Clinical Studies

Myocardial Viability in Cases with Persistent Perfusion Defects on the Dipyridamole Thallium-201 Scintigram
A Comparative Study with Autopsy Findings

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Summary

The aim of this study was to assess the incidence of myocardial infarction among persistent perfusion defects in dipyridamole-stress thallium scintigraphy by inspecting autopsied hearts and to evaluate whether the regional thallium activity of a scintigraphic defect can predict the presence of infarction. Autopsied hearts were compared with dipyridamole myocardial scintigrams undertaken during life in 27 patients (mean age 85 ± 8 years). The time interval from stress testing until death was 428 ± 351 days. Regional thallium uptake of delayed perfusion defect was calculated on the short axis images. The grade of regional myocardial fibrosis in autopsy specimens was also quantified to correlate with the corresponding regional thallium uptake. In 6 of 15 (40%) regions with persistent defects on the scintigram, myocardial infarction was not found at autopsy. Regional thallium-201 uptake of delayed defects < 50% diagnosed infarction with a sensitivity of 82% and a specificity of 80%. A linear correlation \( r = -0.67 \) was observed between percent thallium-201 uptake and the degree of myocardial fibrosis. In conclusion, perfusion defects at 4-hour imaging in dipyridamole-stress testing may overestimate the presence of myocardial infarction and regional thallium-201 activity is helpful in distinguishing between defects with and without infarction. (Jpn Heart J 1996; 37: 301-316)

Key words: Single-photon emission computed tomography Percent thallium-201 uptake Myocardial infarction Myocardial fibrosis Pathology

STRESS myocardial scintigraphy is widely used as a noninvasive method not only in the diagnosis of coronary artery disease1) but also in the assessment of myocardial viability for the selection of therapy2) and for predicting prognosis.3) Perfusion defects persisting 3 to 4 hours after stress testing have been interpreted

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301
as indicating scarred myocardium, whereas defects followed by complete normalization of thallium-201 activity are interpreted as indicating ischemic but noninfarcted myocardium on the basis of a report by Pohost and associates\(^4\) and clinical experience. Perfusion defects with incomplete redistribution of thallium-201 suggest mixed scarred and salvageable components of myocardium. The standard relationship above has not, however, been verified pathologically. Recently, it has been reported that persistent perfusion defects in exercise thallium scintigraphy can be associated with viable myocardium.\(^5\)\(^\text{-}^\text{13}\) Instead of exercise stress, the dipyridamole test has recently been suggested as a promising pharmacological means also applicable to older patients.\(^14\)\(^\text{-}^\text{15}\) However, standard interpretation of the perfusion defects, although considered to be similar to that of the dipyridamole test, has not been confirmed morphologically.

The purpose of this study was to determine the frequency of myocardial infarction at autopsy in persistent perfusion defects with dipyridamole-stress scintigraphy by comparing pathologic findings of autopsied hearts and to clarify how regional thallium-201 activity is correlated with the grade of fibrosis of the corresponding site of ventricular myocardium.

**METHODS**

**Subjects:** Study subjects were 27 patients who underwent dipyridamole-stress thallium scintigraphy for the evaluation of coronary artery disease and on whom autopsies were later performed. There were 12 men and 15 women aged 68 to 97 years (mean 85 ± 8) at death. The average time interval from scintigraphy until death was 428 ± 351 days (range 13 to 1271). Clinical diagnoses at the time of stress testing were old myocardial infarction in 10 patients, ischemic heart disease without clinical evidence of infarction in 10, congestive heart failure without known underlying heart disease in 5, and electrocardiographic ST-T abnormalities in 2. Artificial pacemakers were implanted in 3 patients with bradyarrhythmias due to sick sinus syndrome (2 patients) and atrioventricular block (1 patient). Electrocardiograms demonstrated abnormal Q waves indicative of myocardial infarction in 8 patients. Major causes of death at autopsy were acute infarction in 2 patients, congestive heart failure without acute infarction in 6, malignant tumors in 7, ruptured aortic aneurysm in 2, cerebrovascular diseases in 3, infectious diseases in 3, sudden death of unknown cause in 1, acute renal failure in 1, acute pancreatitis in 1 and suffocation in 1.

