Left ventricular size determines tissue Doppler-derived longitudinal strain and strain rate

Assami Rösner1,2*, Bart Bijnen3,4, Magna Hansen1,2, Ole Jakob How1,2, Erling Aarsæther1,2, Stig Müller1,2, George R. Sutherland5, and Truls Myrmel1,2

1Institute of Clinical Medicine, University of Tromsø, 9037 Tromsø, Norway; 2University Hospital North Norway, Tromsø, Norway; 3Imaging and Cardiovascular Dynamics, KULeuven, Leuven, Belgium; 4Center for Computational Imaging and Simulation Technologies in Biomedicine, Universitat Pompeu Fabra, Barcelona, Spain; and 5St George’s Hospital, London, UK

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Aims Tissue Doppler-derived indices of strain (e) and strain rate (SR) have been developed to assess regional cardiac function. However, the effect of left ventricular (LV) size on e and SR has not been studied in depth. The aim of this study was to assess to what extent heart size influence e or SR.

Methods and results In 21 anaesthetized pigs ranging from 12.5 to 70.0 kg, tissue Doppler-derived e and SR, and haemodynamic parameters, were assessed during controlled heart rates and different loading conditions. dp/dt did not correlate to pig weight, suggesting constant contractility during growth. Longitudinal e and SR were significantly higher in smaller compared with larger hearts. The hyperbolic correlation between pigs weight and e and SR was $r^2 = 0.621$ and 0.372, respectively, both $P < 0.0001$. Afterload elevation induced a reduction in longitudinal e (from $-24.2 \pm 3.2$ to $-12.1 \pm 5.5\%$, $P = 0.001$) and SR (from $-2.3 \pm 0.8$ to $-1.3 \pm 2.4\ s^{-1}$, $P = 0.034$), whereas increasing preload increased e (from $-26.4 \pm 10.3$ to $-38.1 \pm 14.3\%$, $P = 0.006$) and SR (from $-2.3 \pm 0.9$ to $-4.22 \pm 1.8\ s^{-1}$, $P = 0.002$).

Conclusion Longitudinal e and SR decrease with increasing LV dimensions in spite of an unaltered contractility. These results show and confirm that heart size influences e and SR, which are highly load-dependent parameters.

Introduction

Tissue Doppler imaging (TDI)-derived indices can be used for non-invasive measurement of regional myocardial deformation with a high-time resolution (150–180 frames/s). These techniques are now being widely used in research and increasingly introduced into the clinical routine. However, myocardial deformation, although closely related to contractility,1 does not directly represent contractile force. The parameters e (deformation) and SR (rate of deformation) depend on the interaction of contractile force, extrinsic loading conditions, ventricular geometry and the elastic properties of the tissues.2–4

Herbots performing a transverse study on healthy humans demonstrated a decrease in longitudinal strain with maturation and found an inverse dependency on strain on body mass index (BMI) in adolescents and grown-up subjects.5 A mathematical model suggested that ventricular size influences e and SR values unrelated to the changes in contractility or load, such that an increase in chamber size would lead to reduced deformation.6 This proposal is compatible with the influence of ventricular volume on wall tension,7 but the influence of ventricular size on e and SR has not yet been systematically analysed. Therefore, the aim of our study was to quantify the influence of left ventricular (LV) size on TDI-derived e and SR. Furthermore, we aimed to elucidate whether such dependence could be due to changes in contractility, heart rate or wall tension at increasing ventricular size.

Methods

Animals and anaesthesia

The investigation was conducted in accordance with the Guide for the Care and Use of Laboratory Animals published by the US National Institutes of Health (NIH Publication No. 85–23, revised 1996), and the experimental protocol was approved by the local steering committee of the Norwegian Experimental Animal Board. We analysed data from 21 pigs. The body weight and age ranged from 12.5 to 70.0 kg and 43 to 115 days, respectively.

