Regional Cardiac Wall Motion from Gated Myocardial Perfusion SPECT Studies

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Abstract

A method for estimating regional epicardial and endocardial wall motion from gated myocardial perfusion SPECT studies has been developed. The method uses epicardial and endocardial boundaries determined from four long axis slices at each gate of the cardiac cycle. Epicardial and endocardial wall position at each time gate is computed with respect to stationary reference ellipsoids and wall motion is measured along lines normal to these ellipsoids. An initial quantitative evaluation of the method was made using the beating heart from the dynamic mathematical cardiac torso (MCAT) phantom, with and without a 1.5 cm FWHM Gaussian blurring filter. Epicardial wall motion was generally well-estimated within a fraction of a 3.56 mm voxel, although apical motion was overestimated with the Gaussian filter. Endocardial wall motion was underestimated by about two voxels with and without the Gaussian filter. The MCAT heart phantom was modified to model hypokinetic and dyskinetic wall motion. The wall motion analysis method enabled this abnormal motion to be differentiated from normal motion. Regional cardiac wall motion also was analyzed for Tl-201 patient studies. Estimated wall motion was consistent with a nuclear medicine physician’s visual assessment of motion from gated long axis slices for example male and female studies. Additional research is required for a comprehensive evaluation of the applicability of the method to patient studies with normal and abnormal wall motion.

I. INTRODUCTION

Single photon emission computed tomography (SPECT) has been widely used to assess myocardial perfusion with tracers such as thallium-201 and technetium-99m sestamibi [1-5]. The capability to acquire myocardial perfusion SPECT images gated to the cardiac cycle [6, 7] provides the potential to extract additional quantitative information about cardiac function from the time-dependent motion of the myocardium. Several research groups [8-10], including our own [11], have segmented myocardial perfusion scans and estimated global parameters such as left ventricular ejection fraction from cardiac wall motion. The goal of this investigation is to present a method for the more challenging task of extracting information about regional cardiac wall motion from these gated SPECT studies. This task, which is of interest to many researchers [9, 12, 13], is difficult because of the noise and limited spatial resolution of these count-limited studies.

Assessments of endocardial wall motion are routinely made using planar gated blood pool studies [14]. The capability to estimate regional endocardial wall motion from SPECT gated blood pool studies is also being investigated [15-17]. The method proposed in this paper is intended to provide wall motion information supplemental to the three-dimensional perfusion information that is the primary purpose of the perfusion study. Its results will augment qualitative evaluations of wall motion drawn from visual inspection of cine displays of the perfused myocardium. The capability to provide quantitative functional information on regional cardiac perfusion and motion simultaneously from a single SPECT study would be advantageous in a clinical setting.

This paper is organized as follows. The wall motion analysis technique is described in section II. An initial application and quantitative evaluation of this method using mathematical beating heart phantoms is made in section III. In section IV the wall motion analysis method is applied to patient studies for an initial evaluation of its potential use in a clinical setting. A discussion of the method and further work is presented in section V followed by conclusions in section VI.

II. METHODS

A. Myocardial Boundary Determination

The regional cardiac wall motion analysis method uses endocardial and epicardial boundaries from reconstructed 3-D SPECT perfusion images at each time gate of the cardiac cycle. The procedure for determining the boundaries will be briefly described now.

The reconstructed 3-D SPECT images at each time gate were reoriented with the long axis of the heart parallel to the one axis of a Cartesian coordinate system. A line parallel to a second axis passed through the center of the left and right ventricles in short axis slices. Four long axis slices were generated at 45° intervals for each time gate (Figure 1). The slice thickness was set so that a circle circumscribing the heart near its base was divided into eight equal arc segments.

