Longitudinal movements and resulting shear strain of the arterial wall

Magnus Cinthio, Ása Rydén Ahlgren, Jonas Bergkvist, Tomas Jansson, Hans W. Persson, and Kjell Lindström. Longitudinal movements and resulting shear strain of the arterial wall. Am J Physiol Heart Circ Physiol 291: H394–H402, 2006. First published February 10, 2006; doi:10.1152/ajpheart.00988.2005.—There has been little interest in the longitudinal movement of the arterial wall. It has been assumed that this movement is negligible compared with the diameter change. Using a new high-resolution noninvasive ultrasonic method, we measured longitudinal movements and diameter change of the common carotid artery of 10 healthy humans. During the cardiac cycle, a distinct bidirectional longitudinal movement of the intima-media complex, the adventitial region, and the surrounding tissue. For this purpose, the longitudinal movement of the common carotid artery was investigated in 10 healthy normotensive subjects: 6 men (27–62 yr of age) and 4 women (27–47 yr of age). In addition, in a subset of the subjects, measurements were also obtained from the brachial and popliteal arteries and the abdominal aorta. None of the subjects reported previous cardiopulmonary disease, diabetes, or smoking, and none were taking medication. All subjects gave informed consent according to the Helsinki Declaration, and the study was approved by the Ethics Committee of Lund University.

**MATERIALS AND METHODS**

Subjects. Measurements of the common carotid artery were performed on 10 healthy normotensive subjects: 6 men (27–62 yr of age) and 4 women (27–47 yr of age). In addition, in a subset of the subjects, measurements were also obtained from the brachial and popliteal arteries and the abdominal aorta. None of the subjects reported previous cardiopulmonary disease, diabetes, or smoking, and none were taking medication. All subjects gave informed consent according to the Helsinki Declaration, and the study was approved by the Ethics Committee of Lund University.

Ultrasonic measurements of longitudinal movement and diameter change. B-mode ultrasound was used to measure longitudinal movement and distension (i.e., diameter change) over a preselected segment of the arterial wall. All investigations were performed with a commercial ultrasound system (model HDI 5000, Philips Medical Systems, ATL Ultrasound, Bothell, WA) equipped with a 38-mm 5- to 12-MHz linear-array transducer. The image data were transferred to a personal computer for postprocessing and visualized in HDILab (Philips Medical Systems) software designed for offline cine-loop analysis. During the measurements, the vessels were scanned in the longitudinal direction and oriented horizontally in the image.
LONGITUDINAL MOVEMENTS OF THE ARTERIAL WALL

The methods used to measure the longitudinal movement (7) and the diameter change (6) have been described in detail elsewhere. Briefly, the method used to measure longitudinal movement takes advantage of a block-matching technique, which uses a very small region of interest (ROI) that just encloses the preselected echo, ~0.7 \times 0.7 \text{ mm}, with an accompanying kernel size of 0.1 \times 0.1 \text{ mm}. A small kernel was used, because the tissue region we want to investigate is not random in nature and the movement is essentially within the image plane. There is a definite transition in acoustic impedance between blood and the tunica intima, which gives rise to a significant echo. In an ultrasound image, the arterial wall appears as a typical double-line pattern (28) that originates from echoes from the lumen-intima and media-adventitia boundaries, whereas the luminal and medial layers of the arteries are hypoechoic and appear as dark bands. Thus the lumen-intima and media-adventitia boundaries stand out as isolated echoes, and because the symmetry axis of the vessel should be in the image plane for an optimal recording, the same inhomogeneity or irregularity in the arterial wall will be tracked by the algorithm (7). Hence, among the prerequisites for the measurements, the double-line pattern from the boundaries of the lumen-intima and media-adventitia must be clearly visible at the near and far walls. Furthermore, the preselected distinct echo of an inhomogeneity or an irregularity must be visible in all the images during several cardiac cycles. In addition, to ensure that the recording is performed properly, the longitudinal movement must be visible along the visualized vessel wall segment. Therefore, three recordings of four to six cardiac cycles were conducted on each subject. The recording with the best quality from each subject was chosen, and a mean (SD) of the movements were conducted on each subject. The recording with the best quality from each subject was chosen, and a mean (SD) of the movements during the cardiac cycles was estimated. The resolution and reproducibility of the measurements of longitudinal movement were 5 and 12 \text{ \mu m} (7), respectively.

