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Spillover Compensation in the Presence of Respiratory Motion Embedded in SPECT Perfusion Data

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Abstract

Spillover from adjacent significant accumulations of extra-cardiac activity decreases diagnostic accuracy of SPECT perfusion imaging in especially the inferior/septal cardiac region. One method of compensating for the spillover at some location outside of a structure is to estimate it as the counts blurred into this location when a template (3D model) of the structure undergoes simulated imaging followed by reconstruction. The objective of this study was to determine what impact uncorrected respiratory motion has on such spillover compensation of extra-cardiac activity in the right coronary artery (RCA) territory, and if it is possible to use manual segmentation to define the extra-cardiac activity template(s) used in spillover correction. Two separate MCAT phantoms (128^3 matrices) were simulated to represent the source and attenuation distributions of patients with and without respiratory motion. For each phantom the heart was modeled: 1) with a normal perfusion pattern and 2) with an RCA defect equal to 50% of the normal myocardium count level. After Monte Carlo simulation of $64 \times 64 \times 120$ projections with appropriate noise, data were reconstructed using the rescaled block iterative (RBI) algorithm with 30 subsets and 5 iterations with compensation for attenuation, scatter and resolution. A 3D Gaussian post-filter with a sigma of 0.476 cm was used to suppress noise. Manual segmentation of the liver in filtered emission slices was used to create 3D binary templates. The true liver distribution (with and without respiratory motion included) was also used as binary templates. These templates were projected using a ray-driven projector simulating the imaging system with the exclusion of Compton scatter and reconstructed using the same protocol as for the emission data, excluding scatter compensation. Reconstructed templates were scaled using reconstructed emission count levels from the liver, and spillover subtracted outside the template. It was evident from the polar maps that the manually segmented template reconstructions were unable to remove all the spillover originating in the liver from the inferior wall. This was especially noticeable when a perfusion defect is present. Templates based on the true liver distribution appreciably improved spillover correction. Thus the emerging combined SPECT/CT technology may play a vital role in identifying and segmenting extra-cardiac structures more reliably thereby facilitating spillover correction. This study also indicates that compensation for respiratory motion might play an important role in spillover compensation.

Index Terms

Cardiac SPECT; partial volume; respiratory motion; spillover compensation

I. Introduction

In recent Receiver Operating Characteristic (ROC) studies comparing iteratively reconstructed Tc-99m sestamibi slices with combined compensation for attenuation (AC), scatter (SC) and distance dependent resolution (RC) with filtered backprojection (FBP) it was determined that

extra-cardiac activity hampered the accuracy of defect detection particularly in the right coronary artery (RCA) territory [1]. We postulated that spillover compensation could improve defect detection accuracy by removing extra-cardiac activity from the inferior wall of the myocardium and proceeded to perform a preliminary investigation to test this assumption with some success (Fig. 1) [2].

Based on these anecdotal positive results we performed spillover compensation on the 100 patient studies used in our original ROC investigation [1]. For spillover compensation we defined a 3D binary image (or template) of the extra-cardiac activity by manual segmentation of the SPECT perfusion study reconstructed with combined AC, SC, and RC. The binary template was forward projected with the inclusion of distance-dependent spatial resolution and non-uniform attenuation (using the acquired attenuation map) to simulate the imaging in the absence of scatter. The template projections were reconstructed using the same algorithm as the emission slices to yield the fractional spillover for all voxels outside the extra-cardiac template. These fractions were then multiplied by the average count per voxel determined from a central location of the extra-cardiac activity of the patient data set to estimate the spillover. The normalized spillover was then subtracted from the patient data and the extra-cardiac activity set to zero within the segmented binary template.

An ROC comparison between the spillover-corrected data set and the combined compensation (AC+SC+RC) data set using nuclear cardiologists as observers found no additional improvement in detection accuracy (Table I). From Table I it is evident that the area-under-the-curve (AUC) for the RCA territory (inferior wall of the left ventricle) for the spillover corrected data set decreased compared to combined compensation and is now similar to that of FBP. Postulated reasons for this negative result include the influence of respiratory motion embedded in the acquired emission data, and inaccurate manual segmentation of the extra-cardiac activity.

