Assessment of changes in left ventricular systolic function with oesophageal Doppler

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Editor’s key points
- Oesophageal Doppler is becoming more widely used in major surgery to guide fluid therapy.
- It is unclear whether the indices derived from the oesophageal Doppler signal are affected by changes in left ventricular (LV) systolic function.
- Continuous monitoring of LV systolic function may help guide fluid and inotropic therapy during and after surgery.
- This study found that mean acceleration and peak velocity of the aortic flow signal are markers of LV systolic performance.

Background. We tested the ability of mean acceleration (Acc) and peak velocity (V_peak) of the aortic velocity signal measured by oesophageal Doppler to reflect left ventricular (LV) systolic performance.

Methods. We included critically ill patients in whom a fluid challenge (n=25) or the introduction of dobutamine, 5 μg kg⁻¹ min⁻¹ (n=25), was planned by the attending physician. Before and after therapeutic interventions, we measured Acc and V_peak (CardioQ device) and LV ejection fraction (LVEF) using echocardiography.

Results. For all pairs of measurements, the absolute values of Acc and V_peak correlated with LVEF (r=−0.36 and 0.57, respectively). The correlation was significantly higher for V_peak than for Acc. Volume expansion did not significantly change LVEF and Acc, but significantly increased V_peak by 7 (8)% . Dobutamine increased LVEF by 30 (15)%, Acc by 33 (25)%, and V_peak by 20 (10)%. Considering the pooled effects of volume expansion and dobutamine, changes in Acc and V_peak, and those of LVEF were correlated (r=0.53 and 0.67, respectively). When excluding changes <18% (i.e. the least significant change for LVEF), the concordance rate was 96% for Acc and 100% for V_peak.

Conclusions. V_peak and, to a lesser extent, Acc measured by oesophageal Doppler behaved as markers of LV systolic performance as they were almost insensitive to fluid administration and changed to a much larger extent with dobutamine. These indices could be used to estimate LV systolic performance and to assess the effects of inotropic therapy.

Keywords: cardiac output; cardiac output, shock; measurement, equipment; monitors, dobutamine, measurement techniques

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Oesophageal Doppler was developed years ago as a minimally invasive technique allowing haemodynamic monitoring. It has gained increasing popularity, in particular because studies have shown its use in high-risk surgical patients leads to improved outcomes. By measuring blood flow in the descending thoracic aorta, oesophageal Doppler provides reliable estimation of cardiac output.

Beyond aortic blood flow, oesophageal Doppler devices also measure the mean acceleration (Acc) and peak velocity (V_peak) of aortic flow from the Doppler signal. By analogy with the measurements performed at the aortic root level, which are recognized as indices of left ventricular (LV) systolic performance, Acc and V_peak measured by oesophageal Doppler in the descending thoracic aorta have been suggested to be related to LV contractility. Nevertheless, these previous investigations were conducted in a small number of patients or in healthy subjects. Moreover, Acc and V_peak were not compared with the gold standard for the assessment of the LV systolic function at the bedside [i.e. the LV ejection fraction (LVEF) measured by echocardiography].

In the present study, we aimed to confirm that Acc and V_peak are indicators of LV systolic function by comparing these indices to echocardiographic LVEF. We also tested whether Acc and V_peak behave as indicators of LV systolic function; that is, whether they remain unchanged after volume expansion yet increased by dobutamine administration.

Methods

Patients

This study was conducted in a 15-bed medical intensive care unit of a university hospital. It was approved by the Institutional Review Board of our institution (Comité pour la protection des personnes Ile-de-France VII). A deferred informed consent
was obtained from the patient’s surrogate as soon as possible. As he/she recovered consciousness, a deferred informed consent was confirmed from the patient. If the patient or his/her next of kin refused to consent, the patient’s data were not entered into the analyses. All patients had a diagnosis of septic shock and were receiving norepinephrine. Patients were included in the study if they met all of the following criteria:

