

## Exercise thallium-201 myocardial scintigraphy in the follow-up of aortocoronary bypass graft surgery

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**SUMMARY** The predictive accuracy of exercise thallium-201 ( $^{201}\text{Tl}$ ) myocardial scintigraphy in the evaluation of aortocoronary bypass graft surgery was assessed in 48 patients undergoing angiographic investigation 15 months (mean time) after myocardial revascularisation.  $^{201}\text{Tl}$  scintigrams detected 61 out of 77 (79%) patent grafts but only 21 out of 42 (50%) occluded grafts, though, for grafts supplying non-infarcted myocardium, the predictive accuracy of graft patency and graft occlusion was 85 per cent and 81 per cent, respectively. Stress electrocardiography failed to detect 15 out of 21 patients with scintigraphic evidence of regional myocardial ischaemia. Residual ischaemia in the proximal left anterior descending coronary distribution was commonly detected in  $^{201}\text{Tl}$  scintigrams despite a patent, well-functioning left anterior descending graft to the distal coronary segment. Additional residual ischaemia attributable to ungrafted coronary disease was detected by scintigraphy in 32 (67%) patients and most commonly occurred in the distribution of the diagonal branch of the left anterior descending especially in the presence of a patent distal left anterior descending graft. Thus, independent grafts to the diagonal branch of the left anterior descending are recommended at the time of aortocoronary bypass graft surgery.

After aortocoronary bypass graft surgery approximately 20 per cent of patients may need investigation with coronary and graft arteriography to evaluate persistent or recurrent chest pain often not typical of angina pectoris. Though freedom from angina after surgery is positively correlated with vein graft patency<sup>1,2</sup> it does not follow that recurrent angina is always caused by vein graft occlusion, for Di Luzio *et al.*<sup>3</sup> found that patients with occluded grafts may also be free from pain. Thus, chest pain after coronary bypass graft surgery must sometimes depend upon factors other than graft occlusion, including:

- (a) progression of native coronary disease in grafted and ungrafted vessels;
- (b) functional inadequacy of patent grafts caused by graft stenosis or poor graft run-off;
- (c) deterioration of left ventricular function.

In addition, chest pain not directly related to the vascular status of myocardium (for example, non-union of the sternum) may resemble angina pectoris and provoke unnecessary arteriography.

A minimally invasive technique, such as exercise thallium-201 ( $^{201}\text{Tl}$ ) myocardial scintigraphy, which could sensitively distinguish regions of persistent myocardial ischaemia from necrotic and normal myocardium,<sup>4,5</sup> would be a valuable asset in the

preliminary analysis of patients with chest pain after coronary artery surgery, as those patients in whom regional myocardial ischaemia could not be demonstrated might be precluded from further invasive investigation.

This study had two objectives: firstly, to correlate postoperative coronary and graft arteriograms with exercise  $^{201}\text{Tl}$  myocardial scintigrams in patients with previous aortocoronary bypass surgery in order to define scintigraphic criteria which could be used to predict the status of individual coronary bypass grafts; secondly, to analyse the contributions of revascularisation and necrosis to the mechanism of pain relief after surgical revascularisation of the myocardium.

### Patients and methods

Forty-eight patients (44 men and four women; mean age 51, age range 32 to 64 years) who had previous aortocoronary saphenous vein graft surgery were reinvestigated by selective coronary and graft arteriography. Eighteen patients had typical angina pectoris closely resembling their preoperative pain (group A), 13 patients had praecordial pain considered atypical of angina pectoris but which also resembled their preoperative pain (group B), and 17 patients had no chest pain at all (group C).

In the latter group, 10 patients were completely symptomless while the remainder were limited by breathlessness or undue fatigue.

Four patients had a single graft, 18 patients had two grafts, 25 patients had three grafts, and one patient had four grafts (average 2.5 grafts per patient). Forty-six grafts were placed to the left anterior descending coronary artery or its main diagonal branch, 41 to the right coronary artery, and 32 to the left circumflex vessel or its obtuse marginal branch. Autologous saphenous veins were used as grafts and anastomosed to coronary vessels with at least 50 per cent stenosis of the proximal lumen. In addition to coronary artery surgery one patient had an aneurysmectomy and one patient had an aneurysm plicated, but no other procedures, such as valve replacement, were performed.

#### CORONARY ARTERIOGRAPHY

Contrast cineangiography of the native and graft circulation was performed using the Sones or Judkins technique. All patent grafts were demonstrated in at least two views. Occluded grafts were recognised by selective opacification of a graft stump at the aortic anastomosis. If a graft could not be visualised by selective catheterisation a large bolus of contrast medium was rapidly injected into the aortic root. Grafts not opacified by either method were classified as 'not demonstrated' and presumed occluded. All coronary arteriograms were reviewed independently by two observers in ignorance of corresponding myocardial scintigrams. The state of the native coronary vessels and the integrity of the venous grafts were noted. Particular attention was paid to graft stenoses and whether the degree of graft run-off, assessed by the size of the distal vessels and their promptness to opacify, was adequate. Graft run-off was graded as good, moderate, or poor. Narrowing of a coronary artery or graft greater than 50 per cent of the lumen diameter was considered haemodynamically significant.

Although assessment of the arteriographic data was visual and necessarily subjective, the consensus of opinion of at least three observers was always obtained. The mean time between surgery and follow-up arteriography was 14.9 months (range six to 72 months) and the mean time between arteriography and scintigraphy was 48.6 days (range zero days to 13 months). Scintigraphy and arteriography were separated by less than a month in most patients (36).