Therefore, the objectives of this study were to determine what impact uncorrected respiratory motion has on spillover compensation of extra-cardiac activity in the RCA territory, and if it is possible to use manual segmentation of emission reconstructions to define the extra-cardiac activity template for accurate spillover compensation.

II. Materials and Methods

A. Theory

As Nuyts, *et al.* [3] did we approximate the spillover counts (SC_{VY}) at a voxel V outside some extra-cardiac structure Y (such as the liver) from activity within that structure as being the spillover coefficient (SC_{VY}) times the average count per voxel in the structure (A_Y), or as an equation:

$$SC_{VY} = S_{VY} A_Y. \quad (1)$$

SC_{VY} is obtained by forward projecting and reconstructing a template (3D model) for structure Y obtained for example by segmentation. This approximation assumes that the concentration of activity within Y is relatively uniform, and we can obtain a reasonable estimate of A_Y . Next we define the total spillover counts at voxel V in structure X (TSC_{VX}) as the combined spillover from all other structures Y spilling into structure X (myocardium herein). Then the spillover corrected count at a voxel V in structure X (CC_{VX}) is calculated as

$$CC_{vx} = C_v - TSC_{vx} \quad (2)$$

where C_v is the count in voxel V prior to correction.

B. Data Generation

The mathematical cardiac torso phantom (MCAT) was used to simulate a set of data ($128 \times 128 \times 128$) with respiratory motion as previously described [4], [5]. In short, respiratory motion of the heart was simulated with a maximal axial motion of 1.5 cm. Solely axial motion of heart and other structures was simulated. The respiratory motion of the heart was modeled by creating one hundred and ninety two separate phantoms generated as if breathing was tracked over a 40 s period, i.e., eight 5 s respiratory cycles with 24 phantoms per cycle (from end-expiration through end-inspiration and back). The temporal location of the heart was randomly selected within each of the twenty four equally spaced respiratory time frames (respiratory gates) to ensure proper averaging within frames. Phantoms belonging to specific time frames (or gates) were average together. Cardiac motion was simulated with an ejection fraction of 41.1%, while the inferior defect included some thinning and reduced wall motion [6]. Apical thinning was also included as half the thickness of the normal myocardium extending for approximately 1 cm in all other directions. A perfusion defect in the inferior wall was simulated separately employing the improved methodology described in [6] by using a combination of defect sizes to eliminate the sharp transition between normal and diseased myocardium. In the current work, the generation of the defect is fully incorporated in the MCAT source code instead of partially as in [6]. A second MCAT phantom was generated without respiratory motion and a heart model with the same contraction pattern as the first. This MCAT phantom was used as a reference for comparison. The liver was separately simulated as the sole source of extra-cardiac activity using the same methodology as described for the heart to generate respiratory motion gates. With the exception of when the perfusion defect was present, a uniform activity distribution was simulated in both the heart and liver.

The SIMIND Monte Carlo package [7] was used to generate 120 projections through 360 degrees into $120 \times 64 \times 64$ voxel matrices with a spatial sampling of 0.634 cm. This matches the sampling of the clinical studies described in the introduction. Tc-99m sestamibi projections were acquired for both a 15% photopeak energy window centered on 140.5 keV and an 8% photon scatter energy window centered on 123.0 keV. The latter was used for triple energy window (TEW) scatter compensation [8]. Near noise free projections for all data sets (24 respiratory gates and one without respiratory motion added), were simulated separately for the liver, the heart, and the perfusion defect.

