Use of exercise thallium scintigraphy to assess extent of ischaemic myocardium in patients with left anterior descending artery disease

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SUMMARY In this study we attempted to examine the extent of jeopardised myocardium in patients with severe left anterior descending disease. The jeopardised myocardium was defined by the number of perfusion defects in the distribution of the left anterior descending artery depicted on exercise thallium-201 images. One segment in each of the three projections was considered to represent the distribution of this vessel, that is the anterolateral segments in the anterior projection, the anteroseptal segment in the left anterior oblique projection, and the anterior segment in the lateral projection.

We selected 45 patients with ≥ 70 per cent diameter narrowing of the left anterior descending artery and abnormal exercise images in the distribution of this vessel. There were 17 patients with isolated left anterior descending artery disease and 28 patients with multivessel disease including the left anterior descending artery. Fifteen patients (33%) had one perfusion defect (four patients with isolated disease and 11 patients with multivessel disease); 17 patients (38%) had two perfusion defects (six with isolated disease and 11 with multivessel disease); and 13 patients (29%) had three perfusion defects (seven with isolated disease and six with multivessel disease). The anteroseptal segment was the most common segment to become ischaemic during exercise and was abnormal in 40 of the 45 patients (89%). The anterior segment in 27 patients (60%) was next, and the anterolateral segment in 20 patients (44%) the least common. The number of defects did not correlate with the site of disease of the left anterior descending artery (proximal vs. distal to the first septal and first diagonal branches); the degree of left anterior descending narrowing (70 to 99% vs. total occlusion); collaterals; left ventricular asynergy; the presence of isolated left anterior descending disease in contrast with multivessel disease.

We conclude that variations in the extent of perfusion abnormalities seen in patients with disease of the left anterior descending artery suggest that these patients do not constitute a homogeneous group. Future studies designed to study the effect of medical or surgical treatment in such patients may be more meaningful if the extent of jeopardised myocardium, as defined by exercise thallium-201 imaging, is incorporated as a variable in group comparison. Our results also suggest that the number of perfusion defects do not predict the site of the lesion of the left anterior descending artery in relation to its first septal and diagonal branches.

There is still considerable controversy concerning the management of patients with severe disease of the left anterior descending artery. Several studies have reported no improvement in survival in such patients when treated with aortocoronary bypass surgery.¹⁻⁵ Several factors may contribute to this controversy: the location of the narrowing in rela-

tion to the septal perforator and the diagonal branches; the degree of narrowing; and the distribution of this vessel and thus, conceivably, the amount of myocardium nourished by this vessel. We have previously suggested that there is considerable variation in the anatomical distribution of this vessel and in the number of its secondary branches.⁶

Exercise myocardial thallium-201 perfusion imaging can be used to define the diseased vessel or

vessels according to the location of perfusion abnormality in the various projections used for imaging.⁷⁻⁹ The purpose of this study is to examine the extent of jeopardised myocardium in patients with left anterior descending artery disease and to define the factors that could affect it.

Subjects and methods

We selected the patients in this study using the following criteria. First, each patient had exercise-induced perfusion defect(s) in the distribution of the left anterior descending artery (Fig. 1). The defect may involve one or more of the three segments in the distribution of this vessel as seen in the three projections: anterior, left anterior oblique, and left lateral projection. Some of the patients with multivessel disease in this study had, in addition, perfusion defects in the distribution of the remaining two vessels (left circumflex or right coronary artery), but each patient also had perfusion defect(s) in the distribution of the left anterior descending artery. Second, each patient had 70 per cent or greater luminal narrowing of the left anterior descending artery, as shown by coronary arteriography performed within six months of the exercise study. Third, there was no evidence of anterior transmural infarction on the electrocardiogram.

Patients were not included in this study if the following circumstances prevailed. (1) If the exercise thallium-201 images failed to show perfusion defects in the distribution of the left anterior descending artery despite significant disease of this vessel (these patients had perfusion defects in the distribution of the right coronary artery or the left circumflex artery)¹⁰ or (2) if the exercise thallium-201 images showed a perfusion defect in the distribution of the left anterior descending artery but there was no significant disease in this vessel: we have encountered one such patient with mitral

valve prolapse syndrome.¹¹ In our laboratory false positive exercise thallium-201 images are seen in approximately 5 per cent of patients with no significant coronary artery disease. These defects generally involve the inferior segment in the anterior projection; (3) if the exercise images were normal in the presence of isolated disease of the left anterior descending artery (we have also encountered one patient in this category).