#### EXERCISE MYOCARDIAL SCINTIGRAPHY

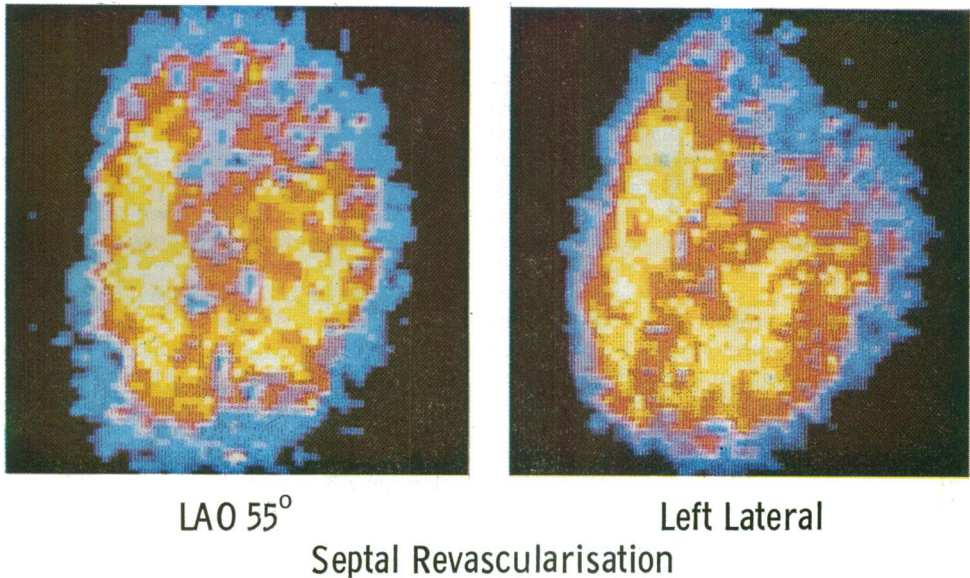
Patients were exercised in the upright position on

a bicycle ergometer, monitoring the V5 lead of the electrocardiogram, until the onset of limiting symptoms. An exercise electrocardiogram was regarded positive for ischaemia if 1 mm or more ST segment depression was seen 0.08 second after the J point in three consecutive beats; 2 mCi of  $^{201}\text{Tl}$  was given through an indwelling intravenous cannula at an exercise end-point which was maintained for approximately one-and-a-half to two minutes after administration of tracer. After a 10-minute recovery period myocardial imaging was performed with subjects in the supine position using an Ohio Nuclear series 100 scintillation camera and a high-sensitivity parallel-hole collimator. Four views of the myocardium were routinely acquired; viz: anterior, left anterior oblique (LAO) 45°, LAO 55°, and a full left lateral projection; 200 000 counts were collected in each image using a 20 per cent window centred on the 72.5 keV mercury  $x$ -ray peak emitted by  $^{201}\text{Tl}$  decay. Each image was acquired in four to eight minutes on transparency film and simultaneously recorded in a 64 × 64 matrix on magnetic disc using a dedicated minicomputer (DEC Gamma II) interfaced to the gamma camera. Digital images were displayed on a TV monitor in 16 grades of colour, linearly related to image intensity, and enhanced by a 50 per cent background erase. Each image was carefully compared with a group of normal myocardial  $^{201}\text{Tl}$  scintigrams previously acquired in our laboratory from patients without evidence of heart disease, including asymptomatic healthy normal volunteers. This comparison was facilitated by using a computer 'area-of-interest' analysis. Segmental areas of interest were outlined on the myocardial images and then expressed quantitatively as average counts per element of the digital matrix. Each segment corresponded approximately to 14 per cent of the myocardial circumference. Segments which fell below our established normal range were regarded as myocardial defects. We have found this semi-quantitative technique sensitive and reproducible; reliance solely on visual interpretation of the images is thus minimal. If necessary, patients had repeat delayed images of the myocardium performed in selected views several hours later in order to distinguish regions of severe myocardial ischaemia, which undergo reperfusion, from the fixed uptake defects of myocardial necrosis.

#### SCINTIGRAPHIC CRITERIA OF GRAFT INTEGRITY

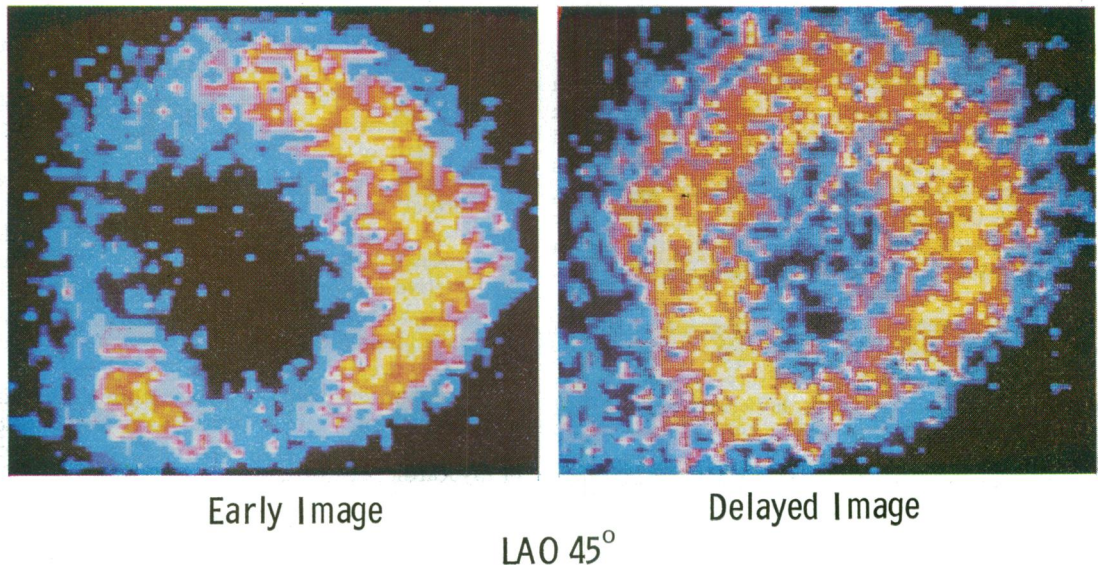
The functional adequacy of venous bypass grafts, and thus, indirectly, whether grafts were likely to be patent or occluded, was determined by using the following *a priori* criteria based on the appearance

