Published in final edited form as:

J Nucl Cardiol. 2009 ; 16(4): 605–613. doi:10.1007/s12350-009-9103-z.

Prognostic validation of an algorithm to convert Myocardial Perfusion SPECT imaging data from a 12-segment model to a 17-segment model

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Abstract

Background—A 17 segment model has become the standard for interpreting myocardial perfusion single photon emission computed tomography (SPECT). Methods for converting preexisting databases from 12 segment models to the 17 segment model are needed for ongoing prognostic studies.

Methods and Results—To develop the conversion algorithm, 150 consecutive SPECT studies (82 abnormal) were read by both a 12 segment and the standard 17 segment models. Summed stress scores (SSS) were calculated from a 17 segment model derived from the 12 segment data and compared to those of the standard 17 segment model. The effect of the conversion algorithm on prognostic data derived from the 12 segment model was evaluated in 25,876 patients from the Duke Nuclear Cardiology Database, including a sample of 3,205 patients with known covariates for adjusted analysis. The derived 17 segment SSS from the 12 segment model was highly correlated ($R=0.99$) to the SSS from the standard 17 segment model. In both unadjusted and adjusted analysis, there was no difference in the prognostic information.

Conclusions—An algorithm for conversion of 12-segment perfusion scores to 17-segment scores has been developed which is highly correlated to visual interpretation by the 17-segment model with nearly identical prognostic information.

Keywords

Myocardial perfusion; single photon emission computed tomography; cardiac death; summed stress score

Introduction

Myocardial SPECT perfusion imaging has been shown to provide important prognostic information in patients with known or suspected coronary artery disease.(1–5) Throughout the

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Disclosures of Conflicts of interests: None

development of myocardial perfusion SPECT stress testing, multiple ventricular segmentation models have been used for assessing myocardial perfusion. Early in the history of SPECT perfusion imaging a 20-segment model was widely employed, but was later felt to over-represent the apical segments of the myocardium.(6,7) Multiple investigators have previously published SPECT perfusion studies using 12 or 14 segment models.(8–22) Since its inception in 1993 the Duke Cardiovascular Databank has used a 12-segment model for assessing myocardial perfusion. Within the last few years a 17-segment model has become the recommended standard for multiple cardiac imaging modalities and has been accepted by the ACC and ASNC for assessment of SPECT myocardial perfusion imaging.(6) In order to adopt this standard while continuing prognostic studies using the greater than 60,000 cases in our database which were previously read using 12-segment model, we needed a method for converting this data to a 17-segment model. A conversion algorithm for converting a 20-segment model to a 17 segment model has been previously described and has been shown to have a high correlation with expert visual reading of the 17-segment model, and equal prognostic information.(23) A similar conversion from a 12-segment to a 17-segment model has not been previously described. The goal of this study was to (1) demonstrate that a conversion algorithm from a 12-segment to a 17-segment model would be highly correlated with expert assessment of the images according to a 17-segment model and (2) to demonstrate that this conversion does not negatively effect the prognostic information that we have previously demonstrated from our 12-segment model data.(2)

Methods

Study Population

All patients in this study were part of the Duke nuclear cardiology database and were also followed in the Duke Cardiovascular Databank. The sample for validation of the conversion procedure consisted of 150 consecutive myocardial rest/stress perfusion studies performed between January 1, 2008 and February 5, 2008. The prognostic study consisted of two cohorts. The primary prognostic analysis was conducted using patients from the Duke Nuclear Cardiology Database (n=25,876) who underwent SPECT imaging at Duke Medical Center between 9/1993 and 7/2008 and for whom patient follow-up was conducted and mortality data was available. Patients are entered in the database as part of routine care and follow-up is ongoing. The prognostic analysis was additionally performed in our previously reported prognostic cohort consisting of 3,205 patients who had complete angiographic and SPECT myocardial perfusion imaging studies.(2) All patients in the second prognostic cohort had coronary angiography within 180 days before or after the SPECT study, and patients were excluded if they underwent subsequent revascularization within 60 days of their myocardial perfusion study. This research was performed with the approval of the institutional review board of the Duke University Medical Center.