Projections with the perfusion defect present were formed by subtracting the perfusion defect projections from the normal heart projections before noise was added. Poisson noise was added such that the scatter free photon projection set of the heart without respiratory motion contained 600 K counts (determined to be in the range of our clinical perfusion studies). Noise added to the scatter-free perfusion-defect-absent respiratory-gated heart data set (24 projection sets in total) was such that the total counts came to 600 K or 25 K counts per projection set. All photopeak energy windows containing scatter as well as the TEW acquired energy windows were scaled using the scaling factors determined from the scatter free normal heart projections described above to obtain the desired number and distribution of photons. The liver projections were scaled to approximately have the same maximal count as the heart wall after reconstruction. This resulted in the total number of counts in the liver being on the order of ~ 2.3 M counts. The same methodology as for the heart was followed when determining liver noise levels for each of the 24 gated respiratory sets (~ 96 K counts per projection set). For phantoms with and without respiratory motion, two separate combined (liver plus heart)

projection data sets were constructed. The first was with a normal perfusion pattern (defect absent in the inferior wall) and the second with an RCA defect equal to 50% of the normal myocardium count level. To evaluate the effect of spillover compensation, similar projection data without the liver present was also generated.

C. Data Reconstruction and Analysis

All data were reconstructed using the rescaled block iterative (RBI) method with 30 subsets of 4 angles and 5 iterations including combined compensation for AC, SC and RC [9]. The TEW method was used during iterative reconstruction to correct for scatter [8] after which a 3D Gaussian post-filter with a sigma of 0.476 cm was applied to suppress noise. These parameters were selected to be the same as was used in the clinical ROC studies mentioned in the introduction. Respiratory gated projection sets were reconstructed separately and combined in two ways, 1) as is, and 2) after respiratory motion correction. Respiratory motion correction was accomplished by simply determining the center of mass of the heart of all 24 gates and aligning all sets with the first gated frame at end expiration. During the alignment process the direction and amplitude of the mismatch are determined and corrected by shifting slices axially with sub-voxel accuracy using linear interpolation.

Filtered data sets were used to manually segment the liver and form 3D binary templates. Manual segmentation was done by displaying slices re-sampled in a 256×256 format for better visualization and drawing regions of interest marking the edge of the extra-cardiac activity. By re-sampling to a larger matrix, segmentation can be done on a sub-voxel level. After segmentation, visual inspection was performed and if a) the segmentation strayed into the heart wall or b) left excessive liver activity fused with the heart, the segmentation was repeated. We elected not to use more sophisticated segmentation algorithms because a study of literature showed that all segmentation algorithms implemented in clinical nuclear cardiology use either some rule based approach to guide the algorithm [10], [11], or using the shape of the heart [12] instead. In most cases these algorithms, as implemented, can fail when the extra-cardiac activity merge with the inferior wall of the heart and manual intervention is needed. The case simulated in this work falls into the latter category.

The above templates were projected using a ray-driven projector [13] to simulate the imaging system with the exclusion of scatter similar to the clinical study mentioned in the introduction. In addition to these segmented templates, the true liver distribution from the MCAT source slices (with respiratory motion included) was also used to generate a set of template projections. The template projections were reconstructed and filtered using the same protocol as for the emission data, excluding scatter compensation. We acknowledge that iterative reconstruction techniques are non-linear with a distinct possibility of a different point of convergence at a given iteration number [14]–[18] when extra-cardiac activity is reconstructed separately. We [19]–[21] have explored the option of calculating the voxel specific spillover coefficients of (1) by adding a fraction of the template projections to the emission projections before reconstruction and then subtracting the emission only reconstructed data from the result as a method of better matching the local convergence of the emission data. Our results indicated that adding a fraction of the template projections to the emission projections before reconstruction and reconstructing the template projections separately [20] are equivalent for normal perfusion distributions for the heart. This result might not hold when a stopping rule rather than a high number of iterations and post filtering is employed.

A region of interest over the liver of the reconstructed emission slices was drawn to determine the average count scaling factor A_V in (1) after which (2) was used to compensate for spillover. After spillover compensation the liver was removed from the emission slices by setting to zero all voxels within the segmented liver mask.

