Quantification of Vessel Wall Motion and Cyclic Strain Using Cine Phase Contrast MRI: In Vivo Validation in the Porcine Aorta

Mary T. Draney,1 Frank R. Arko,3 Marcus T. Alley,2 Michael Markl,2 Robert J. Herfkens,2 Norbert J. Pelc,2 Christopher K. Zarins,3 and Charles A. Taylor1,3

Artery wall motion and strain play important roles in vascular remodeling and may be important in the pathogenesis of vascular disease. In vivo observations of circumferentially nonuniform wall motion in the human aorta suggest that nonuniform strain may contribute to the localization of vascular pathology. A velocity-based method to investigate circumferential strain variations was previously developed and validated in vitro; the current study was undertaken to determine whether accurate displacement and strain fields can be calculated from velocity data acquired in vivo. Wall velocities in the porcine thoracic aorta were quantified with PC-MRI and an implanted coil and were then time-integrated to compute wall displacement trajectories and cyclic strain. Displacement trajectories were consistent with observed aortic wall motion and with the displacements of markers in the aortic wall. The mean difference between velocity-based and marker-based trajectory points was 0.1 mm, relative to an average pixel size of 0.4 mm. Propagation of error analyses based on the precision of the computed displacements were used to demonstrate that 10% strain results in a standard deviation of 3.6%. This study demonstrates that it is feasible to accurately quantify strain from low wall velocities in vivo and that the porcine thoracic aorta does not deform uniformly. Magn Reson Med 52:286–295, 2004. © 2004 Wiley-Liss, Inc.

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Artery wall motion and strain play important roles in vascular remodeling and may be important in the pathogenesis of occlusive and aneurysmal vascular disease (1–6). Quantification of both normal and abnormal wall motion and strain distributions will contribute to our understanding of disease processes and may aid in the clinical evaluation of vulnerable plaques and aneurysms. For example, changes in wall motion and strain patterns may predict which aneurysms are more likely to enlarge and/or rupture. Quantification of vessel wall motion and strain may also have an important role in improving the design and evaluation of intravascular devices.

Previously described noninvasive, in vivo methods to quantify circumferential vessel strain have utilized modalities including ultrasound (7,8) and MRI (9,10). These methods usually calculate strain based on single diameter or vessel circumference measurements over time, neglecting factors which could result in nonuniform strain, including nonuniform wall thickness, nonuniform material properties, and localized disease. In vivo observations of human aortic wall motion have shown that the aorta may not expand uniformly in the radial direction (10–12), and therefore a method is needed to more completely and accurately quantify nonuniform circumferential strain distributions. MR tagging directly acquires displacement data which can be used to calculate strain (13,14). However, tagging requires the image pixel size and tag width to be small compared to the wall displacements, and tag spacing to be small compared to the heterogeneity of the strain field. Current MR tagging techniques are not adequate to measure aortic wall displacements and strain accurately.

We recently described a new method to quantify vessel wall motion and strain using velocity data acquired with cine phase contrast MRI (PC-MRI) (12,15). In this method, forward-backward time integration (16) is used to calculate displacement fields from the measured velocities. Because the displacement information is derived from velocity measurements, motion far smaller than the pixel size can be measured. These displacement fields can then be used to calculate time-dependent, circumferentially varying strain fields which do not require an assumption of axisymmetric deformation. The time integration and tracking algorithm was validated in vitro by comparing embedded marker displacements to those calculated from the velocity data (12). These phantom experiments demonstrated a mean displacement error of 0.22 mm (pixel size 0.39 mm). However, this validation study was performed under ideal conditions possible only in vitro. In vivo imaging is more challenging for numerous reasons, including the need for cardiac and respiratory gating and physiologic variability, which can cause changes in heart rate and blood pressure. Generally, in vivo images have a lower signal-to-noise ratio (SNR) and larger pixels than in vitro images due to scan time and coil limitations. The purpose of the current study was to determine whether accurate displacements and strain fields can be calculated from velocity data acquired in the more complex in vivo situation. A porcine thoracic aorta animal model was used for this study. Markers were embedded in the aortic wall for validation and a custom-built implanted coil was used to improve image quality. Imaging parameters were chosen in the range between the previously validated in vitro case and typical in vivo values.
and secured to ensure that it did not interfere with aortic movement. The thoracotomy incision was closed in layers and the animal was transported to the MRI suite. Respiratory bellows were placed around the thorax to monitor respiratory movement and stainless steel ECG electrodes were inserted into the animal hide. After the animal was positioned in the bore of the magnet in the right decubitus position, variable capacitors on the coil tuning board (located several feet from the coil) were adjusted to tune the coil to 63.86 MHz. Arterial pressure, SPO₂, heart rate, and blood gas levels were monitored continuously throughout the surgery. Anesthesia, fluids, and inotropic drugs (dopamine, epinephrine) were adjusted and administered as necessary to maintain the animal in a stable physiologic state.

