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Unique Contraction Pattern in Patients After Coronary Bypass Graft Surgery by Gated SPECT Myocardial Perfusion Imaging

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Purpose: Although left ventricular systolic function seems to be accurately represented on gated SPECT myocardial perfusion imaging, specific patterns of wall motion (WM) and thickening after coronary bypass graft surgery (CABG), demonstrated by other imaging methods, have not been characterized for gated SPECT myocardial perfusion imaging.

Methods: Gated SPECT myocardial perfusion imaging was studied in 30 consecutive patients after CABG (group 1) and 40 non-CABG patients—30 with normal stress perfusion studies (group 2) and 10 with known previous anterior wall infarction (group 3). Two expert readers evaluated epicardial and endocardial systolic WM. Regional WM and the thickening percentage were obtained using CEQUAL 20 segment bull's eye analysis and compiled into regional values.

Results: Qualitatively, the post-CABG patients had hypokinetic septum, a hyperdynamic lateral wall, and preservation of anterior WM. In 25 of 30 (83.3%) patients, an anterior systolic epicardial “swing” was evident and was different from the inward endocardial and epicardial motion seen in groups 2 and 3. Septal WM was decreased in group 1 compared with group 2 (2.9 vs. 6.0 mm, $P < 0.001$), with no significant difference in septal thickening. This was not different from the reduced septal motion seen in group 3 (4.1 mm), which was accompanied by both reduced thickening and abnormalities of anterior WM. Lateral WM was increased in group 1 compared with group 2 (9.9 vs. 8.2, $P < 0.001$), with no significant difference in lateral wall thickening (34.6% vs. 39%).

Conclusions: There is a characteristic contraction pattern on gated SPECT myocardial perfusion imaging in post-CABG patients distinguished by apparent septal hypokinesis with preservation of septal wall thickening, apparent increase in endocardial lateral WM, and an anterior epicardial “swing,” different from the contraction pattern seen in normal patients and those with previous anterior wall infarction. The related perfusion pattern aids in evaluation of the mechanism of these findings.

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Key Words: Contraction Pattern, Coronary Bypass Graft Surgery, Gated SPECT, Perfusion Imaging.

WALL MOTION ABNORMALITIES are generally assumed to be caused by intrinsic myocardial disease. They can also relate to abnormalities of conduction (1–3) and extramyocardial conditions, which must be recognized. A characteristic pattern of left ventricular wall motion after coronary artery bypass graft surgery (CABG) has been described using non-nuclear imaging methods (4–7). These findings, previously documented and long recognized on echocardiography and blood-pool scintigraphy (4,5), demonstrate the development of septal wall motion abnormalities and even paradoxical septal motion, accompanied by augmented motion of the “posterior wall” after coronary bypass and other cardiac surgeries (6). SPECT myocardial perfusion imaging (SMPI) is now a commonly used noninvasive imaging method in the evaluation of coronary artery disease. Current methods of SMPI use electrocardiographic gating, allowing abnormal patterns of wall motion to be potentially identified with gated (G) SMPI. Although anecdotal descriptions exist of left ventricular wall motion in patients after CABG using GSMPI, left ventricular wall motion in the post-CABG setting has not been thoroughly characterized. In addition, the added value of related regional perfusion evaluation to the assessment of such wall motion abnormalities has not been sought.

This study was conceived and initiated during a clinical nuclear cardiology reading session, following the observation that after CABG patients often appeared to have a characteristic contraction pattern on GSMPI.

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This pattern seemed so unique that it often permitted the reader to correctly predict a patient history of CABG based solely on the appearance of left ventricular wall motion without previous knowledge of the clinical history. The focus of this study was to determine whether a characteristic and even unique contraction pattern indeed exists on GSMPI in patients who have had CABG.

Methods

Patients

The University of California San Francisco Human Research Committee approved the study. Three patient groups, formulated from selected patients referred to Moffitt-Long Hospital for gated SPECT myocardial perfusion imaging between October 2000 and April 2001, were included and compared in this retrospective study. Excluded were patients with bundle branch block, which influences apparent regional contraction, or arrhythmia, which prevents gating and potentially alters wall motion analysis. Group 1 consisted of 30 consecutive patients who had previous CABG surgery and underwent Tc-99 sestamibi GSMPI for clinical indications. Excluded were those who had historic or electrocardiographic evidence of previous anterior myocardial infarction.