All measurements were performed in a quiet room with the subject in the supine position after \approx 15 \text{ min of rest}. All subjects were examined by the same experienced ultrasound technician. The transducer was, in all instances, placed in a fixative clamp to avoid introduction of false movements by the operator. Care was taken to minimize the pressure of the transducer. Electrocardiogram was monitored during all the experiments. Blood pressure was measured at the wrist with an oscillometric sphygmomanometer (BPM Wrist 2300, TOPCOM Europe, Heverlee, Belgium) immediately after the two-dimensional arterial wall movements were measured.

**Common carotid artery.** The longitudinal movement and diameter change of the common carotid artery were measured 2–3 cm proximal to the bifurcation. ROIs were positioned around a distinct echo from inhomogeneities in the intima-media complex, the adventitial region, and the surrounding tissue, respectively, of the far wall. The ROI at the adventitia was positioned 0.47–0.89 mm deeper into the vessel wall than the ROI at the intima-media complex. The ROI at the adventitia was positioned 0.03–0.69 mm deeper into the vessel wall than the ROI at the adventitia.

In one subject, a 47-yr-old woman, immediately after measurement of arterial wall movements, blood flow velocity was measured at the same location by means of pulse-wave Doppler close to the far wall. The length of the sample volume was 1 mm, and the Doppler frequency was 6 MHz. The maximum velocity at systole and diastole is presented.

**Abdominal aorta.** The longitudinal movement and diameter change of the abdominal aorta were measured in two men (27 yr of age) and one woman (47 yr of age) \approx 3 \text{ cm proximal} to the aortic bifurcation. ROIs were positioned around a distinct echo from inhomogeneities in the intima-media complex. In addition, in two subjects, ROIs were positioned around an echo of the adventitial region and the surrounding tissue, respectively. The ROIs at the adventitial echo were positioned \approx 1 \text{ mm} deeper into the vessel wall than the ROI at the intima-media complex. The ROI in the surrounding tissue was positioned \approx 1.5 \text{ mm} deeper into the vessel wall than the ROI at the adventitia.

**Brachial artery.** The longitudinal movement and diameter change of the brachial artery were measured in three men (32–62 yr of age) \approx 5 \text{ cm above} the antecubital crease. ROIs were positioned around a distinct echo from inhomogeneities in the intima-media complex of the far wall. In addition, in two subjects, ROIs were positioned in the adventitial echo of the far wall and around an echo in the surrounding tissue. The ROI at the adventitia echo was positioned \approx 0.4 \text{ mm} deeper into the vessel wall than the ROI at the intima-media complex. The ROI in the surrounding tissue was positioned 0.51–1.1 mm deeper into the vessel wall than the ROI at the adventitia.

**Popliteal artery.** The longitudinal movement and diameter change of the popliteal artery were measured in two men (27 and 34 yr of age) and one woman (47 yr of age) at the midpoint of the popliteal fossa. ROIs were positioned around a distinct echo from inhomogeneities in the intima-media complex of the far wall.

**Calculation of internal shear strain within the arterial wall.** To estimate the internal shear strain of the arterial wall from the measurements of longitudinal movement at different positions within the wall, i.e., the intima-media, the region of the adventitia, and the surrounding tissue, the shearing angle (in rad) (21) was calculated as follows:

\[
\begin{align*}
\text{shear strain}_{I-M/Adv} &= \arctan \left( \frac{L\text{Mov}_{I-M} - L\text{Mov}_{I-M,\text{endo}} - [L\text{Mov}_{\text{Adv}} - L\text{Mov}_{\text{Adv,\text{endo}}}]}{\text{radial distance between ROI of I-M and Adv}} \right) \quad (1) \\
\text{shear strain}_{Adv,ti} &= \arctan \left( \frac{L\text{Mov}_{\text{Adv}} - L\text{Mov}_{\text{Adv,endo}} - [L\text{Mov}_{\text{ti}} - L\text{Mov}_{\text{ti,endo}}]}{\text{radial distance between ROI of Adv and ti}} \right) \quad (2)
\end{align*}
\]

where \text{LMov} is longitudinal movement, I-M is intima-media, Adv is adventitia, \text{ti} is surrounding tissue, and \text{ed} is end diastole. The absolute value of the maximum shearing angle during the cardiac cycle is presented.