* Corresponding author. Tel: +47 95990071; fax: +47 77628298. E-mail address: assami.rosner@unn.no

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The pigs were housed in the animal facilities for 3–7 days, and fasted overnight before experiments with free access to water. Intramuscular ketamine (20 mg/kg) and atropine (1 mg) were used as premedication. Anaesthesia was induced with boluses of pentobarbital-Na and fentanyl, and maintained by continuous intravenous infusions of pentobarbital-Na, fentanyl, and midazolam as described previously.8 The pigs were tracheotomized and intubated, and ventilation was maintained with an air–oxygen mixture (FiO2 = 0.5) using a volume-controlled respirator (Servo 900, Elema-Schönander, Stockholm, Sweden). Tidal volume was adjusted according to arterial blood gas samples (Elema-Schönander, Stockholm, Sweden). Tidal volume was adjusted measured continuously in the aortic arch. Finally, a balloon was served to control the systolic pressure-increments of the ascending aorta was inflated for 30 s. Measurements of aortic pressure and transdiaphragmatic echocardiographic 2D, TDI, M-mode and SR data were acquired in the presence of an after- and preload. Radial M-mode and TDI data were acquired using a S5–1 probe (5–1 MHz) in a parasternal long axis view. Radial M-mode and TDI data were acquired using a transesophageal (TEE) probe (5 MHz). A trans-venous electrode lead was started via an introducer into the right jugular vein and positioned in the right atrium to allow controlled right atrial pacing.

The central venous pressure was measured in the superior caval vein and held constant at 4 ± 1 mmHg by infusion of 0.9% NaCl enriched with glucose (1.25 g/l). The arterial pressure was measured continuously in the aortic arch. Finally, a balloon was inserted from the left femoral artery into the descending aorta for afterload alterations (balloon inflation). Heparin was administered once intravenously during catheterization (2500–5000 IE depending on weight). The abdominal wall was incised in the midline 10–15 cm below the xyphoid without opening either the peritoneum or the intra-thoracic space for the transdiaphragmal echocardiography access.

**Echocardiography**

Echocardiographic images were recorded during a short period of apnea using a Philips IE33 ultrasound scanner. Longitudinal data were acquired using a transesophageal (TEE) probe (5 MHz). A transdiaphragmal longitudinal view of the lateral wall was obtained from which TDI images from the mid-part of the lateral wall were taken. Doppler data from the transaortic and mitral valve blood flow were acquired. To reduce near field artefacts, we used a 2 cm thick jelly pad fixed to the TEE-probe. Radial M-mode and TDI data were acquired using a S5–1 probe (5–1 MHz) in a parasternal long axis view at the mid-part of the inferior wall.

All TDI images were obtained at frame rates of 180–220 frames/s. An image-sector angle of 25° and an optimal depth of imaging were used to increase temporal resolution. For optimizing velocity resolution, pulse repetition frequency was set at the lowest possible rate. A 5-Fr, high-fidelity pressure catheter (CD Leycom, the Netherlands) was inserted via the left common carotid artery and placed in the left ventricle. LV pressure was sampled and stored at 200 Hz. A 4-Fr unipolar pacemaker lead was inserted via an introducer into the right jugular vein and positioned in the right atrium to allow controlled right atrial pacing.

**Experimental protocol**

Smaller and larger pigs were investigated alternately. In order to avoid reverberation artefacts, the intraventricular pressure catheter was retracted before echocardiographic imaging. Parasternal and transdiaphragmal echocardiographic 2D, TDI, M-mode and blood pool Doppler measurements were performed at paced HRs of 120, 140, and 160 beats/min.