## GRAFT PATENCY



**Fig. 1**  $^{201}\text{Tl}$  myocardial scintigram LAO 55°. Normal tracer uptake in the septum is preserved by a patent left anterior descending graft despite total proximal occlusion of the artery (left). Left lateral view. A separate graft to the left anterior descending diagonal branch in the same patient accounts for normal tracer uptake in the anterolateral wall (right).

## GRAFT CLOSURE



**Fig. 2**  $^{201}\text{Tl}$  myocardial scintigram LAO 45°. A severe deficit of tracer in the septum is seen in the early post-exercise image (left) which is not evident in the delayed image (right) because of tracer redistribution. This septal ischaemia was caused by significant proximal left anterior descending disease and unrelieved by a severely stenosed but patent left anterior descending graft.

of tracer in appropriate regions of the myocardial scintigram.

(a) Exercise-induced tracer defects resulting from severe coronary disease would not be expected to appear in corresponding regions of the  $^{201}\text{Tl}$  scintigram if patent venous grafts provided an alternative route for tracer. Thus, the absence of a tracer defect in the territory of a previously grafted coronary artery with severe proximal disease is regarded in this study as a scintigraphic indication of graft patency (Fig. 1).

(b) Conversely, venous grafts which are stenosed, occluded, or have poor run-off will allow underlying coronary disease to cause ischaemic tracer defects in the myocardial scintigram. Thus, tracer deficit in the territory of a previously grafted, severely diseased coronary artery is regarded as a sign of graft insufficiency (Fig. 2). Clearly, the anatomical pathology of functionally inadequate grafts cannot be distinguished by myocardial scintigraphy alone but must be determined by graft angiography.

(c) Finally, judgement cannot be made about graft integrity if a graft subtends necrotic myocardium. Uptake defects attributable to necrosis are permanent and therefore preclude normal uptake of tracer or reversible ischaemic defects despite restoration of myocardial perfusion through patent grafts. In areas of necrosis, graft occlusion or graft

patency may be equally likely.<sup>6</sup> Grafts subtending these regions must, therefore, be classified as equivocal or of indeterminate patency (Fig. 3). In addition, grafts subtending suspicious uptake defects seen in only one view were also classified as equivocal.

#### SCINTIGRAPHIC LOCALISATION OF GRAFTS

These criteria were used to analyse individual coronary bypass grafts whose myocardial territories of supply were identified by our knowledge of the scintigraphic anatomy of ungrafted coronary disease.<sup>7</sup> The regions relevant to each graft are seen in Fig. 4, and listed below.

##### *Left anterior descending graft*

Anterior view; anterior wall and apex.

Left oblique view; septum.

Left lateral view; anterolateral wall and apex.

##### *Right coronary graft*

Anterior view; posteroinferior wall.

Left oblique view; inferior wall.

Left lateral view; no contribution.

##### *Left circumflex graft*

Anterior view; no contribution.

Left oblique view; posterolateral wall.

Left lateral view; inferolateral wall.

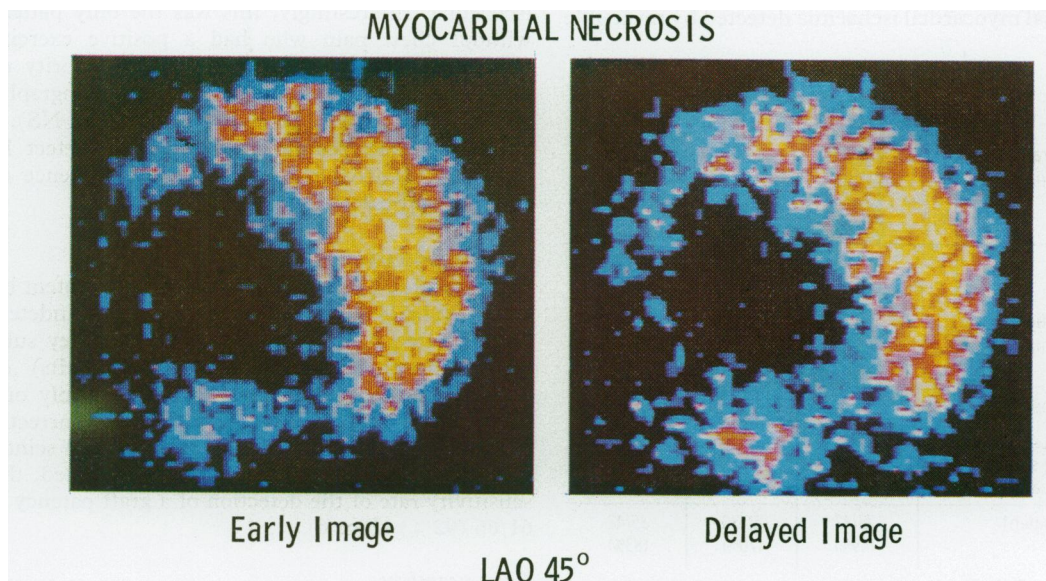


Fig. 3  $^{201}\text{Tl}$  myocardial scintigram LAO 45°. The severe septal tracer defect seen in the left image persists in the delayed image (right) indicating necrosis of the septum. In this situation patent or occluded left anterior descending grafts are not distinguishable.