Values are means (SD) unless otherwise stated. Results in Tables 1 and 2 are mean values based on data from four to six cardiac cycles; in Figs. 2–7, we have chosen to show only two to three of the original four to six cardiac cycles for the sake of clarity. Therefore, in some cases, the values in Tables 1 and 2 do not exactly correspond to the values in Figs. 2–7.

**RESULTS**

**Longitudinal movement of the common carotid artery.** During the cardiac cycle, a distinct multiphasic bidirectional longitudinal movement of the intima-media complex could be observed in all the subjects (Figs. 1 and 2). A distinct antegrade longitudinal movement, i.e., in the direction of blood flow \[0.39 \text{ mm} (SD 0.26), \text{range} -0.03 \text{ to} 0.69\], in early systole was followed by a distinct retrograde longitudinal movement, i.e., in the direction opposite blood flow \[-0.52 \text{ mm} (SD 0.27), \text{range} -0.13 \text{ to} -0.97\], later in systole and a second distinct antegrade longitudinal movement \[0.41 \text{ mm} (SD 0.33), \text{range} 0.10 \text{ to} 1.12\] in diastole (Tables 1 and 2). Thereafter, the intima-media complex gradually returned to its initial position. In 6 of the 10 subjects, the retrograde longitudinal movement in systole was larger than the first antegrade longitudinal movement (Table 2). The corresponding diameter change was of the same magnitude as the longitudinal movements \[0.65 \text{ mm} (SD 0.19), \text{range} 0.43 \text{ to} 1.05\]; Tables 1 and 2, Fig. 2]. The first antegrade longitudinal movement and the distension started almost simultaneously (Figs. 2 and 3). The retrograde longitudinal movement started at almost the same time as the change in the
positive slope of the diameter (Figs. 2 and 3). The second antegrade movement started at almost the same time as the change in the negative slope of the diameter (Figs. 2 and 3). This relation between the longitudinal movement and the diameter change was the same in all the subjects. The direction of blood flow at the vessel wall was antegrade throughout the length of systole and diastole. The maximum velocity of the blood at systole and diastole was 60 and 20 cm/s, respectively, in the 47-yr-old woman in Fig. 3.

The adventitial region showed the same basic pattern of longitudinal movement; however, the magnitude of the move-
arteries during four to six consecutive cardiac cycles.

Table 2. Vascular data of the common carotid artery, abdominal aorta, and brachial and popliteal arteries during four to six consecutive cardiac cycles.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age, yr</th>
<th>1st Antegrade</th>
<th>Retrograde</th>
<th>2nd Antegrade</th>
<th>Strain, rad</th>
<th>ΔD, mm</th>
<th>DA, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>27</td>
<td>0.06 (SD 0.03)</td>
<td>−0.97 (SD 0.01)</td>
<td>1.12 (SD 0.05)</td>
<td>0.94 (SD 0.03)</td>
<td>0.85 (SD 0.01)</td>
<td>5.56 (SD 0.01)</td>
</tr>
<tr>
<td>F</td>
<td>47</td>
<td>0.67 (SD 0.02)</td>
<td>−0.25 (SD 0.02)</td>
<td>0.11 (SD 0.03)</td>
<td>0.35 (SD 0.02)</td>
<td>0.62 (SD 0.01)</td>
<td>6.31 (SD 0.01)</td>
</tr>
<tr>
<td>M</td>
<td>62</td>
<td>0.18 (SD 0.03)</td>
<td>−0.35 (SD 0.04)</td>
<td>0.14 (SD 0.01)</td>
<td>0.11 (SD 0.01)</td>
<td>0.43 (SD 0.01)</td>
<td>5.76 (SD 0.01)</td>
</tr>
<tr>
<td>F</td>
<td>47</td>
<td>0.69 (SD 0.03)</td>
<td>−0.82 (SD 0.02)</td>
<td>0.50 (SD 0.03)</td>
<td>0.45 (SD 0.01)</td>
<td>0.56 (SD 0.01)</td>
<td>5.58 (SD 0.02)</td>
</tr>
<tr>
<td>M</td>
<td>32</td>
<td>0.33 (SD 0.03)</td>
<td>−0.73 (SD 0.04)</td>
<td>0.73 (SD 0.06)</td>
<td>0.49 (SD 0.03)</td>
<td>1.05 (SD 0.02)</td>
<td>6.56 (SD 0.03)</td>
</tr>
<tr>
<td>M</td>
<td>34</td>
<td>0.43 (SD 0.08)</td>
<td>−0.32 (SD 0.10)</td>
<td>0.10 (SD 0.04)</td>
<td>0.29 (SD 0.09)</td>
<td>0.56 (SD 0.02)</td>
<td>6.59 (SD 0.07)</td>
</tr>
<tr>
<td>F</td>
<td>34</td>
<td>0.26 (SD 0.04)</td>
<td>−0.13 (SD 0.02)</td>
<td>0.11 (SD 0.03)</td>
<td>0.53 (SD 0.00)</td>
<td>6.78 (SD 0.01)</td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>39</td>
<td>0.69 (SD 0.05)</td>
<td>−0.58 (SD 0.05)</td>
<td>0.35 (SD 0.02)</td>
<td>0.17 (SD 0.03)</td>
<td>0.64 (SD 0.02)</td>
<td>6.12 (SD 0.04)</td>
</tr>
<tr>
<td>F</td>
<td>27</td>
<td>0.54 (SD 0.05)</td>
<td>−0.64 (SD 0.04)</td>
<td>0.47 (SD 0.05)</td>
<td>0.30 (SD 0.05)</td>
<td>0.50 (SD 0.02)</td>
<td>5.42 (SD 0.04)</td>
</tr>
<tr>
<td>M</td>
<td>27</td>
<td>0.03 (SD 0.02)</td>
<td>−0.39 (SD 0.02)</td>
<td>0.45 (SD 0.03)</td>
<td>0.73 (SD 0.08)</td>
<td>6.22 (SD 0.05)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Vascular data of the common carotid artery, abdominal aorta, brachial artery, and popliteal artery during four to six consecutive cardiac cycles.