1. The presence of acute circulatory failure defined by (i) systolic arterial pressure ≤ 90 mm Hg (or decrease in systolic arterial pressure of > 50 mm Hg in known hypertensive patients) or need for norepinephrine administration, (ii) urinary flow ≤ 0.5 ml kg⁻¹ min⁻¹ for > 2 h, (iii) tachycardia ≥ 100 bpm, or (iv) presence of skin mottling.
2. Need for fluid expansion or dobutamine administration, as decided by the attending physician. For fluid administration, volume expansion was administered either as a fluid challenge or because of positivity of fluid responsiveness tests (pulse pressure¹³ and stroke volume variations, respiratory variations of the descending aortic blood flow,¹⁶ passive leg raising,¹⁵ or end-expiratory occlusion tests).¹⁶ ¹⁷ These tests were not assessed during echocardiography. Infusion of dobutamine was used if LV contractile impairment was thought to account for the haemodynamic failure.
3. Monitoring by a transpulmonary thermodilution device (PiCCO2, Pulsion Medical System, Munich, Germany).

Patients were excluded if echogenicity was not sufficient for a proper assessment of the LVEF by transthoracic echocardiography or if they had a contraindication for the use of oesophageal Doppler monitoring (i.e. known or suspected oesophageal ulcer, malformation, varicose, or tumour).

Measurements

Immediately after inclusion, an oesophageal Doppler device (CardioQ, Deltex Medical, Chichester, UK) was set up. For this purpose, a 90 cm Doppler probe was inserted through the mouth or nose and advanced into the oesophagus to the mid-thoracic level. The probe position was then adjusted to obtain the highest Doppler velocity signal from the descending aorta. The probe position was re-adjusted during the course of the study if the aortic blood velocity signal deteriorated. Three investigators (J.-M.R., M.J., and X.M.) trained in this technique performed all measurements. The time required to obtain an optimal signal was 5 (1) min. We recorded the values of cardiac index, Acc, V_{peak}, and flow time corrected for heart rate that were automatically measured by the oesophageal Doppler device. The flow time is the aortic ejection time. It is related to preload and afterload.¹⁸ ¹⁹

Transpulmonary thermodilution was used for assessing the effects of therapeutic interventions on cardiac index as such effects might be underestimated by the CardioQ device when arterial pressure changes to a significant extent.²⁰ Echocardiography was performed by the transthoracic apical four- and two-chamber apical views (EnVisor Philips version B.0, Philips Medical System, Andover, MA, USA). The LVEF was obtained using the biplane or monoplane Simpson method. The echographic examinations were performed by a cardiologist (X.M.).

Study design

Before all therapeutic interventions, we performed a first set of haemodynamic measurements, including heart rate, systemic arterial pressure, cardiac index measured by oesophageal Doppler and by the transpulmonary thermodilution, flow time corrected for heart rate, Acc, V_{peak}, and LVEF.

After this first set of haemodynamic measurements, volume expansion was done using 500 ml of saline > 10 min, or dobutamine infusion was commenced at 5 μg kg⁻¹ min⁻¹, according to the decision of the clinician in charge of the patient. All other treatments were kept unchanged during the study period.

A second set of haemodynamic measurements was done after the therapeutic intervention (i.e. at the end of fluid administration for patients who received it and 15 min after stabilization of cardiac index in patients in whom dobutamine was used). This set included heart rate, systemic arterial pressure, cardiac index measured by oesophageal Doppler and by the transpulmonary thermodilution, flow time corrected for heart rate, Acc, V_{peak}, and LVEF. Investigators were not blinded to the therapeutic interventions. Echocardiographic measurements were performed after oesophageal Doppler measurements in all instances.

Statistical analysis

The normality of data distribution was tested with the Anderson–Darling test. Variables were summarized as frequencies and percentages for categorical variables, means, and standard deviations (SDs) for continuous normally distributed variables or medians, mean [95% confidence interval (CI)] for sensitivities and specificities, and inter-quartile ranges otherwise. Data were compared using χ², Fisher exact, two-tailed Student’s or Mann–Whitney U-tests, as appropriate. Correlation between variables was tested by the Spearman’s coefficient of rank correlation and correlation coefficients were compared using the Fisher transformation.²¹ The reproducibility of LVEF (inter- and intra-observer) was evaluated by calculating the coefficient of variation (i.e. the ratio of the SD to the mean).

