Regional left ventricular deformation and geometry analysis provides insights in myocardial remodelling in mild to moderate hypertension

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Aim In the early stages of hypertension (HTN), when global left ventricular (LV) function is still unaffected, localized geometrical changes suggest changes in regional function. We investigated regional geometry and systolic deformation (using strain/strain rate (S/SR) imaging) in HTN.

Methods and results We studied 74 untreated mild to moderate HTNs and 34 matched normotensives (NTN). All had a standard echo including myocardial velocity data for regional radial and longitudinal deformation. Despite the absence of abnormalities in standard functional indices and LVH, non-uniform changes in regional geometry and deformation were observed. Besides a significant increase in wall thickness (WT) in all HTN segments, there was a gradual increase in WT from apex to base resulting in prominent basal septal hypertrophy. In HTN, regional longitudinal peak systolic SR (SSR) and end-systolic S (ESS) were significantly ($P$, 0.0001) reduced in the basal septum. In the lateral wall there was an increase in peak SSR and ESS ($P$, 0.05) basally. The basal septal ESS correlated both with mean arterial pressure and basal septal WT, with lower ESS for higher BP and thicker septum.

Conclusion Regionally differing geometrical remodelling occurs early in HTN. Longitudinal ESS and peak SSR are sensitive markers of early changes occurring in HTN.

KEYWORDS Hypertension; Hypertrophy; Echocardiography; Remodelling; Strain imaging

Introduction Hypertension (HTN) is a recognized risk factor for cardiovascular disease.1 A longstanding increase in blood pressure (BP) results in ventricular remodelling2 and may ultimately cause heart failure.3 With proven benefits from prevention strategies and pharmacological BP lowering, early detection of cardiac involvement is important.

In advanced stages, HTN is characterized by an increase in left ventricular (LV) mass and relative wall thickness (RWT),2 which is associated with a non-favourable outcome.4 Localized basal septal hypertrophy (‘sigmoid septum’) has been described as part of the cardiac involvement.5,6 BP control can lead to its regression,4 but its origin and relevance have not been fully investigated.

Besides changes in mass and geometry, global systolic function was shown to be reduced as a result of longstanding pressure overload. However, in early stages, ejection fraction (EF) and short axis endocardial shortening (ES), standard indices of global systolic function, can be supernormal1,7 due to the increased LV radial pump performance associated with hypertrophy.8 Midwall radial shortening fraction (MSF) was suggested as more sensitive in early stages of HTN.8–10

In contrast to global radial function (which can be normal in HTN when assessed by current standard parameters), global longitudinal performance was shown to be reduced in HTN (by echocardiography9 or magnetic resonance tagging).11 Additionally, ultrasound strain/strain rate (S/SR) imaging6 demonstrated this reduction independently on associated diastolic dysfunction.12,13 Impaired global long axis function was shown to be associated with the changes induced by hypertrophy and not with the increased wall stress caused by increased afterload.6

All current approaches to the clinical evaluation of both systolic and diastolic performance in HTN assess global myocardial indices. Abnormalities in global measurements are frequently only found in advanced stages when clear LV remodelling/hypertrophy is present.14,15
In this study we wished to assess if the evaluation of regional, radial and longitudinal, myocardial deformation, using S/Strain imaging, could be more sensitive for detecting early changes in systolic function in HTN where there are no detectable reductions in standard radial functional indices and whether these measurements provide more insight in the remodelling process in the early stages of increased afterload.

Methods

We studied 74 (48 males, 26 females, mean age 48.9 ± 1.4 years) uncomplicated, never treated, individuals with mild to moderately elevated blood pressure (BP) (140–179/90–109 mmHg) and compared these to 34 (22 males, 12 females, mean age 44.2 ± 2.1 years) age-matched normotensive healthy subjects. All patients had a routine physical examination (including office BP measurement), excluding the presence of secondary hypertension, coronary and/or valvular heart disease or heart failure. Additionally, blood tests for serum cholesterol and glucose, a 24-h ambulatory BP monitoring and a standard echocardiographic evaluation (including a complete myocardial velocity imaging (MVI) study) were performed. The study protocol was approved by the local Ethics Committee and a written informed consent was obtained from each participant.

