Measurement of Vessel Wall Strain Using Cine Phase Contrast MRI

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Purpose: To determine the feasibility of using magnetic resonance imaging (MRI) to non-invasively measure strain in the aortic wall.

Materials and Methods: Cine phase contrast MRI was used to measure the velocity of the aortic wall and calculate changes in circumferential strain over the cardiac cycle. A deformable vessel phantom was used for initial testing and in vitro validation. Ultrasonic sonomicrometer crystals were attached to the vessel wall and used as a gold standard.

Results: In the in vitro validation, MRI-calculated wall displacements were within 0.02 mm of the sonomicrometer measurements when maximal displacement was 0.28 mm. The measured maximum strain in vitro was 0.02. The in vivo results were on the same order as prior results using ultrasound echo-tracking.

Conclusion: Results of in vivo studies and measurement of cyclic strain in human thoracic and abdominal aortas demonstrate the feasibility of the technique.

Key Words: vessel wall motion; vessel wall strain; velocity measurements; phase-contrast MRI; human aorta


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ARTERIAL WALL MOTION and strain are hypothesized to affect the development of arterial disease (1–4). Arterial wall motion can be detected non-invasively with an ultrasonic echo-tracking technique (5). However, this technique is effective only where an appropriate acoustic window is present, precluding its use in the human thoracic aorta. A non-invasive method with more complete coverage will enable the comparison of the deformation of the thoracic and abdominal aorta, which may relate to relative differences in pulse pressure or wall compliance. Such a comparison is of clinical relevance because of the differential incidence of disease between the thoracic and abdominal aorta (6).

The purpose of the current investigation was to assess the feasibility of using magnetic resonance imaging (MRI) for the in vivo measurement of cyclical changes in strain in the wall of the aorta. The method was validated on a deformable vessel model and tested in normal human volunteers. Feasibility was judged based on the level of agreement between MRI calculations and sonomicrometer measurements in vitro, and on our ability to repeatedly obtain in vivo results with sufficient image quality to provide strain measurements similar to results in the literature.

MATERIALS AND METHODS

MRI is well suited to the task of measuring vessel wall strain non-invasively. The problem of measuring vessel wall strain is analogous to measurements of myocardial motion except that the tissue displacements are smaller and the walls are thinner. Time-resolved conventional magnitude images could be used to quantify wall motion, but this method suffers from a need for sub-millimeter image resolution to track the small displacements of material points in the aorta, and is prone to errors due to out-of-plane vessel motion. Two alternate MRI-based methods that can be used to measure tissue displacement are tagging (7–9) and phase-contrast velocity imaging (10–13).

MRI tagging was used successfully for in vitro studies of a model aorta (14) where high resolution images across a small field of view could be obtained using a small coil; however, surface coils do not penetrate deeply enough in vivo to evenly image the aorta. For this reason and the difficulty of producing an appropriate (radial) array of thin (< 1 mm) tags, we chose not to use tagging for our study.

In a typical cross-sectional MR image, the maximal frame-to-frame displacement of the aortic wall in vivo is between one and three pixels, and the average frame-to-frame displacement was less than half of one pixel. Phase contrast velocity imaging can detect sub-pixel
motion by using in-plane velocities to calculate displacements. Phase contrast velocity imaging, therefore, was thought to be an ideal technique for aortic imaging in vivo.

The theoretical basis for using in-plane velocity measurements to calculate vessel wall cyclic strain was first outlined by Draney et al (15).

**Strain Formulation**

Strain is a measure of deformation. Aortic radial deformations in vivo have been reported to be greater than 5% over the cardiac cycle (16,17). We therefore chose a finite strain measure, the Green-Lagrange strain tensor (18). Under the simplifying assumptions of plane strain (i.e., we ignore through-plane deformation), uniform radial expansion, and approximating the vessel wall as a membrane, the circumferential strain, $E_{cc}$, can be expressed in terms of the deformations in cylindrical coordinates (see appendix),

$$ E_{cc} = \frac{1}{2} \left( \frac{r}{R_0} \right)^2 - 1 $$

where $R_0$ is the vessel radius at a reference time point, chosen to be the smallest radius observed over the cardiac cycle, and $r$ is the radius measured at any other time in the cardiac cycle. As described below, $R_0$ was automatically determined directly from the magnitude data by averaging the distance from the vessel center to all points in the vessel wall. All other radii, $r$, were calculated from $R_0$ and the in-plane velocities measured in the vessel wall. Calculating radii from in-plane velocities allowed us to detect sub-pixel changes in the radius of the vessel.