Groups 2 and 3 were comprised of 40 randomly selected patients without previous CABG from among those studied by GSMPI for clinical indications during the same period. Group 2 included 30 control patients with normal findings of electrocardiogram and normal GSMPI, with a low to intermediate likelihood of coronary artery disease based on the criteria of Diamond et al. (8) and calculated by the Cadenza computer program (Advanced Heuristics, Bainbridge Island, WA). Group 3 consisted of 10 patients with known previous anterior myocardial infarction based on their clinical history, baseline electrocardiogram results, and the presence of fixed perfusion abnormalities. The presence of infarction in other regions was neither sought nor excluded.

Gated SPECT Imaging Protocol

All gated studies were acquired after stress testing, with poststress image acquisition, approximately 30 minutes after exercise and 1 hour after dipyridamole infusion. Images were acquired over 64 angles in a 180° circular orbit from the 30° right anterior oblique to the 30° left posterior oblique, with a dual-head Philips-ADAC (Milpitas, CA) Forte camera, using a VXGP collimator (Philips-ADAC) with 32 25-second stops after the intravenous administration of 20 to 30 mCi Tc-99m sestamibi at the peak of dynamic exercise or pharmacologic stress. Rest images were obtained after the intravenous administration of 3 to 4 mCi Tl-201. Images were processed using a Butterworth filter, order 5, with a 0.4 cutoff, analyzed, and displayed using the quantitative CEQUAL AutoQuant software package (Philips-ADAC) on a Sun-based Solaris UltraSPARC Pegasys UltraHT (high tier) computer (Sun Microsystems, San Jose, CA) for visual interpretation. All studies were screened for artifact before analysis.

Qualitative and Quantitative Analysis of Left Ventricular Function

The computer-generated CEQUAL surface rendering of epicardial and endocardial systolic wall motion was evaluated

by agreement of two expert readers. In each case, the endocardial systolic wall motion was evaluated in the regions of the left ventricular septum and the anterior, lateral, and inferior walls. Endocardial and epicardial left ventricular wall motion was read as hyperdynamic (demonstrating exaggerated systolic inward motion); normal; hypokinetic (with reduced systolic motion); akinetic (without systolic motion); or dyskinetic (demonstrating outward movement in systole). The presence or absence of anterior systolic epicardial motion, best seen in the left anterior oblique projection, was also noted for each patient study. Wall thickening was visually expressed as normal or abnormal based on the visual impression of wall intensity change during the cardiac cycle.

Quantitative parameters of wall motion and wall thickening were also calculated for each of these evaluated regions, generated from the composite values in component regions, obtained from the 20 AutoQuant segment polar map. These segments were grouped into regions, and the average values in summed component segments were used to calculate regional wall motion in millimeters and wall thickening as a percentage of end-diastolic thickness. These regions are shown in Figure 1, with values for the septal, anterior, lateral and inferior walls as shown, acquired in a normal group 2 patient. Although recent work has revealed the heterogeneous nature of regional systolic wall motion and thickening (9), for this study, the gross values representing the normal limits of systolic wall motion $\geq 30\%$ and wall thickening, $\geq 40\%$, established initially for the AutoQuant analytic method, are applied in all myocardial regions.

Statistical Analysis

A mean value and standard deviation was calculated for wall motion and wall thickening for each of the four wall regions in each of the three groups. Standard two-tailed t tests were used for all two-way comparisons between each of the three groups. Probability values of ≥ 0.05 were used to determine the statistical significance of the observed difference between means.

Results

Patient Characteristics

Group 1 patients included 22 men and 8 women (average age, 64 years) a mean of 7.5 years after CABG. The mean left ventricular ejection fraction (LVEF) was 54%. Treadmill exercise testing was used in 14 patients, whereas 16 patients had dipyridamole pharmacologic stress testing. The average age of group 2 patients was 58 years and included 20 men and 10 women. The mean LVEF was 62%, with treadmill exercise and pharmacologic stress testing each performed in 15 patients. The average age of group 3 patients was 57 years and included 6 men and 4 women. Three patients had exercise treadmill testing and 7 had dipyridamole stress. This group had a mean LVEF of 42%.