Polar maps were generated by using the known location of the heart derived from the MCAT parameter file. All data were also re-orientated to the short-axis slices of the heart for visual evaluation. Also, circumferential profiles were drawn for selected short-axis slices to quantitatively visualize the effect of respiratory motion on perfusion defect size and contrast.

III. Results and Discussion

In Fig. 2 the polar maps in row (a) and (c) depict normal perfusion, and those in row (b) and (d) an inferior perfusion defect of 50%. Rows (a) and (b) are the results of the MCAT phantom generated without respiratory motion present, while rows (c) and (d) give the results of the MCAT phantom with respiratory motion present. The polar maps in the left column (1) were obtained without the liver present, and thus represent the best case scenarios as far as spillover from the liver. The liver was present when generating the polar maps of the next 3 columns. Polar maps in column (2) were left uncorrected for spillover, and the maps of column (3) were obtained by using the manually segmented templates for spillover compensation. From a comparison of the polar maps in the two central columns (2 and 3) with the maps of the first column (1), it is evident that the manually segmented templates were unable to remove all the spillover from the liver into the inferior wall. Note the arrows pointing to the residual spillover from the liver after spillover compensation in column (3) which was especially noticeable with the perfusion defect present (rows b and d). The polar maps in the right column (4) were obtained by using the true liver distribution as the template for spillover compensation, and thus represent the use of the ideal template which includes respiratory motion and is free from segmentation errors. Note that subtracting the true liver distribution during spillover compensation eliminates most of the unwanted counts in the inferior wall. Using the true liver distribution for spillover compensation is similar to i) measuring the respiratory motion and correcting for it prior to spillover compensation (a4 and b4), or ii) modeling the respiratory motion using some prior information (c4 and d4). The influence of respiratory motion when a defect is present in the inferior wall [compare rows (b) and (d)] manifest itself as an apparent increase in defect size. Also, it must be stressed that using the known location of the heart wall to generate the polar maps instead of only searching for the maximum in the heart wall (as commercially available packages do) reduced the apparent influence of the extra-cardiac activity considerably.

The vertical line of high counts in the septal region in the polar maps of Fig. 2 represents the thickening in the left ventricle wall where the right ventricle attaches, while the slight decrease in counts at the center of the polar maps (apex) are indicative of apical thinning. Also, the apparent decrease in inferior wall counts in the polar maps in row (c) is due to the influence of uncorrected respiratory motion.

Fig. 3 shows short-axis slices of the heart for a subset of the slices used to generate the polar maps in Fig. 2. Rows (a) and (b) are normal myocardium without and with respiratory motion and without the liver present (polar maps a1 and c1). The next two rows (c and d) depict the inferior wall defect for the same cases as rows (a) and (b) (polar maps b1 and d1). The effect of respiratory motion manifests itself as a slight decrease in counts in the inferior wall in (b) and an apparent increase in defect size in (d). In rows (e) and (f) respiratory motion is present, the inferior defect is absent, and spillover compensation has been applied using manual segmentation (e) and the true liver (f) (compare c3 and c4, Fig. 2). The last three rows represent spillover compensation with respiratory motion and the inferior defect present using manual segmentation (g), the true liver distribution (h) (compare d3 and d4, Fig. 2), and compensation for respiration with manual segmentation (i). Note that with using the true liver distribution an excellent correction is obtained [compare to (d)]; however, the effects of respiratory motion persist. The remnant of liver activity visible close to the apex in rows (e) and (g) is indicative of the difficulty of accurately segmenting the liver from the reconstructed slices where the LV

and liver are fused due to respiratory motion. It is also hampered by the spatial sampling of 6.34 mm. When respiratory motion compensation is attempted, the manual segmentation improves, the visibility of the defect improves compared to (g), but is not fully restored [compare Fig. 3(c)].