In 11 experiments, additional intraventricular pressure and ultrasound data were acquired in the presence of an after- and preload increase. For the afterload increase, the aortic balloon in the descending aorta was inflated for 30 s. Measurements of aortic pressure served to control the systolic pressure-increments of ~30 mmHg. The balloon was repeatedly inflated during the pressure measurements and for each M-mode and TDI acquisition in the transdiaphragmal and parasternal views. Lastly, preload was increased by infusing dextrane amounting to 20% of the calculated total blood volume (20% of 7% of body weight). Thereafter, TDI and M-mode measurements were again performed and after reinserting the pressure catheter, intraventricular pressure was acquired. All registrations during load alterations were done at a HR of 120 beats/min.

**Data analysis**

Based on the echocardiographic images, LV mass was calculated (M-mode) using the ASE-recommended formula:

\[
LV mass = 0.8 \times (1.04(\text{LVEDD} + \text{PWTd} + \text{SWTd})^{\frac{2}{3}}) - (\text{LVEDD})^{\frac{2}{3}} + 0.6g.
\]

where PWTd and SWTd are the posterior and septal wall thickness at end diastole, respectively, and EDD the end-diastolic diameter.9

M-mode measurements from the parasternal view were used to assess LV end-diastolic diameter (EDD), end-diastolic volume (EDV), end-systolic volume (ESV), end-systolic wall thickness (ESWT) and ejection fraction (EF). Volumes were calculated from the modified Teichholz formula,10 using the mean ratio of transversal (trans)/longitudinal (long) LVEDD from our datasets (measured on apical 2D grey scale images):

\[
\text{EDV} = \frac{\pi}{6} \times \text{EDD}_{\text{long}} \times \text{EDD}_{\text{trans}}^{2} \times \text{PWT}_{\text{d}} \times \text{PWT}_{\text{d}}
\]

with an average ratio

\[
\text{EDD}_{\text{long}}/\text{EDD}_{\text{trans}} = 1.37.
\]

this becomes:

\[
\text{EDV} = \frac{\pi}{6} \times 1.37 \times \text{EDD}_{\text{trans}}^{2} \times \text{PWT}_{\text{d}}
\]

From the parasternal M-mode and intracardiac pressure measurements, end-systolic and -diastolic wall stresses were calculated. The meridional wall-stress of the posterior wall was estimated assuming an ellipsoid LV geometry: 

\[
s = \frac{P \cdot D^2 / 4 \cdot h \cdot (D - h)}{0.133},
\]

where \(s\) is wall stress (in kPa), \(P\) the LVP (in mmHg), \(h\) the posterior wall thickness (in mm), \(D\) the LV cavity minor axis (in mm) and 0.133 a conversion factor from mmHg to kPa.11

LV diastolic function was assessed from mitral pulsed-wave Doppler measuring the maximal velocity of E- and A-wave, the deceleration time (DT) and the isovolumetric relaxation time (IVRT). The timings of aortic valve opening and closure (AVC) were determined from Doppler acquisitions at HRs of 120, 140, and 160 beats/min and were used to calculate ejection time (ET) and TDI data.

TDI data were analysed off-line using dedicated software (QLAB, Philips). The region of interest was chosen to be 8–15 mm long, while a maximal width of 2 mm was used. Curves were processed with a spatial SR smoothing of 10 mm. The temporal SR smoothing was performed in a QLAB function based on a Fast Fourier transformation. To ensure high data quality, only curves adhering to the following criteria were retained: \(e\) curves had mainly to follow the time-course and shape of greyscale M-mode registrations, \(e\) curves had to show clear E- and A-waves and frame by frame manual tracking had not to deteriorate curve quality. Both curve shape and values had to be reproducible over several beats after manual tracking. In longitudinal \(e\) measurements, at least two neighboured regions 2.5 mm apart (when applying SR smoothing over 5 mm) had to show a similarity in curve shape and values to be included. Values from 4 to 6 beats, fulfilling these quality criteria, were averaged. The opening and closure of the aortic valve were measured in blood pool Doppler acquisitions through the aortic valve to define the ET period. Thereafter, peak \(e\) and SR values during ET were extracted by using additional software from our laboratory.
Statistical analysis