Visual Analysis

Qualitatively, all 30 post-CABG patients (group 1) had hypokinetic septal endocardial wall motion, a hyper-

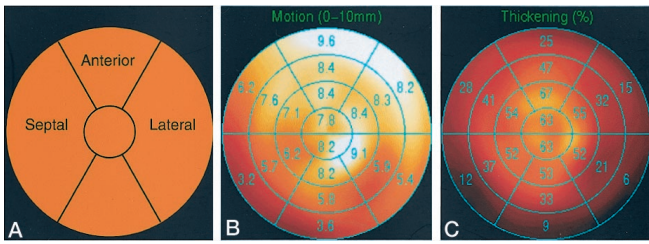


Fig. 1

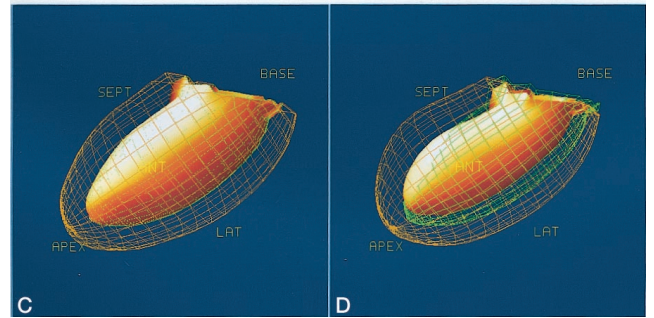
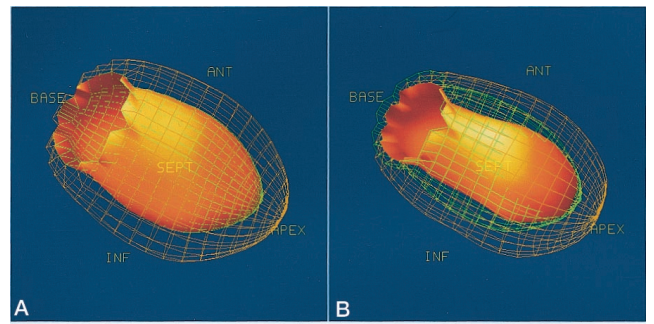


Fig. 4

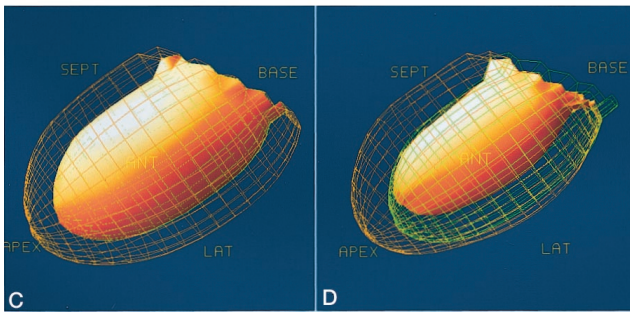
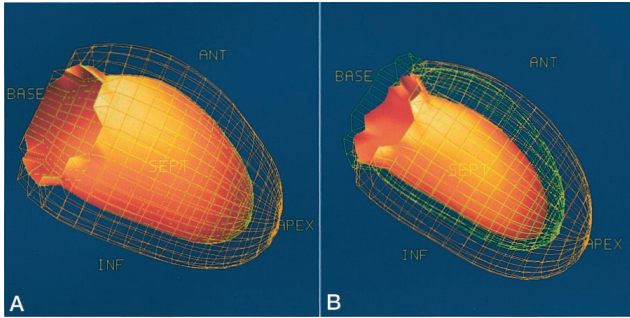