The cardiac boundaries in these long axis images were found using the boundary detection algorithm of Brigger et al. [11]. In this algorithm the long axis slices were transformed to an elliptical coordinate system and a first derivative edge-

Figure 1. (a) Diagram (short axis cut) showing the orientation of the four long axis slices used for segmentation of the epicardial and endocardial boundaries. (b) Diagram of a long axis cardiac slice from position 2 of (a).
Cardiac wall motion is measured along lines that are normal to reference ellipsoids (Figure 2). Different ellipsoids are used for epicardial and endocardial motion and are determined as follows. First, the three-dimensional coordinates of the epicardial and endocardial boundary points are computed using the angular orientation of the long axis slices (Figure 1) and the two-dimensional coordinates of the boundary curves within the slices. Then the epicardial (or endocardial) reference ellipsoid is found by a least squares fit to the epicardial (or endocardial) boundary points from all time gates. The long axis of the heart is usually approximately aligned with the long axis of the reference ellipsoids.

At each time gate the epicardial and endocardial boundary points are projected normally to the appropriate stationary reference ellipsoid. The projection distances (e.g. in Figure 2) are mapped to the projection points on the ellipsoid. These distances are then interpolated onto a uniform grid on the surface of the ellipsoid. Thus a map of the distance of the epicardial or endocardial boundary from the desired reference ellipsoid is obtained at each time gate. Optional smoothing along ellipsoid latitude and longitude lines is performed.

C. Bullseye Plots and Additional Analyses

Bullseye plots of epicardial and endocardial wall positions at each time point are generated from the ellipsoid maps (Figure 3). The radial coordinate of the bullseye plot represents the distance along the surface of the ellipsoid from its pole near the apex of the heart. The bullseye plots are normalized for different heart sizes so that the distance from apex to base is represented by a circle of the same radius for all hearts.

Temporal smoothing of the distance maps may be performed on a pixel-by-pixel basis using the bullseye plots from all time gates. There are options to filter with a boxcar function or a truncated Fourier expansion of the time series. Regional wall motion with respect to end-diastole can be computed by subtraction of appropriate pairs of bullseye plots. Additional processing, e.g. computation of the phase and amplitude of cardiac motion, may be performed. Regional wall motion also may be estimated using the results from...
A. EPICARDIAL MOTION

No Blurring 1.5 cm FWHM Filter

Figure 6. Bullseye plots and profiles of estimated (a) epicardial and (b) endocardial wall motion of the dynamic MCAT phantom with and without a 1.5 cm FWHM Gaussian filter. Motion estimates were obtained by subtracting the wall distances from the reference ellipsoid at the end systolic time gate from the distances at the end diastolic time gate.

Fourier amplitude and phase analysis. This is important if cardiac contraction is not synchronous and if hypokinetic or dyskinetic wall motion is being evaluated.

III. APPLICATION TO MATHEMATICAL CARDIAC PHANTOMS

A. Model for Normal Motion and Myocardial Boundary Determination

The beating heart from the dynamic mathematical cardiac torso (MCAT) phantom [18] was used for an initial evaluation of the wall motion analysis method. The heart model had a left ventricular chamber volume of 126 ml, left ventricular myocardial volume of 154 ml and an ejection fraction of 61.5%. Three-dimensional images of the beating heart were generated at 16 time gates on a 3.56 mm voxel grid. The blood pool activity concentration was 5% of the myocardial activity concentration.

Data sets were analyzed with and without application of a 1.5 cm full-width at half-maximum (FWHM) 3-D Gaussian filter. With the filter, the myocardial/ventricular blood pool activity concentration ratio approximated that of SPECT thallium-201 patient studies. The discretized heart model was used directly in the analysis; projection data generation and image reconstruction were not performed. Noise was not added to the heart images for this initial analysis. Long axis slices were generated for the unblurred and blurred heart models at each time gate (Figure 4). Epicardial and endocardial boundaries were computed using the algorithm described in section II.A. Reference ellipsoids then were fit to these boundary points (Figure 5).

Figure 7. Region of interest locations in the bullseye plots for average wall motion estimates. The label "A" denotes an apical ROI while the prefix "B" denotes a basal ROI.