**Stress protocol:** Antianginal or theophylline-containing medications were withheld for 24 hours before the test. After the patient had relaxed in the supine position for a while, dipyridamole was infused intravenously at a rate of 0.568 mg/kg for 4 minutes. The patient rested for the next 2 minutes and then
started low level exercise of repetitive knee bends in the supine position. Two minutes after the beginning of exercise, 148 MBq of thallium-201 was administered intravenously. Exercise was continued for 1 minute after the injection of the isotope. During the stress testing the patient was closely monitored each minute with respect to blood pressure, heart rate and 12-lead electrocardiogram.

**Single-photon emission computed tomography:** Data acquisition for initial imaging started within 10 minutes after termination of the exercise. Delayed imaging was repeated after 4 hours to evaluate thallium-201 redistribution. The data were obtained on a large field-of-view rotating gamma camera (ZLC-7500 Digitrac, Siemens Medical Systems Inc., Iselin, NJ, USA) equipped with a parallel hole, high-resolution collimator. Each $64 \times 64$ matrix was collected for 25 seconds at each of 32 steps during 180-degree rotation from the 45-degree right anterior oblique to the 45-degree left posterior oblique projections. A computer system (Scintipac-2400, Shimadzu Corp., Kyoto, Japan) was used for data processing to reconstruct short axis, vertical long-axis and horizontal long-axis tomographic images of the left ventricle every 6-mm.

Abnormal perfusion on the initial images and its reversibility on the delayed images were judged by 3 experienced observers without knowledge of other clinical data. The interpretation was based primarily on the background-subtracted (15% uniform) gray-scaled images on transparency films and initial and delayed color bull’s-eye images as well as wash-out rate images. An initial perfusion defect followed by complete or incomplete normalization was categorized as

![Figure 1](image-url)

**Figure 1.** A: The transverse extent of a perfusion defect is expressed as the angle ($\alpha$ in the figure) made by the lines drawn from the left ventricular center to both sides of the defect on the delayed axial tomogram at the level with the largest defect in the myocardium. Regional percent thallium uptake was calculated by dividing the counts per pixel of the most severe portion of a perfusion defect (square 1) by the maximal counts per pixel of the myocardium (square 2) on the serial delayed axial image. B: The transverse extent of infarction is expressed as the angle ($\beta$) of the sector that contains the infarcted area on a transverse section of the autopsied myocardium.
a reversible defect. Incomplete redistribution refers to a partial fill-in of thallium-201 and incomplete recovery of radioactivity with residual hypoperfusion. A defect without subsequent thallium-201 redistribution was defined as a persistent defect, even when the defect was mild in terms of radioactivity. The locations of defects were categorized into anteroseptal, inferoposterior and lateral regions of the left ventricle.

The size of the perfusion defect was expressed in maximal longitudinal and transverse dimensions to compare with the size of myocardial infarction at autopsy. The former extent was calculated by multiplying 6 mm, the thickness of 1 tomographic slice, by the number of slices that included the perfusion defect as visually identified on axial tomograms. The latter was measured as the angle between the two lines drawn from the center of the left ventricular cavity to both edges of the maximal defect area on axial images as demonstrated in Figure 1A.

In order to obtain a regional percent thallium-201 uptake, a small square region-of-interest was placed on regions of a perfusion defect and of maximal thallium uptake on short-axis delayed tomograms (Figure 1A). Percent uptake was expressed as the ratio of counts per pixel at a perfusion defect to the maximal counts per pixel in the myocardium.