Fig. 2

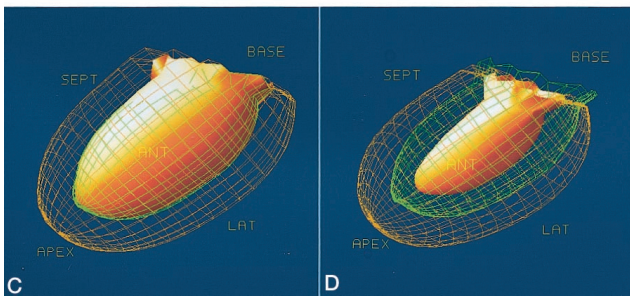
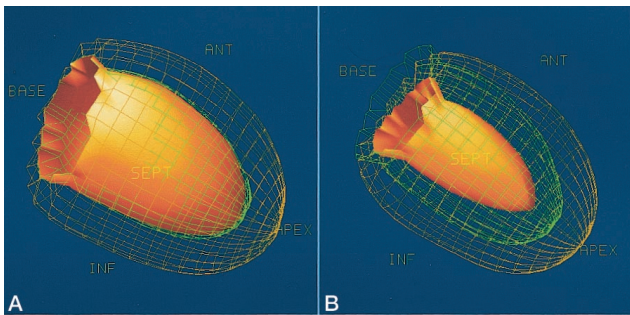
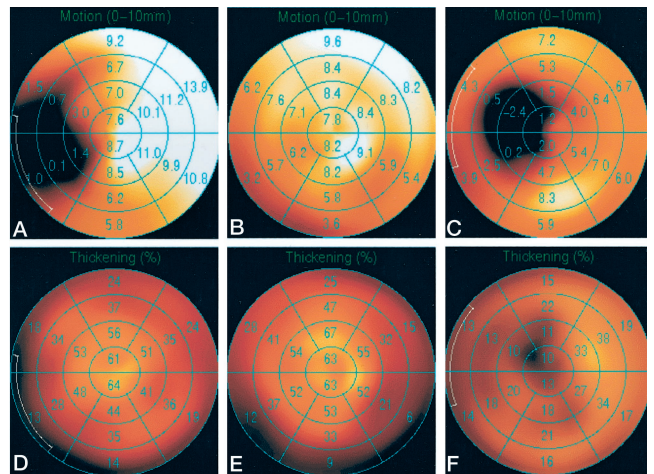


Fig. 3



Post-CABG

Normal Perfusion

Ant Infarct

Fig. 7

← Fig. 1. (A) A polar map display of septal, anterior, and lateral wall regions. (B,C) Polar maps of segmental wall motion and thickening, respectively, in a normal patient. Wall motion and thickening of septal, anterior, and lateral regions were analyzed and presented here for all study groups.

← Fig. 2. Wall motion in a patient after CABG (group 1). These computer-generated, surface-rendered images show left ventricular wall motion in a typical post-CABG patient. The anterior view is shown in the upper panels, whereas the left anterior oblique view is presented in the lower panels. In each case, the end-diastolic frame is shown at left and the end-systolic frame at right. In each panel, the endocardial volume is painted in solid orange, whereas the epicardial boundaries are shown in orange mesh. In the end-systolic frame, the end-diastolic boundaries are shown in green mesh. Preserved anterior endocardial motion is clearly shown in the anterior view. Reduced septal and augmented lateral endocardial motion is seen best in the left anterior oblique view. The apparent anterior epicardial swing is also evident in the left anterior oblique view.

← Fig. 3. Wall motion in a patient with normal perfusion (group 2). The computer-generated, surface-rendered images of left ventricular wall motion in a typical patient with normal perfusion are shown according to the format used in Figure 1. Normal systolic endocardial motion and normal, concentric, and inward epicardial motion are evident.

← Fig. 4. Wall motion in a patient with an anterior infarction (group 3). The computer-generated, surface-rendered images of left ventricular wall motion in a typical patient with a previous anterior infarction are presented in the same format used in Figure 2. Severely hypokinetic to akinetic endocardial motion of the apex and distal anterior wall is seen in the anterior view, with similar abnormalities of the septum seen in the left anterior oblique view. Epicardial motion was generally preserved, with somewhat reduced inward septal motion.

