_{\text{EEO}}$ were higher in responders compared with non-responders (Table 1). Volume expansion increased SAP, SV, CO, and significantly decreased ΔrespSV only in responders (Table 1).

During the EEO test, SV (and CO) increased only in volume responders. The mean increase in SV with EEO was 3% (95% CI: -5 to 12). $\Delta\text{SV}_{\text{EEO}}$ and $\Delta\dot{V}_{\text{CO}_{2\text{EEO}}}$ were not correlated ($r=0.27$, $P=0.088$).

The mean increase in SV with fluid administration was 25% (95% CI: 18–32). Variations of \dot{V}_{CO_2} and SV with fluid responsiveness were not correlated ($r=0.07$, $P=0.97$). ΔrespSV , $\Delta\text{SV}_{\text{EEO}}$, and increase in SV with fluid administration were significantly correlated ($r=0.67$, $P<0.001$, $r=0.39$, $P=0.01$).

The ΔrespSV predicted fluid responsiveness with an AUC of 0.89 (95% CI: 0.76–0.97, $P<0.001$) (Table 2). The predictability of $\Delta\text{SV}_{\text{EEO}}$ was fair with an AUC of 0.78 (95% CI: 0.63–0.89,

Table 1 Cardiovascular variables in responders and non-responders expressed as mean (SD) or mean (95% CI). HR, heart rate; DAP, diastolic arterial pressure; EEO, end-expiratory occlusion; SAP, systolic arterial pressure; SV, stroke volume; CO, cardiac output; ΔrespSV , respiratory stroke volume variation; $\Delta\text{SV}_{\text{EEO}}$, variation of stroke volume during an end-expiratory occlusion; $\Delta\text{E}'_{\text{CO}_2}$, variation of end-tidal carbon dioxide during an end-expiratory occlusion test. * $P < 0.05$ between the groups at base 1 and EEO. $^{\dagger}P < 0.05$ within the groups between base 1 and EEO. $^{\ddagger}P < 0.05$ between the groups at base 2 and after volume expansion. $^{\S}P < 0.05$ within the groups between base 2 and volume expansion

	Base 1	EEO	Base 2	Volume expansion
HR (beats min ⁻¹)				
Responders	73 (19)	73 (19)	73 (20)	72 (16)
Non-responders	68 (19)	66 (19)	67 (19)	68 (20)
SAP (mm Hg)				
Responders	100 (22)	106 (23)	101 (21)	108 (18) [§]
Non-responders	103 (21)	103 (21)	100 (19)	103 (16)
DAP (mm Hg)				
Responders	58 (12)	62 (14)	58 (12)	61 (14)
Non-responders	53 (13)	53 (12)	53 (14)	56 (14)
SV (ml)				
Responders	69 (18)*	75 (21)* [†]	71 (17) [‡]	90 (22) [§]
Non-responders	102 (27)	99 (30)	103 (28)	104 (32)
CO (litre min ⁻¹)				
Responders	4.9 (1.5)*	5.3 (1.7) [†]	5 (1.5) [‡]	6.4 (2.2) [§]
Non-responders	6.8 (2.3)	6.5 (2.7)	6.7 (2.3)	6.8 (2.2)
ΔrespSV (%)				
Responders	21 (9)*		21 (9) [‡]	11 (9) [§]
Non-responders	9 (3)		11 (5)	9 (4)
E'_{CO_2} (mm Hg)				
Responders	31 (4)	36 (5) [†]	31 (4)	32 (4)
Non-responders	31 (3)	35 (4) [†]	31 (4)	30 (3)
$\Delta\text{SV}_{\text{EEO}}$ (%)				
Responders	9 (4–13)			
Non-responders	–3 (–9 to 3)*			
$\Delta\text{E}'_{\text{CO}_2}$ (%)				
Responders	17 (11–23)			
Non-responders	11 (5–16)			

Table 2 Comparison of AUC for the fluid responsiveness indices studied. ΔrespSV , respiratory stroke volume variation; $\Delta\text{CI}_{\text{EEO}}$, variation of cardiac index during an end-expiratory occlusion; $\Delta\text{E}'_{\text{CO}_2}$, variation of end-tidal carbon dioxide during an end-expiratory occlusion test