For assessing the ability of Acc and V_{peak} to track changes in LVEF, we constructed a four-quadrant plot.²² This allowed calculating the percentage of total data points for which the direction changes of Acc and of V_{max} (increase or decrease) were concordant with LVEF. As the least significant change of LVEF is 18%,²³ we applied an 18% exclusion zone to this four-quadrant analysis.²² Multivariable regressions were performed by entering Acc and V_{peak} to estimate LVEF absolute values and relative changes. We performed a receiver operating characteristic (ROC) curve analysis in order to test the ability of Acc and V_{peak} to detect an LVEF of < 35%. Areas under ROC curves were compared by the Hanley–McNeil test. Statistical significance was defined by a P-value of < 0.05.
Statistical analysis was done using the MedCalc 11.6.0 software (MedCalc Software, Mariakerke, Belgium).

Results

Patient characteristics at baseline
Six patients were excluded because of poor echocardiographic views that did not allow assessment of LVEF. Fifty patients were eventually included in the study. Twenty-five patients received volume expansion and another 25 patients received dobutamine infusion. Patient characteristics at baseline are summarized in Table 1. No patient exhibited mitral or aortic regurgitation above 2/4 grade or important tricuspid regurgitation.

Relationship between absolute values of Acc, $V_{peak}$ and LVEF

The inter- and intra-observer reproducibility of LVEF were 6.8 (sd: 1.7) and 6.5 (1.5)%, respectively. Considering all measurements performed before and after therapeutic interventions (n = 100), the correlation between the absolute values of Acc and LVEF was $r = 0.36$ (P = 0.0002) and the correlation between the absolute values of $V_{peak}$ and LVEF was 0.57 (P < 0.0001). This correlation was significantly higher than that between the absolute values of Acc and LVEF (P = 0.04). When entering Acc and $V_{peak}$ in multivariable regression analysis for estimating absolute values of LVEF, the regression coefficient was significant for $V_{peak}$ (P < 0.0001) but not for Acc (P = 0.90).

An Acc ≤ 8 m s$^{-2}$ could detect an LVEF of ≤ 35% with a sensitivity of 80% (95% CI 63 – 92) and a specificity of 62% (95% CI 49 – 73) (Fig. 1). A $V_{peak}$ ≤ 55 m s$^{-1}$ allowed to detect an LVEF of ≤ 35% with a sensitivity of 89% (95% CI 73 – 97) and a specificity of 80% (95% CI 68 – 89). The area under the ROC curve constructed for Acc was significantly lower than for $V_{peak}$ [0.74 (0.04) vs 0.88 (0.03), respectively, P = 0.001] (Fig. 1).

Ability of Acc, $V_{peak}$ to detect changes in LVEF induced by therapeutic interventions

Volume expansion increased cardiac index measured by oesophageal Doppler by 11 (10)% and cardiac index measured by transpulmonary thermodilution by 17 (12)% (Table 2). Dobutamine increased cardiac index measured by oesophageal Doppler by 16 (12)% and cardiac index measured by transpulmonary thermodilution by 22 (18)% (Table 2). The difference between changes in cardiac index measured by oesophageal Doppler and that measured by transpulmonary thermodilution were correlated with changes in mean arterial pressure induced by therapeutic interventions (r = 0.55, P < 0.001). The flow time corrected for heart rate increased during volume expansion [+11 (8)%] but was not significantly changed by dobutamine infusion (Table 2).