Blood pressure was measured using an automatic oscillometric device (Omron HEM 705 CP, Omron Healthcare, UK). A mean of three readings obtained at 2 min interval were taken. Essential hypertension was defined as a sustained increase in systolic BP (BPs) ≥140 mmHg and/or diastolic BP (BPd) ≥90 mmHg12 and the absence of laboratory and/or clinical findings suggesting secondary hypertension. Mean arterial pressure (MAP) was calculated as follows: BPd + 1/3(BPs–BPd).

Twenty-four hour ambulatory BP monitoring was performed using a SpaceLabs 90207 monitor (Space-Labs, Inc., Washington). Measurements were started in the morning and taken every 30 min during day-time and every hour during night-time. Nondippers were identified by a lack of night-time BP fall of 10%.

A transthoracic echocardiographic examination including the acquisition of MVI data was performed in all individuals. Data were acquired from parasternal and apical views using a GE Vivid 7 scanner. For each acquisition, three heart cycles were stored in cine-loop format for postprocessing.

LV internal dimensions in end-diastole (LVEDD) and end-systole (LVESD) and interventricular septum (IVS) and posterior wall thickness (LVPW) were measured on parasternal long axis M-mode.18 LV volumes were measured using the Teichholz formula. Regional wall thickness (WT) was measured on apical 2D images at basal, mid and apical level of the septum, lateral, anterior and inferior walls. Diastolic function was assessed by the early (peak E) and late (peak A) trans-mitral flow, isovolumic relaxation time (IVRT) and deceleration time (DT).

LV mass was calculated19 and normalized by body surface area. A cut-off value of 125 g/m² was used to indicate LV hypertrophy. End-diastolic relative wall thickness (RWT) (the sum of posterior and septal wall thickness divided by the internal diameter) was calculated as an index of LV concentric remodelling.

Global radial function was assessed by both endocardial and mid-wall fractional shortening.9 Meridional end-systolic stress (MESS)20 and circumferential end-systolic stress (CESS)21 were calculated using cuff systolic BP (BPs) taken at the end of the echocardiographic examination as an estimate of intraventricular systolic pressure.

Global longitudinal function was assessed by mitral ring displacement (MRD) at septal, anterior, lateral and inferior sites from apical views. Additionally, an index for long axis shortening (LAS) was calculated by normalising MRD by LV end-diastolic long axis length (LAX).22

All measurements were performed using an Echo-Pac (GE) workstation and averaged over three individual measurements.

Myocardial velocity imaging data were recorded for the assessment of longitudinal deformation. Data from the septum, lateral, anterior and inferior LV walls were obtained using a narrow sector (typically 12°) and an optimal depth. Frame rates of 200-300 frames/s were used to optimize temporal resolution. Special attention was paid to the velocity range setting, avoiding any aliasing while still maximizing velocity resolution.

Radial and longitudinal SR were calculated over a computation area of 5 mm and 10 mm, respectively. The region of interest within the LV wall was continuously tracked during the cardiac cycle. End-diastole (onset of isovolumic contraction) and end-systole (aortic valve closure) were determined from trans-mitral and aortic velocity profiles. SR and S were analysed using dedicated software (Speqle, K.U. Leuven). Three consecutive cardiac cycles were averaged and radial and longitudinal end-systolic strain (ESS), peak systolic strain rate (peak SSR), total deformation (TD) and post-systolic thickening/shortening (PSS) were measured. The reproducibility of this approach has been studied by several authors and is always reported as less than 10% intra- and inter-observer variability.12,23

Statistical analysis was performed using StatView 5.0 (SAS Institute, Inc.). All results were expressed as mean ± SEM. Comparisons of continuous variables between the HTN and NTN were performed by a Student’s t-test for independent groups. Pearson’s correlation was used to examine the associations between MFS and CESS and MESS, and between MAP and regional deformation parameters, basal septal thickness and longitudinal A-V ring displacement. A value of P < 0.05 was considered statistically significant. All tests were two-sided.

