

# Left ventricular ejection time: a potential determinant of pulse wave velocity in young, healthy males

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**Objective** Pulse wave velocity (PWV) is a classic marker of vascular stiffness. Recent studies showed that heart rate is an important determinant of PWV. The purpose of this study was to evaluate the role of myocardial function in determining PWV under resting conditions and under adrenergic stimulation.

**Design and methods** Hemodynamic parameters were investigated under resting conditions in 102 young, healthy males and under stimulation of either  $\beta$ - or  $\alpha_2$ -adrenoceptors in six young, healthy males. PWV was determined from pressure tracing over the carotid and femoral artery. Central hemodynamics were assessed by impedance cardiography and systolic time intervals. Simple ( $r$ ) and multiple ( $\beta$ ) regression analyses were used to assess the relationships between PWV and hemodynamic parameters.

**Results** Under resting conditions, PWV was correlated to age ( $\beta = 0.259$ ,  $P = 0.0052$ ), diastolic blood pressure ( $\beta = 0.279$ ,  $P = 0.0072$ ) and left ventricular ejection time ( $\beta = -0.314$ ,  $P = 0.0277$ ). Under  $\alpha_2$ -adrenergic stimulation PWV was only correlated to diastolic blood pressure (DBP) ( $\beta = 0.806$ ,  $P = 0.0020$ ). Under  $\beta$ -adrenergic stimulation

PWV was only correlated to left ventricular ejection time index ( $r = -0.52$ ,  $P = 0.0325$ ).

**Conclusions** Left ventricular ejection time may be an important determinant of pulse wave velocity under resting and adrenergic conditions in young, healthy males. Further studies are needed to evaluate this relationship in other populations including females and patients with cardiovascular disease. *J Hypertens* 21:2125–2132 © 2003 Lippincott Williams & Wilkins.

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## Introduction

Pulse wave velocity (PWV) is a classical marker of arterial stiffness and is widely used to assess the elastic properties of the arterial tree [1–3]. Several anthropometric and hemodynamic factors have been shown to influence pulse wave velocity including age and blood pressure (BP) [4–6]. Only recently, an association between heart rate and pulse wave velocity has been recognized and a positive correlation between PWV and heart rate has been consistently reported [7–10]. Furthermore, Drinnan *et al.* concluded from their experiments in 15 normal healthy subjects that changes in pulse transit time, from which PWV is calculated, follow changes in heart rate suggesting PWV may be influenced by myocardial function [11].

The pulse wave velocity is the speed with which the pulse wave radiates from the semilunar valves over the arterial system, gradually disappearing in the smaller branches. The pulse wave is generated during left ventricular ejection by transferring a static pressure into

a travelling pressure wave. Hence, it is conceivable that myocardial function may influence characteristics of the pulse pressure wave. However, the relationship between myocardial function and PWV has not been fully elucidated.

The purpose of the present study was to further characterize the relationship between PWV and myocardial function in a homogenous study population of young, healthy males. Hemodynamic relationships were evaluated under resting conditions and under adrenergic stimulation using systolic time intervals and impedance cardiography.

## Materials and methods

### Study population

The study population included young, male volunteers who gave informed, written consent. All subjects were drug-free and judged to be healthy on the basis of medical history, physical examination, electrocardiogram and routine laboratory screening. Investigations

were carried out in the Department of Nephrology at the University of Essen, Germany. The study was approved by the Ethics Committee of the University of Essen Medical School and was in accordance with the principles laid down in the Declaration of Helsinki.

Measurements were performed in a quiet, temperature-controlled (24°C) room. On the study day, volunteers reported to the laboratory at 0700–0800 h after an overnight fast to eliminate postprandial hemodynamic changes. During the investigation subjects remained in the supine position. Each subject was instrumented with a blood pressure (BP) cuff, circular tape electrodes to measure transthoracic impedance and a microphone for phonocardiographic recordings. The characteristics of all study subjects are summarized in Table 1.

### Study protocol

#### **Study I: Hemodynamics under resting conditions**

We analyzed hemodynamic data measured under resting conditions that were obtained from 102 young, healthy males investigated in our laboratory. Measurements of hemodynamics were performed after a period of 30 min of complete rest after an overnight fast.