TDI data were obtained from 20 animals of which nine were studied during preload increase and eight during afterload increase. Statistical analysis was done using SPSS 14.0 (SPSS headquarter, Chicago, IL, USA). All results are reported as means ± SD (standard deviation). We chose linear mixed-models, a statistical method combining regression analysis and repeated measurements. This method was used to assess the association between ϵ, SR and ET with HR, and weight and EDV as fixed effects and subject as random effects. Additionally, mixed-models were used to test fixed effects of HR (160 against 120 and 140 beats/min; and 120 against 140 beats/min). To define the best non-linear model to describe the relation between weight and longitudinal deformation (ϵ and SR), a logarithmic, a one and two component exponential, and an inverse transformation were tested, using $r^2$ as optimizer. To determine the relation between weight and haemodynamic parameters, diastolic parameters, dimensions, and cardiac mass, a linear regression analysis was used and the Pearson’s correlation coefficient and $r^2$ were calculated. $P < 0.05$ was considered statistically significant. A two-tailed paired Student’s t-test was used to compare ϵ and SR parameters at different loading conditions.

Results

From the acquired TDI data did 5.5% not meet the quality criteria and had to be discarded. Table 1 shows the cardiac dimensions, haemodynamics and functional parameters, correlated to body weight. Compared with small pigs, large pigs had bigger cardiac dimensions, but there were no changes in intracardiac pressures, contractility, parameters, diastolic parameters, dimensions, and cardiac mass, a linear regression analysis was used and the Pearson’s correlation coefficient and $r^2$ were calculated. $P < 0.05$ was considered statistically significant. A two-tailed paired Student’s t-test was used to compare ϵ and SR parameters at different loading conditions.

Table 1 The correlation between pig weight and various cardiac parameters

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD (n = 21)</th>
<th>Range</th>
<th>Pearson’s correlation coefficient r</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (days)</td>
<td>82.4 ± 23.5</td>
<td>43–115</td>
<td>0.676</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>EDDlong (cm)</td>
<td>4.32 ± 0.79</td>
<td>3.0–5.7</td>
<td>0.812</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>EDDtrans (cm)</td>
<td>5.8 ± 1.1</td>
<td>4.2–7.5</td>
<td>0.888</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>EDDlong/EDDtrans</td>
<td>1.37 ± 0.21</td>
<td>1.04–1.79</td>
<td>0.224</td>
<td>0.405</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>99.7 ± 43.7</td>
<td>42.1–169.0</td>
<td>0.855</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LV EDV (ml)</td>
<td>63.3 ± 26.4</td>
<td>24.1–109.9</td>
<td>0.686</td>
<td>0.002</td>
</tr>
<tr>
<td>LV ESV (ml)</td>
<td>28.8 ± 12.8</td>
<td>11.0–50.9</td>
<td>0.373</td>
<td>0.007</td>
</tr>
<tr>
<td>EF (%)</td>
<td>54.8 ± 4.6</td>
<td>46.8–62.0</td>
<td>0.308</td>
<td>0.213</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>4.0 ± 3.0</td>
<td>–0.8–11.8</td>
<td>0.385</td>
<td>0.115</td>
</tr>
<tr>
<td>IVPes (mmHg)</td>
<td>113.1 ± 9.4</td>
<td>95.5–133</td>
<td>0.207</td>
<td>0.380</td>
</tr>
<tr>
<td>IVPed (mmHg)</td>
<td>11.2 ± 5.2</td>
<td>4.3–27.4</td>
<td>0.025</td>
<td>0.913</td>
</tr>
<tr>
<td>$\sigma_{sys}$ (kPa)</td>
<td>8.28 ± 0.66</td>
<td>4.65–15.23</td>
<td>0.186</td>
<td>0.432</td>
</tr>
<tr>
<td>$\text{dP/dt}_{\text{max}}$ (mmHg/s)</td>
<td>1606 ± 15</td>
<td>1116–2237</td>
<td>0.254</td>
<td>0.267</td>
</tr>
<tr>
<td>ET (ms)</td>
<td>207.4 ± 30.8</td>
<td>151–269</td>
<td>0.137</td>
<td>0.002</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>0.7 ± 0.4</td>
<td>0.1–4.0</td>
<td>0.165</td>
<td>0.526</td>
</tr>
<tr>
<td>IVRT (ms)</td>
<td>47.8 ± 14.5</td>
<td>21–74</td>
<td>0.045</td>
<td>0.863</td>
</tr>
<tr>
<td>DT (ms)</td>
<td>99.4 ± 28.6</td>
<td>58–144</td>
<td>0.093</td>
<td>0.798</td>
</tr>
</tbody>
</table>