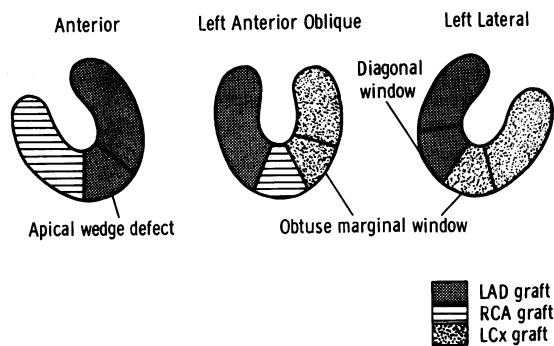


Fig. 4 Diagrams of  $^{201}\text{Tl}$  myocardial scintigrams in three views indicating the territories supplied by coronary grafts.

In addition to quantification all scintigrams were reviewed independently by at least two observers without knowledge of the corresponding coronary angiograms. Observer agreement in the interpretation of scintigrams by visual and quantitative methods was excellent (> 90%) but consensus of a third observer was sought when necessary.

Statistical comparison of data was performed using the  $\chi^2$  test with Yates correction or the Fisher exact test.

## Results

Fig. 5 shows the relation between chest pain and regional myocardial ischaemia detected by reversible

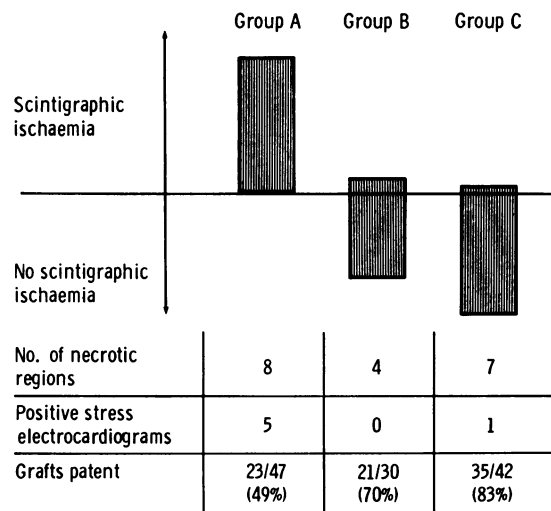


Fig. 5 Relation between chest pain and scintigraphic evidence of myocardial ischaemia in three groups of patients. Group A had typical angina pectoris; group B had atypical angina pectoris; group C had no chest pain.

tracer defects appearing in the  $^{201}\text{Tl}$  scintigram. All 18 patients with typical angina pectoris (group A) had at least one tracer defect in their scintigrams attributable to myocardial ischaemia. In comparison, only five of these patients had a positive stress electrocardiogram. Ischaemic defects were found in regions of the myocardium subtended by occluded grafts in 11 patients and functionally inadequate grafts in six patients (two grafts stenosed, four grafts with poor distal run-off). In one patient tracer deficit was caused by severe ungrafted coronary disease. Angiography showed 24 out of 47 (51%) grafts occluded in this group.

In contrast, only two out of 13 patients with atypical chest pain (group B) had evidence of myocardial ischaemia in the  $^{201}\text{Tl}$  scintigram ( $P < 0.0005$ ). Both patients had reversible uptake defects in the left anterior descending territory. Left anterior descending graft occlusion was confirmed by angiography. The remaining patients had normal tracer uptake or fixed defects of tracer attributable to myocardial necrosis. No patient had a positive stress electrocardiogram. In this group nine out of 30 (30%) grafts were occluded on angiography (group A and B, NS).

Finally, 16 out of 17 patients without chest pain (group C) had no evidence of myocardial ischaemia in the  $^{201}\text{Tl}$  scintigram. One patient had a reversible uptake defect in a small area of the posterolateral wall corresponding to an occluded obtuse marginal graft. Presumably this was a region of painless ischaemia. Interestingly, this was the only patient without chest pain who had a positive exercise electrocardiogram. In this group the majority of grafts 35/42 (83%) were patent at angiography (group A and C,  $P < 0.005$ ; group B and C, NS).

Exercise electrocardiography failed to detect 15 out of 21 patients with scintigraphic evidence of regional myocardial ischaemia.

## PATENT GRAFTS

Of 119 grafts, 77 (65%) were found to be patent by contrast angiography. Eleven grafts were of indeterminate patency by scintigraphy because they subtended necrotic myocardium (seven grafts) or because suspicious defects were seen in only one view (four grafts). Sixty-one grafts were correctly predicted patent from the  $^{201}\text{Tl}$  myocardial scintigram. If the 11 equivocal grafts are excluded, the sensitivity rate of the detection of a graft patency is 61/66 (92%) (Fig. 6).