B. Cardiac Wall Motion Estimation

Epicardial and endocardial boundary motion was estimated by subtracting the cardiac wall distance from the reference ellipsoid at the end systolic time gate from the distance at the end diastolic time gate (Figure 6). Average motion from end diastole to end systole also was computed in regions of interest in the bullseye plots (Figure 7). These results are compared with the true MCAT motion values from the equations describing the beating heart model in Table 1.

Epicardial wall motion is generally well-estimated within a fraction of a 3.56 mm voxel with and without the blurring filter. Apical motion is slightly overestimated with spatial blurring because the contour extends too far from the epicardium at end-diastole. Endocardial motion is underestimated by about 2 voxels (7 mm) with and without blurring because the boundary contours do not move far enough inward as the myocardium contracts (Figure 4). This effect is more severe for the blurred cardiac model near the apex, which is why motion underestimation is greater with blurring than without.
Table 1.
Estimated cardiac wall motion from end-diastole to end-systole in apical and basal ROIs from bullseye plots. The true MCAT motion values are from the equations describing the beating heart model.

| No Spatial Blurring | 1.5 cm FWHM 3-D Gaussian Filter |
|---------------------|---------------------------------|
| **ROI**             | **Epicardium**                  | **Endocardium**                  |
|                     | Estimate | MCAT Error | Estimate | MCAT Error | Estimate | MCAT Error |
| A                   | 1.2      | 1.3        | -0.1     |            | 1.0      | 1.6        | -0.6     |
| B1                  | 0.3      | 0.3        | 0.0      |            | 0.2      | 0.8        | -0.6     |
| B2                  | 0.3      | 0.3        | 0.0      |            | 0.1      | 0.8        | -0.7     |
| B3                  | 0.3      | 0.3        | 0.0      |            | 0.0      | 0.8        | -0.8     |
| B4                  | 0.3      | 0.3        | 0.0      |            | 0.1      | 0.8        | -0.7     |
| Ave. of B1-B4       | 0.3      | 0.3        | 0.0      |            | 0.1      | 0.8        | -0.7     |
|                     | 1.5 cm FWHM 3-D Gaussian Filter |
| **ROI**             | **Epicardium**                  | **Endocardium**                  |
|                     | Estimate | MCAT Error | Estimate | MCAT Error | Estimate | MCAT Error |
| A                   | 1.8      | 1.3        | +0.5     |            | 0.7      | 1.6        | -0.9     |
| B1                  | 0.3      | 0.3        | 0.0      |            | 0.2      | 0.8        | -0.6     |
| B2                  | 0.4      | 0.3        | +0.1     |            | 0.2      | 0.8        | -0.6     |
| B3                  | 0.3      | 0.3        | 0.0      |            | 0.2      | 0.8        | -0.6     |
| B4                  | 0.2      | 0.3        | -0.1     |            | 0.3      | 0.8        | -0.5     |
| Ave. of B1-B4       | 0.3      | 0.3        | 0.0      |            | 0.2      | 0.8        | -0.6     |

Figure 8. Estimated (a) epicardial and (b) endocardial wall motion (amplitude) from Fourier analysis of the dynamic MCAT phantom. The profile locations are indicated in the bullseye displays.

Figure 9. Estimated regional cardiac phase from Fourier analysis of the dynamic MCAT phantom. Profile locations are indicated.
C. Cardiac Wall Motion from Fourier Amplitude and Phase Analysis

In clinical studies myocardial contraction may not be globally synchronous and so subtraction of distance maps from two time gates may not yield an accurate estimate of myocardial motion. The time series of wall motion also may be noisy. For these reasons Fourier amplitude and phase analysis of wall motion will be employed for analysis of clinical studies. In this section the Fourier analysis technique is applied to the MCAT phantom and its results are compared to those from motion estimation using the end diastolic and end systolic time gates.