Discussion

Deformation in hearts of varying sizes

The present study shows the influence of heart size on longitudinal deformation in maturing pigs. This effect of LV size on TDI-derived ϵ and SR was unrelated to contractility and end systolic wall stress. Both longitudinal ϵ and SR were found to decrease with increasing body size and thus cardiac dimension. Owing to an unchanged ratio of intraventricular diameter and wall thickness in growing hearts, end-diastolic and systolic wall stress was not altered with different LV dimensions. The unchanged wall stress was observed...
whether the calculations were done using Laplace’s law or a modification of the 3D ellipsoid model.\textsuperscript{6,12}

We found that the LV size determined both longitudinal $\epsilon$ and SR.\textsuperscript{13} This is an important observation, as longitudinal deformation is the only dimension being used for TDI analyses in the usual 16 segments employed in a clinical setting. Furthermore, longitudinal strain is reported to be more sensitive to pathology than radial parameters.\textsuperscript{14,15} The dependency of longitudinal $\epsilon$ values on body size is consistent with the observations made by Herbots.\textsuperscript{5} In their study of longitudinal $\epsilon$ and SR in healthy humans, an inverse correlation of $\epsilon$/SR and BMI and age was observed.

One possible explanation for a higher $\epsilon$ and SR in smaller pigs might be a lower systemic vascular resistance and a higher cardiac index, resulting in a lower wall stress time integral. This would not contradict an unaltered end-systolic wall stress after a prolonged ET in smaller individuals. Data from our laboratory using differently sized open chest pigs and measuring continuous CO and MAP demonstrated an inverse correlation of $\epsilon$/SR and BMI and age was observed.\textsuperscript{16}

Higher SV indices in smaller individuals indicate a slightly higher EF in smaller individuals. However, in our study, the radial M-Mode-derived EF was not sensitive enough to reflect a possible EF change dependent on heart size.

Assessment of $dP/dt$ indicated no difference in contractility between small and large hearts. $dP/dt$ is known to be highly dependent on loading and HR,\textsuperscript{17} but by keeping HR and loading conditions equal in small and large hearts, we could show that $dP/dt$ was not influenced by varying cardiac sizes. We therefore conclude that the unchanged $dP/dt$ in our experiments indicates unchanged contractility in left ventricles of different sizes.

The effect of HR and ejection time on strain and strain rate

Weidemann et al.\textsuperscript{18} have shown in a closed chest model that increasing HR reduces $\epsilon$, while SR does not change. This finding was reproduced in the present study, probably related to a reduced LV-filling at high heart rates. A clinical study in healthy children suggested that the changes in $\epsilon$ and SR were mainly due to the alterations in ET, and that

![Figure 1](image_url)

Figure 1  Representative longitudinal strain and strain-rate curves for pigs with small (left) and large (right) hearts. The 2D images demonstrates the view from subdiaphragmal with the transesophageal probe in small and big pigs. The insonation angle did not change significantly with body size. AVC, aortic valve closure.
prolongation of ET was a function of HR reduction only. In contrast to these observations, in the present study, we found that ET at a constant HR was altered dependent on heart size (body size) alone. Our findings are also in this aspect consistent with the study by Herbots, who described a similar effect when comparing deformation parameters and ET in men and women. The prolonged ET in smaller individuals may be a result of a lower vascular resistance index. With a longer ET, peak increases to a certain extent. However, SR is not expected to change in dependency on ET, because peak SR is highest at the first third of the ET and low at the end of the ET.