MRI and Measurements
All imaging was performed using a 1.5 T MRI system (Signa CV/i, GE Medical Systems, Waukesha, WI). Two-dimensional fast gradient echo images were obtained using the body coil to localize the aorta in the region of the implanted coil. A series of oblique axial images (single or double oblique to ensure the slices were perpendicular to the aorta) were obtained using the implanted coil to determine the plane which best captured all five markers in the wall of the vessel.

Three-component velocity of the vessel wall was measured in this plane using a 2D cine phase contrast sequence (a version of the GE product cine phase contrast sequence, modified to include multiple velocity encoding values and RF spoiling). The field of view (8–24 cm) and matrix size (256 × 256, 512 × 256) were varied to obtain images with variable pixel sizes and SNR (see Tables 1, 2). With these values, other imaging parameter ranges were: TR 27.9–29.8 ms, TE 10.7–11.1 ms, and BW 15.6–31.25 kHz. Flip angle was held constant at 20° and two sequence averages were acquired. Total acquisition time was 512 heart beats. Separate velocity encoding values were used for the in-plane directions (2 cm/sec) and the through-plane direction (800 cm/sec) to improve velocity resolution in-plane and minimize flow artifacts through-plane. RF spoiling was used to minimize motion artifacts and spatial saturation pulses were used to minimize flow effects and to enhance visualization of the vessel wall.

Respiratory compensation and oversampling to prevent spatial aliasing (no phase wrap) options were also used. Twenty-four time frames through the cardiac cycle were reconstructed.

Through-plane blood flow velocity was quantified using a 2D cine phase contrast sequence (with single direction velocity encoding). Imaging parameters for this scan include 8 cm FOV, matrix size 256 × 256, TR 16.8–19.3 ms, TE 5.0–6.7 ms, BW 15.6 kHz, and flip angle 20°. Flow compensation and RF spoiling were used in some scans to minimize motion artifacts and 24 time frames through the cardiac cycle were reconstructed.

Heart rate and SPO₂ were monitored continuously throughout imaging and arterial pressure and blood gases were monitored at regular intervals between scans. As during surgery, anesthesia level and administration of fluids and inotropic drugs were adjusted as necessary to maintain the animal in a stable physiologic state.
Data Processing

Image processing, including velocity wall segmentation, velocity analysis, and calculation of wall strain, was done using a series of custom programs written in Matlab (MathWorks, Natick, MA). For each reconstructed time frame, magnitude and velocity data were read into Matlab. A contour around the vessel circumference at the approximate center of the vessel wall was manually defined and used to calculate a first-order background velocity correction. This calculation assumes periodicity of wall motion and conservation of momentum in each direction. The stored wall pixel velocity values were then updated using this background correction.

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The vessel wall in the first frame, also known as the reference time, was divided into 36 overlapping 30° sectors, and the average (x,y) coordinates and velocity values for each sector were calculated. These sectors, roughly equivalent to half the wall thickness, were extracted to yield a set of potential vessel wall pixels. Pixels in the neighborhood of the updated center were again extracted and subjected to magnitude and velocity tests. Pixels determined to be in the wall subsequent to these tests were stored with their corresponding magnitude and velocity values. The convex hull of the wall points and the lumen points (defined as all nonwall pixels lying interior to the wall) were also stored for marker segmentation. The wall points (black plus signs), convex hull (solid black line), and lumen points (white plus signs) are illustrated in Fig. 2a. Other symbols in this figure are described in the context of the marker tracking, described at the end of this section.