**Data Acquisition**

Using a 1.5-T Signa CV/i MRI scanner (GE Medical Systems, Milwaukee, WI) with a maximum slew rate of 150 T/m/second, two time-resolved, cross-sectional scans of the vessel were acquired. The first acquisition, the strain scan, used a modified cine phase contrast pulse sequence to quantify the in-plane motion of the vessel wall. Radiofrequency (RF) spoiling was implemented to reduce artifacts due to motion-induced fluctuations in the transverse steady state. Our method requires only two in-plane velocity components. However, removing the sensitivity to the third component in the product sequence was technically difficult. Therefore, velocity was measured along all three directions using a four-point technique, but with different velocity sensitivity for the two in-plane directions vs. the through-plane direction. A low in-plane venc (2–5 cm/s) was used to achieve a reasonable velocity signal-to-noise level. A high through-plane venc (100-200 cm/second) was chosen to reduce pulsatility ghosting. Pre-saturation slabs above and below the slice of interest reduced the signal of the fluid, improving segmentation of the wall. Parameters used for both the phantom and volunteers are listed in Table 1. The second cross-sectional acquisition collected through-plane velocity data of the fluid or blood with a standard cine phase contrast pulse sequence with venc set to 100 cm/second (in vitro) or 200 cm/second (in vivo). Neither saturation pulses nor in-plane velocity encoding were used, resulting in a TR of 18–19 msec. Temporal resolution was further improved by measuring only the through-plane velocity.

Phantom scans were performed in the head coil. Volunteer scans used the body coil for excitation and a quadrature abdominal coil or a phased array coil (either torso or cardiac) for reception. Even with the phased-array coils, the image signal was relatively uniform over the vessel of interest.

The total scan time depended on the heart rate. At 70 beats per minute, each scan took 7.3 minutes. In vivo data were acquired during free breathing with a standard respiration compensation algorithm.

**Strain Calculation**

All strain analysis was performed using algorithms implemented within Matlab (MathWorks, Inc., Natick, MA). User input was needed to identify 12–18 points in the middle of the vessel wall, which is visible as a locally bright structure, at each time frame on the magnitude images. All subsequent analysis was performed automatically.

For the purpose of identifying which pixels to use for the analysis, a cubic spline was fit to the user-defined points. All image pixels that intersected the spline were considered to be within the vessel wall. To account for the wall thickness, four pixels (one in each Cartesian direction) adjacent to each spline pixel were also considered to be within the vessel wall. This approach is supported by the visual appearance of the vessel images and by the expected thickness of the vessel wall. Pixels with magnitude values less than 30% of the maximum wall value were then removed from further analysis. The remaining pixels were divided into 16 sectors (Fig. 1). The mean spatial velocity over all sectors, which estimates the global motion of the vessel, was calculated (Fig. 1a) and subtracted from the velocity in each sector (Fig. 1b). The radial component of the velocity in each sector was then determined (Fig. 1c).

After the radial velocities were calculated for all the time frames, the temporal average radial velocity in each sector was subtracted, forcing the average velocity to be zero, as expected for periodic motion. Non-zero mean velocity was primarily caused by eddy currents, though motion ghosting occasionally contributed addi-
tional phase offsets. The corrected sector velocities were used to calculate an average radial velocity for the entire vessel at each point in time.

Radial displacements were calculated from radial velocity data as follows. First, a reference radius was automatically chosen by identifying the last time frame containing negative radial velocities (i.e., final point of contraction) and calculating the average radius from the spline fit of the next time frame (which should be the smallest radius, $R_0$). The radii of subsequent time frames were calculated using a forward-backward integration scheme (19) to reduce the effect of accumulating errors on the calculated radii. These radii were used in the strain calculations (Eq. 1). Note that in this velocity-based calculation, displacements that are far smaller than the pixel spacing can be measured.

**In Vitro Validation**

Our method to quantify cyclic strain using in-plane velocities was validated using a model vessel made of polyvinyl alcohol (PVA) cryogel (14), a deformable material with excellent tissue-mimicking properties for MR and ultrasound imaging. The model was 20 cm long and 2.5 cm in diameter, with 0.3-cm-thick walls. The ends of the model vessel were attached to the inlet and outlet of a custom built flow chamber, which was then filled with water to provide signal outside the model vessel (Fig. 2). A five-inch surface coil was placed directly on top of the water-filled chamber.