Volume expansion did not significantly change LVEF [+2 (5)%, P = 0.07] and introducing or increasing the dose of dobutamine increased LVEF by 30 (15)% (P < 0.0001). Acc increased during dobutamine infusion [+33 (25)%], P < 0.0001] but not during volume expansion [+6 (14)%, P = 0.10]. $V_{peak}$ increased during dobutamine infusion [+20 (10)%, P < 0.0001] and volume expansion [+7 (8)%], P = 0.001].

| Table 1 Patient characteristics at baseline. SAPS, simplified acute physiology score |
|-----------------|-----------------|-----------------|-----------------|
| Patients receiving volume expansion (n = 25) | Patients receiving dobutamine (n = 25) |
| Age [mean (sd), yr] | 66 (10) | 71 (10) |
| Gender [male/female, number of patients] | 11/14 | 18/7 |
| SAPS II [mean (sd)] | 54 (12) | 55 (16) |
| Acute respiratory distress syndrome [number of patients (%)] | 12 (48%) | 8 (32%) |
| Patients receiving norepinephrine [number of patients (%)] | 20 (80%) | 18 (72%) |
| Dose of norepinephrine [median (inter-quartile range), μg kg$^{-1}$ min$^{-1}$] | 0.66 (0.18 – 0.93) | 1.03 (0.31 – 1.65) |
| Type of shock [number of patients (%)] | | |
| Septic | 25 (100%) | 9 (36%) |
| Cardiogenic | 0 (0%) | 16 (64%) |
Considering the pooled effects of volume expansion and dobutamine \((n=50)\), the correlation between the relative changes in Acc and LVEF was \(r=0.52\) \((P<0.0001)\) and the correlation between the relative changes in \(V_{peak}\) and LVEF was \(r=0.67\) \((P<0.0001)\) (Fig. 2). This correlation was not significantly different from that between the relative changes in Acc and LVEF \((P=0.25)\). The relative changes in Acc and in \(V_{peak}\) were significantly correlated \((r=0.68, P<0.0001)\). The concordance rate between the changes in Acc and LVEF was 90\% (Fig. 2), meaning that in 90\% of instances, Acc and LVEF changed in the same direction. When excluding changes <18\% (i.e. the least significant change for LVEF), the concordance rate was 96\% (Fig. 2).

For the pooled effects of volume expansion and dobutamine \((n=50)\), the concordance rate between the changes in \(V_{peak}\) and LVEF was 92\% (Fig. 2). When excluding changes <18\% (i.e. the least significant change for LVEF), the concordance rate was 100\% (Fig. 2).

When entering Acc and \(V_{peak}\) in multivariable regression analysis for estimating relative changes in LVEF, the regression coefficient was significant for \(V_{peak}\) \((P=0.0001)\) but not for Acc \((P=0.37)\).

For the pooled effects of volume expansion and dobutamine \((n=50)\), there was no significant correlation between the changes in mean arterial pressure and Acc \((P=0.84)\) or \(V_{peak}\) \((P=0.67)\).

**Discussion**

We found that \(V_{peak}\) and, to a lesser extent, Acc measured by oesophageal Doppler behave as indicators of LV systolic function. Acc, and particularly \(V_{peak}\), were correlated with absolute values of LVEF and being able to track the changes in LVEF induced by therapeutic interventions. Acc and \(V_{peak}\) were more sensitive to dobutamine than to volume expansion.

Detecting LV systolic dysfunction and assessing the effects of inotropic treatment is of utmost importance for haemodynamic monitoring, both in the operating theatre and in the intensive care unit. Although echocardiography is the gold standard for evaluating LV contractility at the bedside, it does not allow for continuous monitoring. In this regard, echocardiography is not easy to use for systematic detection of contractile dysfunction or for repeated assessment of LV contractility during inotropic therapy. The advantage of oesophageal Doppler would be to allow such continuous monitoring by an automatic analysis of the features of the aortic velocity signal.