**Pathologic examinations:** After fixation in 10% formalin, the heart was weighed and cut along the planes perpendicular to the long axis spaced about 1 cm apart. Myocardial infarction, defined as an area of scarred tissue more than 1 cm in diameter, was inspected in terms of the location, size, and type of extension, i.e., transmural or nontransmural infarction. The infarction was termed transmural when the infarcted area extended from the endocardium to the epicardium at least partly within the entire lesion. In nontransmural infarction, necrosis was limited to the subendocardial region and there was no subepicardial involvement on any cut surface. Epicardial coronary arteries and branches were serially cut every 5 mm to examine their distribution and the degree of stenosis to determine infarct-related coronary arteries. Coronary stenosis was evaluated semiquantitatively using a 5-point scoring system, where the most severe stenosis in each major coronary artery was scored as follows: 100% obstruction = 5, 75 to 99% reduction of the cross-section area = 4, 50 to 74% stenosis = 3, 25 to 49% stenosis = 2, less than 25% stenosis = 1, and no stenosis = 0. A score of 4 or 5 was regarded as a significant stenosis.

Infarction size was measured directly in longitudinal length, transverse length and thickness. To compare the size of the scintigraphic perfusion defect, the transverse extent was expressed by the angle of the sector that contained the infarcted area and whose center was placed at the middle of the left ventricular cavity on the transverse section with the maximal infarcted area (Figure 1B). Pathologic findings were recorded with illustrations and photographs of the ante-
rior and posterior aspects of the heart, the serial transverse myocardial sections and coronary arteries.

Myocardial tissue was embedded in paraffin for microscopic examination. Sections 5-μ-thick were stained with hematoxylin-eosin and by the Azan method to confirm the pathologic diagnoses and to estimate the degree of myocardial fibrosis. Fibrosis was quantified as follows. Preparations stained by the Azan method were magnified 15-fold. A representative portion was selected for measurement and displayed on the monitor with further magnification to ×30. Fibrosis and myocytes were identified by a computer-assisted image-analyzing system (IBAS 2000, Zeiss, Germany) for the calculation of fibrosis index: Fibrosis index = \(\frac{\text{fibrosis area}}{\text{(area of fibrosis + myocytes)}}\) × 100. Papillary muscles and large vessels were excluded manually from the measurement. An area of 3.1 mm² was covered by one procedure. The measurements were repeated consecutively from the epicardium to endocardium, providing a rectangular area of 2.6 mm in width located transmurally. In each of the myocardial regions perfused by major coronary arteries, 1 to 3 rectangular transmural areas were selected for calculation of the fibrosis index usually at the most severely fibrotic part in the preparation.

Comparison of scintigraphic and pathologic findings: The photographs and illustrations of myocardial transverse sections were compared with the serial axial scintigrams in each case to correlate the perfusion defects with myocardial infarction and with related coronary arteries. The degree of fibrosis in a myocardial area was also correlated with the percent thallium-201 uptake at the corresponding region. Acute and old myocardial infarctions that had occurred after the stress testing, old infarctions without significant coronary stenosis at the time of autopsy and abnormal fibrosis caused by etiologies other than coronary artery disease, such as myocarditis, were omitted from the comparison. The data obtained on percent thallium uptake were divided according to the presence or absence of infarction at the corresponding myocardium to see whether this parameter could act as an indicator of infarcted myocardium. Furthermore, percent uptake at the infarcted area was compared between defects with and without electrocardiographic abnormal Q waves and between transmural and nontransmural infarcts.

Statistical analyses: The results are expressed as mean values ± standard deviation or proportions. Differences in numerical variables between groups were evaluated by Student’s t-test. The correlation between percent thallium-201 uptake and the fibrosis index in corresponding myocardial areas was determined using linear regression analysis. Statistical significance was defined as \(p < 0.05\).
RESULTS

Scintigraphic findings: Three patients experienced chest pain during the test. Electrocardiographic ST-segment depression of more than 1mm was induced in 5 patients. All adverse effects disappeared spontaneously soon after the exercise.