The inlet and outlet of the chamber were connected to a flow system controlled by a computerized flow pump (Shelley, Ltd. London, Ontario). The pump was programmed to produce a sinusoidal flow pattern with a 19.8 mL/second peak-to-peak amplitude at the pump output orifice and an average flow of 5.2 mL/second. The prescribed flow rate was lower than that expected in the human aorta in vivo, and resulted in small vessel motions. This provided a test of our method in detecting and quantifying small deformations.

Ultrasound sonomicrometry was used to provide an independent measure of the change of model vessel radius. We attached four small (3 mm diameter) sonomicrometer crystals (Triton Technology, Inc., Wellesley, MA) to opposing sides of the model vessel. The transit time of an ultrasound pulse across the vessel was recorded as a function of time and converted

**Figure 1.** Steps involved in determining mean radial velocity superimposed on a detail from an in vivo magnitude image. The wall is divided into 16 segments (shown in black). The mean in-plane velocity for each sector is shown by the white arrows before any processing (a), after subtracting rigid body motion (b), and after removing tangential components (c).

**Figure 2.** Model vessel (made from PVA) in the chamber used for measurement. Ultrasound sonomicrometers (US) are attached to the side of the vessel. The chamber is filled with water, and the inlet is connected to a computerized flow pump.
into a measure of distance using the known speed of sound in water at room temperature. The same flow waveform was used to drive the flow system with the model vessel in the MR imager. Phase contrast data were acquired according to the previously described protocol. The change in vessel diameter over time as measured by sonomicrometry was compared to the change in diameter as calculated from phase contrast data.

**RESULTS**

**In Vitro**

The model vessel, with its thick wall and close proximity to the receive coil, produced images with very high signal-to-noise ratios (SNR ~ 125), measured as the signal in the vessel wall divided by the standard deviation (SD) in the background. The change in radius as a function of time, as measured by sonomicrometry (circles) and as calculated from the MRI data (squares), is shown in Figure 3a. The 0.28-mm change in diameter was readily measured. The largest discrepancy between the displacements was less than 0.02 mm, as shown in the Bland Altman style plot (20) in Figure 3b. The 0.02-mm discrepancy is less than 6% of peak-to-peak amplitude of motion and only 1/32 of the pixel width.

The relative circumferential strain calculated from the MRI data is plotted in Figure 4 along with the through-plane flow as measured by MRI during a separate acquisition. The compliance of the tubing caused damping of the waveform such that the peak flow at the site of MRI flow measurements was less than that in the prescribed flow. The mean flow rate was conserved. The strain was smaller than typical in vivo strain because of the limited output of the mechanical flow pump. The mean and maximum circumferential strain in vitro were 0.01 and 0.02, respectively.

Error bars on the strain data in Figure 4 depict the SD of the strain. We examined the SD in the mean radial velocity as a function of time and found that it was roughly uniform across time frames, so we combined these into a single estimate of the SD of the mean radial velocity. This was propagated through the calculations of radius (11) and strain. Since they are based on an experimentally-measured variance, these error bars reflect all sources of variability.

**In Vivo**

Images from two of the in vivo scans are shown in Figures 5 and 6. Figure 5 shows an example of the thoracic aorta at the level of the right pulmonary artery.
(thoracic 1), while Figure 6 shows the abdominal aorta mid-way between the renal arteries and the iliac bifurcation (abdominal 2). Four time frames within the cardiac cycle are shown. The SNRs of all in vivo images (measured as the signal in the vessel wall divided by the SD in the background) were between 8 and 15.

The circumferential strain calculations and through-plane flow measurements for all volunteers are plotted in Figures 7 (thoracic waveforms) and 8 (abdominal waveforms). Note that the scale used for flow measurements (right-hand axis) in the abdominal data (Fig. 8) is half of that used for the thoracic data (Fig. 7). The mean strain values were all within a range of 0.01–0.04, and maximum strains were within 0.04–0.08. Table 2 lists the mean and maximum strain for all the experiments along with the age of the volunteers.

The error bars on the strain measurements in Figures 7 and 8 reflect the SD of the strain, which was derived from the variance of the mean radial velocity as described above for the in vitro case.