At each bullseye pixel the time series of wall distance from the reference ellipsoid was filtered in the frequency domain, retaining the zero-frequency and first harmonic terms. Regional wall motion and phase were computed for each pixel. The phase at each pixel with respect to global phase is reported.

Estimated wall motion from Fourier analysis of the dynamic MCAT phantom (Figure 9) is in excellent agreement with the motion results obtained from the end-diastolic and end-systolic time gates (Figure 6). Since regional phase (Figure 9) is measured with respect to mean cardiac phase, values of zero are expected for a heart with synchronous contraction as is the case for the MCAT phantom. The epicardial phase values are generally near zero. There are greater fluctuations in the endocardial and basal values due to greater errors in wall boundary estimates at these locations.

D. Models for Hypokinetic and Dyskinetic Motion

Since the myocardial contraction of the heart in the dynamic MCAT phantom is synchronous, modified forms of this phantom were developed to test the wall motion analysis method for hypokinetic and dyskinetic motion. The left ventricular epicardial and endocardial walls at each of the 16 time gates were modeled by parts of surfaces from four different ellipsoids. Each ellipsoid contributed to the cardiac wall over a 90° range in short axis view (Figures 3, 10), corresponding to the surfaces in sectors from 1) anterior to lateral wall, 2) lateral wall to inferior wall, 3) inferior wall to septal wall and 4) septal wall to anterior wall. The myocardial surfaces were constrained to be continuous at the sector boundaries (Figure 10). The ellipsoid semi-axis lengths for the long axis of the heart were the same for each sector and changed with time as for the dynamic MCAT phantom, modeling normal apical motion (Figure 11). The ellipsoid semi-axis lengths in short axis view were varied to
Table 2.

Estimated cardiac wall motion from end-diastole to end-systole in ROIs near the base of the heart for hypokinetic and dyskinetic motion models. The model values are from the equations describing the beating heart model.

**Hypokinetic Motion Model**

| ROI Location       | Epicardium | Endocardium |
|--------------------|------------|-------------|
|                    | Estimate Model | Error (cm) | Estimate Model | Error (cm) |
| Anterolateral Wall | 0.1 0.0 0.1 | 0.0 0.0 0.0 |
| Inferoseptal Wall  | 0.2 0.3 -0.1 | 0.3 0.8 -0.5 |

**Dyskinetic Motion Model**

| ROI Location       | Epicardium | Endocardium |
|--------------------|------------|-------------|
|                    | Estimate Model | Error (cm) | Estimate Model | Error (cm) |
| Anterolateral Wall | -0.3 -0.4 0.1 | -0.1 -0.7 0.6 |
| Inferoseptal Wall  | 0.2 0.3 -0.1 | 0.3 0.8 -0.5 |

Model hypokinetic and dyskinetic motion. The right ventricle was not modeled.

For the hypokinetic motion model, the myocardium in the inferoseptal quadrant (sector 3) contracted normally, i.e., as in the dynamic MCAT phantom (Figure 10). The ellipsoid semi-axis lengths in the short axis plane did not change with time for the anterolateral quadrant (sector 1). Due to the shortening of the long axis, wall motion was hypokinetic rather than akinetic in the anterolateral region. The ellipsoid semi-axis lengths for sectors 2 and 4 were calculated in accord with the continuity constraints on the epicardial and endocardial boundaries. The ejection fraction for this model was 43.5%.

For the dyskinetic motion model, contraction in the inferoseptal quadrant again was normal. In the anterolateral quadrant the ellipsoid semi-axis lengths in short axis view increased from end-diastole to end-systole, reflecting dyskinetic motion (Figure 10). The magnitude of the semi-axis increase at each time gate was 0.35 times the magnitude of the decrease in the normally contracting inferoseptal region. The ejection fraction for this model was 23.1%.

