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# Continual measurement of arterial $dP/dt_{max}$ enables minimally invasive monitoring of left ventricular contractility in patients with acute heart failure

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## Abstract

**Background:** Continuous, reliable evaluation of left ventricular (LV) contractile function in patients with advanced heart failure requiring intensive care remains challenging. Continual monitoring of  $dP/dt_{max}$  from the arterial line has recently become available in hemodynamic monitoring. However, the relationship between arterial  $dP/dt_{max}$  and LV  $dP/dt_{max}$  remains unclear. This study aimed to determine the relationship between arterial  $dP/dt_{max}$  and LV  $dP/dt_{max}$  assessed using echocardiography in patients with acute heart failure.

**Methods:** Forty-eight patients (mean age 70.4 years [65% male]) with acute heart failure requiring intensive care and hemodynamic monitoring were recruited. Hemodynamic variables, including arterial  $dP/dt_{max}$ , were continually monitored using arterial line pressure waveform analysis. LV  $dP/dt_{max}$  was assessed using continuous-wave Doppler analysis of mitral regurgitation flow.

**Results:** Values from continual arterial  $dP/dt_{max}$  monitoring were significantly correlated with LV  $dP/dt_{max}$  assessed using echocardiography ( $r = 0.70$  [95% confidence interval (CI) 0.51–0.82];  $P < 0.0001$ ). Linear regression analysis revealed that LV  $dP/dt_{max} = 1.25 \times$  (arterial  $dP/dt_{max}$ ) ( $P < 0.0001$ ). Arterial  $dP/dt_{max}$  was also significantly correlated with stroke volume (SV) ( $r = 0.63$ ;  $P < 0.0001$ ) and cardiac output (CO) ( $r = 0.42$ ;  $P = 0.0289$ ). In contrast, arterial  $dP/dt_{max}$  was not correlated with SV variation, dynamic arterial elastance, heart rate, systemic vascular resistance (SVR), or mean arterial pressure. Markedly stronger agreement between arterial and LV  $dP/dt_{max}$  was observed in subgroups with higher SVR ( $N = 28$ ;  $r = 0.91$ ;  $P < 0.0001$ ), lower CO ( $N = 26$ ;  $r = 0.81$ ;  $P < 0.0001$ ), and lower SV ( $N = 25$ ;  $r = 0.60$ ;  $P = 0.0014$ ). A weak correlation was observed in the subjects with lower SVR ( $N = 20$ ;  $r = 0.61$ ;  $P = 0.0004$ ); in the subgroups with higher CO ( $N = 22$ ) and higher SV ( $N = 23$ ), no significant correlation was found.

**Conclusion:** Our results suggest that in patients with acute heart failure requiring intensive care with an arterial line, continuous calculation of arterial  $dP/dt_{max}$  may be used for monitoring LV contractility, especially in those with higher SVR, lower CO, and lower SV, such as in patients experiencing cardiogenic shock. On the other hand, there was only a weak or no significant correlation in the subgroups with higher CO, higher SV, and lower SVR.

**Keywords:** Left ventricle, Contractility,  $dP/dt$ , Systemic vascular resistance, Cardiac output, Stroke volume, Acute heart failure

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## Background

Left ventricular (LV) contractility is one of the most important parameters determining LV performance and cardiac function and, therefore, directly influences global hemodynamic status [1]. Clinical conditions with impaired LV contractility, such as heart failure or septic cardiomyopathy, are frequent subjects of intensive and acute cardiology care [1]. There is, therefore, an apparent clinical need for bedside measurement or even monitoring of contractility. However, current options for the assessment of LV contractility are significantly limited. The reference method for the measurement of LV contractility (i.e., LV end-systolic elastance [2]) cannot be used in routine clinical practice due to invasiveness and technical issues [3, 4]. The maximum rate of LV pressure rise during ventricular contraction (LV  $dp/dt_{max}$ ) has been adopted as a surrogate marker of LV inotropic state and contractility [5]. This parameter is determinable in clinical settings; however, it requires direct LV pressure measurement, which is impractical and too invasive for LV contractility monitoring. LV  $dp/dt_{max}$  can also be estimated non-invasively using echocardiographic techniques [6, 7]. However, although this measurement can be performed repeatedly, it is not feasible for continuous monitoring and is frequently limited by low-quality signal.