**DISCUSSION**

The in vitro experiments produced excellent results, showing uniform expansion and contraction over the course of the cardiac cycle. The agreement between the
ultrasound data and the MRI-calculated radii validated our ability to use in-plane velocity measurements to track the expansion and contraction of vessels in an ideal setting. The in vitro experiment included a thick vessel wall, clear separation of the vessel wall from its surroundings, high signal due to the close proximity of the phantom to the receiving coil, lack of background motion, and reproducible periodic motion of the phantom. The lag between the strain and flow curves of Figure 4 is expected because the vessel changes shape proportional to pressure and the peak flow is known to precede the peak pressure (21).

The results in humans demonstrate the feasibility of using cine phase-contrast MRI for non-invasive in vivo measurement of circumferential strain in the thoraco-abdominal aorta. All curves show smoothly varying strain that lags the through-plane flow measurements, though not often by very much. The smaller lag (when compared to the in vitro results) is likely a result of the differences in downstream flow conditions, geometry, and vessel wall compliance between the phantom and in vivo studies. Our data suggest that this method may be able to measure the expected reduction of vessel strain with age, which is a factor of 50% or more (16,17,22).

Vessel strain can also be studied using ultrasound echo-tracking (5,23). Using a linear definition of strain as the difference in diameter between systole and dias-

**Figure 6.** Magnitude images of the abdominal aorta at four points in the cardiac cycle. The labels Ab1–Ab4 refer to the corresponding time points labeled in Figure 8. The contrast and brightness levels have been set to highlight the aortic wall.
tolerates over the diastolic diameter, two studies found that abdominal strain was $0.10 \pm 0.02$ and $0.076 \pm 0.024$, respectively, in normal volunteers under 35 years of age (16,17). Another study, primarily of older patients with heart disease, measured strain in two older normal volunteers, age 47 and 58, of $0.033$ and $0.038$, respectively (22). Transcutaneous ultrasound relies on an absence of bone or air between the transducer and the vessel of interest. This makes investigation of the thoracic aorta very difficult, whereas MRI can be used to image the entire length of the aorta. Further, the intra-observer variability with ultrasound was high (16% in the abdominal aorta) (24).

Our measurements of maximal abdominal strains calculated from the MRI data of $0.04 - 0.08$ (Table 2) were lower than those determined by ultrasound echotacking, but the measurements are of the right order of magnitude. There are differences in the two methods that may help explain the discrepancies. First, MRI averages data, spatially and temporally, and an average measurement will always be lower than the maximum instantaneous value whenever there is underlying noise and variation. Second, the ultrasound technique corrects for bulk motion only along the beam direction. With the MRI technique, we were able to subtract all in-plane motion. Movement of the vessel perpendicular to the ultrasound beam can mimic diameter changes and cause overestimation of strain. Finally, our calculation method (Eq. 1) also differs from the linear (small strain) method used in ultrasound. However, this is not a major source of discrepancy for the ranges of strains in this study.

When comparing the in vivo MRI measurements to our phantom data, the lower SNR and increased motion artifacts presented some difficulty in the analysis. The phased array coils (mean SNR = 12) performed better than the quadrature coil (mean SNR = eight) as expected, producing more reproducible strain measurements. The SNR was occasionally degraded by respiration artifacts that were superimposed over the area of interest. The SNR for one such subject was only seven, despite the use of a phased array coil. In all cases, placing the phase-encode axis in the right-left direction reduced the effects of ghosting on the depiction of the aorta.

The abdominal aorta proved more challenging to image than the thoracic aorta. In the thoracic region (Fig. 5), very little tissue surrounds the aorta. When the blood is successfully saturated, the aortic wall is well depicted. In the abdomen, the aorta is smaller in diameter and located more centrally in the body. Therefore, the signal of the vessel wall is lower. Further, more tissue surrounds the abdominal aorta, making the outer edge of the vessel wall more difficult to discern.

Manual user input to identify the vessel wall is a potential source of error, but the velocity in neighboring points is expected to be correlated. Although the wall may be mis-identified, the velocities of the tissue or

![Figure 7. Relative circumferential strain (closed circles) and through-plane flow (open boxes) from four volunteers at various levels of the thoracic aorta. The error bars reflect the SD of the strain measurements. The labels Th1–Th4 are the time points at which the images in Figures 5 and 9 were acquired.](image-url)
blood adjacent to the wall in high resolution images may give a reasonable approximation of the velocity of the wall itself. A bigger concern is the increase in the variance of the velocity in voxels with very low signal.