Results

Table 1 shows the clinical characteristics. In comparison to NTN, the HTN individuals had a higher BMI (P < 0.0001). Glucose and plasma total cholesterol did not differ significantly.
significantly. However, HTN patients had a less favourable lipid profile with an increased total cholesterol/HDL ratio (4.2 ± 0.1 vs 3.3 ± 0.2, P < 0.001).

Global remodelling was present in HTN (Table 2) with both the septal and posterior walls significantly thicker compared to NTN and an increase in RWT (0.43% ± 0.01 vs 0.33 ± 0.01, P < 0.0001), resulting in a significantly increased LV mass (189.5 ± 5.7 g vs 136.9 ± 7.1 g) and LV mass index (96.4 ± 2.6 g/m² vs 74.6 ± 2.9 g/m²). However, changes in wall thickness were not uniform in HTN (Table 3 and Figure 1). Although wall thickness was increased in all segments, there was a gradual increase in the degree of hypertrophy from apex to base in all walls with the most prominent increase in thickness at the basal septum (12.1 ± 0.1 mm HTN vs 8.1 ± 0.1 mm NTN, P < 0.0005) (Table 3). This resulted in the characteristic basal septal bulge in HTN. LV cavity diameters and volumes did not differ. Only LVEDVI (volume to BSA index) was reduced in HTN. Also, although not statistically significant, global and regional LV remodelling was more pronounced in non-dippers.

Although significantly different from the control group (Table 2), the diastolic measurements for the HTN patients would not be considered as diastolic dysfunction\(^\text{21}\) in most of the patients (IVRT\(_{30-50y} < 100\) ms, DT\(_E\) < 50y < 220 ms, \(E/A\) ratio\(_{30-50y} > 1\)).

Global radial LV systolic function in HTNs, assessed by endocardial motion (EF) or mid-wall fractional shortening (MFS), did not differ from NTN (Table 2). There was a significant correlation between MFS and wall stresses (MESS: \(R = -0.47\), P < 0.0001 and CESS: \(R = -0.46\), P < 0.0001) in both HTN and NTN (Figure 2). However, we did not observe the downward shift in MFS which was previously described.\(^{21}\) There was no correlation between BP and both EF (\(R = 0.01\), NS) and MFS (\(R = 0.04\), NS).

Global longitudinal function (averaged ring displacement, Table 4) was significantly reduced in HTN compared to NTN (1.20 ± 0.47 cm, P < 0.0001). LAS was also significantly reduced (14.12 ± 0.27 vs 16.59 ± 0.35\(\%\), P < 0.0001). However, this reduction showed marked regional differences. MRD of the lateral wall did not differ between HTN and NTN whereas there was a clear reduction in the septal MRD (Table 4). Additionally, there was an inverse relation between longitudinal MRD at the different sites and MAP. The correlation between septal longitudinal MRD displacement and MAP was the strongest (\(R = -0.56\); P < 0.0001) (Figure 3). However, there was no correlation between global longitudinal MRD and LV wall stress (both MESS and CESS).

Regional radial deformation of the mid and basal posterior wall is shown in Table 5. Radial peak SSR and peak SS did not differ between HTN and NTN. However, in HTN, there was a significant increase in post-systolic thickening in the basal segment.

Regional longitudinal velocities (Table 6) were only significantly reduced in the mid and basal segments of the lateral wall in HTN.

Figure 4 shows typical traces for regional longitudinal myocardial velocities, strain rate and strain for HTN (left) and NTN (right). While in NTN, the traces did not differ for apical, mid and basal segments, there was a clear inhomogeneity in regional longitudinal deformation when BP was elevated, with reduced systolic shortening in the basal septum followed by marked post-systolic deformation.