## False negatives

Sixteen patient grafts were incorrectly predicted occluded from the myocardial scintigram, that is apparent false negatives for graft patency. Though

patent, five grafts were functionally inadequate because of significant graft stenosis (two grafts) or poor run-off (three grafts), thus mimicking graft occlusion. In this false negative group only one patent graft had angiographically good distal run-off compared with 47 patent grafts with good distal run-off in the true positive group ( $P < 0.001$ ).

Eleven occluded grafts were incorrectly predicted patent by  $^{201}\text{Tl}$  scintigraphy and thus classified as false positives for graft patency or, alternatively, as the false negatives for graft occlusion discussed below. Thus, the predictive accuracy of  $^{201}\text{Tl}$  scintigraphy in the determination of graft patency is  $61/61 + 11$  (85%).

#### OCCLUDED GRAFTS

At angiography, 42 (20 graft stumps and 22 grafts not shown) out of 119 (35%) grafts were presumed occluded. Twenty-one of these grafts were correctly predicted occluded from analysis of appropriate regions of the myocardial scintigram. Ten occluded grafts were classified equivocal by scintigraphy because they subtended necrotic areas (eight grafts) or were associated with moderate tracer reduction seen in only one view (two grafts). The latter two grafts supplied the left anterior descending artery which, in one case, showed considerable hypertrophy of its first septal branch, serving as a major collateral channel; in the other case, the native left anterior descending was only moderately diseased (60% stenosis). If these 10 occluded grafts are excluded, sensitivity rate for graft occlusion is  $21/32$  (66%) (Fig. 6).

#### False negatives

In contrast, 21 occluded grafts were predicted patent by  $^{201}\text{Tl}$  scintigraphy because no tracer defects were seen in regions corresponding to the territory of graft supply, that is apparent false

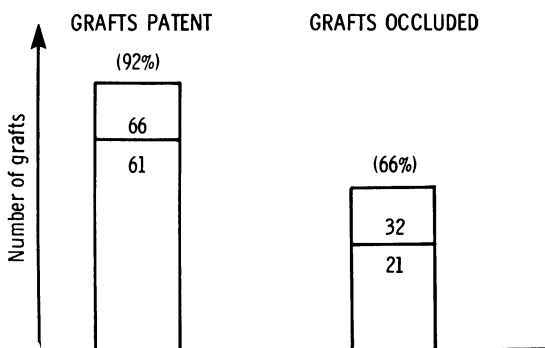


Fig. 6 Number of grafts predicted patent and occluded by  $^{201}\text{Tl}$  scintigraphy.

negatives for graft occlusion. It was interesting that in 11 occluded graft territories three regions were well supplied by collateral vessels, three regions were supplied by native vessels with less than 70 per cent narrowing of the coronary lumen, and one region was in the territory of a recessive right coronary artery and, therefore, very small. Only four occluded grafts incorrectly thought patent from the scintigram subtended regions supplied by severely diseased native vessels with inadequate development of coronary collateral vessels.

The protective role of coronary collaterals in preventing the scintigraphic detection of graft occlusion could be extended to partial protection in four patients with graft occlusion who had well-developed collateral channels and less profound tracer defects than expected from the severity of corresponding coronary artery disease. One patient had partial scintigraphic protection in the territory of a severely diseased obtuse marginal artery left ungrafted but supplied by plentiful collaterals. However, no protection was seen in the scintigrams of two patients with severe disease of the right coronary artery and an occluded right coronary artery graft despite retrograde collateral filling from the left coronary artery. One of these patients had severe disease in all collateral donor vessels and no patent grafts.

Five patent grafts predicted occluded by scintigraphy were classified as false positives for graft occlusion and have already been discussed as the false negatives for graft patency. Though anatomically patent these grafts all had poor function. Thus, the predictive accuracy of graft occlusion determined by scintigraphy is  $21/21 + 5$  (81%).

#### Mid-septal tracer transition of left anterior descending graft patency

Several patients had scintigrams showing deficit of tracer in the upper half of the septum, with preservation of normal tracer accumulation in the lower half of the septum, that is a mid-septal transition of tracer uptake as illustrated in Fig. 7. This is paradoxical, for the scintigraphic criteria of graft patency and graft occlusion are both present in the same territory of one grafted vessel, the left anterior descending artery. The majority of patients with this appearance had no other defects in the distribution of this artery when other scintigraphic projections were analysed. This septal pattern of tracer distribution was attributed to better perfusion of the lower, more distal septum in preference to the upper, more proximal septum, and was thought most likely to occur with a patent left anterior descending graft which had good distal run-off. Subsequent angiographic correlations confirmed

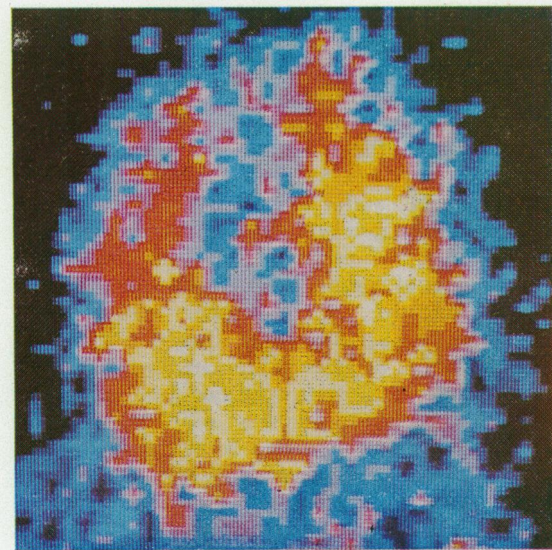


Fig. 7 Tracer deficit in the upper septum coexists with normal tracer accumulation in the lower septum causing mid-septal transition of tracer in a patient with a patent left anterior descending graft and good distal run-off.