<table>
<thead>
<tr>
<th>Vascular data from common carotid artery, abdominal aorta, and brachial and popliteal arteries during four to six consecutive cardiac cycles.</th>
<th>CCA</th>
<th>Aorta</th>
<th>Brachial Artery</th>
<th>Popliteal Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔD, mm</td>
<td>ΔD/Δa, %</td>
<td>Internal Wall Shear Strain, rad</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st Antegrade displacement, mm</td>
<td>0.39 (SD 0.26)</td>
<td>1.08 (SD 0.77)</td>
<td>0.08 (SD 0.07)</td>
<td>0.27 (SD 0.24)</td>
</tr>
<tr>
<td>2nd Antegrade displacement, mm</td>
<td>−0.52 (SD 0.27)</td>
<td>−0.76 (SD 0.49)</td>
<td>−0.08 (SD 0.04)</td>
<td>−0.28 (SD 0.04)</td>
</tr>
<tr>
<td>Shear strain, rad</td>
<td>0.41 (SD 0.33)</td>
<td>0.65 (SD 0.19)</td>
<td>1.12 (SD 0.55)</td>
<td>0.11 (SD 0.03)</td>
</tr>
<tr>
<td>Intima-media-adventitial region</td>
<td>0.36 (SD 0.26)</td>
<td>0.34d</td>
<td>0.22d</td>
<td>e</td>
</tr>
<tr>
<td>Adventitial region</td>
<td>0.49 (SD 0.20)</td>
<td>0.20d</td>
<td>0.08d</td>
<td>e</td>
</tr>
</tbody>
</table>

Values are means (SD); n, number of subjects. CCA, common carotid artery; 1st antegrade longitudinal displacement, antegrade longitudinal displacement of the intima-media complex during early expansion of the artery; retrograde longitudinal displacement, retrograde longitudinal displacement of the intima-media complex; 2nd antegrade longitudinal displacement, antegrade longitudinal movement of the intima-media complex during diastole; ΔD, diameter change; DA, diameter at diastole; ΔD/Δa, arterial strain in radial direction. *Not distinct enough to allow measurement. †Not detectable. ‡No distinct echo in the adventitial region could be followed throughout the cine loop, resulting in inaccurate tracking. *There were no distinct echoes in the adventitial region or the surrounding tissue that could be followed throughout the cine loop, resulting in inaccurate tracking.
Longitudinal movements of the arterial wall.