← Fig. 7. Shown from the related CEQUAL QGS display are the polar wall motion maps (upper row) and the polar thickening maps (lower row) in typical patients after CABG (left), patients with normal perfusion (center), and patients after anterior infarction (right). Reduced septal wall motion with preserved thickening after CABG and reduced wall motion and thickening after anterior infarction are evident.

dynamic lateral epicardial wall, and preservation of normal anterior endocardial motion (Fig. 2). This was seen despite preserved septal perfusion in all cases. In 25 of 30 patients in group 1, a “swinging” anterior systolic epicardial septal motion was identified, as shown in Figure 2, in contrast to the inward, or posterior, septal systolic motion shown in 29 of 30 patients in group 2 (Fig. 3). Twenty-nine of the 30 patients with normal perfusion (group 2) had normal concentric inward motion of both endocardial and epicardial walls (Fig. 3). In all the patients with anterior infarction (group 3), hypokinetic septal endocardial wall motion and hypokinetic anterior endocardial wall motion were seen. In these patients, anterior wall motion abnormalities were generally confirmed to the distal half of the anterior wall with preserved basal wall motion (Fig. 4). Epicardial septal motion in 10 of 10 patients in group 3 was inward or posterior in direction, similar to but relatively reduced compared with that seen in group 2. Visual assessment revealed preserved thickening (brightening) of the septum, anterior and lateral walls in the post-CABG group 1 patients, but septal, anterior, and lateral wall thickening appeared reduced compared with normal in the postinfarction group 3 patients.

Quantitative Analysis

Wall Motion. Figure 5 shows quantitative results for endocardial septal wall motion. The average inward septal wall motion in the post-CABG patients (group 1) was 2.9 mm, which was less but not significantly different compared with the average septal wall motion of 4.1 mm for the anterior infarct group (group 3). The septal wall motion of both groups 1 and 3 was significantly decreased from the average inward septal wall motion of 6 mm ($P < 0.001$) obtained for the normal perfusion group (group 2). Endocardial motion of the lateral wall (9.9 mm) was significantly increased in the post-CABG group compared with the other two groups ($P < 0.001$). Compared with the anterior wall motion of the normal perfusion group (8.5 mm), anterior wall motion was modestly reduced in the post-CABG group (7.4 mm, $P < 0.01$) and markedly decreased in the anterior infarction group (5.6 mm, $P < 0.001$), where basal and distal segments were included.

Wall Thickening. Figure 6 shows the quantitative evaluation of average wall thickening. Unlike average wall motion, septal wall thickening in the post-CABG group was normal (31.4%) and not significantly different compared with that of the normal group at 37.4% (group 2). However, average septal wall thickening in the postinfarction patients (group 3), like the average septal wall motion in this group, was markedly reduced at 18.1% compared with that in the other two groups ($P < 0.001$).

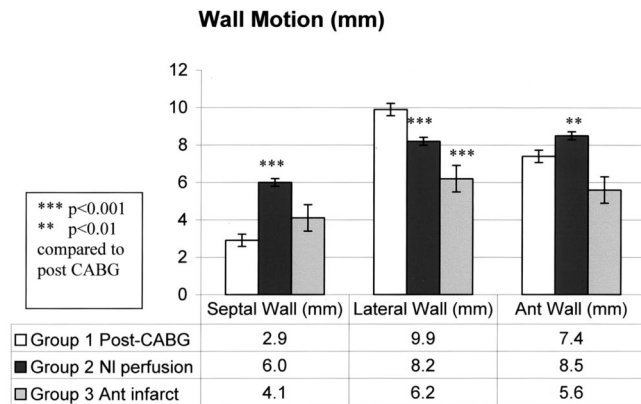


Fig. 5. Measurements of regional septal wall motion are presented. Septal wall motion in post-CABG patients (white bars) was significantly reduced compared with that in patients with normal perfusion (black bars) but not significantly different from those with previous anterior infarction (gray bars). Lateral wall motion in the post-CABG patients was increased compared with that in other groups, whereas anterior wall motion was reduced in this group compared with normal patients and was more reduced in the patients with anterior infarction (group 3).