These three-dimensional models were blurred with a 1.5 cm FWHM Gaussian filter and long axis slices were generated at each time gate (Figure 11). Wall motion was analyzed as for the normally contracting MCAT cardiac phantom. Bullseye plots of amplitude and phase from Fourier analysis are shown in Figure 12. Decreased motion is evident in the anterolateral wall for the hypokinetic model, as expected (Figure 12a). For the dyskinetic model, the amplitude of motion is normal near the apex. In the anterolateral region, the amplitude passes through zero then increases again toward the base of the heart. As this occurs, the phase changes from near 0° to near 180° or -180°, indicating dyskinetic motion.

Wall motion was computed in anterolateral and inferoseptal ROIs near the base of the heart using the bullseye plots of wall position at end-diastole and end-systole. The anterolateral ROI was located between the B2 and B3 ROIs of Figure 7 while the inferoseptal ROI was between the B1 and B4 ROIs. Epicardial wall motion is well-estimated in the inferoseptal (normally-contracting) and anteroseptal (abnormally-contracting) regions for both the hypokinetic and dyskinetic models (Table 2). For the inferoseptal wall, endocardial motion is underestimated by about 5 mm in both models, roughly the same amount as for the normally contracting MCAT model (Table 1).

Motion of the endocardial wall in the anterolateral region is estimated accurately for the hypokinetic cardiac model but is underestimated by 5 mm for the dyskinetic cardiac. Errors in motion estimation are due in part to spatial smoothing and to the fact that the ROI was not exactly at the base of the heart. An error of 5 mm is about 1.5 times the voxel dimension in the original model and one-third of the width of the 1.5 cm FWHM Gaussian blurring filter applied to simulate SPECT resolution.

IV. APPLICATION TO CLINICAL MYOCARDIAL PERFUSION STUDIES

The regional wall motion estimation method was applied to a challenging clinical imaging situation, the case of gated TI-201 SPECT perfusion studies. The SPECT studies were performed on patients referred for suspected coronary artery disease. Three mCi of TI-201 were injected at peak stress, followed by a 30 minute acquisition on a triple camera SPECT system. The projection matrix was 128x64 with 3.56 mm pixels. A 20% photopeak energy window was used and the cardiac cycle was gated into 10 time intervals. Filtered backprojection reconstruction was performed using an order 3 Butterworth filter with a cutoff frequency of 0.50 cycles/cm. Regional wall motion estimation was performed using Fourier amplitude and phase analysis.

Two example applications of regional wall motion analysis are shown. Patient 1 was a 63 year old male who had a normal rest Tc-99m gated blood pool study with an ejection fraction of 52%. Long axis slices at end systole and end diastole from the TI-201 SPECT study are shown in Figure 13a. Estimated wall motion (Figure 13b) was consistent with the a nuclear medicine physician's visual assessment of motion from cine displays of the gated long axis slices. Motion is greater in the lateral wall and small to near zero in the septal wall.

Patient 2 was a 58 year old female who had a normal rest gated blood pool study with an ejection fraction of 67%. The long axis slices at end diastole and end systole from the gated TI-201 study are shown in Figure 14a. The estimated wall motion (Figure 14b) agreed with the physician's assessment of motion from ciné views of the long axis slices. For this patient the wall motion is more uniform with greater epicardial wall motion in the inferior wall. The phase plots are smoother over the myocardial surface than for patient 1.
Figure 13. Patient example 1. (a) Long axis slices at end-diastole and end-systole showing epicardial and endocardial boundaries. (b) Regional wall motion amplitude and phase. The lateral wall shows the most vigorous contraction (epicardial max. 0.88 cm; endocardial max. 0.59 cm). Motion is diminished and near zero in parts of the septal wall. Regional phase is well determined where there is greatest motion, but less so in apical and septal regions where motion is small. Discontinuities in the phase plots are due to phase transitions between +180° and -180°.