The initial scintigrams showed perfusion defects in 30 regions of the ventricular wall in 17 patients; 11 in the inferoposterior, 12 in the anteroseptal and 7 in the lateral wall of the left ventricle. The delayed images showed redistribution of thallium-201 to the defects in 13 regions of 9 patients: 6 inferoposterior, 5 anteroseptal and 2 lateral regions. Redistribution was complete in 5 regions and incomplete in the remaining 8 regions. The other 17 defects were persistent and showed no redistribution.

Autopsy findings: Heart weights ranged from 240 to 490 g (mean 366 ± 73). Pathologic diagnoses were old myocardial infarction in 13 patients (19 areas), acute infarction in 3 (3 areas), myocarditis in 2 and amyloidosis in 1. The 19 areas of old infarction consisted of 8 transmural and 11 nontransmural infarcts. Abnormal Q waves were considered to be due to infarction in 7 cases and to amyloidosis in one case. Significant coronary stenosis was recognized in 56 (69%) arteries. In extent of stenosis, 20 patients (44%) were classified as triple-vessel (involving 5 left main trunk lesions), 8 (30%) as double-vessel and 4 (15%) as single-vessel disease; 3 (11%) had normal coronary arteries. Coronary stenosis was not found in 1 patient with clinically evident old myocardial infarction.

Figure 2. Serial delayed scintigrams and the corresponding transverse sections of the autopsied heart of an 80-year-old woman with anteroseptal myocardial infarction. A: The upper direction indicates the anterior wall of the left ventricle. A perfusion defect is observed in the anteroseptal region. Percent thallium uptake of the defect was 20%. B: A massive transmural anteroseptal infarction is observed with myocardial wall thinning. Complete occlusion was found in the left anterior descending artery.
Figure 3. Anterior and posterior perfusion defects are associated with the scattered fibrosis of nontransmural infarcts in this 80-year-old male patient. Regional percent thallium-201 uptakes were 42 and 41%, respectively. All 3 coronary arteries were totally occluded and the left main trunk had a stenosis score of 4.

Figure 4. An 81-year-old man with clinical diagnoses of ischemic heart disease and second-degree atrioventricular block. A: The axial tomograms reveal a definite perfusion defect in the inferoposterior region extending partially to the lateral wall without subsequent redistribution of thallium on the delayed images. The patient died suddenly 79 days after stress testing. B: At autopsy, a relatively small area of nontransmural infarction was found (arrow). Critical stenosis was proved for all 3 coronary arteries.
Table I. Relationship between Myocardial Infarction and Scintigraphic Perfusion Defects in 56 Regions with Coronary Stenosis

<table>
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<th></th>
<th>Old MI*</th>
<th>No MI</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent defects*</td>
<td>9[4]/60%</td>
<td>6/40%</td>
<td>15</td>
</tr>
<tr>
<td>Reversible defects</td>
<td>4[2]/31%</td>
<td>9/69%</td>
<td>13</td>
</tr>
<tr>
<td>Complete RD</td>
<td>2[0]/40%</td>
<td>3/60%</td>
<td>5</td>
</tr>
<tr>
<td>Incomplete RD</td>
<td>2[2]/25%</td>
<td>6/75%</td>
<td>8</td>
</tr>
<tr>
<td>No perfusion defects</td>
<td>3[3]/11%</td>
<td></td>
<td>28</td>
</tr>
</tbody>
</table>

*Perfusion defects due to MI without CAD and to myocarditis were excluded from the comparison. #Old MI which occurred between stress testing and death and MI without CAD were excluded. CAD = coronary artery disease; MI = myocardial infarction; NTMI = nontransmural myocardial infarction; RD = redistribution of thallium-201.

Comparison of scintigraphic and pathologic findings: Figure 2 shows a case with transmural anteroseptal infarction and persistent perfusion defect concordant in location and extent. Figure 3 presents the case of an 80-year-old male. Anterior and posterior nontransmural infarcts were associated with perfusion defects on the delayed images in the corresponding regions. Figure 4 shows a representative case with significant discrepancy between the sizes of the perfusion defect and myocardial infarction at autopsy. The axial images following stress showed a perfusion defect in the inferoposterior region. Thallium-201 redistribution was not observed on the delayed images. At autopsy, although subendocardial infarction was identified in the posterolateral wall, its longitudinal and transverse extents were smaller than those of the perfusion defect. This discordance indicates the presence of myocardial viability at the ischemic zone.