Regional longitudinal deformation for all LV segments is shown in Table 7. As already observed in the traces (Figure 4), a clear inhomogeneity in the changes in regional longitudinal deformation was observed.

While apical and mid ventricular segments in HTN showed no significant difference in deformation compared to NTN, the basal septum of HTN patients showed a significant reduction in peak SSR (\(-1.34 ± 0.05\) vs \(-1.61 ± 0.09\)\(\%\), P < 0.006), ESS (\(-13.9 ± 0.7\) vs \(-21.1 ± 0.8\)), and maximal S (\(-17.9 ± 0.6\) vs \(-22.9 ± 0.8\), P < 0.0001). On the other hand, there was an increase in deformation in the basal lateral wall (Table 7). The anterior and inferior walls did not differ although there was a slight, significant decrease in ESS in the basal anterior segment.

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### Table 2 | Global LV geometry and systolic and diastolic function in HTN and NTN subjects

<table>
<thead>
<tr>
<th></th>
<th>HTN</th>
<th>NTN</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDD (cm)</td>
<td>4.83 ± 0.06</td>
<td>4.89 ± 0.08</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>3.03 ± 0.06</td>
<td>3.09 ± 0.07</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>110.58 ± 3.2</td>
<td>113.72 ± 4.4</td>
</tr>
<tr>
<td>LVEDVI (ml/cm²)</td>
<td>56.4 ± 1.5</td>
<td>62.5 ± 1.8</td>
</tr>
<tr>
<td>LV EF (%)</td>
<td>66.66 ± 0.89</td>
<td>66.67 ± 0.99</td>
</tr>
<tr>
<td>MFS (%)</td>
<td>22.96 ± 0.54</td>
<td>23.32 ± 0.56</td>
</tr>
<tr>
<td>IVS (cm)</td>
<td>1.10 ± 0.02</td>
<td>0.84 ± 0.03</td>
</tr>
<tr>
<td>LVPW (cm)</td>
<td>1.03 ± 0.02</td>
<td>0.80 ± 0.02</td>
</tr>
<tr>
<td>LAX (cm)</td>
<td>8.48 ± 0.09</td>
<td>8.37 ± 0.13</td>
</tr>
<tr>
<td>RWT</td>
<td>0.43 ± 0.01</td>
<td>0.33 ± 0.01</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>189.5 ± 5.7</td>
<td>136.9 ± 7.1</td>
</tr>
<tr>
<td>LV mass index (g/m²)</td>
<td>96.4 ± 2.6</td>
<td>74.6 ± 2.9</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.14 ± 0.06</td>
<td>1.53 ± 0.09</td>
</tr>
<tr>
<td>IVRT, ms</td>
<td>99.2 ± 2.8</td>
<td>82.2 ± 2.4</td>
</tr>
<tr>
<td>E DT, ms</td>
<td>203.9 ± 7.5</td>
<td>176.1 ± 6.53</td>
</tr>
</tbody>
</table>

\(^*P < 0.05\) and \(^{1}P < 0.0005\).

### Table 3 | Regional LV wall thickness (mm) in HTN and NTN

<table>
<thead>
<tr>
<th></th>
<th>HTN</th>
<th></th>
<th>NTN</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Basal (mm)</td>
<td>12.1 ± 0.3</td>
<td>8.1 ± 0.3</td>
<td>10.3 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>Mid (mm)</td>
<td>9.6 ± 0.2</td>
<td>8.0 ± 0.2</td>
<td>8.7 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>Apex (mm)</td>
<td>7.7 ± 0.2</td>
<td>7.1 ± 0.1</td>
<td>7.1 ± 0.1</td>
</tr>
</tbody>
</table>

\(^{1}P < 0.05\); \(^{2}P < 0.005\) and \(^{3}P < 0.0005\).
Post-systolic deformation was significantly increased in HTN compared to NTN in the septal (20.0 ± 2.6 vs 4.0 ± 1.5%, \(P < 0.0001\)) and anterior (15.2 ± 2.6 vs 2.5 ± 0.8%, \(P < 0.001\)) basal segments. In most individuals with increased BP (56% vs 13% in NTN), there was a clearly identifiable peak of post-systolic shortening (-10%) in the basal septum.