#### **Study II: Hemodynamics under myocardial and vascular stimulation**

Six healthy, young male volunteers received an increasing dose of  $\alpha$ -methylnoradrenaline (AMN) with concomitant blockade of either  $\alpha_2$ -adrenoceptors with yohimbine or  $\beta$ -adrenoceptors with propranolol [12]. AMN was administered by intravenous (i.v.) infusion in incremental dose steps of 0.1, 0.2, and 0.4  $\mu\text{g}/\text{kg}$  per min for 10 min at each dose level. At the end of each dose level measurements of the hemodynamic parameters were performed. For safety reasons infusions of AMN were terminated if systolic blood pressure (SBP) rose by more than 50 mmHg, diastolic blood pressure (DBP) by more than 30 mmHg or if heart rate (HR) increased by more than 50 bpm or fell (with propranolol pretreatment) below 40 bpm. Subjects received i.v.

infusions of AMN after pretreatment with  $\alpha_2$ -adrenoceptor blockade with yohimbine (given as a loading dose of 32  $\mu\text{g}/\text{kg}$  by slow i.v. injection over 10 min, followed by an i.v. maintenance dose of 1  $\mu\text{g}/\text{kg}$  per min) or  $\alpha_2$ -adrenoceptor blockade with propranolol (given as an i.v. loading dose of 62.5  $\mu\text{g}/\text{kg}$  over 10 min followed by a maintenance infusion of 0.45  $\mu\text{g}/\text{kg}$  per min). The i.v. loading dose injections of yohimbine and propranolol were administered 45 min prior to the start of the AMN infusion. The maintenance infusions of yohimbine and propranolol were started immediately following the administration of the loading dose and were maintained until the end of the AMN infusion. The chosen doses had been shown to be safe and to produce a significant degree of adrenoceptor blockade in young, healthy volunteers [13,14]. With propranolol pre-treatment, infusion of AMN resulted in an increase of DBP with a secondary reflex bradycardia. With propranolol only three subjects completed the last dose of 0.4  $\mu\text{g}/\text{kg}$  per min AMN. In one subject, measurement of the femoral pulse wave was not possible at the highest dose of AMN for technical reasons.

We used relative values for all hemodynamic parameters for data analysis under adrenergic stimulation. That is, parameters were analyzed as changes from baseline. Baseline measurements were taken immediately prior to the infusion of  $\alpha$ -methylnoradrenaline. Subjects were studied on separate occasions, and study days were at least 1 week apart to ensure no carry-over effects of  $\alpha_2$  and  $\beta$ -adrenoceptors blockade [12].

The cardiovascular effects of AMN are mediated by both  $\alpha_2$ -adrenoceptors and  $\beta$ -adrenoceptors [12]. Thus, concomitant blockade of  $\alpha_2$ -adrenoceptor would allow selective targeting of  $\beta$ -adrenoceptors by AMN thereby stimulating myocardial function. With simultaneous inhibition of  $\beta$ -adrenoceptors, AMN selectively stimulates  $\alpha_2$ -adrenoceptors thereby causing vasoconstriction [12]. Further details of the study protocol have been previously described [12].

**Table 1 Hemodynamic and anthropometric characteristics of the study population**

Parameter	Mean	SEM
Age (years)	27.4	0.4
Height (cm)	183.2	0.7
Weight (kg)	80.5	0.9
Body mass index ( $\text{kg}/\text{m}^2$ )	24.0	0.2
Pulse wave velocity (m/s)	5.88	0.07
Systolic blood pressure (mmHg)	120.3	0.8
Diastolic blood pressure (mmHg)	73.4	0.8
Heart rate (bpm)	57.3	0.9
Stroke volume (ml)	96.0	2.5
Cardiac output (l/min)	5.4	0.1
Total peripheral resistance ( $\text{dyne s}/\text{cm}^5$ )	1406	37.5
Electromechanical systole index ( $\text{QS}_2\text{I}$ )	497.4	3.8
Left ventricular ejection time index (ms)	407.4	1.3
Pre-ejection period index (ms)	134.8	1.2