Among 56 coronary arteries with significant stenosis, 28 showed corresponding perfusion abnormalities on the initial scintigrams, while scintigraphic defects were identified in 2 regions among 25 normal coronary arteries. The latter 2 perfusion defects were persistent on the delayed images and were pathologically attributed to an old infarction without coronary stenosis and confluent fibrosis due to myocarditis in the anterior wall. In 28 stenosed arteries not associated with perfusion abnormality, 4 (14%) had infarction that was either nontransmural or small-sized (<2 cm in the maximum diameter) and were accompanied by triple-vessel disease.

Clinical records revealed that the onset of 2 old transmural infarcts was after stress testing. Including these 2 cases, the following lesions were excluded from the comparison between perfusion abnormality and infarction at autopsy: 3 acute infarctions and 2 perfusion defects on the scintigram due to myocarditis and old infarction without significant coronary stenosis. In the remaining cases (Table I), 9 old infarcts (60%) were found in 15 areas with persistent defects on the scintigram, and 4 old infarcts (31%) in 13 areas with reversible defects. Of the latter 4 areas with infarction, 2 were nontransmural and showed incomplete
Table II. Scintigraphic and Pathologic Findings in Cases with Delayed Perfusion Defects

<table>
<thead>
<tr>
<th>Reversibility of defect</th>
<th>Related coronary artery</th>
<th>Pathology</th>
<th>Coronary stenosis score</th>
<th>MI Size, longitudinal (mm)</th>
<th>MI Size, transverse (degree)</th>
<th>Perfusion defect size, longitudinal (mm)</th>
<th>Perfusion defect size, transverse (degree)</th>
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<td>48</td>
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<td>70°</td>
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<td>93°</td>
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<tr>
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AMI = acute myocardial infarction; LAD = left anterior descending artery; LCX = left circumflex artery; NT = nontransmural; OMI = old myocardial infarction; RCA = right coronary artery; TM = transmural.

* OMI without significant stenosis.

Redistribution in perfusion. Conversely, no myocardial infarction was found in 40% of persistent perfusion defects or in 75% of delayed defects with incomplete redistribution. These incidences indicate that persistent defects detected by visual analysis do not predict the presence of infarction.

Table II shows coronary lesions, infarct size and the size of perfusion defects in cases with delayed perfusion defects, i.e., persistent defects and defects with incomplete redistribution. The presence or absence of infarction in persistent perfusion defects showed no definite relationship to the severity of coronary stenosis, number of diseased arteries or location of infarction. In some cases, the sizes of the perfusion defects were larger than the actual infarct sizes. However, there were no clinical or pathological data that could predict the discordance in the sizes.

**Regional thallium-201 activity (Figure 5):** Percent thallium-201 uptake was obtained for 21 defects on delayed images including both persistent and incom-
Figure 5. Percent thallium-201 uptake. A: Comparison of different degrees of perfusion. Percent uptake was higher in the case of a partially reversible defect (incomplete RD). B: The presence of a pathological infarct was associated with lower thallium uptake; 50% uptake could discriminate between perfusion defects with and without infarction. C: Comparison of transmural and nontransmural infarction. The latter morphology yielded a higher thallium uptake. D: Comparison of myocardial infarction with (Q-MI) and without (NonQ-MI) abnormal Q waves. The difference was not statistically significant (NS).