The reduction in septal ESS correlated significantly with BP (\(R = 0.48; P < 0.0001\)) (with lower ESS for higher BP) as well as with the amount of regional hypertrophy (\(R = 0.35, P < 0.0004\)) (Figure 5).

Discussion

As shown previously, our results demonstrated that HTN leads to global LV remodelling with an increase in RWT and mass. Diastolic parameters were impaired compared to NTN, but this would clinically not be interpreted as prominent diastolic dysfunction.

Global radial function and regional radial deformation, parameters traditionally used to assess systolic function, did not differ between normals and hypertensives. Although mid-wall shortening fraction (MSF) was suggested as more sensitive to detect hypertensive heart disease and predict cardiovascular risk,\(^8\)\(^9\)\(^10\) we could not find any change in this index despite the presence of concentric remodelling (increase in RWT), probably due to the mild to moderate hypertension. However, we did observe a similar relationship between MSF and end-systolic LV wall stress.\(^11\)\(^12\)

In contrast, there was a clearly detectable and wall-dependent change in both global and regional longitudinal function. There was a significant reduction of longitudinal shortening.\(^8\)\(^9\)\(^25\)\(^26\) Despite the fact that longitudinal myocardial velocities were virtually identical, regional longitudinal deformation was clearly different in the basal myocardial segments in HTN. There was a significant reduction in longitudinal ESS and peak SSR in the basal septum with a concomitant increase in the basal lateral wall. These findings agree with MRI data reporting non-uniformity in septal longitudinal shortening with a predominant reduction of basal septal shortening.\(^11\) Interestingly, in our study, in HTN, maximal total longitudinal deformation in this region was not only reduced but also shifted towards early diastole. This post-systolic contribution to total deformation increased significantly with increased afterload.

Regionally differing patterns of changes in morphology and deformation in the basal LV segments are determined by the fibre structure of the myocardium and its interaction with local wall stress. The sub-endocardial fibres are mainly longitudinally oriented, while mid-wall fibres are circumferential.\(^27\) Local wall stress is determined by curvature and is increasing with increasing radius of curvature. While in the apex, the radii of curvature are similar in all directions, at the base, they are substantially smaller in the radial compared to the longitudinal direction. Both fibre orientation and local geometry result in a non-uniform wall stress distribution, with a decrease from endo- to epicardium and from equator to apex.\(^28\)\(^30\) Due to a greater local radius of curvature, stress in basal segments will increase more than in mid and apical segments when LV pressure increases. Since hypertrophy is directly related to wall stress\(^31\) there must be a gradual increase in the hypertrophic response from apex to base, as we observed. Furthermore, the septum is more flattened (both in the radial and longitudinal direction) compared to the lateral wall. Expected higher septal wall stress is compensated by the pressure in the right ventricle which normalises transmural pressure across the
septum (Figure 6). However, with a unilateral rise in LV pressure, when this compensation becomes insufficient, local stress increases more in the septum (particularly in the basal third). This will result in greater degree of septal wall thickening, leading to the development of the characteristic basal septal bulge. While in normal hearts the postero-lateral wall is thicker than the anterior wall, we found an increase in thickness in the anterior and septal walls compared to the lateral and inferior walls. As previously reported, localized basal septal hypertrophy was most prominent in those with elevated BP.

Regional remodelling of the myocardium paralleled changes in local longitudinal deformation. The most hypertrophied basal septal segment showed a marked reduction in systolic deformation with a concomitant development of marked postsystolic shortening. The contralateral free wall basal segment compensated for this septal reduction in deformation with increased systolic S/SR. These findings suggest that the regional indices of LV geometry and function might be more optimal for the detection of early changes in systolic performance in hypertension.