Recently, a new surrogate has been proposed—arterial  $dp/dt_{max}$ . This parameter can be calculated from the arterial pressure waveform, obtained minimally invasively from a peripheral arterial line [8–10] or even non-invasively [11]. Arterial  $dp/dt_{max}$  is, therefore, available bedside and in patients with an arterial line already used for pressure monitoring and blood gas analyses; it does not require any additional invasive access. Moreover, arterial  $dp/dt_{max}$  can be measured on a beat-by-beat basis and, therefore, continually monitored. On the other hand, arterial  $dp/dt_{max}$  is not only determined by LV contraction but is also influenced by various peripheral arterial factors and load conditions [8–11]. Recently, several experimental studies demonstrating a significant relationship between arterial  $dp/dt_{max}$  and LV contractility have been published [8–10]. To date, however, clinical studies focusing on the relationship between arterial and LV  $dp/dt_{max}$  in patients with acute heart failure requiring intensive care and an arterial line are lacking. The aim of our study was, therefore, to assess the relationship between arterial and LV  $dp/dt_{max}$  in this patient population.

## Methods

### Study population

Consecutive patients admitted between January and September 2018 to the cardiology intensive care unit due to acute heart failure requiring an arterial line

for invasive blood pressure monitoring and central venous catheter were eligible for the study. Patients with moderate to severe aortic stenosis, those who required mechanical circulatory support, and those with absence of mitral regurgitation enabling measurement of LV  $dp/dt_{max}$  were excluded. All patients have to be at the time of measurement on stable doses of inotropes/vasopressors, on stable ventilation support, and with regular cardiac rhythm.

### Hemodynamic measurement

Arterial blood pressure (mean), central venous pressure (mean), heart rate, cardiac output (CO), stroke volume (SV), dynamic arterial elastance, and systemic vascular resistance (SVR) were measured using a clinical monitoring platform (EV1000, equipped with HPI software, Edwards Lifesciences, Irvine, CA, USA) connected to the arterial and central venous lines. SVR was calculated using the formula:  $SVR = 80 \times (MAP - CVP)/CO$ . The dynamic arterial elastance was defined as the ratio of pulse pressure variation and stroke volume variation. All parameters were calculated in the 20-s interval that contains few respiratory cycles. An arterial catheter was inserted into the left or right radial artery, and the left or right jugular vein was used for central venous access, with the tip of catheter in the superior vena cava.

### $dp/dt_{max}$ measurement

Arterial  $dp/dt_{max}$  was measured from the arterial pressure curve by the EV1000 system and HPI software. The system calculates  $dp/dt_{max}$  for each beat in a 20-s cycle, then the median value of all the  $dp/dt_{max}$  values in the 20-s interval is displayed; values obtained at the time of LV  $dp/dt_{max}$  were used in the analysis. LV  $dp/dt_{max}$  was measured at the end of expiration using transthoracic echocardiography (Phillips CX50, Amsterdam, The Netherlands) from the analysis of the mitral regurgitation jet by continuous-wave Doppler; calculation was based on the time interval (T) between blood flow of 1 m/s and 3 m/s using the formula:  $LV dp/dt_{max} = 32/T$  [6, 7]. Three measurements were performed at one time, and the mean values were used for analysis. The echocardiographic measurements were performed by an experienced physician who was blinded to the arterial  $dp/dt_{max}$  values.

### Statistical analysis

Gaussian distribution of the measurement data was tested using the Shapiro-Wilk normality test. Correlation was tested using the Spearman test by calculating the Spearman correlation coefficient. LV and arterial  $dp/dt_{max}$  were compared using the Bland-Altman analysis. Linear regression was used to derive the equation representing the relationship between LV and arterial  $dp/dt_{max}$ . The analyses were performed using GraphPad Prism version 7 (GraphPad

Software, Inc., La Jolla, CA, USA) and MedCalc (MedCalc Software, Ostend, Belgium);  $P < 0.05$  was considered to be statistically significant.

## Results

Forty-eight patients were enrolled in the study; baseline characteristics of the study population are summarized in Table 1. The mean age was 70.4 years, the majority were males (65%), and the main cause of acute heart failure was ischemic cardiomyopathy (65%). Eighty-five percent of patients were treated with intravenous inotropes, and the majority required vasopressors (73%).