Figure 14. Patient example 2. (a) Long axis slices at end-diastole and end-systole showing epicardial and endocardial boundaries. (b) Regional wall motion amplitude and phase. Estimated contraction is greatest in the inferior wall (epicardial maximum 0.75 cm; endocardial maximum 0.44 cm). Regional phase varies smoothly on the myocardial surfaces.
V. DISCUSSION

A. Limitations of the Cardiac Wall Motion Estimation Method

A method for estimating regional cardiac wall motion from gated myocardial perfusion studies has been presented. This is a difficult problem for SPECT cardiac imaging, particularly with TI-201, where the wall motion is on the same order as or less than the image resolution. The studies with the mathematical heart phantoms show that the wall motion analysis method enables abnormal motion to be differentiated from normal motion, however a calibration procedure appears necessary to quantify the relationship between measured and true wall motion. Factors contributing to measurement uncertainties include the smoothing that occurs with the generation of the long axis slices and the difficulty of accurate epicardial and endocardial boundary detection.

A limitation of this wall motion analysis method is that it does not model time dependent motion of the reference ellipsoid. Such motion may occur if the center of mass of the heart or its overall spatial orientation change with time. The current analysis method could be enhanced to model such motion.

The cardiac wall motion measured by our method is that of the endocardial or epicardial surface with respect to the reference ellipsoid, not the absolute motion of points at particular anatomic locations on the endocardium or epicardium. Tagged, gated MRI images of the heart allow accurate, high resolution measurement of myocardial motion during the cardiac cycle [19]. Viewed from the apex there is a counterclockwise rotation of the left ventricular myocardium during contraction. For eight normal volunteers the twisting of the epicardium ranged from an average of 2° at the base to 11° at the apex while that of the endocardium was greater and ranged from an average of 4° at the base to 19° at the apex [20]. Inferences about motion of a particular anatomic region of the heart from gated SPECT must take such rotation into consideration. We are not aware of any current SPECT or PET analysis or display methodologies that take this twisting motion into account. It may eventually be possible to use information from a morphological deformation map derived from gated SPECT data [21] to estimate this rotation, which would enable more accurate association of function with anatomic location.

The effect of statistical noise on wall motion estimation is important for low count TI-201 studies and remains to be investigated. The mathematical phantom studies could be extended by using reconstructed images from simulated projection data that include the physical effects of scatter, attenuation, collimator response and Poisson noise.

The capability of the method to estimate wall motion will be compromised if there are perfusion defects. As relative perfusion decreases the signal-to-noise ratio in that part of the image will decrease and boundary and motion estimates will become more uncertain. For a transmural defect for which there is no uptake of TI-201, it will not be possible detect epicardial or endocardial boundaries or to estimate wall motion. It should be kept in mind that the goal of the wall motion estimation method presented in this paper is to extract further functional information from gated cardiac perfusion studies, not to replace other methods for motion assessment such as gated MRI. The capability of the method to estimate motion for regions with low perfusion remains to be studied.

B. Comparison with Other Approaches to Regional Wall Motion Estimation

The evaluation of regional wall motion estimation from gated SPECT studies is a difficult task. Faber et al. [12] compared regional motion for gated sestamibi studies in four patients with gated MRI results. Endocardial and epicardial surfaces were detected using a gradient-based method and the mid-myocardial surface motion was calculated. The SPECT-derived motions in eight large regions of the heart were generally within 3-4 mm (1 pixel) of the MRI motion. This error level is consistent with the errors obtained by averaging the epicardial and endocardial errors of Table 1. The higher count level of the sestamibi studies made boundary detection easier than with our TI-201 patient images. The use of the mid-myocardial boundary, which averages the epicardial and endocardial boundaries, may be more robust for estimating gross wall motion than using either boundary alone.