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For both calculations, the mean $\lambda/L$ value (peak stretch/original length) among sequences for a given animal was used. The SDs of the displacement differences in the x and y component directions ($\epsilon_x$ from Table 1) are used in the retrospective propagation of error calculation, and the in-plane $v_{\text{enc}}$ 2cm/sec is used in the prospective propagation of error calculation. The shaded sequences have further results shown in Figures 4, 5, and 7.
the curve approximating the center of the wall (dotted white line), and the sector-average velocity vectors (drawn at the sector-average spatial coordinates) are illustrated in Fig. 2b. Note that signal dropout can cause shifting of the vector away from the geometric center of the sector. The radial extent of the sectors exceeds the wall thickness and is large for illustrative purposes only. The sector boundaries and corresponding velocity vectors are shown in an alternating pattern of black, white, and gray for illustrative purposes. A complete description of the tracking scheme used to calculate wall displacements from the wall velocities has previously been described (12). Briefly, the position of the spatial center of each sector at each subsequent time was calculated using simple forward integration and simple backward integration, both starting from the same point in the reference image. Examples of the trajectories of one sector are shown in Fig. 2c (forward integration) and Fig. 2d (backward integration), along with the wall centers and sector locations at all time points. The start point is indicated with a black dot and the shade of gray of the trajectory vectors indicates location in the cardiac cycle, with black being the first frame in the cardiac cycle. Note that the start point is the same for both forward and backward time integration, and equivalent time frames in Fig. 2c,d are represented with the same shade of gray. Note that the sectors are elongated in the radial direction for illustrative purposes and do not represent the thickness of the wall. The positions from the forward and backward trajectories were then combined using a time-dependent weighting function, which simultaneously enforces periodicity and minimizes the effects of eddy currents (16). The forward (shades of gray), backward (shades of gray), and combined trajectories (solid black, closed contour) of a single sector are shown in Fig. 2e. All combined trajectories (black lines) for one of the acquisitions (sequence 2b in Tables 1 and 2) are shown in Fig. 2f, along with the outlines of the trajectory points at the reference time (the first time frame; solid white line) and the time of maximum expansion (dotted white line). Note that the starting points of the trajectories are not evenly spaced around the circumference due to signal dropout from the markers: the sector centers are calculated using the wall
pixels; significant shift can occur when dropout is located within a sector.

For each time frame the trajectory points are used as endpoints to construct 36 overlapping linear segments around the vessel wall. These segments, at the reference time and the time of maximum expansion, are shown in Fig. 3. Green-Lagrange cyclic strain around the vessel wall was then calculated in each segment over time, using principal values of the strain tensor, \( E \), where:

\[
E = \frac{1}{2} (\mathbf{F}^T \mathbf{F} - I), \tag{1}
\]

and \( \mathbf{F} \) is the deformation gradient (17). Assuming that the vessel wall displacements vary linearly around the circumference and that there is no strain in the direction perpendicular to the vessel cross-section, the principal strain corresponding to the local circumferential direction (oriented along the length of the segment), \( E_1 \), is piecewise constant, and can be calculated in terms of the stretch, \( \lambda \), of the segment:

\[
E_1 = \frac{1}{2} (\lambda^2 - 1), \tag{2}
\]

where the stretch, \( \lambda = \frac{l}{L} \), is calculated from the original segment length (i.e., at the reference time), \( L \), and the final segment length (i.e., at any other time), \( l \). Actual vessel wall strain cannot be determined without knowledge of the unloaded state of the vessel, thus the strains referred to herein as “\( E \)” (from Eq. 2) are cyclic principal strains, relative to a reference configuration.

The center of each validation marker in each time frame was approximated by calculating the center of intensity of the signal dropout (Fig. 2a). The region of signal dropout was initially determined by a user-defined rectangle (white square). This region was refined by excluding pixels outside of the convex hull of the wall points, points contained in the wall, and points contained within the lumen; the remaining points are shown as white circles in Fig. 2a. The region intensities were inverted and scaled such that the pixel of lowest original intensity corresponded to a value of 1, and then the center of intensity was computed (shown as a white star). The original images were not temporally independent (the number of reconstructed images was greater than the actual temporal resolution based on the heart rate and the TR); however, the marker center was independently determined on each frame. Thus, each marker trajectory, as a function of time, was Fourier filtered, with the number of terms included in the Fourier expansion equal to the number of temporally independent time frames acquired in the cine PC study. This effectively made the temporal resolution of the images and the marker-tracking the same. The marker centers in the first time frame were used to initialize single-sector displacement calculations as shown in Fig. 2c–e, with the 30° sector centered around the marker center. Each marker trajectory was then compared to the corresponding trajectory determined by the velocity data.