The values from continual arterial  $dP/dt_{max}$  monitoring were significantly correlated with LV  $dP/dt_{max}$  assessed using echocardiography ( $r = 0.70$  [95% confidence interval (CI) 0.51–0.82];  $P < 0.0001$ ) (Fig. 1). Linear regression revealed that  $LV\ dP/dt_{max} = 1.25 \times (\text{arterial } dP/dt_{max})$  ( $P < 0.0001$ ). Arterial  $dP/dt_{max}$  was significantly correlated with SV ( $r = 0.63$  [95% CI 0.41–0.78];  $P < 0.0001$ ) and CO ( $r = 0.42$  [95% CI 0.14–0.63];  $P = 0.003$ ). In contrast, arterial  $dP/dt_{max}$  was not correlated with SV variation, dynamic arterial elastance, heart rate, SVR, or mean arterial pressure (Table 2).

The correlation between arterial and LV  $dP/dt_{max}$  was calculated also in subgroups above and below the mean value of the recorded variables. Marked differences in the correlation between arterial and LV  $dP/dt_{max}$  were observed in the subgroups based on the mean SVR, CO, and SV; on the other hand, similar correlation was observed in the subgroups based on SV variation ( $r = 0.54$

vs.  $r = 0.59$ ), dynamic arterial elastance ( $r = 0.70$  vs.  $r = 0.73$ ), heart rate (0.68 vs. 0.72), and mean arterial pressure ( $r = 0.80$  vs.  $r = 0.76$ ).

## SVR subgroups

The study population was divided into two groups according to the mean level of SVR ( $>$  or  $<$  900 dyn-s/cm<sup>5</sup>). In the subgroup of patients with lower SVR ( $<$  900 dyn-s/cm<sup>5</sup> [ $n = 28$ ]), a statistically significant correlation between arterial  $dP/dt_{max}$  and LV  $dP/dt_{max}$  was found ( $r = 0.61$  [95% CI 0.31 to 0.80];  $P = 0.0004$ ). However, in the subgroup with higher SVR ( $>$  900 dyn-s/cm<sup>5</sup> [ $n = 20$ ]), the correlation between arterial and LV  $dP/dt_{max}$  was markedly stronger and highly statistically significant ( $r = 0.91$  [95% CI 0.78 to 0.97];  $P < 0.0001$ ) (Fig. 2). Linear regression analysis revealed that in subgroup with higher SVR, LV  $dP/dt_{max}$  could be calculated according to the equation:  $LV\ dP/dt_{max} = 1.08 \times (\text{arterial } dP/dt_{max})$ .

## CO subgroups

In the subgroup of patients with lower CO ( $<$  6 L/min [ $n = 26$ ]), a strong and highly statistically significant correlation between arterial  $dP/dt_{max}$  and LV  $dP/dt_{max}$  was found ( $r = 0.81$  [95% CI 0.60 to 0.91];  $P < 0.0001$ ). In contrast, in the subgroup with higher CO ( $>$  6 L/min [ $n = 22$ ]), the correlation between arterial and LV  $dP/dt_{max}$  was not statistically significant ( $r = 0.29$  [95% CI -0.16 to 0.64];  $P = 0.18$ ) (Fig. 3). Linear regression revealed that in the subgroup with lower CO, LV  $dP/dt_{max}$  could be calculated according to the equation:  $LV\ dP/dt_{max} = 1.21 \times (\text{arterial } dP/dt_{max})$ .

## SV subgroups

In the subgroup of patients with lower SV ( $<$  65 mL [ $n = 25$ ]), a statistically significant correlation between arterial  $dP/dt_{max}$  and LV  $dP/dt_{max}$  was found ( $r = 0.60$  [95% CI 0.26 to 0.81];  $P = 0.0014$ ). In contrast, in the subgroup with higher SV ( $>$  65 mL [ $n = 23$ ]), the correlation of between arterial and LV  $dP/dt_{max}$  was not statistically significant ( $r = 0.38$  [95% CI -0.05 to 0.69];  $P < 0.07$ ) (Fig. 4). Linear regression revealed that in the subgroup with lower SV, LV  $dP/dt_{max}$  could be calculated according to the equation:  $LV\ dP/dt_{max} = 1.33 \times (\text{arterial } dP/dt_{max})$ .