### Anthropometric and hemodynamic parameters

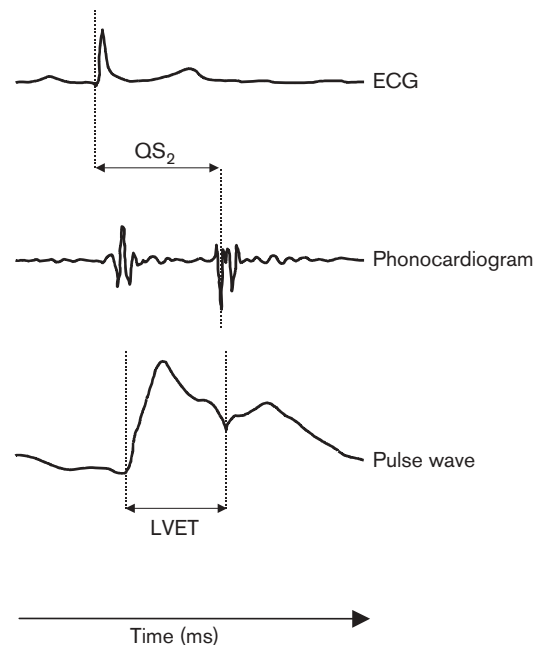
Height and weight were measured and body mass index (BMI) was calculated as weight to height squared. BP (mmHg) was measured with a standard mercury sphygmomanometer with the disappearance of Korotkoff's sound defined as DBP. Mean arterial pressure (MAP) was defined as DBP plus one-third of pulse pressure (PP), which was calculated by subtracting DBP from SBP.

Pulse transmission time (ms) was obtained non-invasively from pressure tracings over the carotid and femoral artery that were recorded at high paper speed (100 mm/s) using a Siemens-Cardirex<sup>®</sup> multichannel ink jet recorder (Siemens Medizintechnik, Erlangen, Germany) as previously described [15–17]. The foot of the pulse waves was identified as the beginning of the initial upstroke. This initial upstroke was determined by the first derivative, which was graphically obtained by a tangent. Pulse transmission time was determined from the time delay between the two corresponding feet of the carotid and the femoral pulse waveforms. PWV (m/s) was calculated as the ratio between the distance travelled by the pulse wave and pulse transmission time [15,16]. The intra-day variability of PWV in our laboratory was only 3.8% as expressed by the coefficient of variation [12]. All measurements were taken by the same investigator.

Systolic time intervals (STI) were measured according to standard techniques [18,19] from simultaneous recordings of an electrocardiographic lead, a phonocardiogram and a carotid pulse tracing at high paper speed (100 mm/s) using a Siemens-Cardirex<sup>®</sup> multichannel ink jet recorder as previously described [12,14,20,21]. From these recordings we determined the duration of the RR-interval to calculate HR (bpm), the duration of the electromechanical systole (QS<sub>2</sub>), and the duration of the left ventricular ejection time (LVET) (ms). Figure 1 illustrates how these cardiac time intervals are obtained. The duration of the pre-ejection period (PEP) (ms) was calculated by subtraction of LVET from QS<sub>2</sub>. QS<sub>2</sub>, PEP and LVET were corrected for HR under resting conditions [18] and under stimulation [20] to yield QS<sub>2</sub>I, PEPI and LVE TI. The formula used to correct LVET for HR under resting conditions was: LVE TI = 1.7 HR + LVET [18]. Using increasing doses of atropine, we determined in our laboratory the formula to correct LVET for HR under stimulation in young, healthy males: LVE TI = 1.1 HR + LVET [20]. The duration of the diastole (ms) was calculated by subtraction of QS<sub>2</sub> from the RR-interval.