**Propagation of Error Analysis**

The SD of the distance between the velocity-based and marker based trajectories (\( \sigma_x \)) can be used in a retrospective propagation of error analysis (18) to analytically estimate the SD in calculated strain (\( \sigma_E \)) due to errors in displacement estimation:

\[
\sigma_E = \sqrt{2} \frac{\sigma_x \lambda}{L}. \tag{3}
\]

The SD in calculated strain is a function of the original length, the amount of stretch, and the SD of the displacements. A complete derivation of this result is given in Ref. 12. Physically, longer original lengths distribute uncertainty in position and stretch, thus lowering uncertainty in strain.

Retrospective propagation of error requires a means to calculate the position error, \( \sigma_x \). While this quantity can provide valuable information regarding the accuracy of the data, it is only feasible in the setting of a validation study.
which provides reference positions to compare with the calculated trajectories. A propagation of error analysis can also be performed to prospectively analyze the quality of the data, using an estimate for $\sigma_x$ rather than a measured $\sigma_x$. A relationship between the maximum SD of displacement error and the SD of velocities, $\sigma_v$, can be derived from the forward-backward integration scheme (16):

$$\sigma_x^2 = \sigma_v^2 \frac{T^2}{4M}, \quad [4]$$

where $T$ is the period of the cardiac cycle and $M$ is the number of temporally independent time frames. This can also be expressed in terms of repetition time, TR, and heart rate (HR):

$$\sigma_x^2 = 60 \cdot \sigma_v^2 \frac{TR}{HR}, \quad [5]$$

where TR is in seconds and HR is in beats/minute. Eq. 5 also illustrates that an increased heart rate (which degrades temporal resolution) lowers uncertainty in position; this is because positional error from the time integration has less time to accumulate. From the calculation of the phase contrast velocity image (19), $\sigma_v$ is

$$\sigma_v = \sqrt{ \frac{2}{\pi} \left( \frac{v_{enc}}{SNR} \right) }, \quad [6]$$

The velocity encoding value determines the maximum velocity which can unambiguously be measured, but it can also be thought of as a measure of velocity resolution. The higher the $v_{enc}$, the lower the velocity resolution, and consequently, the higher the uncertainty in the measured velocity. Substituting Eq. 6 into Eq. 5, and the result into Eq. 3, the theoretical $\sigma_E$ is:

$$\sigma_E = \left( \frac{4 \sqrt{15}}{\pi} \right) \left( \frac{\sqrt{12M}}{L} \right) \left( \frac{TR}{HR} \right) \left( \frac{1}{SNR} \right). \quad [7]$$

Equation (7) demonstrates that the SD in strain can be reduced by increasing the segment length relative to the amount of stretch, lowering the $v_{enc}$, increasing the temporal resolution (lowering the TR), and increasing image SNR. Also, for a given set of imaging parameters and estimated stretch and length values, SNR can be used to nominally predict the precision of the calculated strain.

**RESULTS**

A magnitude image (FOV 16 cm$^2$, 512 $\times$ 256; image shows center 256 $\times$ 256) and a close-up image of the vessel with superimposed velocity vectors are shown in Fig. 4 for both animals (top row: sequence 1(d) in Tables 1 and 2; bottom row: sequence 2(b)). The velocities are shown at the time of maximum expansion rate and are scaled such that the length corresponds to a 20x magnification of the velocity in pixels/frame. In both cases the vessel wall and the five markers are very clearly visualized and the velocity vectors are spatially consistent.
A comparison of the marker motion and the PC-derived trajectories is shown in Fig. 5, using the same series for both animals as shown in Fig. 4. Each marker and velocity-based trajectory is shown at actual size (white) and at a 15× magnification (black). The orientations of the trajectories are very similar and the distance between any two corresponding trajectory points was less than a pixel (pixel size 0.3125 mm). For the imaging sequence of the first animal [1(d)], shown on the left, the mean distance between marker and velocity-based trajectory points for all five markers was $0.08 \pm 0.05$ mm. For the imaging sequence of the second animal (with a distal arterio-venous fistula) [2(b)], shown on the right, the mean distance between marker and velocity-based trajectory points for all five markers was $0.13 \pm 0.06$ mm. The mean distance between trajectory points for all sequences and all markers in both pigs was 0.12 mm and the maximum distance was 0.42 mm. Further details of the displacement comparisons, including differences between the x and y components of the marker and velocity-based trajectory points and the distances, are shown in Table 1. Sequences with lower spatial resolution had larger differences between the trajectory points. Spatial resolution, however, was not the only factor. Sequences 1(a) and 1(b) had the same imaging parameters, but the higher heart rate of the animal during the three repetitions of sequence 1(b)—per Table 2—resulted in a greater difference between the trajectory points.