The adventitial region showed the same basic pattern of longitudinal movement, but, as in the common carotid artery, the magnitude of the movement of the adventitial region was smaller than that of the intima-media complex (Fig. 6A; see supplemental video 2 at http://ajpheart.physiology.org/cgi/content/full/00988.2005/DC1), thus introducing shear strain within the wall of this vessel as well (Fig. 6B). In the two subjects, the maximum shear strain was 0.34 rad (range 0.19–0.50) between the intima-media complex and the adventitia and 0.20 rad (range 0.06–0.33) between the adventitia and the surrounding tissue during the cardiac cycle (Tables 1 and 2).

Longitudinal movement of the brachial artery. Also in the brachial artery, a distinct bidirectional longitudinal movement of the intima-media complex was observed during the cardiac cycle (Fig. 7A). However, the pattern of movement differed among the subjects. In two subjects, an antegrade longitudinal movement that started almost simultaneously with distension of the artery (0.13 mm, range 0.11–0.14) was followed by a retrograde longitudinal movement (−0.06 mm, range −0.06 to −0.06; Table 2). Thereafter, the intima-media complex gradually returned to its initial position. In the third subject, a first antegrade movement could not be detected, and a retrograde movement, which started almost simultaneously with distension of the artery, was seen (−0.24 mm; Table 2). Thereafter, the intima-media complex gradually returned to its initial position. The corresponding diameter change was 0.38 mm (SD 0.08) (range 0.29–0.44; Tables 1 and 2).

**DISCUSSION**

Using a new noninvasive ultrasonic method, we clearly demonstrate, for the first time, a distinct longitudinal movement of the arterial wall during the cardiac cycle that is of the same magnitude as the diameter change in the common carotid artery of healthy humans, in contrast to current conjecture. The longitudinal movement shows a distinct multiphasic bidirectional pattern, with a distinct antegrade movement in early systole, i.e., in the direction of blood flow, that is followed by a distinct retrograde movement, i.e., in the direction opposite blood flow, later in systole. In diastole, a second distinct antegrade longitudinal movement is seen before the vessel wall gradually returns to its initial position. In the present study, we also clearly demonstrate that the inner parts of the vessel wall, the intima-media complex, show a larger longitudinal movement than the outer part of the vessel wall, the adventitial region. Thus there is shear strain and, accordingly, shear stress within the vessel wall, which has not previously been described.

Because of the striking longitudinal arterial wall movements found in the easily accessible common carotid artery, a predominantly elastic artery, we were curious to find whether such longitudinal movements are present in other arteries. Therefore, although at the limit of the capacity of this new method and any other technique that is available today, the longitudinal movements of the abdominal aorta and the muscular brachial...
or popliteal arteries were also investigated in a subset of the healthy individuals. Also in these vessels, a distinct bidirectional longitudinal movement of the intima-media complex of the same magnitude as the diameter change was clearly demonstrated and a distinct retrograde movement in systole was seen. However, as expected, because of physiological differences in the arterial tree, the pattern of movement differed and a second distinct antegrade movement during diastole was not as obvious as in the common carotid artery, although it seems to be present in several cardiac cycles. Apart from physiological differences in the arterial tree, methodological factors may, however, also contribute to this finding. In the aorta, the deeper location of the vessel resulted in a lower frame rate, making small movements more difficult to detect and measure; in the brachial artery, the smaller movements resulted in noisier recordings. Importantly, the magnitude of the longitudinal movements of the intima-media complex is larger than that of the adventitial region also in the abdominal aorta and brachial artery. Accordingly, there is also shear strain and, thus, shear stress within the wall of these arteries, indicating that this might be a general phenomenon.

The pattern of the longitudinal movement in the common carotid arterial wall implies that the mechanisms responsible for the longitudinal movement are not easily clarified, and several interacting factors seem to be involved. In vivo, at least two structures provide arterial fixation to the surrounding structures, the perivascular connective tissues and the arterial side branches, causing tethering (21). Furthermore, arteries in the body are naturally under a condition of longitudinal tension (21). The circumferential elastic modulus of the arterial wall has been shown to differ in the arterial tree, with central arteries being more elastic than peripheral arteries because of differences in the collagen-to-elastin ratio. Furthermore, in predominantly elastic arteries, such as the aorta and common carotid artery, the circumferential elastic modulus has been shown to be age and sex dependent (14, 31). Studies on the longitudinal elastic modulus of arteries are sparse, and the results from earlier in vitro studies are not clear-cut (21). Further studies are needed to show whether the pattern and magnitude of the longitudinal arterial wall movements and/or the longitudinal elastic modulus are influenced by age and/or sex.