Lateral wall thickening in the post-CABG group (36%) also was not significantly different from the normal controls at 39%, but lateral wall thickening was reduced in the anterior infarct group (26%) compared with the other two groups ($P < 0.01$). Anterior wall thickening, at 36%, was normal in the post-CABG group. This is not statistically different from the 39% in the normal perfusion group but was markedly reduced and less than normal in the anterior infarct group (18%, $P < 0.001$) compared with both of the other groups. Figure 7 shows examples of polar maps with related data for wall motion and wall thickening in patients from each of the groups.

Discussion

Findings

The phenomenon of reduced or even paradoxical interventricular septal motion after coronary artery bypass surgery is well known but poorly characterized by GSMPI but previously well reported using other non-nuclear imaging methods (4–6). In this study, we wanted to characterize the regional systolic wall motion and thickening of patients with previous CABG, studied for clinical indications, using gated stress myocardial perfusion scintigraphy. We did so to objectively characterize the pattern of gross visual abnormality often seen in such patients on gated studies. We also wanted to determine whether the pattern of systolic motion and thickening, previously described in this group when studied with echocardiography and other methods, was also evident

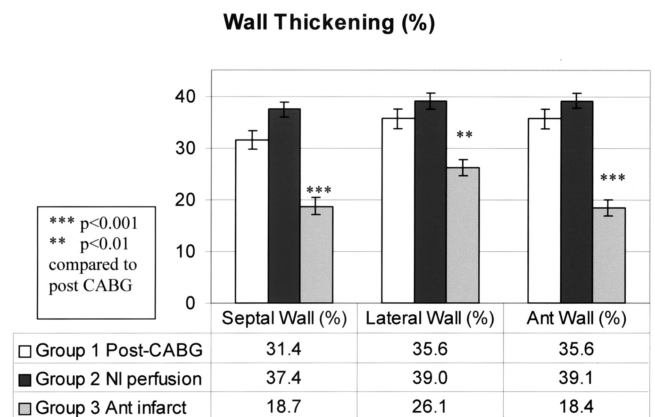


Fig. 6. Measurements of regional septal wall thickening are presented. Post-CABG patients (white bars) had preserved septal thickening compared with patients with normal perfusion (black bars), but septal thickening was significantly reduced in those with previous anterior infarction (gray bars). Lateral wall thickening was preserved in the post-CABG patients but was reduced in the patients with anterior infarction (group 3), in whom anterior wall thickening was also reduced.

on gated perfusion imaging, a newer method with more modest spatial resolution.

Our findings confirm, with a new method, gated perfusion scintigraphy, those documented and long recognized in the echocardiography and blood-pool scintigraphy literature relating apparent septal hypokinesis and augmented motion of the “posterior wall” after coronary bypass and other surgeries (4–6). Less complete descriptions indicate accompanying “anterior movement of the whole heart” and a transient reduction in septal thickening after coronary bypass (5). In 25 of the 30 post-CABG patients evaluated, the contraction pattern on gated SPECT-MPI showed hypokinesis of the septal wall with preservation of septal wall thickening, significantly increased lateral wall motion, and visual evidence of an anterior “swing” in epicardial motion. This is in contrast to the normal, concentric, inward epicardial and endocardial motion, identified in 29 of the 30 patients with normal perfusion on stress MPI, and the blunted, inward septal and anterior endocardial wall hypokinesis and reduced wall thickening seen in all of the patients with anterior infarction. Anterior WM and thickening in the anterior infarction patients (group 3) was clearly reduced compared with the other groups despite the inclusion of basal anterior segments, where contraction was generally preserved.

These findings closely parallel those on echocardiography, which have been attributed to the loosening of the heart in the pericardium during cardiac surgery with resulting anterior cardiac motion during systole. This anterior motion or “swing” results in visual blunting of

the posterior, septal contractions while augmenting posterior or lateral wall motion (7). The anterior motion of the entire heart, within the chest cavity during systolic contraction, cancels the posteriorly directed septal wall contraction, which may produce apparent septal dysnergy compared with other cardiac wall segments. Such an anterior epicardial swing could result in apparent septal hypokinesis and a hyperdynamic lateral wall in the setting of normal wall thickening on GSMPI, as identified in our post-CABG patients. These findings on echocardiography are said to vary with other factors, including baseline cardiac function, the effects of revascularization, and the extent of pericardial loosening but do not appear to relate to the regional state of perfusion (7). Instead, just as we found, perfusion-related changes were distributed in the pattern of vascular supply, where isolated septal abnormalities were rare (7). Resolution of these wall motion findings is said to occur with the formation of restrictive pericardial adhesions (10).