A graph illustrating the propagation of error relationship (Eq. 3) between the SD of displacements (i.e., the distance between the actual and measured position of a point), relative to the original segment length, and the SD of strain, relative to a given value of strain (related to stretch through Eq. 2), is shown in Fig. 6. These curves are valid for both retrospective and prospective propagation of error analyses. The shaded gray area represents results from a retrospective propagation of error analysis, using a measured $\sigma_x$ of 0.096 mm (the mean $\sigma_x$ for all markers at all time points for all sequences listed in Tables 1 and 2) and an original segment length range of 3.6–4.2 mm. The resulting $\sigma_E$ range is $0.033–0.041$ mm/mm. Note that even for zero actual strain, any uncertainty in displacement will result in uncertainty in calculated strain.

Strain SD results from both retrospective and prospective propagation of error analyses, for all imaging sequences, are shown in Table 2. Also shown are the imaging parameters and values needed for the prospective propagation of error calculation. An average value of peak stretch and original segment length was calculated for each animal (animal 1 peak stretch = 1.08 mm/mm, mean $L = 4.5$ mm; animal 2 peak stretch = 1.1 mm/mm, mean $L = 3.6$ mm). For all but one imaging sequence the prospective propagation of error analysis is equal to or underestimates the calculated strain SD from the retrospective propagation of error analysis. The degree to which the prospective propagation of error analysis accurately predicts $\sigma_E$ is related to pixel size (see Table 1) and heart rate; smaller pixels and lower heart rates generally produce better predictions. However, smaller pixels reconstructed

FIG. 5. Comparison of velocity-integrated and marker trajectories for one series (FOV 16 cm, 512 × 256) of two pigs (1(d) on the left, 2(b) on the right). Each marker and velocity-based trajectory is shown actual size on the vessel wall (white lines) and enlarged ~15-fold, in line with the actual trajectory, to illustrate differences. The black dotted paths are the marker trajectories and the black vector paths are the velocity-based trajectories. The size of the trajectory arrowheads relates to the magnitude of displacement within a time interval.

FIG. 6. The relationship between the SD of measured displacements and the SD of calculated strain, calculated using a propagation of error analysis (Eq. 3). The shaded region represents results from a retrospective propagation of error, with the region along the x-axis corresponding to a measured $\sigma_x$ of 0.096 (calculated from XY SDs shown in Table 1) and an original segment length range of 3.6–4.2 mm. The resulting $\sigma_E$ range, shaded along the y-axis, is $0.033–0.041$ mm/mm.
from nonsquare acquisition matrices degrade the prediction. For example, sequences 1(a) and 2(a)—0.3125 true spatial resolution—have a better predicted strain variation than sequences 1(d) and 2(b), with 0.3125 reconstructed spatial resolution.

Motion, strain, and physiology results are shown in Fig. 7, with the top row corresponding to sequence 1(d) and the bottom row to 2(b). On the left, trajectories of the vessel wall are shown with the vessel centerlines. The extent of the trajectories are consistent with the location of the centerlines. Note that the motion of the vessel is not axisymmetric, as evidenced by the orientation of the trajectories. Although the amount of motion is less than has been seen in the human thoracic aorta (10–12), the pattern of motion is very similar, with increased motion around approximately 2/3 of the circumference and minimal motion in the remainder of the wall.

Contour plots illustrating the temporal and spatial distribution of principal strain are shown in the middle of Fig. 7. Time increases with increasing radius, with the reference frame being the innermost radius. A close-up of the vessel is shown inside the plot to illustrate the relationship between the marker locations and strain. For the first animal strain ranged from −0.4 to 0.4 mm/mm, and for the second animal strain ranged from −0.2 to 0.12 mm/mm.