Our technique for exercise thallium-201 imaging has been described elsewhere (Iskandrian *et al.*, 1980).¹⁰⁻¹³ In brief, each patient was exercised in the fasting state; we used the multistage Bruce protocol for treadmill exercise testing (stages of three minutes' duration, each of a different exercise intensity level determined by changing the speed and the grade of the motor-driven treadmill). At peak exercise 2 mCi thallium-201 was injected intravenously and flushed with dextrose and water. The end points of exercise were as follows: 2 mm or more ST segment depression of the horizontal or downsloping variety with or without angina; extreme fatigue; leg weakness or shortness of breath; hypotension; dizziness; frequent arrhythmias; or attainment of at least 85 per cent of the maximum predicted heart rate for age and condition.

We performed exercise imaging within 15 minutes of injection using a commercially available scintillation camera with a high-resolution parallel-hole collimator 4 cm thick. Exercise images were performed in the anterior, left anterior oblique, and lateral projections. Four-hour delayed images were performed in the projection or projections showing perfusion abnormalities during stress. The count density was kept constant in the delayed images in each patient.

We performed left and right heart catheterisation, left ventriculography, and selective coronary arteriography in each patient using standard techniques.

Fig. 1 Schematic presentation of the distribution of the left anterior descending artery, as seen in three projections of exercise thallium-201 images. The lightly shaded areas represent the distribution of the left anterior descending artery.

The degree of diameter narrowing of the left anterior descending artery and the location of the narrowing in relation to the major septal and diagonal branches were determined in each patient. Because of the known observer variability in assessing the obstructive disease, the degree of narrowing was divided into either complete occlusion or subtotal occlusion (that is 70 to 99% narrowing). The disease of the left anterior descending artery was considered proximal if the site of the narrowing was before the first septal or first diagonal vessels (or both), or there were separate narrowings involving one or both of these branches in addition to the disease of the left anterior descending artery. The left ventriculogram was assessed for wall motion abnormalities. Collaterals were judged to be present when there was delayed contrast filling of the diseased left anterior descending artery beyond the narrowing. The consensus of two independent observers was used in the analysis of data of the exercise images and of angiography. Statistical analysis was performed with the χ^2 test and Student's *t* test.

Results

There were 36 men and 9 women between 30 and 75 years of age (mean age, 50 years). Seventeen patients had isolated disease of the left anterior descending artery; 28 patients had disease of one or two additional vessels in addition to the left anterior descending artery. There were no significant differences in the resting heart rate between patients with one defect (82 ± 17 beats/min); two defects (79 ± 13 beats/min); and three defects (71 ± 15 beats/min). Similarly, there were no significant differences in the resting systolic blood pressure (127 ± 14 mmHg) in patients with one defect; (129 ± 14 mmHg) in

patients with two defects; and (119 ± 16 mmHg) in patients with three defects. Eight of the 15 patients (53%) with one defect exercised to stage III or more according to a standard Bruce protocol, compared with 10 of 17 patients (59%) with two defects and seven of 13 patients (54%) with three defects ($P = \text{NS}$). The remaining patients stopped exercising in stages I or II. The results of exercise testing are shown in Table 1. There were no statistically significant differences between patients with one defect compared with those with two or three defects in the maximum heart rate during exercise, the duration of exercise, the functional aerobic impairment, the presence of angina during exercise, and the results of the exercise electrocardiogram. On the other hand, the maximum systolic pressure during exercise and the double product (heart rate $\times$ systolic blood pressure) during exercise were higher in patients with one defect compared with those with multiple defects.

The large number of patients with non-diagnostic exercise electrocardiogram in this study is the result of the presence of resting ST abnormalities or failure to achieve at least 85 per cent of the maximum predicted heart rate.

There was a total of 88 perfusion defects in the three projections in these patients. The antero-septal segment was most commonly involved followed by the anterior segment and then the anterolateral segment (Table 2). Of the patients with single perfusion defects, the antero-septal area was involved in 10 patients, the anterolateral area in four patients, and the anterior area in only one patient. Of the patients with isolated disease of the left anterior descending artery, four had one perfusion defect, six had two perfusion defects, and seven had three perfusion defects. Of the patients with multivessel