Stress and Myocardial SPECT Imaging Protocol

All patients who could exercise underwent treadmill stress testing using the modified Bruce protocol, unless a different protocol was requested by the attending physician. Patients who were unable to exercise underwent pharmacological vasodilator stress testing with either adenosine or dipyridimole. If the patient had a contraindication to vasodilator stress, dobutamine stress testing was performed.

The protocol for performing SPECT myocardial perfusion imaging studies has been previously described.(2,3) In summary, SPECT data were obtained with multihead detectors using a step-and-shoot protocol. Images at rest were obtained for 30 seconds/projection and those during stress were obtained for 20 seconds/projection.

For validation of the conversion algorithm, a rest-stress imaging protocol with technetium-99m was used for all studies. In the prognostic study, either a technetium-99m rest-stress protocol, or a dual-isotope protocol with thallium-201 for rest and technetium-99m for the stress images was used. For obese patients weighing > 280 lb a 2- day stress-rest protocol was used.

Image Interpretation

To validate the conversion algorithm 150 sets of rest and stress images were read by consensus of two reviewers with expertise in nuclear cardiology. The images were first read by the previously reported (2) 12-segment model and then read (unblinded) according to the standard ACC 17 segment model (Figure 1). The reviewers were blinded to the patients' clinical information. The relative perfusion to each segment was quantified using a 4 point scale with 0 representing no defect, 1 representing a mild defect, 2 representing a moderate defect, and 3 representing a severe defect.

All of the studies in the prognostic cohort were independently reviewed as part of routine clinical care, by physician specialists in nuclear cardiology with the availability of accessory clinical data. The 12-segment model was used to quantify the location of the perfusion abnormalities, and the above 4 point severity score was utilized.

Conversion of 12-segment to 17-segment Model and Validation

The data from the 12-segment model was converted to the 17-segment model as shown in figure 1. Eight of the segments are identical between both models (segments 1, 4, 5, 6, 7, 10, 11, and 12 of the 17 segment model). The scores from segments 2 and 3 on the 12-segment model were assigned to segments 2 and 8 and segments 3 and 9 of the 17-segment model respectively. Scores from segments 11 and 12 of the 12-segment model were assigned to segments 13 and 15 respectively of the 17 segment model.

The remaining 3 segments of the 17 segment model (segments 14, 16, and 17) required weighted averages of segments from the 12 segment model. Two different weighting algorithms of neighboring segments were evaluated. In algorithm 1 the apical cap (segment 17) was taken as the average of the two apical segments (11 and 12) of the 12-segment model. The apical septum (segment 14) was assigned the average of segments 2 and 3, and the apical lateral wall (segment 16) was assigned to the average of segments 9 and 10 of the 12-segment model. In algorithm 2, the apical cap (segment 17) was calculated as in algorithm 1 above. The apical septum (segment 14) was assigned the average of segments 2, 3, 11, 12, and the apical lateral wall (segment 16) was assigned the average of segments 9, 10, 11, 12. The above weighting schemes were chosen for both their simplicity, and to preserve the same regionality of the perfusion defects.

A cumulative summed stress score (SSS), which has been shown to be highly predictive of cardiovascular outcomes in a 12-segment model (2) as well as the summed rest score (SRS) were obtained from the scores of both the original 12 segment model data, the visually scored 17 segment model data, and the 17-segment model score derived from the 12 segment data using our algorithm.

Additionally, as a reference, the 17 segment model SSS was predicted from the segment scores of the 12 segment model using a linear least squares fit. This results in a weighting scheme for the 12 segment model which will minimize the mean square error of the given data, but will result in a complex weighting of the segments from the 12-segment model, will not necessarily preserve coronary territories, and may not be consistent with how an expert would weight the segments visually. The resulting weights are also shown in the appendix.

Volumetric SSS and SRS were also derived by weighting each segment of the 12 segment model by $1/12^{\text{th}}$ of the ventricular volume, and each segment of the 17 segment model by $1/17^{\text{th}}$ of the ventricular volume. This is equivalent to weighting each segment of the 12 segment model by $17/12$ or 1.42. This weighting scheme is also shown in the appendix.