Flow, pressure, and average strain waveforms are shown at the bottom of Fig. 7. The length of the variation bars on the strain waveforms are the ± SD of the strain around the circumference of the vessel. The slight notches preceding the primary peaks on the flow and pressure waveforms of the second animal are likely due to flow and pressure changes induced by the distal arterio-venous fistula.

**DISCUSSION AND CONCLUSIONS**

Validation studies were conducted to evaluate, in vivo, the accuracy of displacement fields calculated from cine PC-MRI velocity data. Nitinol wire markers were inserted into the wall of porcine aortas and motion trajectories of the markers were compared to those calculated from the velocity-derived trajectories. Propagation of error methods were then used to retrospectively and prospectively evaluate the precision of strain calculated from the displacement fields.

The vessel wall was successfully imaged in both pigs, using a variety of imaging parameters. Qualitatively, velocity data was locally uniform (Fig. 4, right), suggesting the measured velocities are not random. Further, the trajectories calculated from the velocity data are consistent with the motion of the vessel observed in the magnitude images (Fig. 7, top) and the orientation of the velocity trajectories match the marker trajectories (Fig. 5). Quantitatively, the average time-matched distance difference between trajectory points (all markers, all sequences, both animals) was 0.12 mm, relative to an average pixel size of
0.44 mm. This difference may be due to errors in the velocity data or in the marker tracking; in most cases, the wall moved less than the diameter of the marker artifact. Additionally, although significant effort was made to align the markers with the axis of the vessel, there was no way to ensure that they were inserted perfectly parallel. Thus, motion of the vessel could produce changes in the orientation of the marker with respect to the magnetic field, altering the relationship between the center of the marker signal void and the actual marker center. Although the marker validation technique has some limitations, this method still has many advantages. The wire markers produce a signal dropout large enough to image (>1 pixel), yet themselves are very thin. Additionally, this method provides a direct comparison, acquired in the same scan, to the velocity-based trajectories. In the setting of physiologic variability, same-scan validation is very advantageous.

The nonaxisymmetric deformation of the vessel, illustrated in Fig. 7 (left), is consistent with what was observed in earlier human studies (10–12), where a portion of the wall appears to be relatively fixed. The results of this study suggest that the porcine aorta, like the human, deforms nonuniformly around the circumference. This motion pattern may be the result of tethering, variations in wall thickness, and/or variations in material properties. This nonuniform motion may or may not result in nonuniform strain fields, but does justify the need for, and the use of, a more general method to quantify strain around the vessel circumference.

The average circumferential strain waveform shown in Fig. 7 (right) illustrates a temporal similarity to the pressure and flow waveforms, with the strain and pressure peaks occurring at nearly the same time. The variation bars on the strain waveforms are the ±SD of the strain around the circumference. The peak SD in the first animal was 0.03, in the second animal, 0.01; both values are in the range of the SD which may be explained by errors in displacement calculation rather than true variation in circumferential strain. The color strain plots shown in the middle of Fig. 7 illustrate both the temporal and circumferential variation in strain. In these plots it is evident that the overall strain difference through time (i.e., the maximum strain minus the minimum strain) does have some variation around the circumference greater than what is predicted by the propagation of error analysis (e.g., Fig. 7, top middle, 12 o’clock vs. 6 o’clock), thus indicating that there may in fact be a true circumferential variation in strain. Conclusions about circumferential strain variation in vivo need to be made cautiously, however, since several experimental factors may influence the results. These factors include the physiologic and imaging effect of the markers (which are thin relative to the thickness of the wall, but may still invoke a physiologic reaction, and additionally may distort velocity data immediately adjacent to the region of signal dropout) and the effects of the surgery, including anesthesia and exposure of the vessel. The primary purpose of this study was to determine whether velocity obtained with PC-MRI could be used to calculate accurate time-dependent, circumferentially varying strain fields. Future studies without surgical exposure or marker implantation will be needed to explore the issues of circumferential strain variation.