that this interpretation was probably correct, since the sign of mid-septal tracer transition was most commonly seen in patients with patent left anterior descending grafts and good distal run-off (18 out of 33 (54%)). Moreover, all these patients had severe proximal left anterior descending disease, with very poor filling of the proximal segment from native coronary and graft injections of contrast medium. It was remarkable that completely normal uptake of tracer in the septum occurred in only eight patients with patent left anterior descending grafts. Fixed uptake defects extending down the whole septum resulting from myocardial necrosis were seen in seven patients with patent left anterior descending grafts and prevented the scintigraphic recognition of graft patency. Only one patient with a patent left anterior descending graft had reversible tracer reduction in the whole septum mimicking a graft occlusion. This patient had severe stenosis of the left anterior descending graft at its distal anastomosis causing complete septal ischaemia.

Mid-septal tracer transition was not a specific sign of a patent left anterior descending graft with good distal run-off, since four patients with an occluded graft also exhibited this septal distribution of tracer, though three of these patients did have evidence of left anterior descending graft occlusion in other views.

The usual sign of left anterior descending graft occlusion was a reversible tracer deficit extending

down the whole septum. Five patients with an occluded left anterior descending graft had this appearance which was irreversible in one patient because of septal necrosis. Only one patient with left anterior descending graft occlusion had completely normal accumulation of tracer in the septum. These findings are summarised in Fig. 8.

**APICAL WEDGE DEFECT IN ANTERIOR VIEW**  
 Fig. 9 shows an example of abnormal tracer reduction at the cardiac apex causing a wedge-shaped defect. In our experience this defect is usually attributable to left anterior descending coronary disease and often indicates that the distal left anterior descending lumen is of poor calibre.<sup>7</sup> In the context of a patent left anterior descending graft the appearance of this apical defect usually meant that the graft had poor distal run-off, as shown by the following analysis.

(a) *Patent left anterior descending graft*

Four patients with a patent left anterior descending graft but necrosis in the left anterior descending territory all had fixed apical tracer deficit in the anterior view. In the absence of myocardial necrosis 21 out of 28 patients (75%) with patent left anterior descending grafts had completely normal tracer accumulation in the cardiac apex. All these patients




|  | Number of Grafts Patent | Number of Grafts Occluded |
|--|-------------------------|---------------------------|
|  Normal septum          | 8                       | 1                         |
|  Mid-septal transition  | 18                      | 4                         |
|  Whole septal reduction | 7                       | 5                         |

Fig. 8 The relation between left anterior descending graft integrity and corresponding patterns of tracer distribution in the septum.

had good distal run-off from their left anterior descending graft. The remaining seven patients all had abnormal apical reduction of tracer. Two of these patients had very poor graft run-off and two patients had significant stenosis of the graft at its distal anastomosis. In a further two patients the apical deficit in the anterior view was not specific for left anterior descending disease but could be attributed to left circumflex disease producing severe tracer deficit in the lower posterolateral wall of the myocardium (left circumflex territory) when seen in the left anterior oblique view. These false positive left anterior descending apical defects were easily identified when all four views of the myocardial scintigram were compared. Finally, the distal run-off from one left anterior descending graft supplied a large obtuse marginal vessel cross-filling through prominent collateral channels seen on the angiogram. In this patient dynamic exercise may have precipitated a steal phenomenon whereby graft flow was shunted away from the cardiac apex along collateral channels to the obtuse marginal territory, thus producing the relative deficit of apical tracer accumulation shown in Fig. 9.

*(b) Occluded left anterior descending graft*

Two patients with occluded left anterior descending grafts subtending myocardial necrosis had significant apical tracer reduction similar to those patients with patent left anterior descending grafts and myocardial necrosis.

Five patients with an occluded left anterior

descending graft but persistence of a good calibre distal left anterior descending lumen all had completely normal tracer accumulation in the cardiac apex seen in the anterior view. Conversely, three out of four occluded left anterior descending grafts which subtended a poor calibre distal left anterior descending lumen did have significant tracer reduction in the cardiac apex.

*(c) No left anterior descending graft*

Two patients had significant proximal left anterior descending disease left ungrafted because the distal lumen of this vessel was of poor calibre. Both patients had apical defects of tracer accumulation.

In contrast, three patients with no left anterior descending disease and no left anterior descending graft, but grafts to other coronary vessels, had completely normal apical accumulation of tracer.

Thus, in summary, anterior view deficit of tracer at the cardiac apex, in the absence of myocardial necrosis, was a highly specific sign of impaired run-off from a patent left anterior descending graft but could not distinguish stenoses in the graft from poor calibre native distal vessels.