The data were additionally transformed into a percent of the maximal score for each of the models to allow for direct comparison of the scores from models with different numbers of segments as proposed by Berman et al.(23) This effectively converts the stress scores into units of percentage of maximal score which may be easier to understand and facilitates direct comparison of scores derived with different numbers of segments. This was done by dividing the SSS or SRS by the maximum possible stress score given the number of myocardial segments in the model. Mathematically, this conversion does not effect the correlation between the stress scores from which it was derived.

Comparison of Weighting Schemes

The 17-segment scores derived from the weighting algorithms of the 12-segment model as described above were compared to the visually read 17-segment model with linear regression, correlation coefficients, and determination of the mean-squared error between the model and the visual interpretations. The model with the highest correlation coefficient, lowest mean-squared error, with a regression equation with a slope close to 1 and with an intercept near zero was chosen for the prognostic validation.

Follow Up

The follow-up methodology for the prognostic cohort has been previously described.(2) Briefly, information was collected on death, cardiovascular death, non-fatal myocardial infarction and date of last known status. An independent clinical events committee reviewed and classified all events without knowledge of the patient's clinical, cardiac catheterization, or SPECT myocardial perfusion results. Follow-up was greater than 93% complete.

Statistical Analysis

In the validation sample (n=150) we assessed agreement between scores based on the 12 segment method, 17 segment method, and the derived 17 segment score using correlation coefficients and linear regression. For each of the segments in the derived 17-segment model which involved a weighted average of multiple segments of the 12-segment model (segments 14, 16, 17), the derived scores were correlated with the visually read 17 segment model scores. A sensitivity analysis was conducted after the removal of all normal studies to see if this markedly affected the regression coefficients and correlation coefficients between the models.

We conducted prognostic analysis using the 12 segment score and compared the results to an analysis based on the derived 17 segment score. For the primary prognostic cohort (n=25,876) we used logistic regression to predict 1 year mortality and 1 year cardiac death (CD). Logistic regression was used, as opposed to survival analysis, because 1 year censoring was low (3%) and censoring is unrelated to the comparison of stress scores. We compared the resulting odds ratios and survival probability plots. A Cox proportional hazards model was use to evaluate all-time mortality and all-time CD. Hazard ratios and C-index were obtained along with survival plots. These analyses were unadjusted so that the prognostic value of the stress scores could be compared directly.

To address the possibility that prognostic information might differ after controlling for covariates, we conducted an adjusted prognostic analysis in the previously published cohort of 3,205 patients who had completed angiographic and SPECT myocardial perfusion imaging studies. We repeated our analysis of mortality and cardiac death accounting for important

covariates that were identified and previously published study for this cohort by Borges et. al.. (2)

Results

Validation of Conversion Algorithm

In our validation cohort, 82 of the 150 studies analyzed had perfusion defects, 46 of which demonstrated stress induced ischemia. The average SSS using the 17 segment model was 5.7 overall and 10.4 when only including positive studies corresponding to 11% and 20% of the maximum score respectively. Table 1 shows the correlation coefficients, regression slopes, and intercepts for the 4 weighting schemes. In all cases, the derived 17 segment model parameters were highly correlated with the visually read 17 segment model SSS. Algorithm 1 had the highest correlation coefficient, a slope closest to one, and a lower mean-squared error than the volumetric SSS or algorithm 2. The derived-17 segment data from algorithm 1 and the visually read 17 segment SSS are shown in figure 2. When the negative studies were removed the correlation coefficient was 0.991 (95% CI 0.978–0.991) with a regression equation of $1.01 \times - 0.49$. As the regression equations have a slope very close to 1 and an intercept near zero, the derived 17 segment scores are nearly equivalent to those of the visually read 17 segment scores. The average error between the derived 17 segment SSS and the visually assessed 17 segment SSS is 0.60 units, and in 80% of cases the two scores varied by less than one unit (the equivalent of changing a single segment by a single severity grade). There were only 4 cases where the difference between the two scores was greater than 5% of the maximal score. Furthermore, all of these outliers had a percent max score > 20% of the maximal possible score, meaning that they would all remain in a high risk group with either segmentation algorithm. The discrepancies occurred primarily in the apex due to the slight difference in weighting of the apex when the apical lateral or apical septal walls are unaffected.