Another factor likely contributing to this characteristic appearance is the artifact introduced by our assessment of region wall motion from a fixed reference point where movement of the entire ventricle affects the quantitative assessment of regional wall motion (10). Alternatively, others have suggested that the apparent blunting of septal motion and associated findings could relate to abnormalities of ventricular contraction because of the contributing effect of hemodynamics, with variation of blood pressure resulting in postoperative changes (11). This cause, however, would not well explain the widely evident anterior cardiac swing seen in most of our post-CABG patients.

The pattern of systolic septal function seen on GSMPI in the post-CABG group demonstrated abnormal wall motion but preserved thickening. This combination, accompanied by preserved septal perfusion, supports abnormal motion based not on the presence of ischemia or scar but rather by other factors, perhaps as suggested in the echocardiography literature noted already, related to unrestrained movement in the absence of the pericardium in the post-CABG patient. The essential normal or nonpathologic origin of these findings is important in contrast to a decrement in wall motion on an ischemic basis. Recognition of this difference is important, given its possible confounding role when seeking improved wall motion due to revascularization of ischemic or "hibernating" myocardium (5,12,13).

Our findings confirm the ability of the newer method of gated perfusion imaging to recognize these documented findings and characterize them in terms of systolic wall motion and wall thickening. These findings point to a clinical role of the thickening measurement on gated MPI and show the added value of related perfu-

sion in the assessment of such wall motion abnormalities. They can alert many practitioners who may be new to or inexperienced with the evaluation of systolic left ventricular wall motion to this important and visually impressive finding.

Limitations

A chief limitation is that this study was based on the evaluation of selected patients who were broadly assessed according to group characteristics. Imperfect patient selection may explain reduced systolic motion of the lateral wall in patients with previous infarction compared with those with normal perfusion. Some of these patients with anterior infarction likely had multivessel disease and possibly infarction in other areas. The demographics of the patient groups studied were not thoroughly analyzed. Although similar, it would not be surprising to find differences between them. This would, however, have no bearing on the image patterns we have identified here.

To the extent that the selection of regional boundaries did not well parallel the coronary distribution, the between-group differences may have been blunted. This is may be especially true for anterior wall analysis. However, given the large differences seen between the established groups and the parallel between visual findings and quantitative segmental analysis, it is unlikely that a redefinition of segmental boundaries would change the findings in important ways. There was no attempt to differentiate anterior from posterior septal motion.

Other populations, as those with left bundle branch block or artificially paced rhythms, may also have abnormal patterns of systolic function different from normal and similar to that in the post-CABG group. Such populations were not included in this preliminary study. The AutoQuant software has been shown to be accurate and reproducible. Yet flaws in its calculation of LVEF, especially in small ventricles and possibly in the presence of left bundle branch block and with previous infarction or CABG, have been noted, and quantitation of wall thickening is based on an indirect measure. Although these issues await resolution, they likely have an insignificant influence on the results.

Clinical Implications

The apparent septal hypokinesis with normal septal wall thickening in the post-CABG group is compared with the septal and anterior wall hypokinesis and accompanying decreased wall thickening identified in the anterior infarct patients. These findings suggest that, in the evaluation of regional left ventricular function with GSMPI, examination of wall thickening in addition to the pattern of wall motion and perfusion is important in

patients after CABG. If not properly recognized, septal disease, ischemia, or infarction may be erroneously diagnosed in patients after CABG.

Conclusions

There is a characteristic contraction pattern of GSMPI in patients after CABG that is distinguished by apparent hypokinesis or akinesis of the septal wall, preservation of septal wall thickening, apparent increase in endocardial lateral wall motion, and the presence of an anterior epicardial swing. This specific pattern is seen commonly after CABG and is different from the contraction pattern seen in non-CABG patients with previous anterior infarction and those with normal findings perfusion studies. Its recognition is important for the correct interpretation of systolic left ventricular function with GSMPI.

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