We used impedance cardiography to measure central hemodynamics. Impedance cardiography agrees closely with measurements obtained by Doppler echocardiography or thermodilution [22] and is acceptable for

Fig. 1



The parameters of systolic time intervals. The total electromechanical systole (QS<sub>2</sub>) and the left ventricular ejection time (LVET) are derived from simultaneous recordings of the electrocardiogram (ECG), phonocardiogram, and pulse wave. QS<sub>2</sub> is the time interval from the beginning of the electrical myocardial depolarization displayed in the ECG to the closure of the aortic valve as determined by the second heart sound displayed on the phonocardiogram. LVET is the time interval between the foot and the incisure of the carotid pulse wave. The duration of the pre-ejection period (PEP) was calculated by subtraction of LVET from QS<sub>2</sub>.

clinical use particularly in studies investigating young subjects free from any cardiovascular disease [23]. Stroke volume (SV) (ml) was measured using the standard approach with circular tape electrodes and graphical signal analysis according to Kubicek's equation [24,25]. A 'Kardio-Dynagraph' was used to record changes in transthoracic impedance (Heinz Diefenbach Elektromedizin, Frankfurt, Germany). Cardiac output (CO) (l/min) was calculated as  $CO = HR \times SV/1000$ . Total peripheral resistance (TPR) (dyne/s per cm<sup>-5</sup>) was calculated as MAP divided by CO.

For each time point, five measurements were taken of each hemodynamic parameter and the mean of these five measurements was used for the analysis under resting conditions and adrenergic stimulation.

### Statistical analysis

#### Study I: Hemodynamics under resting conditions

Resting hemodynamic parameters from the 102 subjects were used for statistical analysis. Simple linear regression analysis (Pearson) was applied to detect and describe strength and direction of correlations of PWV

to hemodynamic parameters. Multiple and stepwise regression analysis was applied to those parameters that were found to be significantly correlated to PWV using simple regression analysis.

### Study II: Hemodynamics under myocardial and vascular stimulation

Hemodynamic parameters under stimulation were analyzed as changes from baseline values. Baseline measurements were taken immediately prior to adrenergic stimulation by AMN. A dose-response curve was obtained to evaluate the effect of adrenergic stimulation on PWV. Data points of all doses for all six subjects were pooled for regression analysis. Simple linear regression analysis (Pearson) was applied to detect correlations of pulse wave velocity to hemodynamic parameters. Multiple regression analysis was applied to those parameters that were found to be significantly correlated to PWV using simple regression analysis.

The strength of correlation was quantified by the correlation coefficients of simple ( $r$ ) and multiple ( $\beta$ ) regression analysis. A  $P$  value less than 0.05 was considered to be significant. All values have been expressed as means  $\pm$  SEM. Statistical analysis was performed with GB-Stat V6.0 for MS Windows (Dynamic Microsystems, Inc., Silver Spring, Maryland, USA).

Even though pulse wave velocity is influenced by other anthropometric and hemodynamic parameters including age and diastolic blood pressure [17], we did not correct pulse wave velocity for these parameters as they were included in the multiple regression analysis.

## Results

### Study I: Hemodynamics under resting conditions

Table 2 shows the results of simple regression analysis between PWV and hemodynamics under resting conditions. PWV was significantly correlated to age, DBP, HR,  $QS_2I$ , and LVETI. These relationships were further analyzed by multiple regression analysis using PWV as dependent variable and age, DBP, HR,  $QS_2$ , and LVET as independent variables.  $QS_2$  and LVET were used instead of  $QS_2I$  and LVETI because HR was also submitted to multiple regression analysis. PWV remained significantly correlated only to age, DBP, and LVET (Table 2). The relationships between PWV and age, DBP, and LVET, respectively, are shown in Figure 2. Stepwise multiple regression showed that age, DBP and LVET were independent predictors PWV.

A positive relationship between HR and PWV has been consistently reported [7–10] and was also found in this study when simple regression analysis was applied. In order to determine the time interval of the cardiac

**Table 2 Results of simple and multiple regression analyses for hemodynamic parameters under resting conditions using pulse wave velocity as dependent variable. Correlation coefficients of simple ( $r$ ) and multiple regression analysis ( $\beta$ ) and  $P$  values ( $P$ ) are displayed**