Completely reversible defects resulted in 49.7 ± 17.0% uptake. Uptake was lower in 14 persistent perfusion defects (42.7 ± 13.5%) than in 7 defects of incomplete redistribution (63.7 ± 14.9%) (p = 0.0043) (Figure 5A). The mean percent uptakes of 11 infarcted regions and 10 regions without infarction were 38.7 ± 10.4 and 61.8 ± 14.4%, respectively, showing a statistically significant difference (p = 0.0004) (Figure 5B). Nontransmural infarcts showed higher uptakes
Figure 6. Thallium-201 uptake correlated with the grade of fibrosis measured on the myocardial specimen at a site corresponding to the perfusion defect. Low thallium uptake with severe fibrosis featured transmural infarcts (closed squares). The linear correlation was statistically significant in all cases ($p<0.005$). Note that cases without infarction (open circles) are distributed over a wide range of thallium-201 uptake.

Quantification of myocardial fibrosis: The pathological grade of fibrosis was expressed as the fibrosis index. Its normal range determined in 17 coronary areas without significant stenosis was 7.5 ± 4.5%. The mean value of the index in 12 non-infarcted areas with coronary stenosis was 12.2 ± 8.1% and 42.4 ± 20.7% in 10 infarcted areas with coronary stenosis. Among 21 areas where percent thallium-201 uptakes were measured on delayed perfusion defects, the corresponding fibrosis indices were obtained in 16 areas (29.1 ± 24.0%). A comparison revealed an inverse relationship with a correlation coefficient of $r = -0.67$ ($p < 0.005$) (Figure 5D).
However, lower thallium-201 uptake did not always indicate severe fibrosis, while higher percent thallium-201 uptake invariably corresponded to little fibrosis. The fibrosis index of 5 nontransmural infarcts was 33.7 ± 17.3% and that of 5 transmural infarcts was 51.1 ± 21.8% with no statistically significant difference. Although 4 old myocardial infarcts associated with abnormal Q waves had larger fibrosis indices (49.8 ± 11.1%) than 6 infarcts without abnormal Q waves (37.5 ± 25.0%), the difference was not significant. Among stenosed coronary arteries uncomplicated by myocardial infarction, the fibrosis index was not correlated with either the severity of stenosis or the extent of stenosed arteries.

**DISCUSSION**

In stress perfusion scintigraphy, an irreversible defect generally implies transmural myocardial infarction, whereas a partially transient defect suggests mixed biological components with scarred and salvageable myocardium. Recently, the presence of viable myocardium was demonstrated in a substantial number of persistent perfusion defects in exercise-stress thallium-201 scintigraphy in comparison with other imaging procedures \(^5-^8\) or interval changes in cardiac function \(^9,^{10}\) or myocardial perfusion \(^11-^{13}\) before and after coronary revascularization therapy. Gibson et al \(^11\) found that 45% of myocardial segments with persistent defects on exercise-stress thallium-201 scintigraphy showed improved perfusion after coronary bypass grafting. Imaging 18 to 24 hours after thallium-201 injection reportedly shows reversibility of perfusion in 21% of 4-hour persisting defects. \(^7\) Kiat and associates \(^13\) also found that imaging 18 to 72 hours after thallium-201 perfusion showed redistribution in 61% of persistent defects seen at 4 hours and that this late redistribution can predict improvement in cardiac function after coronary intervention. It is also reported that 49% of persistent defects observed 4 hours after exercise stress testing were improved on additional images obtained following a second thallium-201 injection after delayed imaging. \(^9\) With respect to myocardial metabolic activity, 47% of irreversible defects on thallium-201 images showed glucose metabolism in positron emission tomography. \(^17\)

Myocardial viability is assessed mainly by clinical observations. There are few pathological reports on the myocardial viability of persistent perfusion defects. \(^18,^{19}\) Bulkley et al \(^19\) demonstrated that quantified thallium-201 defects obtained at rest are larger in some patients than the infarcted myocardial area identified at necropsy, suggesting that thallium-201 defects include ischemic but viable myocardium. Our results demonstrate pathologically that 40% of persistent defects in dipyridamole scintigraphy are not related to infarction. Also, defects followed by incomplete redistribution do not necessarily indicate mixed
pathology of scarred and viable myocardium because 75% were not associated with infarction. This result is in agreement with the report of an exercise-stress study by Cloninger et al. In view of previous studies, the persistent defects without myocardial infarction observed in our patients would probably have shown reversibility in perfusion with later imaging or with reinjection of thallium-201. In addition, the presence of myocardial viability is relevant in patients with myocardial infarction when the infarct is nontransmural or the perfusion defect is larger than the infarct size, as shown in Figure 4. Functional improvement would, therefore, be anticipated after revascularization in such cases.