	Area under the ROC curve	95% confidence interval	Cut-off value (%)	Sensitivity (%)	Specificity (%)	Positive likelihood ratio	Negative likelihood ratio	Positive predictive value (%)	Negative predictive value (%)
ΔrespSV	0.89	0.76–0.97	13	93	86	6.5	0.08	93	86
$\Delta\text{SV}_{\text{EEO}}$	0.78	0.63–0.89	2.3	82	71	2.87	0.25	85	67
$\Delta\text{E}'_{\text{CO}_2\text{EEO}}$	0.68	0.51–0.81	7.4	82	57	1.92	0.31	79	62

$P=0.003$). $\Delta\text{E}'_{\text{CO}_2\text{EEO}}$ did not predict fluid responsiveness with an AUC of 0.68 (95% CI: 0.51–0.81, $P=0.07$). The AUC of ΔrespSV was greater than that of $\Delta\text{E}'_{\text{CO}_2\text{EEO}}$ ($P < 0.05$) (Fig. 1).

Discussion

The objective of this study was to evaluate the predictive value of EEO in the operating theatre. $\Delta\text{SV}_{\text{EEO}}$ measured by ODM was

unable to accurately predict fluid responsiveness. Similarly, variations of E'_{CO_2} were unable to predict fluid responsiveness or track changes of SV with EEO or fluid administration.

Predicting fluid responsiveness at the bedside remains an everyday challenge for anaesthesiologists, and various complementary approaches to dynamic indices have been studied. Passive leg raising (PLR) has been widely validated in mechanically or spontaneous ventilated patients in various

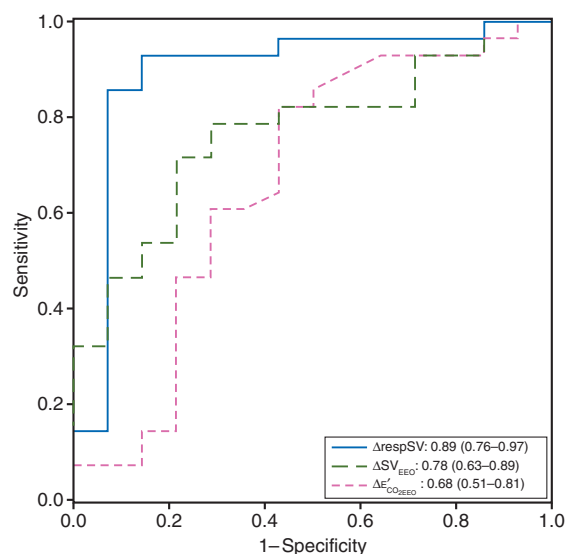


Fig 1 ROC curves of respiratory variation in SV (ΔrespSV), EEO variation in SV ($\Delta\text{SV}_{\text{EEO}}$), and EEO variation in exhaled end-tidal carbon dioxide ($\Delta\text{E}'_{\text{CO}_2\text{EEO}}$) to discriminate responders and non-responders to volume expansion. Area under ROC appears in cartouche with 95% CI.

clinical settings.¹⁴ However, PLR is difficult to perform in the operating theatre. We therefore evaluated the EEO test as an easy-to-use manoeuvre. Monnet and colleagues⁵ recently demonstrated the ability of the variation in CO during EEO to accurately predict fluid responsiveness in critically ill patients. These authors also confirmed the accuracy of this manoeuvre compared with respiratory-derived dynamic indices.⁶ In a population of 47 critically ill patients, Monnet and colleagues⁵ demonstrated that the EEO test had the highest AUC to predict fluid responsiveness. However, our study did not confirm this good accuracy of EEO in the operating theatre. Several explanations can be proposed for these discordant results. Haemodynamic characteristics of the study population differed between the study by Monnet and colleagues⁵ and our study. They studied critically ill patients with acute circulatory failure (mostly related to sepsis), whereas we included operated patients with no clinical signs of shock. Our patients might have had a higher baseline preload reserve than those in the study by Monnet and colleagues. The increase in SV after EEO and fluid administration would therefore be higher in the study by Monnet and colleagues than in our study.⁵ Similarly, the cut-off value to predict fluid responsiveness was lower in our study. The preload reserve status of our subjects might have affected the accuracy of EEO. Another explanation could be the difference between strategies of mechanical ventilation in intensive care unit (ICU) and operating theatre. In the study by Monnet and colleagues,⁵ most patients suffered from acute respiratory distress syndrome (ARDS), and were ventilated with low protective strategy with higher PEEP. This ventilation strategy could decrease venous return more than in our