Pulse wave velocity	Simple regression		Multiple regression	
	$P$	$r$	$P$	$\beta$
Age (years)	<b>0.0026</b>	<b>0.301</b>	<b>0.0052</b>	<b>0.259</b>
SBP (mmHg)	0.1069	0.160	–	–
DBP (mmHg)	<b>&lt;0.0001</b>	<b>0.421</b>	<b>0.0072</b>	<b>0.279</b>
HR (bpm)	<b>0.0125</b>	<b>0.245</b>	0.5672	–0.077
SV (bpm)	0.4903	–0.083	–	–
CO (ml/min)	0.5050	0.067	–	–
TPR (dyne s/cm <sup>5</sup> )	0.5748	0.056	–	–
$QS_2I$ (ms)	<b>0.0333</b>	<b>–0.210</b>	–	–
LVETI (ms)	<b>0.0132</b>	<b>–0.245</b>	–	–
PEPI (ms)	0.1816	0.133	–	–
$QS_2$ (ms)	<b>0.0070</b>	<b>–0.264</b>	0.8311	0.039
LVET (ms)	<b>0.0001</b>	<b>–0.371</b>	<b>0.0277</b>	<b>–0.314</b>
PEP (ms)	0.5733	0.056	–	–

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance;  $QS_2I$ , electromechanical systole index; LVETI, left ventricular ejection time index; PEPI, pre-ejection period index; LVET, left ventricular ejection time; PEP, pre-ejection period.

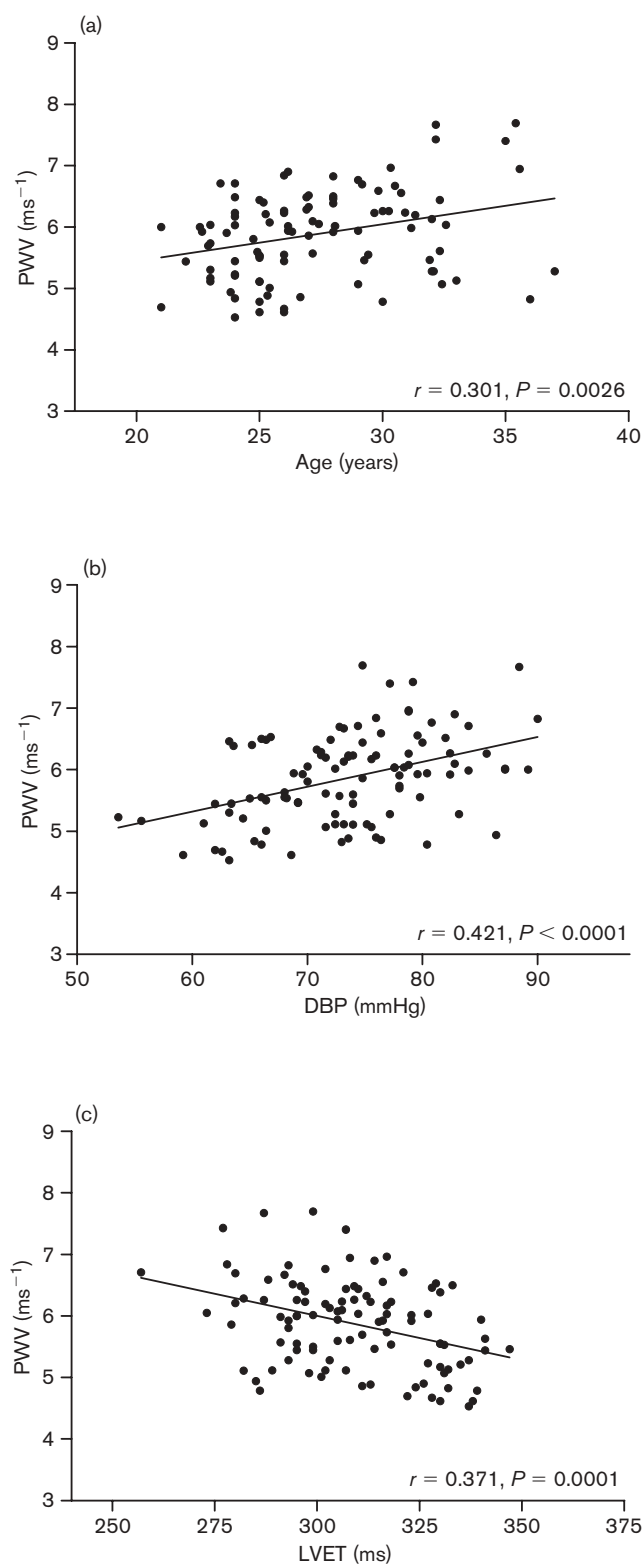
cycle responsible for this relationship, we performed a multiple regression analysis using PWV as dependent variable and the time intervals of the heart cycle PEP, LVET and diastole duration as independent variables under resting conditions. PWV was significantly correlated only to LVET ( $P = 0.0078$ ,  $\beta = -0.353$ ) but not to PEP ( $P = 0.4701$ ,  $\beta = 0.071$ ) or duration of the diastole ( $P = 0.8599$ ,  $\beta = 0.024$ ).