Several authors have reported that relative thallium-201 concentrations may be an indicator of myocardial viability. In our study, quantification of regional myocardial thallium-201 uptake was helpful in distinguishing noninfarcted from infarcted myocardium with a sensitivity of 82% and a specificity of 80%. Thallium-201 uptake of less than 50% yielded false-positive results for diagnosing myocardial infarction in 18%. This incidence approximates the clinical observations of Gibson et al. who reported that 21% of severe defects with semiquantified thallium-201 uptake of less than 50% converted to normal uptake after coronary bypass surgery. Furthermore, the percent uptake in nontransmural infarctions was significantly higher than in transmural infarctions. When myocardial infarction is nontransmural, myocardial viability is expected. In this context, percent thallium uptake is informative when intervention is under consideration in a patient with myocardial infarction.

The significant correlation between the grade of thallium-201 activity and that of fibrosis suggests that regional thallium uptake can characterize myocardial pathology to a certain extent. However, lower myocardial thallium-201 activity did not necessarily indicate severe fibrous replacement in some cases. The grade of fibrosis is not the only factor that determines the thallium-201 activity at the lesion. Accumulation and washout of thallium-201 in ischemic myocardium are influenced by regional myocardial blood flow, development of collateral vessels, severity and extent of coronary stenosis, achieved level of stress and blood concentration of thallium-201. Severity of stenosis, multiplicity of stenotic lesions in a coronary artery and number of diseased arteries can affect regional myocardial blood flow. We could not specify which morphologic factor played the most significant role in diminishing the thallium-201 uptake of persistent perfusion defects without infarction, as no relationship was proved between these factors and perfusion defects. It is possible that collateral flow prevented fibrotic changes in regions with persistent perfusion defects in the present study as reported by Schwarz and associates, leading to the discrepancy between fibrosis index and percent thallium uptake. In the case shown in Figure 3, collateral circulation must have been present because the 3 major coronary arteries were
totally occluded.

Q-wave infarction grossly suggests transmural necrosis, while non-Q infarction indicates a nontransmural necrosis. The fibrosis index of Q-wave infarction was actually higher than that of non-Q infarcts, though the small number of cases did not reveal statistical significance. Non-Q infarction showed a widely varying fibrosis index. The same propensity was observed in percent thallium uptake. The absence of abnormal Q waves in a patient with myocardial infarction may not be a reliable finding for smaller degrees of fibrosis.

**Limitations:** The skewing of our patients toward an older age may have influenced some of the results, for example, accuracy of stress testing or prevalence of multivessel disease. In addition, the autopsied patients might have represented a group with poor prognoses. There is a possibility that coronary and myocardial pathology might have been changed during the period between the stress testing and autopsy.

Minor topographic discrepancies are inevitable when scintigraphic and pathologic findings are compared. We measured the grade of fibrosis at the most severe and representative region in the specimen under investigation; on the other hand, we estimated percent thallium-201 uptake at the most severe defect in serial axial tomograms. The two values were thus supposed to be comparable.

**Conclusion:** Our study demonstrates that persistent perfusion defects seen in dipyridamole scintigraphy tend to overestimate irreversibly damaged myocardium on the basis of autopsy. Regional thallium-201 activity is beneficial for distinguishing infarcted from noninfarcted perfusion defects in patients without clinical evidence of infarction, and transmural from nontransmural infarcts in patients with myocardial infarction.

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**References**


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