That Acc and \(V_{peak}\) could be considered as markers of LV systolic performance is supported by the fact that their absolute values were correlated with LVEF and that they tracked the dobutamine-induced changes in LVEF. Moreover, Acc and \(V_{peak}\) behaved as indicators of LV systolic performance (i.e. they were minimally changed by volume expansion while they were very sensitive to dobutamine infusion). Of note, their relative changes were not correlated with the simultaneous changes in mean arterial pressure, suggesting that they were not influenced by LV afterload.\(^{24,25}\) An animal study has already demonstrated a good correlation between Doppler peak aortic velocity and electromagnetic catheter measured LV contractility.\(^{26}\) In normal subjects, Singer and colleagues\(^{12}\) have shown that dobutamine increased \(V_{peak}\) in a dose-dependent manner while esmolol exerted the opposite effect. In 15 patients, Singer and colleagues\(^{1}\) have observed that inotropic drugs increased the area under the aortic velocity signal primarily through changes in \(V_{peak}\). Our results confirm these previous findings and even strengthen them through the direct comparison of Acc and \(V_{peak}\) with the echocardiographic LVEF. This suggests that oesophageal Doppler is able to assess, at the level of the descending aorta, the features of the aortic velocity signal that are known to indicate the LV systolic performance when they are obtained at the level of the aortic annulus.\(^{27-30}\)

Theoretically, both \(V_{peak}\) and Acc should be indices of the LV ejection phase. Nevertheless, in the present study, \(V_{peak}\) was a better indicator of LV systolic performance than Acc. Indeed, the correlation of absolute values of \(V_{peak}\) with LVEF was significantly better than that of Acc. Moreover, using multiple regression analysis, Acc did not significantly contribute to estimating absolute values or relative changes in LVEF. Also, the area

### Table 2: Haemodynamic changes induced by volume expansion and dobutamine. \(V_{peak}\), peak velocity; Acc, mean acceleration. *P<0.05 after vs before volume expansion; †P<0.05 after vs before dobutamine; ‡P<0.05 dobutamine vs volume expansion

<table>
<thead>
<tr>
<th></th>
<th>Volume expansion ((n=25))</th>
<th>Dobutamine ((n=25))</th>
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<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Heart rate (\text{[mean (so), bpm]})</td>
<td>86 (14)</td>
<td>84 (13)</td>
</tr>
<tr>
<td>Mean arterial pressure (\text{[mean (so), mm Hg]})</td>
<td>73 (11)</td>
<td>79 (21)</td>
</tr>
<tr>
<td>Cardiac index (thermodilution) (\text{[mean (so), litre min}^{-1} \cdot \text{m}^{-2})}</td>
<td>2.7 (0.7)</td>
<td>3.2 (0.9)*</td>
</tr>
<tr>
<td>Cardiac index (oesophageal Doppler) (\text{[mean (so), litre min}^{-1} \cdot \text{m}^{-2})}</td>
<td>2.9 (1.2)</td>
<td>3.2 (1.1)*</td>
</tr>
<tr>
<td>Global end-diastolic volume (\text{[mean (so), ml m}^{-2})}</td>
<td>706 (150)</td>
<td>784 (183)*</td>
</tr>
<tr>
<td>Cardiac function index (\text{[mean (so), s}^{-1})}</td>
<td>4.4 (0.8)</td>
<td>4.4 (0.8)</td>
</tr>
<tr>
<td>Flow time corrected (\text{[mean (so), ms]})</td>
<td>300 (61)</td>
<td>332 (66)*</td>
</tr>
<tr>
<td>LVEF (\text{[mean (so), %]})</td>
<td>55 (13)</td>
<td>56 (12)</td>
</tr>
<tr>
<td>(V_{peak}) (\text{[mean (so), m s}^{-1})}</td>
<td>69 (19)</td>
<td>74 (19)*</td>
</tr>
<tr>
<td>Acc (\text{[mean (so), m s}^{-2})}</td>
<td>10.3 (4.1)</td>
<td>10.7 (3.8)</td>
</tr>
</tbody>
</table>
under the ROC curve for detecting LVEF ≤ 35% was significantly better for $V_{\text{peak}}$ than for Acc. This relatively poorer ability of Acc to reflect the LV systolic performance has been already observed in animals. It could be explained by the fact that oesophageal Doppler measures the Acc of the aortic velocity signal while catheter-tipped velocity probes found maximal acceleration to be the best marker of LV contractility, even better than $V_{\text{peak}}$. In the present study, $V_{\text{peak}}$ was not a pure marker of LV systolic function, because it also significantly increased during volume expansion. Nevertheless, dobutamine infusion was associated with a much larger increase in $V_{\text{peak}}$ than volume expansion.