The stress difference between circumferential and longitudinal fibres also explains why we observe a clear change in longitudinal function, both global (ring displacement) and regional. Since the longitudinal fibres show a larger radius of curvature, the increase in stress on the longitudinally oriented endocardial fibres is more pronounced compared to the circumferential fibres. This means that the circumferential fibres, generating the radial deformation of the myocardium, are much better in generating higher pressure and will retain normal functional parameters for a much longer time. As we, and others, have observed, assessing ventricular function using any technique based on measuring mainly radial function (EF, FAC, mid-wall shortening) will be very insensitive in detecting the transition from compensated hypertrophy to intrinsic myocardial dysfunction in patients with hypertension.

In order to maintain its stroke volume despite the presence of any regional dysfunction there must be either dilatation of the ventricle or local increase in deformation. In

### Table 4 Longitudinal mitral ring displacement in HTN and NTN

<table>
<thead>
<tr>
<th></th>
<th>HNT</th>
<th>NTN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septum (mm)</td>
<td>10.1 ±0.2</td>
<td>13.0 ±0.3</td>
</tr>
<tr>
<td>Anterior wall (mm)</td>
<td>11.2 ±0.3</td>
<td>13.5 ±0.4</td>
</tr>
<tr>
<td>Lateral wall (mm)</td>
<td>13.8 ±0.4</td>
<td>14.8 ±0.4</td>
</tr>
<tr>
<td>Inferior wall (mm)</td>
<td>12.7 ±0.3</td>
<td>14.4 ±0.5</td>
</tr>
<tr>
<td>Average over the four walls (mm)</td>
<td>12.0 ±0.3</td>
<td>13.9 ±0.3</td>
</tr>
</tbody>
</table>

*P < 0.005 and †P < 0.0001.

### Table 5 Infero-lateral radial peak systolic strain rate/strain and post-systolic thickening index in HTN and NTN

<table>
<thead>
<tr>
<th></th>
<th>Peak SSR (s⁻¹)</th>
<th>End SS (%)</th>
<th>Post-SSI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HNT</td>
<td>NTN</td>
<td>HNT</td>
</tr>
<tr>
<td>LAX basal</td>
<td>5.37 ± 0.20</td>
<td>6.09 ± 0.58</td>
<td>68.63 ± 3.47</td>
</tr>
<tr>
<td>LAX mid</td>
<td>4.64 ± 0.27</td>
<td>4.74 ± 0.25</td>
<td>59.45 ± 3.99</td>
</tr>
<tr>
<td>SAX basal</td>
<td>5.13 ± 0.28</td>
<td>5.09 ± 0.32</td>
<td>58.81 ± 3.30</td>
</tr>
<tr>
<td>SAX mid</td>
<td>4.99 ± 0.25</td>
<td>5.18 ± 0.32</td>
<td>65.73 ± 3.93</td>
</tr>
</tbody>
</table>

*P < 0.01.

### Table 6 Longitudinal LV peak systolic velocities (cm/s) in HTN and NTN

<table>
<thead>
<tr>
<th></th>
<th>Basal</th>
<th>Mid</th>
<th>Apical</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HNT</td>
<td>NTN</td>
<td>HNT</td>
</tr>
<tr>
<td>Septum (cm/s)</td>
<td>5.56 ± 0.15</td>
<td>5.59 ± 0.22</td>
<td>3.78 ± 0.12</td>
</tr>
<tr>
<td>Anterior (cm/s)</td>
<td>5.11 ± 0.21</td>
<td>5.64 ± 0.38</td>
<td>3.01 ± 0.20</td>
</tr>
<tr>
<td>Lateral (cm/s)</td>
<td>5.46 ± 0.21*</td>
<td>6.27 ± 0.33</td>
<td>3.84 ± 0.22*</td>
</tr>
<tr>
<td>Inferior (cm/s)</td>
<td>5.65 ± 0.15</td>
<td>5.82 ± 0.23</td>
<td>4.10 ± 0.13</td>
</tr>
</tbody>
</table>