Figure 3 shows a plot of the percentage of maximal score by both the 12 segment model and the 17 segment model by weighting each segment proportional to its volume. The regression equation is $0.97 \times - 0.30$ with a correlation coefficient of 0.98 (95% CI 0.97–0.98). This correlation coefficient is identical for directly comparing the 12 segment and 17 segment models and weighting the 12 segment model data by 12/17, only the regression equations differ. The correlation coefficient for this method is just slightly less than for our conversion algorithm, however the mean-square error is greater as shown in table 1.

In looking specifically at the 17-segment model segments which are calculated from a weighted average of 12-segment model segments (14, 16, and 17) the stress scores for these segments are highly correlated ($R=0.79$ (95%CI 0.72–0.84), 0.62 (95%CI 0.51–0.71), 0.93 (95%CI 0.91–0.95) respectively).

Risk stratification with 12-segment and 17 segment models

Table 2 shows a comparison of the unadjusted odds ratio (OR) estimates based on logistic regression for overall mortality and cardiovascular mortality in the primary prognostic cohort ($n=25,876$) for the 12 segment SSS and the derived 17 segment SSS. Table 3 shows an unadjusted comparison of the hazard ratio (HR) estimates for the same cohort using a Cox-proportional hazard model for all time mortality. All OR and HR are expressed for a 5% increase in the percent maximum score for each segmentation model. The results of both of these analyses are nearly identical for both the original 12 segment scores and the derived 17 segment scores.

In the prognostic cohort, the average difference between the 12 segment model %SSS, and the derived 17-segment model %SSS was $1.9 \pm 2.2\%$. In 90% of the patients, there was less than

a 5% difference between the two %SSS, and of those with a greater than 5% difference between the models, the mean %SSS was 40%. Thus, the largest deviations between the models were in patients with markedly abnormal %SSS.

Figure 4 shows the event rate of cardiac death per year as a function of the percent abnormal myocardium for both the original 12 segment scores and the derived 17 segment scores for each 5% increase in %SSS. There are no significant differences between the event rates at each degree of abnormality by independent t-tests. In this unselected population, a SSS less than 5% of the ventricle was predictive of less than a 2% risk of cardiac death per year.

Table 4 shows the adjusted logistic regression model for overall and cardiovascular specific mortality for the prognostic cohort with known covariates (n=3205) for the 12 segment SSS and the derived 17 segment SSS expressed as a percentage of the maximum possible scores. Table 5 shows the adjusted analysis based on the Cox proportional hazard model for the same cohort. Both of these adjusted analyses have nearly identical results and c-indices which are both greater than 0.7.

Figure 5 shows unadjusted and adjusted 5 year survival curves for the 12 segment and 17 segment SSS for the prognostic cohort (n=25,876).

Discussion

We have demonstrated a conversion algorithm to convert 12-segment model data to 17 segment data which is highly correlated with expert reading of the same studies by the 17 segment model. Thus we have a robust method for converting 12 segment data into 17 segment data. Our conversion algorithm performed better than merely converting each of the scores to a percentage of the ventricular volume, although our validation data suggests that this methodology would also result in highly correlated scores as shown in (Table 1, Volumetric). A least squares fit of the validation data would also provide highly correlated scores, but does not necessarily result in weights which intuitively correlate to how an expert would visually assign the weightings.

When we look at the prognostic value of the converted data, it is nearly identical to the prognostic information derived from our 12 segment model data. This was true for both logistic regression models and Cox proportional hazard models. Additionally, the results were nearly identical in both adjusted and unadjusted models. Thus the conversion of the 12 segment model data to 17 segments does not disrupt the prognostic utility of this data. In the unadjusted analysis, the c-indices were greater than 0.6, and in the adjusted cohort they were greater than 0.7.