The primary hemodynamic forces that act on the arterial vessel wall consist of pressure acting perpendicular to the vessel wall, resulting in cyclic diameter change and a circumferential or radial strain, and pulsatile blood flow acting as a shear force in the direction parallel to the vessel wall. A first obvious hypothesis is that the shear force from the blood flow is an important factor in the longitudinal arterial wall movements, although this alone does not explain the large longitudinal movement in the direction opposite blood flow during systole or the distinct multiphasic pattern of movement in the common carotid artery. The longitudinal tension and the elastic recoil of the arterial wall may, however, be contributing factors. Moreover, it is well known that the base of the heart moves toward the ventricular apex in systole (30), which may be an additional factor. Furthermore, in a study of exposed vessels in pigs, it was recently reported that the length of a segment of the common carotid artery changes during the cardiac cycle (32), which may also contribute to the longitudinal movement of the arterial wall. Furthermore, during their

![Fig. 4: Longitudinal movement of the intima-media (solid line), adventitial region (bold dashed line), and surrounding tissue (dashed line) of the common carotid artery in a 27-yr-old woman. Longitudinal movement gradually decreased as depth into the arterial wall increased. B: cyclic shear strain between the intima-media and the adventitial region of the common carotid artery in the subject in A. ECG bottom trace as reference.]
travel in the arterial tree, the pulse waves are reflected when they meet a transition from one vascular impedance to another (21). In the common carotid artery, the timing of the different phases of the longitudinal movement in relation to the distension of the artery, i.e., the diameter change, shows good agreement with the expected arrival of the pulse wave, reflected pulse wave, and rereflected pulse wave, respectively (Figs. 2 and 3), thus suggesting that a longitudinal shear force from the pulse wave might be present. Moreover, an active component from the wall itself cannot be excluded. In our study, the arterial wall movements were recorded immediately after the subject stopped breathing; thus the longitudinal movements of the arterial wall are not secondary to movements of the chest during respiration (7). In conclusion, further research is required to clarify the mechanisms responsible for the longitudinal movements of the arterial wall.

Fig. 5. Longitudinal movement of the intima-media complex of the far wall (solid line) and diameter change (dashed line) in relation to ECG (bottom trace) in the abdominal aorta of a 27-yr-old man. For longitudinal movement, a positive deflection denotes movement in the direction of blood flow. Longitudinal movement is of the same magnitude as diameter change.

Fig. 6. A: longitudinal movement of the intima-media (solid line), adventitial region (bold dashed line), and surrounding tissue (dashed line) of the abdominal aorta in a 27-yr-old man. Longitudinal movement gradually decreases as depth into the arterial wall increases. B: cyclic shear strain between the intima-media and adventitial region of the abdominal aorta in the subject in A. ECG bottom trace as reference.

Fig. 7. A: longitudinal movement of the intima-media complex of the far wall (solid line) and diameter change (dashed line) in relation to ECG (bottom trace) in the brachial artery of a 34-yr-old man. B: longitudinal movement of the intima-media complex of the far wall (solid line) and diameter change (dashed line) in relation to ECG (bottom trace) in the popliteal artery of a 47-yr-old woman. For longitudinal movement, a positive deflection denotes movement in the direction of blood flow. Longitudinal movement is of the same magnitude as diameter change.
The demonstration of a significant longitudinal movement of the arterial wall during the cardiac cycle and the presence of shear strain and, thus, shear stress within the arterial wall is new information that not only has the potential to improve our knowledge of the elastic properties of the arterial wall and improve the ability to detect early abnormalities in arterial wall function, but it may have a wide range of implications for vascular biology and hemodynamics and, thus, for the study of vascular disease. Different vascular beds show substantial variation in their susceptibility to vascular disease. Yet the underlying mechanisms of this heterogeneity are not clear. Among the predisposing factors, the hemodynamic forces shear stress and tensile stress generated by the flowing blood are considered of utmost importance. However, the longitudinal movement of the arterial wall and the resulting shear stress within the wall have been overlooked, and they cannot be excluded as additional factors of vital importance.