The results of the prospective propagation of error analysis indicate that the precision of the strain results, given adequate spatial and temporal resolution, can be accurately predicted from known imaging parameters (TR, venc, and SNR) and known or quantifiable physiologic parameters (heart rate and vessel diameter over time [to estimate original segment length and stretch]). The required spatial resolution is dependent on the wall thickness, but this experiment suggests that 3–4 pixels through the wall thickness (1.5–2.0 mm) is adequate. Clearly, more pixels through the wall thickness will facilitate wall segmentation and minimize selection of wall boundary pixels which may be contaminated by higher velocity blood or lower velocity surrounding tissue. Increased heart rate decreases the predicted σy, since there is less time for error to accumulate through the cardiac cycle. However, increased heart rate also decreases true temporal resolution, relative to the period of the cardiac cycle, which may result in image and velocity blurring. The ability to prospectively predict the quality of the strain data will allow future noninvasive validation studies to evaluate circumferential strain variations.

Potential sources of error in calculating cyclic strain from PC-MRI data principally arise from errors in the calculated displacement fields. In this method, such errors can be introduced during the acquisition of the velocity data, the segmentation of the vessel wall, the integration of the velocity data, and sector tracking. Velocity acquisition can be optimized by maximizing SNR and temporal resolution, minimizing artifacts, and using the lowest possible velocity encoding value which does not result in velocity aliasing or unreasonably long TRs. Temporal resolution determines whether rapid velocity changes can be captured; any velocity changing more rapidly than the actual temporal resolution (for a three-component velocity acquisition, this is 4“TR) will not be accurately measured. This type of error will lead to underestimation of strain during peak systole (20).

Segmentation of the vessel wall can be facilitated with higher spatial resolution, but smaller pixels also degrade SNR and temporal resolution. Segmentation errors are largest in time frames where the boundaries of the vessel wall are blurred, which may be the result of low SNR or inadequate temporal resolution.

In this implementation of combined forward-backward time integration, it is assumed that velocities are constant during any given time frame; this may result in error in time frames where the velocity is changing rapidly in comparison to the temporal resolution. It is also assumed that the vessel does not move perpendicular to the imaging plane. Although the magnitude data is used to segment the vessel wall and extract velocities, only the velocities are used in the tracking algorithm, and thus it is possible that the extent of the trajectories are larger or smaller than would be expected from examination of the magnitude data alone. Although an increased number of sectors would produce a more detailed map of circumferential strain, the propagation of error analysis illustrates that a finer discretization of the wall also results in increased variation in the calculated strain. The choice of the number of sectors is thus a trade-off between accuracy and completeness, and determination of the maximum accept-
able sector size to capture strain nonuniformities will potentially depend on the vessel being analyzed. The use of phase contrast velocity data to quantify vessel strain has several advantages. First, because three components of velocity can be measured, general displacement fields and strain tensors can be calculated. The Lagrangian strain theory underlying the method described can be extended to utilize 4D (time-resolved 3D) velocity data, thus producing complete 4D strain fields. Second, the velocity data is extracted directly from the wall, not the vessel lumen, and thus is less vulnerable, given adequate spatial resolution, to motion-related artifacts. Although the method presented is based on velocity measurements, cyclic Lagrangian strain is ultimately calculated from displacements. Therefore, if tagging methods gain resolution sufficient to capture displacements of vessel walls, this method is readily extensible.

The method for quantifying strain presented herein is, in principle, a noninvasive method. However, the implanted coil which was used in this validation study to improve SNR and facilitate acquisition of high spatial resolution images is not, for obvious reasons, feasible for use in human studies. Although intravascular and transesophageal coils are currently available for human use, at this time they are not ideal for acquiring human aortic vessel wall velocity data due to limited imaging range and movement of the coil relative to the vessel wall. If these issues can be resolved, an internal coil would permit the combination of high SNR and high spatial resolution obtained in this study—but would still impose limitations for studies on normal individuals due to their invasive nature. Alternatively, better image quality and temporal resolution may be obtained through sequence optimization and improved hardware (at this time, temporal resolution is principally limited by gradient heating restrictions). The image quality needed for strain quantification will depend on the desired strain accuracy; the propagation of error described (Eq. 7) may be used to approximate the necessary SNR improvements.

This study has validated that, even with physiologic variability, time-dependent, circumferentially varying vessel wall cyclic strain can be accurately calculated from phase contrast velocity data. A propagation of error analysis was developed which can be used to prospectively evaluate the quality of calculated strain directly from known or measurable imaging and physiologic parameters. Further validation and application of this method, using a coil suitable for imaging humans, will enable the investigation of circumferential motion and strain distributions and the relationship between motion, strain, and occlusive and aneurysmal disease processes.

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