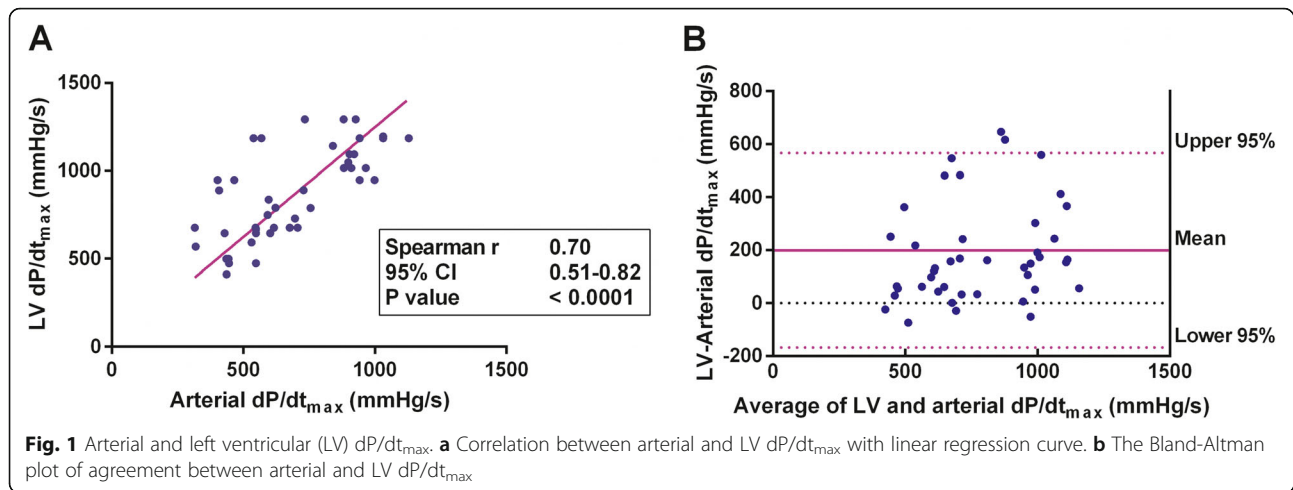
## Discussion

Our results demonstrate that in adult patients with acute heart failure, the values of arterial  $dP/dt_{max}$ , which can be continuously monitored by analysis of the pressure waveform, were significantly correlated with LV  $dP/dt_{max}$ . An even better agreement between arterial  $dP/dt_{max}$  and LV  $dP/dt_{max}$  was observed in subgroups with higher SVR, lower CO, and lower SV. This observation is particularly important because monitoring LV contractility is most desirable in patients with heart failure with critical hemodynamic

**Table 1** Baseline characteristics ( $n = 48$ )

Characteristics	Value
Male sex	31 (65)
Age (years, mean $\pm$ SD)	70.4 $\pm$ 8.1
Decompensated CHF	31 (65)
De novo AHF	17 (35)
Ischemic cardiomyopathy	31 (65)
Dilated cardiomyopathy	7 (15)
Acute myocardial infarction	15 (31)
Severe mitral regurgitation	19 (40)
Left ventricular ejection fraction (% , mean $\pm$ SD)	28 $\pm$ 10
Mechanical ventilation	17 (35)
Inotropes (dobutamine, milrinone)	41 (85)
Vasopressors (norepinephrine, vasopressin)	35 (73)
Heart rate (beats/min, mean $\pm$ SD)	92.7 $\pm$ 11.7
Mean arterial pressure (mmHg, mean $\pm$ SD)	74.0 $\pm$ 5.2
Cardiac output (L/min, mean $\pm$ SD)	6.0 $\pm$ 1.2
Stroke volume (mL, mean $\pm$ SD)	65.5 $\pm$ 10.1
Systemic vascular resistance (dyn-s/cm <sup>5</sup> , mean $\pm$ SD)	891.5 $\pm$ 236.3

Data presented as  $n$  (%) unless otherwise indicated. CHF chronic heart failure, AHF acute heart failure