There are several important limitations to consider when interpreting the results of our study. Ideally one would like to directly compare the visually read 12 segment model data to visually read 17 segment data in the prognostic cohort; however given the enormous number of studies in our database this would not be feasible. Our study does demonstrate that the conversion of our 12 segment model data to a 17 segment model does not disrupt the prognostic utility of this data. This was similar to the approach taken by Berman et al. in creating a conversion algorithm to reduce 20 segment data to 17 segment data.(23) However, this type of analysis does not enable us to determine if directly reading by a 17 segment model would have greater or lesser prognostic information then by reading using the 12 segment model.

The images were read by two reviewers in consensus, and the inter-reader variability in assigning the scores to each segment was not assessed. The inter-reader variability for reading nuclear perfusion images has been well established (24–26), and was not a goal of this study. Our goal was to determine if 12 segment data could reasonably be converted to 17 segment

data by a simple mathematical algorithm, which would agree with visual reading by the 17 segment model.

The images were read by both models in an unblinded fashion. This was done to minimize intra-reader variability. The emphasis of this study was on how specific perfusion defect would be spatially mapped via either segmentation algorithm, rather than in the interpretation of whether a perfusion abnormality exists. Furthermore we wanted to insure consistency in the degree of severity which was read since our goal was primarily to look at the spatial localization of the defects when using 12 versus 17 segments, without the additional variability introduced by reading a defect with differing severity by both models. Similarly, the intra-reader variability of reading nuclear studies has been assessed previously.(24–26)

As no exclusions were applied to our main prognostic cohort, determining cut-offs for the % SSS for a specific event rate, may not be comparable to other prognostic studies which generally exclude patients with known coronary disease or prior revascularization. This is evident in our data as the event rate of cardiac death for a normal nuclear study is 2% in this cohort, and the overall annualized cardiac death rate was 5% for this population. However, in this data set, the event rate was less than 2% for a %SSS of 5% of the ventricular volume. Furthermore, as the angiographic data available for the adjusted cohort was obtained within 180 days before or after the index nuclear study, there may be significant biases in determining an appropriate diagnostic cut-off for the 12-segment model for detecting coronary artery disease.

The advantages of our conversion algorithm include its simplicity and its amenability to being applied to our pre-existing data without any interventions by a reader. Furthermore, as much as possible, the algorithm preserves the coronary distribution of the segments. As the derived 17 segment parameters are highly correlated to the visual reads with a regression equation with a slope near 1 small intercept, the derived scores do not require further modifications to be directly compared to future studies read by the 17 segment algorithm. In the validation study, nearly 80% of the cases differed by less than one unit from the visually assessed 17 segment read. This difference corresponds to changing a single segment by a single severity score, which is considerably less than what would be expected from intra-reader or inter-reader variability. Thus, this conversion is unlikely to affect results of prognostic studies using 17 segment scores derived from originally read 12 segment models. Our analysis in two prognostic cohorts with both adjusted and unadjusted models attest to this fact.

In conclusion, we have developed an algorithm to convert 12 segment myocardial perfusion data to 17 segments which is highly correlated with visual reading according to the 17 segment model. Furthermore, the conversion algorithm has been shown not to adversely affect the prognostic information as demonstrated by our cohort analysis.

Acknowledgments

This work was supported by: None

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Figure 1.
Left ventricular segmentation Bulls-eye plots for the (a) 12 segment model and (b) standard ACC 17 segment model.

Figure 2.

Correlation of the 17 segment SSS derived from the 12 segment data and the visually read 17 segment SSS. The regression equation is $y=0.99x-0.17$ with a correlation coefficient of 0.991 (95% CI 0.988–0.994).

Figure 3. Correlation of the 12 segment and 17 segment SSS expressed as (a) the actual SSS and (b) the percent maximal SSS. Although they have different regression equations, as expected mathematically, they have identical correlation coefficients ($R=0.978$ (95% CI 0.970–0.984)).

Figure 4.

Cardiac death per year for the % abnormal myocardium for original 12 segment scores and the derived 17 segment scores for each 5% increase in %SSS were not significantly different. In this population the cardiac death with a %SSS less than 5% of the ventricle was 2%.