Influence of loading on strain and strain rate

The effects of loading conditions on the regional deformation parameters have previously been studied thoroughly. However, the influence of load on longitudinal \( \varepsilon \) and SR in an experimental closed-chest model has not been investigated previously. Consistent with the measurements on muscle strips, we found a decrease in both radial and longitudinal \( \varepsilon \) and SR with an incremental afterload. As previously shown by Urheim et al. in a sternotomy model, we found that longitudinal \( \varepsilon \) and SR increased significantly with volume loading. \( \varepsilon \) and SR are thus clearly dependent on both pre- and afterload, and the relative effects of loading were similar for \( \varepsilon \) and SR.

Compared with \( \varepsilon \), the load dependency of SR has not been closely investigated. In agreement with physiological studies, but in contrast to a clinical study, we found a pronounced load sensitivity of SR as well. As SR represents the infinitesimal increment of \( \varepsilon \)-curves, the shape of \( \varepsilon \)-curves during pre- and afterload changes correspond to increased SR at preload increase and decreased SR at afterload increase. \( \varepsilon \)-Curves showed a delayed onset, steep increase and a prolonged plateau at high-level preloads, and a delayed peak \( \varepsilon \) was observed during increased afterload. An afterload increment induced an increased post-systolic \( \varepsilon \) and SR. \( \varepsilon \) and SR should therefore not be presented as contractility parameters alone, but must be presented more like functional parameters in an integrated load dependent pump system.

Table 2 The effect of body weight on strain and strain-rate at different heart rates

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ( N = 20 )</th>
<th>Range</th>
<th>( r^2 )</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Longitudinal strain (%) HR 120</td>
<td>(-28.7 \pm 11.7^*)</td>
<td>(-54.7) to (14.36)</td>
<td>0.621</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HR 140</td>
<td>(-28.4 \pm 13.4^t)</td>
<td>(-58.4) to (13.2)</td>
<td>0.375</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HR 160</td>
<td>(-25.4 \pm 11.8)</td>
<td>(-54.6) to (12.7)</td>
<td>0.375</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Longitudinal SR (s(^{-1})) HR 120</td>
<td>(-2.66 \pm 1.17)</td>
<td>(-4.6) to (0.81)</td>
<td>0.375</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HR 140</td>
<td>(-3.40 \pm 2.73)</td>
<td>(-12.8) to (0.77)</td>
<td>0.375</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HR 160</td>
<td>(-3.11 \pm 2.35)</td>
<td>(-10.5) to (0.63)</td>
<td>0.375</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Values are means \( \pm \) SD. Dependency of longitudinal strain (\( \varepsilon \)) and strain-rate (SR) on body weight was tested at HR 120 and 160 and 140 and 160 using mixed models. Additionally, dependency of strain and SR on different HR was also tested with the repeated measurements component of mixed models: *\( P < 0.05; \) \( \varepsilon \) and SR values of all subjects at HR 120 vs. HR 160; †\( P < 0.05; \) values of all subjects at HR 140 vs. values at HR 160. Radial and longitudinal strain showed decreasing values with increasing HR, whereas SR stayed uninfluenced by HR.
Figure 3  Strain and strain-rate (SR) curves examples loading differences in one pig. Left, afterload increase; middle, resting deformation; right, preload increase. Upper row, strain curves; lower row, SR curves. PSS, post-systolic shortening. AVC, Aortic valve closure.