*Ungrafted coronary disease detected in scintigram*

Thirty-two patients had tracer defects in the myocardial scintigram attributable to severe coronary artery disease which had not received venous bypass grafts. Only three patients had ungrafted coronary disease which was not detected by scintigraphy. One patient had a 50 to 60 per cent stenosis

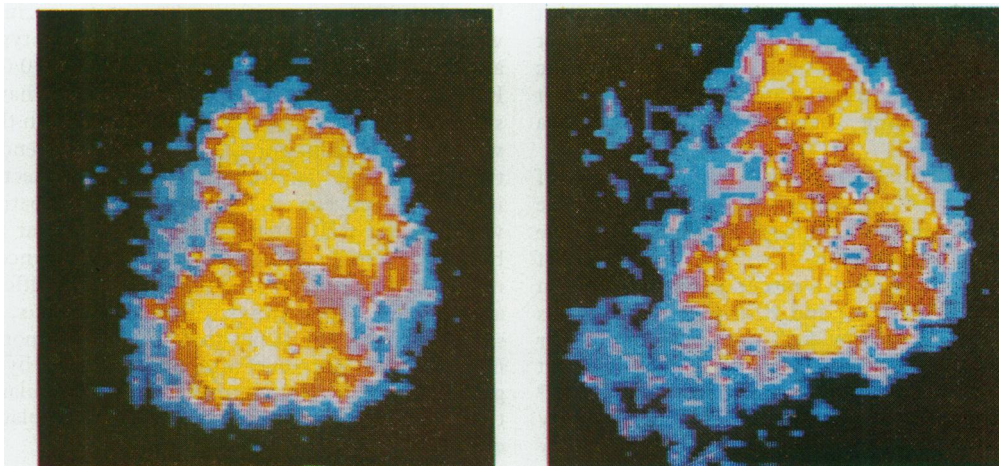


Fig. 9 Anterior view  $^{201}\text{Tl}$  myocardial scintigrams. A significant defect of tracer was seen at the cardiac apex in the early post-exercise scintigram (left) which disappeared in the delayed scintigram (right) indicating apical ischaemia on exercise. This patient had a patent left anterior descending graft filling an obtuse marginal artery through collateral vessels. A steal phenomenon at the cardiac apex may have been precipitated by exercise.



of the left circumflex vessel and a patent left anterior descending graft; one patient had severe disease in a recessive right coronary artery subtending only a small area of myocardium, and the final patient had a 70 per cent stenosis in a trifurcation obtuse marginal artery which subtended myocardium already served by a separate patent obtuse marginal graft.

The diagonal branch of the left anterior descending was the most frequent ungrafted vessel associated with uptake defects in its territory of supply (28 patients). Independent diagonal branch left anterior descending disease was found in 12 patients with scintigraphic evidence of ischaemia and in two patients with evidence of myocardial necrosis in the distribution of this vessel.

Fourteen patients had severe coronary disease affecting the left anterior descending before its main diagonal branch. Though 11 of these patients had a patent left anterior descending graft the main diagonal branch of the left anterior descending could not be visualised, either anterogradely by left coronary arteriography, or retrogradely by selective graft angiography. The remaining three patients had occluded left anterior descending grafts with severe left anterior descending coronary disease arising before its diagonal branch. All these patients had evidence of diagonal branch ischaemia in the  $^{201}\text{Tl}$  scintigram. Thus, in the absence of a separate graft to the main diagonal branch of the left anterior descending, scintigraphic evidence of ischaemia was found in the distribution of this vessel regardless of the status of the main left anterior descending graft.

Conversely, in 14 patients who had excellent opacification of the main diagonal left anterior descending branch the characteristic scintigraphic uptake defects (diagonal windows) caused by left anterior descending diagonal branch ischaemia were never seen.

Only three patients had angiographic evidence of diagonal branch left anterior descending disease with no corresponding 'diagonal window' in the  $^{201}\text{Tl}$  scintigram, that is false negatives. One of these patients achieved a rather low work-load on the bicycle ergometer, and it is possible that this was insufficient to provoke a perfusion defect, for the sensitivity of exercise scintigraphy to detect critical stenoses increases with the level of exercise.<sup>8</sup>

Thus, after aortocoronary bypass graft surgery the scintigraphic diagonal window was a highly sensitive and specific marker of persistent ischaemia in the diagonal branch of the left anterior descending despite a patent graft to the parent vessel (sensitivity rate 28/31 (90%), specificity rate 14/14 (100%).

## Discussion

Thallium-201 ( $^{201}\text{Tl}$ ) behaves as a biological analogue of potassium<sup>9</sup> and is more suitable for imaging with conventional scintillation cameras than other monovalent cations.<sup>10</sup> Its initial distribution within viable myocardium is proportional to the regional coronary blood flow prevailing at the time of intravenous administration.<sup>4</sup> Tracer redistribution is seen later in regions of resolving ischaemia.<sup>11</sup> Several recent studies have shown that exercise  $^{201}\text{Tl}$  myocardial scintigraphy is a sensitive technique to detect and localise obstructive coronary artery disease.<sup>7 12 13</sup> Its value in the assessment of aortocoronary bypass graft surgery by comparing pre- and postoperative myocardial scintigrams has also been reported.<sup>14 15</sup> This study was designed to determine the role of postoperative exercise  $^{201}\text{Tl}$  myocardial scintigraphy alone in the non-invasive evaluation of aortocoronary bypass graft surgery.

The rather low graft patency rate (65%) found in this series accords with the highly selected group under study; nearly two-thirds of patients were evaluated because of chest pain. Nevertheless, these patients are typical of those requiring further assessment after aortocoronary bypass graft surgery.

The quality of chest pain was a useful guide to the degree of myocardial revascularisation; all patients with typical angina pectoris had scintigraphic evidence of persistent regional myocardial ischaemia, whereas most patients who were free from pain did not have ischaemic defects in their scintigrams ( $P < 0.0005$ ). This was consistent with the fact that, at angiography, graft occlusion was found more frequently in patients with typical angina than in those without chest pain ( $P < 0.005$ ). Patients with atypical chest pain did not have a significantly different graft occlusion rate than those without chest pain, nor was there any difference in the prevalence of scintigraphically detectable ischaemia between these groups. Most patients with atypical pain after operation had similar pain before operation. In contrast, the prevalence of myocardial necrosis was not significantly different among these three groups of patients. Thus, this study supports the belief that the most important mechanism of pain relief after aortocoronary bypass graft surgery is effective myocardial revascularisation<sup>16</sup> rather than myocardial necrosis, placebo effects, or denervation.