Germano et al. [9, 22] have used estimates of endocardial and epicardial boundaries to quantitatively estimate global ejection fraction but have reported only qualitative visual assessments of wall motion and wall thickening based on these surfaces [22]. Goris et al. [13] calculated centroid and second moments of count distributions to characterize the midpoints and thickness of the myocardial wall as a function of time. Wall motion amplitude and wall thickening amplitude were compared with preliminary empirical normal limits, but no quantitative assessment of motion was made. The use of moments in the computation of the midmyocardial surface and its thickness is an interesting idea. One might expect that it would make the estimated surface location less sensitive to image noise than derivative-based methods such as that used in this paper. The use of a second moment as a measure of thickness is related to fitting a parametric curve to a profile. It is an alternative to computing the difference between boundary surfaces determined from edge-detection routines.

In comparison to previous work summarized above, a major contribution of the present paper is that cardiac wall motion was estimated quantitatively for a mathematical cardiac phantom whose motion was known exactly. It is important to objectively evaluate analysis programs under different conditions. In this paper wall motion was estimated with and without spatial blurring, and for normal, hypokinetic and dyskinetic wall motion. The method also was applied to gated TI-201 patient studies, demonstrating that regional wall motion information can be extracted even from these noisy studies.

C. Considerations for Clinical Application of the Wall Motion Estimation Method

The myocardial boundary and motion estimates will be dependent on the resolution of the imaging system, which includes hardware such as collimator type, image acquisition parameters such as radius of rotation, and reconstruction parameters such as filter type and cutoff. For application to clinical studies, as many of these parameters as possible
should be held fixed. Then the mean and standard deviation of motion in different cardiac regions can be established for a normal patient population for this particular acquisition, reconstruction and analysis procedure. It may prove useful to normalize motion by the size of the heart. Motion from patients with suspected cardiac disease then can be compared to results from this normal patient database. It would be desirable to classify regional cardiac motion as normal, hypokinetic, akinetic or dyskinetic based on its difference from the mean normal value, taking the standard deviation of normal motion into consideration. Serial studies on a single patient could be used to track cardiac function or to evaluate the effect of a given therapy.

The amount of image noise in clinical TI-201 SPECT patient studies suggests that additional research may be needed to determine the spatial frequencies for which regional wall motion can be reliably determined. It may be the case that regional wall motion can only be well-estimated in four or five large regions corresponding to vascular territories perfused by the major coronary arteries. Analysis of a larger set of patient studies could test this hypothesis.

D. Further Directions

The proposed cardiac wall motion analysis method uses epicardial and endocardial boundaries that were obtained from long axis slices for each time gate. An alternative approach is to use the 3-D reconstructed SPECT images from all time gates simultaneously in the determination of epicardial and endocardial boundaries. Spatial and temporal smoothing could be performed as part of such an analysis in order to diminish noise-related artifacts. A combined spatial-temporal approach to Bayesian SPECT image reconstruction has been previously proposed by Lalush et al. [23].

VI. CONCLUSIONS

A method for estimating regional wall motion from gated myocardial perfusion SPECT studies has been developed. The method uses epicardial and endocardial boundaries determined from long axis slices in each gate of the cardiac cycle. Wall motion with respect to reference ellipsoids is computed.

An initial evaluation of the method was made using the beating heart from the dynamic MCAT phantom, with and without a 1.5 cm FWHM Gaussian blurring filter. Epicardial wall motion was generally well-estimated within a fraction of a 3.56 mm voxel, though apical wall motion was overestimated with the smoothing filter. Endocardial wall motion was underestimated by about two voxels. The MCAT heart phantom was modified to model hypokinetic and dyskinetic wall motion. The wall motion analysis method enabled this abnormal motion to be differentiated from normal motion. A calibration procedure appears necessary to quantify the relationship between measured and true wall motion. Further studies are needed to study the effects of factors such as noise, scatter, and attenuation on the estimation of regional wall motion.

Regional cardiac wall motion was analyzed for TI-201 patient studies. Estimated wall motion was compared with a nuclear medicine physician’s visual assessment of motion from gated long axis slices for example male and female studies. The estimated motion was consistent with the physician’s readings of motion in these studies. Additional research is required to evaluate comprehensively the applicability of the method to patient studies with normal and abnormal wall motion.

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