Table 3  Radial and longitudinal deformation parameters and haemodynamic parameters at

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rest</th>
<th>Loading</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Longitudinal strain (%)</td>
<td>Preload increase</td>
<td>$-26.4 \pm 10.3^a$</td>
<td>$-38.1 \pm 14.3^a$</td>
</tr>
<tr>
<td></td>
<td>Afterload increase</td>
<td>$-24.2 \pm 3.2^b$</td>
<td>$-12.1 \pm 5.5^d$</td>
</tr>
<tr>
<td>Longitudinal SR (s$^{-1}$)</td>
<td>Preload increase</td>
<td>$-2.3 \pm 0.9^a$</td>
<td>$-4.2 \pm 1.8^a$</td>
</tr>
<tr>
<td></td>
<td>Afterload increase</td>
<td>$-2.3 \pm 0.8^a$</td>
<td>$-1.3 \pm 2.4^a$</td>
</tr>
<tr>
<td>$P_{es}$ (mmHg)</td>
<td>Preload increase</td>
<td>$112.1 \pm 10.9^e$</td>
<td>$118.4 \pm 16.7^c$</td>
</tr>
<tr>
<td></td>
<td>Afterload increase</td>
<td>$109.8 \pm 10.0^e$</td>
<td>$141.8 \pm 24.0^c$</td>
</tr>
<tr>
<td>$P_{ed}$ (mmHg)</td>
<td>Preload increase</td>
<td>$12.0 \pm 5.3^c$</td>
<td>$16.1 \pm 7.8^c$</td>
</tr>
<tr>
<td></td>
<td>Afterload increase</td>
<td>$10.5 \pm 3.9^c$</td>
<td>$16.5 \pm 6.9^c$</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>Preload increase</td>
<td>$3.95 \pm 3.01^c$</td>
<td>$9.32 \pm 9.36^c$</td>
</tr>
<tr>
<td></td>
<td>Afterload increase</td>
<td>$3.95 \pm 3.01^c$</td>
<td>$4.67 \pm 2.94^c$</td>
</tr>
</tbody>
</table>

Rest and different loading conditions ($^aN = 10$; $^bN = 8$; $^cN = 11$). Values as means ± SD. P, two tailed, paired t-test between different loading groups; $P_{es}$, end-systolic left ventricular pressure; $P_{ed}$, end-diastolic left ventricular pressure; CVP, central venous pressure. Preload increase significantly increases strain and strain-rate (SR) values, whereas afterload increase decreases strain and SR values.

Figure 4  Longitudinal and radial strain and strain-rate (SR) at different loading conditions. Significant decrease of strain and SR with afterload increase and increase with preload increase.
Limitations of the study

Using a model of growing adolescent pigs, we cannot exclude that maturation did influence tissue properties like elasticity or fibre structure. However, other studies on puppies and mature dogs of different sizes have not shown significant changes in contractility parameters. 12

We observed some very high-longitudinal mid-wall ε values (45–60%) in smaller subjects. These values do not necessarily imply a shortening of the whole heart or one whole myocardial wall to a uniform extent. First, this phenomenon is probably most pronounced in mid-wall segments of the lateral wall and might not be a general phenomenon for the entire ventricle. Secondly, small rotational movements in hyperkinetic myocardium and angular deviations within the myocardial wall during the cardiac cycle might lead to higher TDI-derived longitudinal mid-wall values compared with global longitudinal endo- or epicardial strain.

Implications

Both the mathematical model6,25 and our findings demonstrate that longitudinal ε and heart size show a hyperbolic relationship despite the fact that heart shape and global contractility parameters stayed unchanged. Therefore, calculations of deformation parameters should be corrected for heart size both in the adolescent heart and pathological conditions. Further clinical and modelling studies will have to be performed to determine size and load corrected ε and SR values reflecting normal and pathological cardiac function. However, taking heart-size, dimensions and loading into account, ε and SR measurements may serve as valuable tools to assess regional myocardial function.

Conflict of interest: none declared.

References