Our results support the view that oesophageal Doppler should not be used only for measuring cardiac output as the analysis of the aortic velocity signal provides useful additional information. The velocity signal in the descending aorta can be assimilated to a triangle, whose base is the flow time and height is $V_{\text{peak}}$. The area of the triangle (stroke distance) is proportional to stroke volume. As a marker of preload, the flow time (corrected for heart rate) is the most well-known index derived from the velocity signal and it has been integrated in several algorithms for guiding haemodynamic optimization. In this regard, we observed that flow time increased with volume expansion but that it was not altered.

![Graph](image-url)
by dobutamine. The present study additionally suggests that \( V_{\text{peak}} \), which has been much less studied than flow time, should also be taken into account for assessing the haemodynamic status with oesophageal Doppler. In practice and according to our results, \( V_{\text{peak}} \) should not be considered as a perfect surrogate of LVEF but low values of \( V_{\text{peak}} \) should alert the clinician that LVEF is probably low and should prompt an echocardiographic examination in order to confirm the presence of LV systolic dysfunction. The relatively good trendability of \( V_{\text{peak}} \) suggests that if inotropic therapy is initiated, then the relative changes in \( V_{\text{peak}} \) could allow monitoring of its effects in an easier way than by repeating echocardiography.

Interestingly, this study confirms that the CardioQ device underestimated the changes in cardiac index induced by volume expansion compared with transpulmonary thermodilution. Indeed, the CardioQ device estimates the aortic diameter from nomograms based on patient characteristics (i.e. it considers the aortic diameter as constant in a given patient). However, the aortic diameter physiologically varies along with the mean arterial pressure.\(^{31}\) Our team previously demonstrated that the change in aortic diameter induced by fluid administration actually accounts for a large part of the fluid-induced changes in cardiac output and that considering the aortic diameter as constant leads to an underestimation of the response to fluid therapy.\(^{20}\) In the present study, we confirm that, compared with transpulmonary thermodilution, which was demonstrated to provide a reliable measurement of cardiac output,\(^{32–34}\) the CardioQ system underestimates the response to a treatment that changes the systemic arterial pressure.

The present study has some limitations. First, we used LVEF as an indicator of LV systolic function although this is not a pure marker of LV contractility and it is altered by loading conditions.\(^{35}\) However, LVEF is the conventional gold standard for estimating the LV systolic performance at the bedside. Secondly we used dobutamine in order to change LV contractility. Nevertheless, dobutamine is not a pure inotrope and it modifies the loading conditions of the left ventricle. Thirdly and as discussed above, oesophageal Doppler does not allow recording of maximal acceleration, which should be a better marker of LV contractility than Acc. Nonetheless, the present study investigated the variable that is actually displayed by the commercial oesophageal Doppler devices (i.e. the only one that can be used in clinical practice). Finally, investigators were not blinded to the therapeutic interventions.

In conclusion, this study showed that \( V_{\text{peak}} \) and, to a lesser extent, Acc measured by oesophageal Doppler could be used as indicators to estimate the LV systolic performance and to assess the effects of inotropic therapy.

Authors’ contributions
X.M. conceived the study, performed analysis and interpretation of the data, and drafted the manuscript; J.-M.R. performed the collection of data and contributed to analysis and interpretation of the data; M.J. performed the collection of data and contributed to analysis and interpretation of the data; C.R. participated in the design of the study, contributed to analysis and interpretation of the data, and helped to draft the manuscript; J.-L.T. conceived the study, participated in its design, contributed to analysis and interpretation of the data, and helped to draft the manuscript. All authors read and approved the final manuscript.

Declaration of interest
J.-L.T. and X.M. are members of the Medical Advisory Board of Pulsion Medical Systems. The other authors have no financial competing interest to disclose.

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