Figure 5.
(a) Unadjusted and (b) Adjusted 5 year survival curves for the 12 segment and 17 segment models for the prognostic cohort.

Table 1
Performance of Weighting Algorithms

Correlation coefficients, regression slope and intercept, and mean-squared error (MSE) for each of the weighting algorithms.

Criteria	Volumetric	Algorithm 1	Algorithm 2	Least Squares
R (95%CI)	0.978 (0.970–0.984)	0.991 (0.988–0.994)	0.991 (0.988–0.994)	0.993 (0.990–0.995)
Slope	0.97	0.99	0.96	1.00
Intercept	−0.30	−0.17	−0.16	−0.06
MSE	0.311	0.045	0.284	0.003

Table 2
Unadjusted Logistic Regression

Unadjusted OR based on logistic regression for overall mortality and cardiovascular mortality from the whole prognostic cohort (n=25,872) for the 12 segment SSS and derived 17 segment SSS.

Predictor	Outcome	OR (95% CI)	C-index
12 segment SSS	Mortality	1.14 (1.13–1.16)	0.63
derived 17 SSS	Mortality	1.14 (1.13–1.16)	0.63
12 segment SSS	CV Mortality	1.17 (1.15–1.19)	0.66
derived 17 SSS	CV Mortality	1.16 (1.15–1.18)	0.66

note: OR are expressed for each 5% increase in %SSS

Table 3
Unadjusted Cox Proportional Hazard Model

Unadjusted HR based on a Cox proportional hazard model for overall mortality and cardiovascular mortality from the whole prognostic cohort (n=25,876) for the 12 segment SSS and derived 17 segment SSS.

Predictor	Outcome	HR (95% CI)	C-index
12 segment SSS	Mortality	1.10 (1.09–1.11)	0.63
derived 17 SSS	Mortality	1.09 (1.09–1.10)	0.64
12 segment SSS	CV Mortality	1.12 (1.12–1.13)	0.63
derived 17 SSS	CV Mortality	1.13 (1.12–1.14)	0.63

note: HR are expressed for each 5% increase in %SSS

Table 4
Adjusted Logistic Regression

Adjusted OR based on logistic regression for overall mortality and cardiovascular mortality from the prognostic cohort with known covariates (n=3,205) for the 12 segment SSS and derived 17 segment SSS.

Predictor	Outcome	OR (95% CI)	C-index
12 segment SSS	Mortality	1.11 (1.05–1.18)	0.73
derived 17 SSS	Mortality	1.11 (1.05–1.17)	0.73
12 segment SSS	CV Mortality	1.15 (1.08–1.23)	0.79
derived 17 SSS	CV Mortality	1.14 (1.06–1.21)	0.79

note: OR are expressed for each 5% increase in %SSS

Table 5
Adjusted Cox Proportional Hazard Model

Adjusted HR based on a Cox proportional hazard model for overall mortality and cardiovascular mortality from the prognostic cohort with known covariates (n=3,205) for the 12 segment SSS and derived 17 segment SSS.

Predictor	Outcome	HR (95% CI)	C-index
12 segment SSS	Mortality	1.08(1.05–1.11)	0.77
derived 17 SSS	Mortality	1.07 (1.04–1.10)	0.77
12 segment SSS	CV Mortality	1.11 (1.08–1.15)	0.78
derived 17 SSS	CV Mortality	1.10 (1.07–1.14)	0.78

note: HR are expressed for each 5% increase in %SSS

Appendix 1

Weighting Schemes

Segment	Volumetric	Algorithm 1	Algorithm 2	Least Squares
1	1.42	1	1	0.87
2	1.42	2.5	2.25	2.32
3	1.42	2.5	2.25	2.26
4	1.42	1	1	0.42
5	1.42	1	1	1.40
6	1.42	1	1	0.96
7	1.42	1	1	1.14
8	1.42	1	1	1.50
9	1.42	1.5	1.25	1.15
10	1.42	1.5	1.25	1.01
11	1.42	1.5	2	1.19
12	1.42	1.5	2	2.19