The innermost layer of the arterial wall, the intima, consists of vascular endothelial cells that line the inner surface of the blood vessels. This metabolically active monolayer is constantly exposed to biochemical and biomechanical stimuli, and it is well established that the vascular endothelium has a crucial role in vascular biology and the development of atherosclerosis. It has been shown in a variety of studies that shear stress at the lumen-wall interface is an important determinant of endothelial cell function, which is among the factors that influence the production of vasoactive substances such as nitric oxide (29), prostacyclin (10), and endothelin (18). Our study demonstrates that there is also a cyclic shear stress within the arterial wall. Furthermore, the intima-media complex and, thus, the endothelial cells have been shown to be displaced longitudinally in a bidirectional manner in relation to blood flow. Little is known about how this may influence endothelial cell function and the function of the media. The smooth muscle cells of the media, which have long been considered a mainly structural and contractile cell, and the extracellular matrix components have proven to be capable of numerous functions considered important in the pathogenesis of vascular diseases such as atherosclerosis (12). In this context, the increasingly used MRI (22) and ultrasound (5) methods for in vivo estimation of blood flow shear stress are based on the assumption that the longitudinal movements of the arterial wall are negligible. Moreover, the adventitia, the outermost layer of the arterial wall of larger arteries, contains small blood vessels, the vasa vasorum (“vessels of vessels”), which in the largest arteries also penetrate into the media (16). There is increasing evidence suggesting a role for the vasa vasorum in the development of atherosclerotic vascular disease (2, 3, 20). An interesting issue is how the circulation of the vasa vasorum is influenced by cyclic shear stress within the wall and whether it is beneficial.

The longitudinal arterial wall movements and the resulting shear stress within the wall may also have implications for the understanding of the pathophysiology of arterial dissection, such as carotid artery dissection, and play a role in vascular anastomoses and vascular grafts and their long-term outcome. Mismatch between the mechanical properties of native arteries and vascular prostheses has been implicated in the pathogenesis of graft failure (1). Moreover, the longitudinal movements may also have implications for aneurysmal diseases such as abdominal aorta aneurysm. Long-term cyclic stress may weaken a material and increase its vulnerability to fracture.

Another challenging issue for future studies is whether the new unique opportunity to noninvasively measure the longitudinal movements of the arterial wall at different depths in the wall, including plaque movement and shearing beneath the plaque, may aid in the characterization and assessment of risk of atherosclerotic plaques. The need for new methods to improve risk assessment of atherosclerotic plaques has recently been advocated (19). The risk of plaque disruption is a function of the intrinsic properties of individual plaques and the extrinsic forces acting on the plaque (9). The motion of the carotid atherosclerotic plaque has recently been suggested to be related to the risk of cerebral events (11).

With the present technique and transducers, we cannot with certainty discriminate putative differences in movement of the intima, one cell layer, from the media. Therefore, we have chosen to describe the movement of an echo in the intima-media complex. Thus we cannot tell whether there is shear strain and, thus, shear stress between the intima and the media as well or within the media. It would be of great interest to be able to separate also the intima and the media and to discriminate putative differences in movements between these layers. Further technical improvements and the use of very high-frequency transducers will be needed to achieve this in the future.

It will also be of great interest to measure the longitudinal movement at arterial sites that are known to be predisposed to atherosclerosis, such as the carotid bifurcation, proximal internal carotid artery. However, because of the complex geometry of the bifurcation with turbulent flow and complex differences in the mechanical properties (based on studies of diameter change), for these first in vivo studies of an overlooked phenomenon, we have chosen to observe sites with simple geometry and predictable hemodynamics.

In conclusion, this study clearly demonstrates that in central elastic arteries, as well as in large muscular arteries, in humans there is a distinct longitudinal movement of the arterial wall during the cardiac cycle of the same magnitude as the diameter change, which is contrary to previously reported findings. We also demonstrate that the inner part of the vessel wall, the intima-media complex, show a larger longitudinal movement than the outer part of the vessel wall, the adventitial region, which introduces substantial shear strain and, thus, shear stress within the vessel wall. This completely new information seems to be of fundamental importance for the further study and evaluation of vascular hemodynamics and biology and, accordingly, for the study of atherosclerosis and vascular diseases. Further study is needed to elucidate the underlying mechanisms and the clinical implications of this phenomenon.

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