In both the abdominal and thoracic aorta, there were cine-frames in which the wall signal was too low or too blurred and reduced our confidence in the velocity measurements. We attribute most of this to the limited temporal resolution. The in-plane measurements were made with a four-point technique, meaning four sequence excitations were performed consecutively to make a velocity-sensitized measurement. With a 29 msec TR, the true temporal resolution was four \( \times 29 = 116 \) msec. The bulk of the aortic motion happens during a 300 msec period corresponding to the systolic push. One way to improve the temporal resolution is to acquire each of the four flow encodings in a separate heartbeat. The disadvantage to this scheme is that it converts a 7–9 minute scan into a 28–36 minute scan. Alternatively, acquiring measurements with two (rather than four) flow encodings in each heartbeat would double the temporal resolution by doubling the scan time. A three-point, two-direction interleaved scan would reduce the temporal resolution to 87 msec without changing the scan time. These last two options are feasible, but they were not evaluated in the current study.

While our results are promising, our data indicate that the vessels in vivo appear to expand and contract non-uniformly; our strain calculation algorithm assumed uniform strain. Figures 9 and 10 show velocity vectors from 16 sectors superimposed on magnitude images of the aorta at the thoracic (Fig. 9) and abdominal (Fig. 10) level. The expansion phase is fairly uniform in both cases, but the vessel wall appears to twist in later phases. While we are not aware of reports in the literature supporting this observation, we are also not aware of reports to the contrary. The wall could be experiencing strain terms in addition to the circumferential strain that we calculated. Our method assumes plane-strain and treats the vessel wall as a membrane. It was not possible in the current study to determine the accuracy of these assumptions.

Table 2
Mean and Maximum Circumferential Strain Calculated From Phase-Contrast Velocity Measurements in the Aortic Wall With Age of Volunteers

<table>
<thead>
<tr>
<th>Aorta</th>
<th>Mean strain</th>
<th>Max strain</th>
<th>Age (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic 1</td>
<td>0.03</td>
<td>0.07</td>
<td>25</td>
</tr>
<tr>
<td>Thoracic 2</td>
<td>0.04</td>
<td>0.08</td>
<td>28</td>
</tr>
<tr>
<td>Thoracic 3</td>
<td>0.02</td>
<td>0.04</td>
<td>41</td>
</tr>
<tr>
<td>Thoracic 4</td>
<td>0.03</td>
<td>0.05</td>
<td>36</td>
</tr>
<tr>
<td>Abdominal 1</td>
<td>0.02</td>
<td>0.05</td>
<td>27</td>
</tr>
<tr>
<td>Abdominal 2</td>
<td>0.01</td>
<td>0.04</td>
<td>31</td>
</tr>
<tr>
<td>Abdominal 3</td>
<td>0.02</td>
<td>0.05</td>
<td>21</td>
</tr>
<tr>
<td>Abdominal 4</td>
<td>0.02</td>
<td>0.05</td>
<td>24</td>
</tr>
</tbody>
</table>
sumptions. Improvements in SNRs and temporal resolution, as well as in the analysis algorithms, are needed to more fully investigate the strain of the aorta wall with MRI.

In conclusion, we have demonstrated the feasibility of a new non-invasive method to quantify aortic wall strain in vivo. The technique was validated by sonomicroscopy in vitro. Strain measurements in the abdom-

Figure 9. Velocity vectors of the thoracic aorta at different points in the cardiac cycle. Rigid body motion has been subtracted. Circumferential strain analysis is done only on the radial component of the velocity. The labels Th1–Th4 refer to the corresponding time points labeled in Figure 7.

Figure 10. Velocity vectors of the abdominal aorta at different points in the cardiac cycle. Rigid body motion has been subtracted. Circumferential strain analysis is done only on the radial component of the velocity. The labels Ab1–Ab4 refer to the corresponding time points labeled in Figure 8.
nal aorta were of the same order of magnitude of similar measurements done with ultrasound echo-tracking. The level of agreement is considered good when taking into account fundamental differences between the two techniques. The in vivo measurements quantify, non-invasively, pulsatile circumferential vessel wall strain in both the thoracic and abdominal aorta.