*P < 0.05.
In most of our hypertensive subjects, we also observed the continuation of deformation after aortic valve closure. In NTN, there was a small degree of post-systolic shortening, but this was much more pronounced in HNT. This post-systolic thickening/shortening has previously been explained based on a differential thickening/shorting of neighbouring segments during systole due to an imbalance between locally developed force and wall stress (mainly pressure). After...
the pressure drops, this differential thickness/length is (pas-
sively) equalised, resulting in post-systolic deformation of the
segment that showed the least deformation when pressure
was still high. As in HTN, wall stress is disproportionally
larger in basal segments; we expected and observed a
clear increase in post-systolic deformation in these segments.
This will probably be further exaggerated by the fact that the
base is the last part to be electrically activated.40 Contraction
of the earliest activated segments will result in accentuation
of an already increased wall stress in the basal segments
before local active force development. This will be much
more pronounced in HTN, where the hypertrophied myocar-
dium can generate more force and thus increase ventricular
pressure faster.

These proposed explanations are additionally supported
by the observed strong correlation between deformation
(S/SR) of the basal septum and both the mean arterial
pressure ($R = 0.48$, $P < 0.0001$) and the basal septal thick-
ness ($R = 0.35$, $P < 0.0004$).

**Limitations**

2D echocardiographic measurements do not provide as accu-
rate measurements of LV wall thickness as M-mode. How-
ever, we used this approach not to estimate precise
wall thickness but to compare LV geometry between HTN
and NTN.

Strain(-rate) imaging is inherently angle dependent. How-
ever, we made sure that the deformation to be assessed
was aligned with the ultrasound beam. In order to obtain
reproducible deformation curves, images were taken at
high frame rate, reverberation artefacts were carefully
avoided and the region of interest was tracked throughout
the cardiac cycle.

With the current approach for S/SR analysis, regional
radial deformation can only be analysed in the infero-lateral
segments.

**Conclusions**

The increase in regional wall stress as a result of mild/mod-
erate hypertension results in geometrical remodelling,
expressed in regionally differing degrees of hypertrophy,
most pronounced in the basal septum, which are paralleled
by changes in regional longitudinal deformation. Although
we could not observe any changes in global systolic function
based on radial parameters (EF and midwall shortening),
global longitudinal function (ring motion) was clearly
decreased.

While regional radial deformation was almost unaltered,
there were distinct changes in regional longitudinal defor-
mation, where a decrease in lengthening in the basal
septum (accompanied by the development of pronounced
post-systolic shortening) was compensated by an increase
in deformation in the lateral wall. This suggests that the
measurement of regional longitudinal deformation is a
highly sensitive method for detecting early changes in myo-
cardial function in patients with HTN.

Studying the changes of longitudinal deformation in the
basal septum provides new insight in the disease process
of hypertensive hearts and could prompt early and aggres-
sive antihypertensive treatment to avoid hypertensive
target organ damage. Also, in clinical trials with drugs or a
non-pharmacological approach, aimed at the assessment of

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**Figure 5** Correlation between basal septal peak systolic strain and mean arterial pressure (left) and basal septal thickness (right).

**Figure 6** Non-uniformity of the LV (longitudinal) curvature. $R_{\text{apex}}$: radius of curvature at the apex; $R_{\text{lateral}}$: radius of curvature of the lateral wall; $R_{\text{septal}}$: radius of curvature of the septum; and PRV: pressure in the right ventricle.
subtle changes in myocardial function, regional deformation could be used since it is less dependent on the quality of grey scale images. Additionally, using basal septal longitudinal deformation might provide a sensitive way to monitor blood pressure lowering therapy in individual patients, thus optimising outcome.

References