### Study II: Hemodynamics under myocardial and vascular stimulation

Stimulation of both myocardial function by  $\beta$ -adrenoceptors and vascular function by  $\alpha_2$ -adrenoceptors resulted in a significant dose-dependent increase in PWV [12]. AMN with concomitant  $\beta$ -adrenoceptor blockade also significantly increased SBP, DBP, TPR, and decreased HR and CO, as previously shown [12]. AMN with concomitant  $\alpha_2$ -adrenoceptor blockade increased SBP, HR, CO and decreased DBP, TPR, PEPI, and LVETI [12].

Results of simple and multiple regression analyses used to detect significant correlations between PWV and hemodynamics under vascular stimulation are shown in Table 3. Following stimulation by AMN via  $\alpha_2$ -adrenoceptors, PWV was correlated to SBP, DBP, TPR,  $QS_2I$ , and PEPI when simple regression analysis was applied. DBP, TPR,  $QS_2I$ , and PEPI were subjected to multiple regression analysis using PWV as dependent variable. After multiple regression analysis, PWV remained significantly correlated only to DBP. Figure 3a shows the relationship between PWV and DBP under vascular stimulation. SBP was removed from multiple

Fig. 2



Graph of pulse wave velocity (PWV) as function of (a) age, (b) diastolic blood pressure (DBP) and (c) left ventricular ejection time (LVET) under resting conditions in 102 young, healthy males described by simple regression analysis.

**Table 3** Results of simple and multiple regression analyses for hemodynamic parameters (expressed as changes from baseline) under vascular stimulation via  $\alpha_2$ -adrenoceptors using pulse wave velocity as dependent variable. Correlation coefficients of simple ( $r$ ) and multiple regression analysis ( $\beta$ ) and  $P$  values ( $P$ ) are displayed

	Simple regression		Multiple regression	
	$P$	$r$	$P$	$\beta$
Delta pulse wave velocity				
Delta SBP (mmHg)	< 0.0001	0.888	–	–
Delta DBP (mmHg)	< 0.0001	0.910	0.0020	0.806
Delta HR (bpm)	0.8132	0.067	–	–
Delta SV (bpm)	0.0881	–0.455	–	–
Delta CO (ml/min)	0.9094	0.032	–	–
Delta TPR (dyne $\text{s}/\text{cm}^5$ )	0.0013	0.750	0.1499	0.239
Delta $\text{QS}_2\text{I}$ (ms)	0.0010	0.761	0.2630	0.210
Delta LVETI (ms)	0.2106	0.343	–	–
Delta PEPI (ms)	0.0276	0.567	0.0810	–0.317

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance;  $\text{QS}_2\text{I}$ , electromechanical systole index; LVETI, left ventricular ejection time index; PEPI, pre-ejection period index.

regression analysis because SBP and DBP showed high multicollinearity. That is, only DBP remained significantly correlated to PWV ( $P = 0.0303$ ) when multiple regression analysis was applied using PWV as dependent and SBP and DBP as independent variables.

Results of myocardial stimulation by AMN via  $\beta$ -adrenoceptors for simple regression are shown in Table 4. PWV was inversely correlated to LVETI but was not correlated to any other hemodynamic parameter. Since only one parameter was found to be significantly correlated to PWV, multiple regression analysis was not performed. Figure 3b shows the relationship between PWV and LVETI under myocardial stimulation. There was also an inverse correlation between PWV and LVETI, when the curves of the six subjects were analyzed individually (Fig. 3c).

## Discussion

In this study we found an inverse relationship between pulse wave velocity and left ventricular ejection time in young healthy males. We were able to detect this relationship under resting conditions as well as under  $\beta$ -adrenergic stimulation.