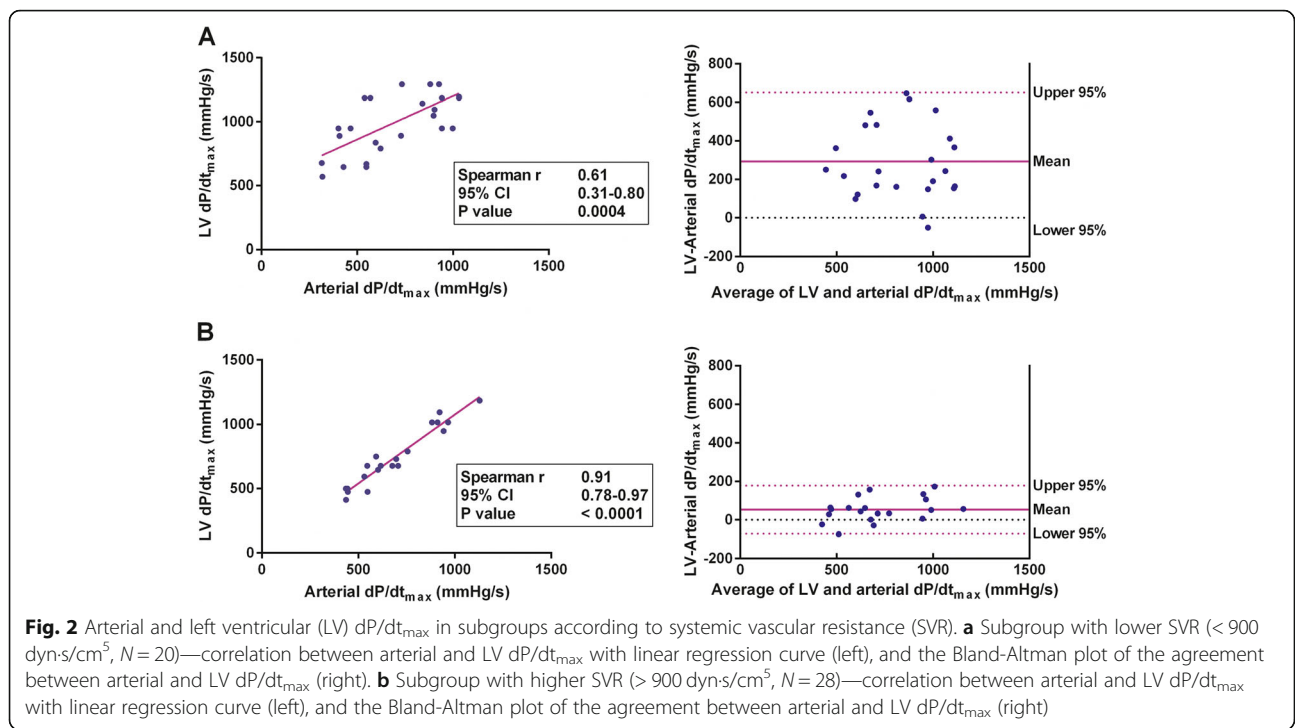
collapse, such as in cardiogenic shock, characterized by increased SVR and decreased CO and SV. In contrast, in the subgroups with lower SVR, higher CO, and higher SV, the correlation was weak or even absent.

Acute heart failure is a critical condition in which LV contractility is commonly depressed, and therapeutic strategies are frequently based on increasing inotropy [12]. Although LV contractility status can be routinely and intermittently assessed using echocardiography in patients with acute heart failure, even with the use of new techniques, the measurement can be inaccurate and interpretation difficult [13–17]. There are currently highly limited options for continuous LV contractility monitoring. In contrast to echocardiographic techniques, the maximal rate of arterial pressure increases during systole (arterial dP/dt<sub>max</sub>) can be easily and continuously calculated from the pressure waveform. It has been shown in animal studies that arterial dP/dt<sub>max</sub> may correlate with LV contractility status under various hemodynamic conditions. In a porcine model of endotoxin-induced shock and catecholamine infusion, Morimont et al. [8] observed that arterial dP/dt<sub>max</sub> was significantly correlated with LV

contractility measured by LV end-systolic elastance (Ees) or LV dP/dt<sub>max</sub>. The authors also found a better correlation when adequate vascular filling according to the arterial pulse pressure variation was achieved. These results are consistent with our observations. We have demonstrated a strong relationship between arterial and LV dP/dt<sub>max</sub> under higher SVR, which could be also a result of increased vascular filling. Monge Garcia et al. [10] analyzed the relationship among arterial dP/dt<sub>max</sub>, LV dP/dt<sub>max</sub>, and Ees in sequential changes of afterload, preload, and contractility in pigs. In this study, arterial dP/dt<sub>max</sub> enabled the tracking of Ees changes, especially during the modification of afterload and contractility, and changes in cardiac contractility (i.e., Ees) were the main determinants of arterial dP/dt<sub>max</sub> changes. Moreover, these observations are in good agreement with our results; Monge Garcia et al. recorded higher values of LV dP/dt<sub>max</sub> in comparison with arterial dP/dt<sub>max</sub>, similar to our study. A good correlation between arterial dP/dt<sub>max</sub> and LV dP/dt<sub>max</sub> in heart failure patients was also reported in the study by Tartiere et al. [11], in which the dP/dt<sub>max</sub> from the radial artery was assessed non-invasively using applanation tonometry and LV dP/