To better appreciate the effects of respiratory motion, we generated circumferential profiles through the average of the 3 short axis slices centered on the lesion location in the inferior wall (Fig. 4). It is obvious that the count density with respiration in the anterior, i.e., start and end of circumferential profiles, and inferior walls is less than without respiratory motion due to the dispersion of counts with respiratory blurring. However counts in part of the lateral wall and the septal wall compare well with the no respiratory motion case. This is due to the modeling of only inferior-superior respiratory motion and in the case of the septal wall also the presence of the RV to make the myocardium to appear thicker than it really is. The second effect already discussed in the previous paragraph regarding the increase in the apparent size of the lesion with respiration is also reinforced. In addition, the differences in perfect segmentation (true liver) and manual segmentation are also visible.

IV. Conclusion

We have determined that it is difficult to successfully correct cardiac SPECT slices for distortions in regional uptake due to spillover using manually segmented extra-cardiac distributions. Correction by manual segmentation is further degraded when respiratory motion is included in simulation of the acquired data. The emerging combined SPECT/CT technology can play a vital role in identifying and segmenting extra-cardiac structures more reliably. However, indications are that compensation for respiratory motion will play an important role in spillover compensation in cardiac imaging.

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Fig. 1. Patient with an angiographic occlusion in the RCA greater than 50%, without (a, and top row) and with (b, and bottom row) spillover correction. Both data sets were corrected for attenuation, scatter and resolution. A decrease in counts in the RCA of 48% was observed with spillover compensation.

Fig. 2.

Polar maps in row (a) and (c) depict normal perfusion, and those in row (b) and (d) an inferior perfusion defect of 50%. Rows (a) and (b) are generated without respiratory motion present, while rows (c) and (d) include respiratory motion. The polar maps in the left column (1) are obtained without the liver present, while the polar maps in columns (2) to (4) are with the liver present. Polar maps in column (2) are uncorrected for spillover and column (3) is obtained by using the manually segmented extra-cardiac activity templates for spillover compensation. The arrows in column (3) indicate where the liver uptake influences the inferior wall. The two arrows in (a3) each point to suspiciously high uptake in the inferior wall. The polar maps in column (4) are obtained by using the true liver distribution as the template for spillover compensation.

Fig. 3.

Subset of short-axis slices of the heart reorientated from the transaxial slices used to generate the polar maps in Fig. 2. Rows (a) and (b) are normal myocardium without and with respiratory motion and without the liver present. The next two rows (c and d) depict the inferior defect for the same cases as rows (a) and (b). In rows (e) and (f) respiratory motion is present, the inferior defect is absent, and spillover compensation has been applied using manual segmentation (e) and the true liver (f). The last three rows represent spillover compensation with respiratory motion and the inferior defect present using manual segmentation (g), the true liver distribution (h), and compensation for respiratory motion and manual segmentation (i).

Fig. 4.

Circumferential profiles with and without respiratory motion generated using the average of the 3 short-axis slices centered on the perfusion defect location. The profiles start in the center of the anterior wall and proceed clockwise through the lateral wall, the inferior wall (with defect), the septal wall, and finally end back in the anterior wall. The circumferential profiles are generated from the data with the liver present. The circumferential profiles with respiratory motion are generated using the short axis slices in Fig. 3(g) and (h) (centered on slice 7 of 10). The short axis slices from which the no respiratory motion profiles were generated are not shown in Fig. 3.

Area Under the ROC Curves for CAD and RCA. Combined Depicts Iterative Reconstruction With Combined Compensation for AC, SC, and RC

TABLE I

	Obs1	Obs2	Obs3	Avg	Std
CAD-Spillover	0.894	0.913	0.892	0.900	0.012
CAD-Combined	0.902	0.882	0.932	0.905	0.025
CAD-FBP	0.812	0.818	0.869	0.833	0.031
RCA-Spillover	0.718	0.775	0.693	0.729	0.042
RCA-Combined	0.771	0.787	0.786	0.781	0.009
RCA-FBP	0.708	0.747	0.794	0.750	0.043