Table 1 Results of exercise testing

	15 patients with one defect			17 patients with two defects			13 patients with three defects			<i>p</i> value
	No.	Per cent	Mean $\pm$ SD	No.	Per cent	Mean $\pm$ SD	No.	Per cent	Mean $\pm$ SD	
Maximum heart rate (beats/min)			150 $\pm$ 19			144 $\pm$ 32			139 $\pm$ 26	NS
Maximum systolic BP (mmHg)			163 $\pm$ 31			161 $\pm$ 24			137 $\pm$ 30	<0.05 (one and two vs. three)
Double product (HR $\times$ systolic BP $\times 10^3$ mmHg beats/min)			25.3 $\pm$ 6			23.5 $\pm$ 8			19.2 $\pm$ 6	<0.02 (one vs. three)
Exercise duration (minutes)			6.7 $\pm$ 3.5			6.8 $\pm$ 3.1			7.3 $\pm$ 3.4	NS
Functional aerobic impairment (%)			+23 $\pm$ 28			+21 $\pm$ 31			+17 $\pm$ 33	NS
Angina during exercise	9	(60)		9	(53)		5	(39)		
Exercise electrocardiogram										
Positive	6	(40)		6	(35)		4	(31)		
Negative	3	(20)		3	(18)		2	(15)		
Nondiagnostic	6	(40)		8	(47)		7	(54)		

Abbreviation: BP, blood pressure.

Table 2 Location of perfusion defects in 45 patients with severe disease of left anterior descending artery

	No. of patients with exercise defects		
	Anterolateral segment	Anteroseptal segment	Anterior segment
Isolated left anterior descending artery (n = 17)	9	15	13
Multivessel disease (n = 28)	12	25	14
Total	21	40	27

disease, 11 had isolated defects, 11 had two defects, and six had three defects (Fig. 2).

The effects of the degree of narrowing, the site of narrowing, extent of the coronary artery disease, collaterals, and left ventricular asynergy on the number of perfusion defects are shown in Table 3. As can be seen, 18 of the 23 patients with disease located proximal to the first septal and diagonal branches, and eight of the 12 patients with distal disease had multiple defects not significant (NS).

Similar conclusions can be drawn when the lesion is considered in relation to the first septal or first diagonal branch separately. Thus, of the 31 patients with disease proximal to the diagonal, 22 had two or three defects, and of the remaining 14 patients with distal disease, eight had multiple defects (NS); 18 of 25 patients with disease proximal to the first septal branch had multiple defects, compared with 12 of 20 patients with distal disease (NS).

The extent of coronary artery disease did not appear to affect the number of perfusion defects since only four of the 17 patients with isolated left anterior descending artery disease had an isolated

Fig. 2 Exercise thallium-201 images in anterior, left anterior oblique (LAO), and lateral projections. (A) Isolated defect of the anterolateral segment (arrow). (B) Two defects (arrows). There is in addition an apical defect seen in the anterior projection. (C) Three defects (arrows). There is in addition a defect involving the inferior surface.

defect; the remaining 13 had two or three defects. On the other hand, 17 of 28 patients with multivessel disease had multiple defects. The degree of left anterior descending artery narrowing did not appear to affect the extent of perfusion abnormalities since 11 of the 15 patients with isolated perfusion defects had 70 to 99 per cent narrowing and in the remaining four there was total occlusion of the vessel. In 23 of the 30 patients with two or more defects the narrowing of the left anterior descending

Table 3 Effects of degree of narrowing of left anterior descending artery, location of narrowing, extent of coronary artery disease, collaterals, and left ventricular function on numbers of exercise perfusion defects

	No. of patients	One perfusion defect	Two or three perfusion defects
Site of disease			
Before S ₁	25	7 (28)	18 (72)
After S ₁	20	8 (40)*	12 (60)*
Before D ₁	31	9 (29)	22 (71)
After D ₁	14	6 (43)*	8 (57)*
Before S ₁ , D ₁	23	5 (22)	18 (78)
After S ₁ , D ₁	12	4 (33)*	8 (67)*
Coronary artery disease			
Isolated LAD only	17	4 (24)	13 (76)
Multivessel disease	28	11 (39)*	17 (61)*
Degree of LAD narrowing			
70 to 99 per cent	34	11 (32)	23 (68)
Total occlusion	11	4 (36)*	7 (64)*
Collaterals: Present	21	7 (33)	14 (67)
Absent	24	8 (33)*	16 (67)*
Left ventriculogram			
Normal or hypokinetic	38	13 (34)	25 (66)
Akinesia or dyskinesia	7	2 (29)*	5 (71)*

Numbers in parentheses represent percentages. S₁, first septal; D₁, first diagonal; LAD, left anterior descending artery; NS, not significant; *, the differences were not significant.

was 70 to 99 per cent, and in the remaining seven patients there was total occlusion. Collaterals were present equally in patients with one or multiple defects, and the left ventricular wall motion abnormalities were also equally distributed.