First, we investigated possible determinants of PWV under resting conditions in 102 young, healthy males. We found that in addition to age only DBP and left ventricular ejection time were correlated to PWV. In a second study, we determined the hemodynamic parameters that mediate the increase in PWV under myocardial and vascular stimulation. The increase in PWV under  $\beta$ -adrenergic stimulation was only correlated to left ventricular time index suggesting an interaction between PWV and myocardial function. To the best of

**Table 4 Results of simple regression analysis for hemodynamic parameters (expressed as changes from baseline) under myocardial stimulation via  $\beta$ -adrenoceptors using pulse wave velocity as dependent variable. Correlation coefficients of simple regression analysis ( $r$ ) and  $P$  values ( $P$ ) are displayed**

Delta pulse wave velocity	Simple regression	
	$P$	$r$
Delta SBP (mmHg)	0.3053	0.264
Delta DBP (mmHg)	0.4280	-0.206
Delta HR (bpm)	0.7008	-0.101
Delta SV (bpm)	0.4533	-0.195
Delta CO (ml/min)	0.2762	-0.280
Delta TPR (dyne s/cm <sup>5</sup> )	0.8395	0.053
Delta QS <sub>2</sub> l (ms)	0.4238	-0.208
Delta LVETI (ms)	<b>0.0325</b>	<b>-0.520</b>
Delta PEPI (ms)	0.2734	0.282

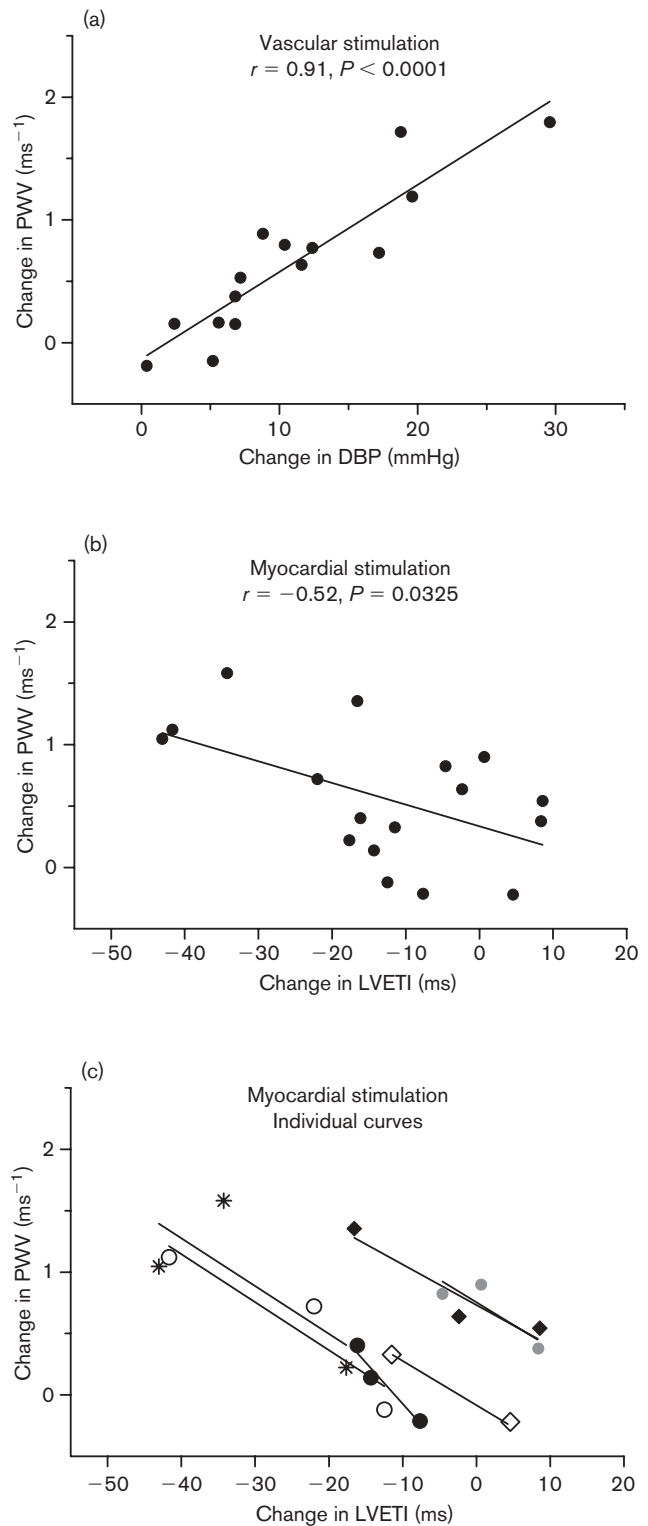
SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance; QS<sub>2</sub>l, electromechanical systole index; LVETI, left ventricular ejection time index; PEPI, pre-ejection period index.