**APPENDIX**

Strain is described by a $3 \times 3$ tensor; in this paper we focus on one term of the tensor. The assumptions used in making this simplification are outlined below.

The Lagrangian Finite Strain tensor, $E$, can be expressed as a function of the Cauchy-Green deformation tensor, $C$, and the identity tensor, $I$, (18)

$$ E = \frac{1}{2} (C - I). \quad (A1) $$

The deformation tensor can be expressed in terms of the deformation gradient, $F$.

$$ C = F^T F. \quad (A2) $$

where $F^T$ is the transpose of $F$. If $F$ is symmetric, $F^T = F$. The deformation gradient, $F$, is determined by the displacement vector, $u$, which we derive from the MRI-velocity data:

$$ F = I + \nabla u. \quad (A3) $$

We express the displacement in cylindrical coordinates, with the $z$-axis aligned with the axis of the vessel:

$$ u = u_r \hat{r} + u_\theta \hat{\theta} + u_z \hat{z}. \quad (A4) $$

The gradient of $u$ in cylindrical coordinates can then be expressed as:

$$ [\nabla u] = \begin{bmatrix}
\frac{\partial u_r}{\partial r} & \frac{1}{r} \left( \frac{\partial u_r}{\partial \theta} - u_\theta \right) & \frac{\partial u_r}{\partial z} \\
\frac{\partial u_\theta}{\partial r} & \frac{1}{r} \frac{\partial u_\theta}{\partial \theta} + u_r & \frac{\partial u_\theta}{\partial z} \\
\frac{\partial u_z}{\partial r} & \frac{1}{r} \frac{\partial u_z}{\partial \theta} & \frac{\partial u_z}{\partial z}
\end{bmatrix}. \quad (A5) $$

We neglect any through-plane displacements ($u_z = 0$) and any $z$ dependence of the in-plane displacements ($\frac{\partial u_r}{\partial z} = \frac{\partial u_\theta}{\partial z} = 0$). This is consistent with an assumption of plane strain. Next, we assume that the vessel expansion will be uniform around the vessel so that $u_\theta = 0$, $\frac{\partial u_r}{\partial \theta} = 0$, and $\frac{\partial u_z}{\partial \theta} = 0$. Finally, the vessel wall is approximated as a membrane, and we assume the radial displacement is uniform, and thus $\frac{\partial u_r}{\partial r} = 0$. Substituting these assumptions into Eq. A5, we get:

$$ [\nabla u] = \begin{bmatrix}
0 & 0 & 0 \\
0 & \frac{1}{r} (u_r - u_\theta) & 0 \\
0 & 0 & 0
\end{bmatrix}. \quad (A6) $$

Using Eq. A6 in Eq. A3, we can calculate

$$ [F] = [F]^T = \begin{bmatrix}
1 & 0 & 0 \\
0 & 1 + \frac{1}{r} (u_r - u_\theta) & 0 \\
0 & 0 & 1
\end{bmatrix}. \quad (A7) $$

Since $F$ is symmetric, $C = F^2$, which yields

$$ [C] = \begin{bmatrix}
1 & 0 & 0 \\
0 & 1 + 2 \frac{u_r}{r} + \frac{u_\theta^2}{r^2} & 0 \\
0 & 0 & 1
\end{bmatrix}. \quad (A8) $$

Finally, the full strain tensor in cylindrical coordinates can be expressed as

$$ [E] = \begin{bmatrix}
0 & 0 & 0 \\
0 & \frac{u_r}{r} + \frac{1}{2} \frac{u_\theta^2}{r^2} & 0 \\
0 & 0 & 0
\end{bmatrix}. \quad (A9) $$

Because of our simplifying assumptions, the only non-zero term is the term describing circumferential strain, $E_{\phi \phi}$. Let $R_0$ be the smallest radius of the vessel. The radial displacement at any point in time, $u_r$, is $r(t) - R_0$.

$$ E_{\phi \phi} = \frac{r(t) - R_0}{R_0} + \frac{1}{2} \frac{(r(t) - R_0)^2}{R_0^2}. \quad (A10) $$

which simplifies to the expression in Eq. 1:

$$ E_{\phi \phi} = \frac{1}{2} \left( \frac{r(t)^2}{R_0^2} - 1 \right). \quad (A11) $$

**REFERENCES**