**Table 2** Correlation between arterial dP/dt<sub>max</sub> and other recorded hemodynamic variables

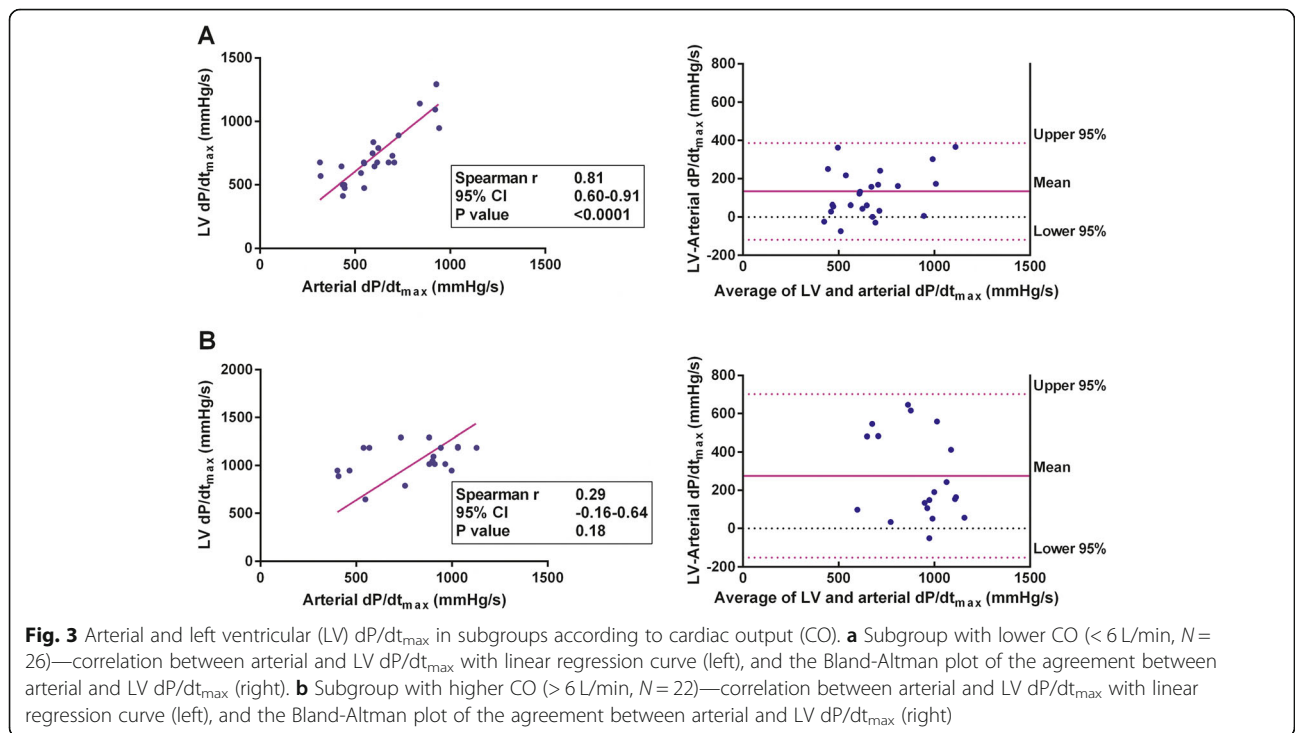
Variable	Spearman's <i>r</i>	95% confidence interval	<i>P</i> value
Stroke volume	0.6297	0.4137 to 0.7786	< 0.0001
Cardiac output	0.419	0.1446 to 0.6336	0.003
Stroke volume variation	-0.2635	-0.5159 to 0.03099	0.0703
Dynamic arterial elastance	0.06733	-0.2293 to 0.3525	0.6493
Heart rate	-0.06501	-0.3505 to 0.2315	0.6607
Systemic vascular resistance	-0.1734	-0.4431 to 0.1251	0.2385
Mean blood pressure	0.345	-0.02172 to 0.6298	0.0574