subjects.¹⁷ Thus, the increase in venous return with EEO might be higher in ARDS patients. In addition, most of our subjects underwent abdominal surgery under balanced general anaesthesia, and the possibility of artifacts due to activation of the sympathetic nervous system related to surgical stress and altered venous return cannot be eliminated.¹⁸ Open abdominal surgery might have altered the effect of the EEO manoeuvre on venous return. Our results partly confirmed those reported by Monnet and colleagues, but assessment of $\Delta\text{SV}_{\text{EEO}}$ by ODM was unable to more accurately predict fluid responsiveness than ΔrespSV during surgery. Furthermore, the cut-off value of $\Delta\text{SV}_{\text{EEO}}$ was close to the limit of reproducibility of ODM.

In contrast to $\Delta\text{SV}_{\text{EEO}}$, the variation of E'_{CO_2} cannot be used to track changes of SV (or CO). Since the development of E'_{CO_2} monitoring, several studies have demonstrated that E'_{CO_2} correlates with tissue CO_2 production, pulmonary ventilation, pulmonary perfusion, or CO.^{9–12} Isserles and Breen,⁹ studying mechanically ventilated dogs, demonstrated a correlation between changes in CO induced by inflation/deflation of a vena cava balloon and E'_{CO_2} . These results were in accordance with those observed in patients undergoing cardiopulmonary resuscitation.¹⁹ Recently, Monnet and colleagues¹³ found similar results in the ICU setting. During preload challenge (i.e. PLR or fluid administration), change in E'_{CO_2} was correlated with change in CO. These authors therefore concluded that measurement of E'_{CO_2} during PLR can predict fluid responsiveness.¹³ Young and colleagues²⁰ also highlighted the value of dynamic changes of E'_{CO_2} as an adjunctive indicator of fluid responsiveness in ICU. Our results in the operating theatre are not in line with these observations. No significant difference was observed between responders and non-responders in terms of baseline E'_{CO_2} . E'_{CO_2} was not significantly increased in response to fluid infusion, whereas SV (and CO) was significantly increased. We also did not observe any correlation between E'_{CO_2} and SV. As discussed below, the SV changes observed in our study were lower than those observed during acute circulatory failure, ARDS, or experimental studies.^{9–13} As minor changes in SV were not associated with changes in E'_{CO_2} , no correlation could be demonstrated between SV and E'_{CO_2} . Moreover, the precision of E'_{CO_2} measurements with our device was close to $\text{E}'_{\text{CO}_2\text{EEO}}$ values. E'_{CO_2} was therefore unable to track changes in SV associated with fluid infusion during surgery.

This study has several limitations. There is a small number of subjects included. While our results might reflect everyday clinical reality in the operating theatre, our population consisted of operated patients with no signs of shock. The most common indications for fluid infusion were optimization of CO, followed by arterial hypotension. Another limitation could be the ODM device (CardioQ™, Deltex Medical) used to measure and track changes in SV (and CO). Although accuracy between ODM device and thermodilution-based CO device can be questioned, there is evidence to support the use of ODM for CO-guided intraoperative fluid optimization.^{21–22} We therefore assume that ODM is able to track changes in SV during various preload challenges.²¹ The reproducibility of SV was close to the $\Delta\text{SV}_{\text{EEO}}$ values observed in this study. Consequently, this might

limit bedside use of ΔSV_{EEO} to assess fluid responsiveness with ODM device.

In conclusion, measurement of variations of SV induced by EEO was poorly predictive of fluid responsiveness. EEO was unable to predict an increase in SV with fluid expansion during surgery more accurately than respiratory-derived indices. In addition, measurement of \dot{V}_{CO_2} during EEO was unable to predict fluid responsiveness. Moreover, measurement of \dot{V}_{CO_2} could track changes in SV induced by fluid infusion or EEO.

Authors' contributions

P.-G.G. conceived, designed, and coordinated the study, and drafted the manuscript. E.B., B.D.B., and J.G. participated in coordination of the study. E.L. and H.D. participated in coordination of the study and helped draft the manuscript.

Declaration of interest

None declared.

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