The correlation between extent of coronary artery disease, the degree of left anterior descending artery narrowing, left ventricular asynergy, collaterals to the site of the disease (proximal or distal to the first septal perforator), and number of perfusion defects is shown in Table 4. As can be seen, 72 per cent of the patients with disease proximal to the first septal perforator had two or three perfusion defects

compared with 60 per cent of patients with distal disease (NS). The degree of narrowing, extent of coronary artery disease, left ventricular asynergy, and collaterals were equally distributed in the two groups.

Table 5 shows a similar correlation analysed in relation to the location of the disease of the left anterior descending artery in relation to the first diagonal branch. Again, 71 per cent of the patients with disease proximal to the first diagonal had two or three perfusion defects compared with 57 per cent of patients with distal disease (NS).

Table 6 shows the location, extent, degree of

Table 4 Correlation between degree of left anterior descending artery narrowing, extent of coronary artery disease, left ventriculogram, collaterals, and number of perfusion defects in relation to site of narrowing before and after first septal perforator

	Site of lesion of LAD	
	Before first septal (n = 25)	After first septal (n = 20)
Coronary artery disease		
Isolated LAD	6 (24)	11 (55)
Multivessel disease	19 (76)	9 (45)
Degree of narrowing of LAD		
70 to 99 per cent	18 (72)	16 (80)
Total occlusion	7 (28)	4 (20)
Left ventriculogram		
Normal or hypokinesia	22 (88)	16 (80)
Akinesia or dyskinesia	3 (12)	4 (20)
Collaterals	13 (52)	8 (40)
No. of perfusion defects		
One	7 (28)	8 (40)
Two or three	18 (72)	12 (60)

Numbers in parentheses represent percentages. The differences were not significant. LAD, left anterior descending artery.

Table 5 Correlation between degree of narrowing of left anterior descending, extent of coronary artery disease, left ventriculogram, collaterals, and number of perfusion defects, and site of narrowing of left anterior descending in relation to first diagonal

	Site of lesion of LAD	
	Before first diagonal (n = 31)	After first diagonal (n = 14)
Coronary artery disease		
Isolated LAD	11 (35)	6 (42)
Multivessel disease	20 (65)	8 (57)
Degree of LAD narrowing		
70 to 99 per cent	25 (81)	9 (64)
Total occlusion	6 (19)	5 (36)
Left ventriculogram		
Normal or hypokinesia	26 (84)	12 (86)
Akinesia or dyskinesia	5 (16)	2 (14)
Collaterals	15 (48)	6 (43)
No. of perfusion defects		
One	9 (29)	6 (43)
Two or three	22 (71)	8 (57)

Numbers in parentheses represent percentages. The differences were not significant. LAD, left anterior descending artery.

Table 6 Correlation between degree of narrowing of left anterior descending, site of narrowing, extent of coronary artery disease, collaterals and left ventriculogram on site of exercise-induced perfusion defect

	Perfusion defect of anterolateral segment		Perfusion defect of antero-septal segment		Perfusion defect of anterior segment	
	Present (n = 21)	Absent (n = 24)	Present (n = 40)	Absent (n = 5)	Present (n = 27)	Absent (n = 18)
Site of LAD disease						
Before S ₁	10 (48)	15 (63)	23 (58)	2 (40)	17 (63)	8 (44)
After S ₁	11 (52)	9 (37)	17 (42)	3 (60)	10 (37)	10 (56)
Before D ₁	14 (67)	17 (71)	28 (70)	3 (60)	21 (78)	10 (56)
After D ₁	7 (33)	7 (29)	12 (30)	2 (40)	6 (22)	8 (44)
Coronary artery disease						
Isolated LAD	9 (43)	8 (33)	15 (38)	2 (40)	13 (48)	4 (22)
Multivessel disease	12 (57)	16 (67)	25 (62)	3 (60)	14 (52)	14 (73)
Degree of narrowing						
70 to 99 per cent	14 (67)	20 (83)	31 (78)	3 (60)	22 (81)	12 (67)
Total occlusion	7 (33)	4 (17)	9 (22)	2 (40)	5 (19)	6 (33)
Collaterals	12 (57)	9 (38)	18 (45)	3 (60)	12 (44)	9 (50)
Left ventriculogram						
Normal or hypokinesia	18 (86)	20 (83)	33 (83)	5 (100)	22 (81)	16 (89)
Akinesia or dyskinesia	3 (14)	4 (17)	7 (17)	0	5 (19)	2 (11)

Numbers in parentheses represent percentages. Differences are not significant. LAD, left anterior descending artery; S₁, first septal; D₁, first diagonal.

narrowing of the left anterior descending, and left ventricular asynergy in patients with or without perfusion abnormalities in each of the three segments. The particular segments involved with perfusion abnormality did not predict the location, the degree of narrowing or the extent of coronary artery disease in these patients. It is interesting to note that though the anterosseptal segment was the most common area to be ischaemic, this segment did not have localising accuracy in determining the site of the disease in relation to the first septal perforator.