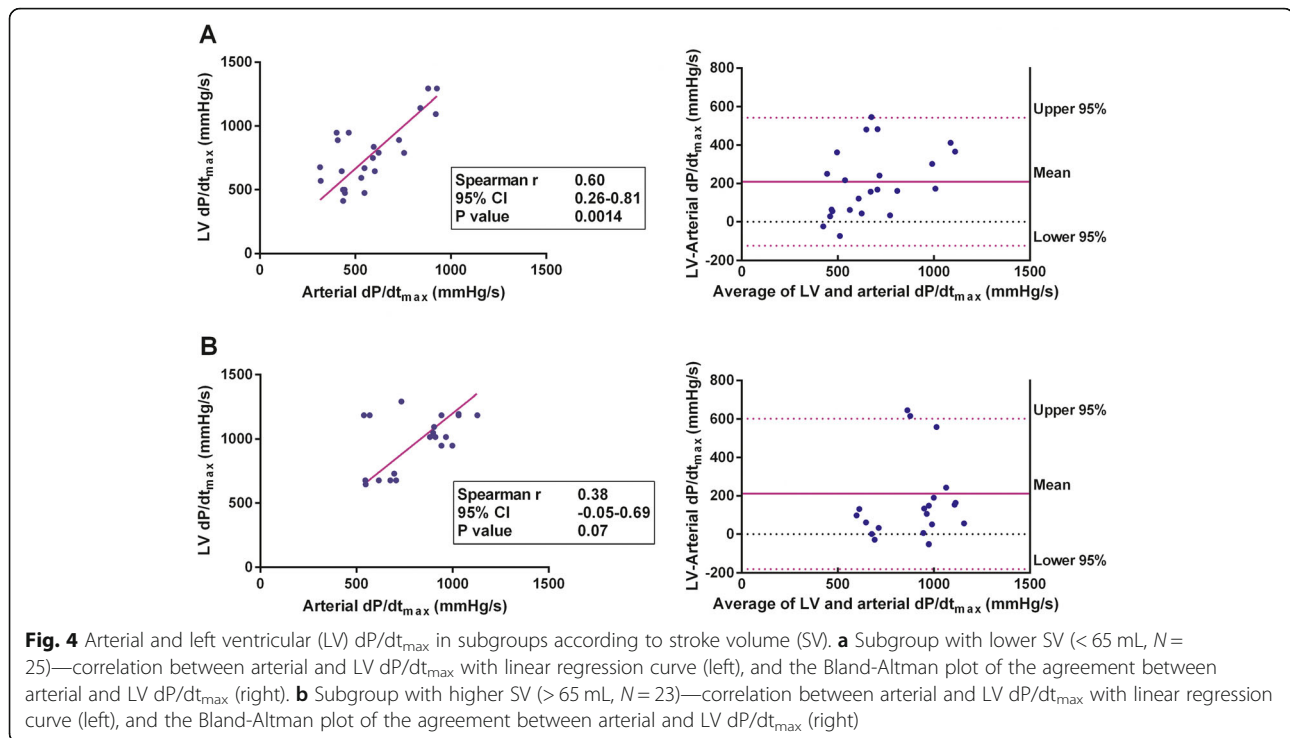


$dP/dt_{max}$ . Our results are also consistent with the observation by Scolletta et al. [18] who reported significant correlation between arterial  $dP/dt_{max}$  and LV  $dP/dt_{max}$  in a group of critically ill patients. Furthermore, and again similar to our results, a very close linear relationship was found between arterial  $dP/dt_{max}$  from the

femoral artery and invasively measured LV  $dP/dt_{max}$  (with a catheter in the LV) in patients scheduled for coronary artery bypass surgery [9]; arterial  $dP/dt_{max}$  also underestimated LV  $dP/dt_{max}$  in this study.

In contrast, Kim et al. [19] studied the relationship between arterial  $dP/dt_{max}$  from the radial artery, aortic  $dP/dt_{max}$





$dt_{max}$ , and selected echocardiographic variables such as LV ejection fraction or LV fractional shortening in children undergoing congenital heart disease surgery. They did not find a significant correlation between arterial  $dP/dt_{max}$  and the other variables, and these observations were explained by differences between radial artery and aortic pressure waveforms. The discrepancy with our results can be elucidated by the other measured parameters in Kim et al.'s study. While we used LV  $dP/dt_{max}$  as a surrogate parameter of LV contractility, Kim et al. measured LV ejection fraction and LV fractional shortening, which are not the accepted markers of LV contractility, because they are influenced by many other factors (e.g., preload, mitral regurgitation). The observations in the study by Kim et al. could be also influenced by CO or SVR (or vascular filling), which were not recorded. Recently, Vaquer et al. [20] published a study focused on femoral arterial  $dP/dt_{max}$  in critically ill patients, predominantly with septic shock. They observed increase in arterial  $dP/dt_{max}$  after administration of dobutamine and norepinephrine but not after volume expansion. The changes in arterial  $dP/dt_{max}$  were strongly correlated with the changes in pulse pressure and systolic arterial pressure in all interventions including volume expansion. Vaquer et al. conclude that femoral arterial  $dP/dt_{max}$  is, therefore, an unreliable estimate of LV systolic function [20]. Our study was not designed to evaluate the relationship between  $dP/dt_{max}$  and LV systolic function; we focused on the comparison of the arterial  $dP/dt_{max}$  and the LV  $dP/dt_{max}$  as a surrogate