Nevertheless, a high proportion of cases did have evidence of significant ischaemia after aortocoronary bypass graft surgery, particularly in the proximal branches of the left anterior descending coronary artery, despite angiographically patent

grafts. Experimental work<sup>17 18</sup> has shown that partition of flow between graft and native vessel is determined by their relative diameters and the degree of stenosis in the proximal coronary artery. In the presence of a 50 per cent proximal coronary stenosis a graft always carried more than 90 per cent of total flow. These authors suggested that complete occlusion of a bypassed proximal coronary segment was likely in view of the poor flow carried in this portion of the vessel. Clinical studies have confirmed these predictions, for Aldridge and Trimble and others<sup>19-21</sup> have all reported progression of significant proximal coronary disease to total occlusion in 17 to 46 per cent of cases after bypass grafting. New occlusions occurred most commonly in grafted coronary arteries.

The pattern of midseptal tracer transition described in the scintigrams of our patients with patent left anterior descending grafts supports the concept that ischaemia in the proximal coronary segment is common and sufficiently severe to be detected scintigraphically. Midseptal transition of tracer is emphasised as a sign of a patent left anterior descending graft with good distal run-off rather than a marker of left anterior descending graft occlusion for which evidence should be obtained from other views in addition to the left anterior oblique projection.

Relative ischaemia was commonly found in the distribution of angiographically normal diagonal branches of the left anterior descending when grafts to the main segment of the distal left anterior descending were patent. In this situation we suggest that if the main diagonal branch of the left anterior descending is a large and important vessel it should receive an independent venous graft at the time of aortocoronary bypass graft even though it appears angiographically free of disease.

The predictive accuracy of postoperative <sup>201</sup>Tl myocardial scintigraphy is remarkably high using the criteria of graft integrity previously outlined. It was interesting that patent grafts could be predicted more accurately than occluded grafts which were usually not detected because tracer uptake was preserved by flow through native and collateral vessels. A similar effect of the coronary collateral circulation is seen in patients with ungrafted coronary artery disease.<sup>22</sup> Flow through patent grafts subtending necrotic myocardium is unlikely to make a significant contribution to pain relief, though it cannot be disputed that in some cases such patent grafts may provide important collateral channels to other regions of the myocardium. Similarly, it is doubtful whether occlusion of grafts subtending regions of myocardial necrosis can be

seriously considered as a cause of recurrent angina pectoris.

Our results confirmed that the occurrence of regional necrosis was unrelated to recurrent chest pain. Equal numbers of occluded and patent grafts subtended necrotic myocardium and indicated that necrosis did not predispose to graft occlusion. In this predictive analysis exclusion of grafts subtending necrosis is justified by their functional irrelevance to the relief or recurrence of angina pectoris. Failure to predict graft integrity in regions of myocardial necrosis is, therefore, not an important limitation of <sup>201</sup>Tl myocardial scintigraphy.

On the basis of these results we suggest that patients with recurrent or persistent chest pain after aortocoronary bypass graft should be investigated initially with exercise <sup>201</sup>Tl myocardial scintigraphy and only referred for coronary and graft arteriography if there is evidence of myocardial ischaemia suggesting occluded or poorly functioning grafts, or significant ungrafted coronary disease. Moreover, presumptive knowledge of individual graft status at coronary arteriography avoids prematurely abandoning attempts to opacify grafts which are patent yet often elusive. In addition <sup>201</sup>Tl myocardial scintigraphy can be used to interpret postoperative coronary arteriograms in which graft identities are uncertain or suspected patent grafts have not been adequately demonstrated.

It is interesting that this high predictive accuracy was achieved by analysing postoperative <sup>201</sup>Tl myocardial scintigrams alone without the refinement of preoperative scintigrams for comparison. We therefore agree with Greenberg *et al.*<sup>23</sup> that postoperative myocardial scintigraphy alone is invaluable in documenting the results of aortocoronary bypass graft surgery non-invasively.

Repeated non-invasive evaluation of graft status is an advantage of <sup>201</sup>Tl myocardial scintigraphy not provided by coronary arteriography. Graft closure may be symptomatically silent and thus go undetected in the absence of routine graft angiography, a procedure not acceptable to all patients. Serial <sup>201</sup>Tl scintigraphy has good reproducibility<sup>8</sup> and may be a more attractive alternative technique to determine graft closure rates, particularly if early postoperative scintigrams and angiograms have been correlated previously.

Finally, in patients with failed aortocoronary bypass graft operations <sup>201</sup>Tl scintigraphy can indicate regions of residual myocardial ischaemia relevant for revascularisation and distinguish necrotic myocardium unlikely to benefit from further surgery.

In conclusion, postoperative exercise <sup>201</sup>Tl scintigraphy alone is a highly sensitive and specific

technique in the preliminary investigation of recurrent angina pectoris after myocardial revascularisation and gives a new cutting edge to the logical planning of further surgery.

We thank Dr Lawson McDonald for referring many of the patients included in this study.

Dr R J Wainwright was supported by a British Heart Foundation grant during the completion of this work.

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