Discussion

Our results in this selected group of patients indicate that there is a wide spectrum of exercise-induced perfusion abnormalities in patients with significant disease of the left anterior descending artery. Approximately one-third of the patients had isolated perfusion defects, one-third had two perfusion defects, and one-third had three perfusion defects, as seen on exercise thallium-201 imaging. It is conceivable that there is some overlap between the representative anatomical segments in the three projections. This overlap may depend not only on the size, shape, and position of the heart but also on the precise degree of the assigned projections.

It is unlikely, however, that the separation of the patients into three groups is fortuitous on the basis of overlap alone. The left ventriculograms in patients with disease of the left anterior descending artery show a range from normal to severe dysfunction with aneurysm formation or large akinetic-dyskinetic segments (or both).¹⁴ This variation in the extent of jeopardised myocardium is not unexpected given the variation in distribution of the left anterior descending artery and in the number of its secondary branches and thus, perhaps, in the amount of the myocardium nourished by this vessel.⁶

The extent of coronary artery disease, that is the presence of isolated left anterior descending artery disease versus two- or three-vessel disease (including the left anterior descending artery); the degree of narrowing (that is 70 to 99 per cent vs. total occlusion); collaterals; the degree of left ventricular asynergy assessed by left ventriculography; the location of the narrowing in relation to the first septal perforator and the first diagonal branches did not appear to influence the extent of perfusion defects. Two important factors should be considered in relation to the number of the defects in our patients. First, the level of exercise, and second, the resolution of the scintillation camera. McLaughlin *et al.*¹⁵ have shown, in a group of 12

patients with coronary artery disease studied at two levels of exercise, that only 16 of 30 (53%) of the ischaemic defects present at maximal exercise were seen during light exercise. In our study there were no significant differences between patients with one defect compared with those with multiple defects in the intensity of exercise (duration or stage according to a standard Bruce protocol); peak heart rate during exercise; the presence of angina during exercise; and the presence of positive exercise electrocardiograms during exercise. In fact, the maximum systolic blood pressure and the double product during exercise were significantly higher in patients with one defect compared with those with two or three defects (Table 1). These findings thus indicate that the level of exercise may not be the only reason for the difference in the size of the ischaemic myocardium in our patients. In patients with multi-vessel disease, it may be assumed that patients may end the exercise because of ischaemia in the distribution of vessels other than the left anterior descending artery. This appears less likely since we have selected for this study only the patients who developed ischaemia in the distribution of the left anterior descending artery, though a few of them had ischaemia in the distribution of the remaining vessels as well. The second problem is related to the resolution of the scintillation camera. It is well known that large defects (more than 2.5 cm in diameter) can be visualised when imaged in any projection but small defects can only be visualised if imaged tangentially or *en face*. Thus, it is conceivable that some small defects may not have been visualised because of the resolution of the camera, overlap with adjacent normal myocardium, or because of improper projection. The effect of missing small defects should not influence the results substantially since we used the same scintillation camera and obtained comparable projections in all patients.

The clinical importance of the variation in the extent of perfusion abnormalities in patients with left anterior descending artery disease could not be answered from our study, but it may be important to determine whether patients with multiple defects have a higher mortality rate or a higher risk of developing severe left ventricular dysfunction in the event of infarction than patients with isolated defects.

The results from this study may also explain the conflicting conclusions obtained from the comparison of medical versus surgical treatment in these patients. It appears appropriate to us that future studies addressing this problem should incorporate the results of exercise thallium-201 imaging as a variable.

Our study also suggests that the antero-septal segment seen in the left anterior oblique projection is the segment most commonly ischaemic in patients with left anterior descending artery disease. This is not unexpected since in this projection there is a variable overlap of the anterior and septal regions, both areas being served by the left anterior descending artery; therefore, the perfusion abnormality in this area may be better projected. This finding appears to be important, since laboratories involved in exercise-gated left ventricular nuclear angiograms (generally performed in a modified left anterior oblique projection) can predict left anterior descending artery disease with reasonable assurance if wall motion abnormalities are noted in the antero-septal area, which is well defined in this projection.

Abnormalities in the perfusion of the antero-septal area, however, did not correlate with the location of the left anterior descending artery disease in relation to the first septal perforator; there are two main reasons for these findings: the first septal perforator may, in fact, not be important; that is, septal perfusion may depend on the total number of septal perforators or, on the contribution of the right coronary artery to septal perfusion via the posterior septal perforator branches.

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