marker of LV contractility. Although the study group characteristics are different, the observation of increase in arterial  $dP/dt_{max}$  after administration of agents with inotropic effect (dobutamine, norepinephrine) in the study by Vaquer et al. [20] is in a good agreement with our results showing significant correlation between arterial and LV  $dP/dt_{max}$ . Our observation that correlation between arterial and LV  $dP/dt_{max}$  depends on SVR is also consistent with the results by Vaquer et al. [20] describing that femoral  $dP/dt_{max}$  is influenced by LV preload and afterload.

Several authors reported that the arterial  $dP/dt_{max}$  is significantly influenced by vascular filling conditions [8, 10, 20]. We observed in our study that the correlation between arterial and LV  $dP/dt_{max}$  was influenced by SVR, which reflects loading conditions; however, SV variation or dynamic arterial elastance had no effect on the relationship between arterial and LV  $dP/dt_{max}$ . This contrast can be at least partly explained by the fact that in our study, the measurement was done in a single time point, while in the other studies, serial measurements were performed enabling evaluation of dynamic changes. In addition, we included entirely patients with acute left heart failure, where the LV preload is usually increased that may or may not be accompanied by changes in other indirect markers of vascular filling. Our study had several limitations, the first of which was possible bias caused by the small sample size. We designed only a pilot study focusing primarily on feasibility; however, a larger trial should be performed to confirm our results. We also acknowledge that arterial  $dP/dt_{max}$  is not only a function of

LV contractility but is influenced by many other factors, at least by arterial vessel wall characteristics (e.g., arterial elasticity and stiffness), which was not assessed in the present study. We did not record pulse pressure enabling to calculate arterial elastance. It can be assumed that there can be marked individual differences in arterial system properties in heart failure patients, who often present with other diseases and various degrees of peripheral atherosclerosis. Patients with moderate to severe aortic stenosis were ineligible for this study; however, we cannot exclude the possibility that even mild aortic stenosis may have influenced the results. Moreover, in our study, we have performed only single measurements at one time point in each patient. Our study, therefore, was not designed to evaluate the trends in arterial  $dP/dt_{max}$  changes. In addition, while the LV  $dP/dt_{max}$  values were obtained at the end of expiration, the arterial  $dP/dt_{max}$  were calculated as a median value from 20-s interval, therefore not at the same period of respiratory cycle. Finally, LV  $dP/dt_{max}$  is only a surrogate marker of LV contractility and measurement of this parameter using echocardiography can be inaccurate.

## Conclusions

Our results suggest that in patients with acute heart failure requiring intensive care with an arterial line, continuous calculation of arterial  $dP/dt_{max}$  may be used for monitoring LV contractility, especially in those with higher SVR, lower CO, and lower SV, such as in patients experiencing cardiogenic shock. On the other hand, there was only a weak or no significant correlation in the subgroups with higher CO, higher SV, and lower SVR.

## Abbreviations

CO: Cardiac output; Ees: End-systolic elastance; LV: Left ventricular; SV: Stroke volume; SVR: Systemic vascular resistance; T: Time interval

## Authors' contributions

PO and DV conceived and designed the study. DV, AK, MJ, and JN contributed to the recruitment of trial participants and were responsible for the acquisition of data and their integrity. PO, MJ, and JN did the statistical analysis and prepared the figures and tables. All authors participated in the interpretation of the results. PO wrote the first draft of the manuscript. DV, AK, MJ, and JN critically reviewed and revised the manuscript for important intellectual content. All authors approved the final version for publication.

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## Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

## Ethics approval and consent to participate

The present study was performed in accordance with the principles of the Declaration of Helsinki, and the study protocol was approved by the Institutional Ethics Committee of the Na Homolce Hospital (Prague, Czech Republic). Informed consent was obtained from conscious patients before measurements. In unconscious subjects at the time of measurement, informed consent was obtained retrospectively. In two deceased patients, informed consent was obtained from next of kin.

## Consent for publication

Not applicable

## Competing interests

PO received speaker's fee from Edwards Lifesciences. The other authors declare that they have no competing interests.

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