our knowledge, this is the first study describing a relationship between PWV and LVET.

A possible explanation for this inverse relationship could relate to myocardial performance. The pulse wave is generated by the contracting heart and the velocity of the pressure wave can be measured intra-ventricularly [26]. When intraventricular pressure reaches aortic diastolic blood pressure, the aortic valve opens and the stroke volume is ejected into the proximal aorta. The initial speed of the pressure wave is mainly determined by the velocity of myocardial shortening which in turn is a function of wall movement and time during left ventricular ejection. In fact, LVET is correlated with shortening velocity [27]. Thus, an inverse relationship between LVET and PWV could be attributed to myocardial function. This idea is further supported to the observation that changes in cardiac afterload do not affect the duration of left ventricular ejection [18,28]. Therefore, it is unlikely that LVET is largely influenced by PWV, which reflects arterial stiffness and consequently ventricular afterload. More studies are needed to further explore the relationship between PWV and myocardial function.

Following vasoconstriction via  $\alpha_2$ -adrenoceptors, PWV was only correlated to DBP. These data suggest that the observed rise in PWV under  $\alpha_2$ -adrenergic stimulation may be mediated by the concomitant rise of blood pressure, which is a result of  $\alpha_2$ -adrenoceptor mediated vasoconstriction [29]. It is also possible that  $\alpha_2$ -adrenergic stimulation may directly affect the compliance of the aorta, thereby altering PWV, which is an estimate of aortic compliance. Recent studies have demonstrated that  $\alpha_2$ -adrenoceptors are involved in aortic vasoregulation [30–32] and endothelial release of nitric oxide

**Fig. 3**



Graph of pulse wave velocity (PWV) as function of diastolic blood pressure (DBP) under (a)  $\alpha_2$ -adrenoceptor stimulation, and (b) left ventricular ejection time index (LVETI) under  $\beta$ -adrenoceptor stimulation. (c) Individual curves of the six subjects for PWV as a function of LVETI under  $\beta$ -adrenoceptor stimulation are shown. Data from each subject are labelled with a different symbol ( $\bullet$ ,  $\circ$ ,  $\blacklozenge$ ,  $\diamond$ ,  $*$ ).

(NO) has been described as the mechanism responsible for this effect *in vitro* [33].

We believe that LVET could be the time interval of the cardiac cycle responsible for the previously reported relationship between PWV and heart rate [7–10]. We found that, although PWV was inversely correlated to LVET, PWV was not related to PEP or the duration of the diastole. These results indicate that the relationship between heart rate and PWV may be attributed to the time of the left ventricular ejection.

The finding of a relationship between LVET and PWV may be of clinical importance. It has been suggested that PWV may be corrected for heart rate [10]. Results of this study indicate that LVET may be the time interval of the cardiac cycle responsible for the relationship between heart rate and PWV. Therefore, it may be more accurate to correct PWV for LVET instead for heart rate. This can be conveniently done because LVET can be determined from the recording of a central pulse wave that is used to measure PWV. Data from this study suggest that PWV may be corrected for LVET under resting conditions and stimulation. However, further investigations are required to determine these relationships in females and older individuals.

In summary, this is the first study describing a relationship between pulse wave velocity and left ventricular ejection time under resting conditions and under myocardial stimulation in young, healthy males. LVET may be the time interval responsible for the frequently observed relationship between PWV and heart rate. Further studies are needed to evaluate the relationship between PWV and myocardial function in health and disease.

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