Diagnostic Methods
Computer-Assisted Tomography

The usefulness of x-ray computed tomography for the diagnosis of myocardial infarction

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ABSTRACT Conventional and enhanced computed tomographic (CT) examinations were performed in 103 patients with myocardial infarction for evaluation of the diagnostic usefulness of CT. After intravenous bolus injection of contrast material, an initial filling defect and late enhancement of the infarcted myocardium appeared on the cardiac CT images. These two findings were direct evidence of myocardial infarction; the former was found mostly in the patient with recent myocardial infarctions, and the latter was recognized both in those with recent and those with "remote" infarctions. Wall thinning at the site of infarction was found by enhanced CT mostly in patients with anteroseptal or extensive anterior infarctions, and was rarely found in patients with inferoposterior infarctions. Left ventricular aneurysms and ventricular thrombi were found by enhanced CT in 39 and 23 of the 103 subjects, respectively, and the sensitivity of CT in detecting intracardiac thrombi was higher than that of two-dimensional echocardiography. Calcification of the myocardium and pericardial effusion associated with myocardial infarction were also detected by conventional nonenhanced CT. Thus, cardiac CT was found to be a useful test in evaluating patients with myocardial infarction.


RECENTLY x-ray computed tomography (CT) has been validated as a useful noninvasive diagnostic method for use in patients with various cardiac diseases. As far as the application of CT in patients with myocardial infarction is concerned, since the report by Adams et al.,1 which demonstrated that the CT findings in infarcted myocardium differed from those in normal myocardium whether or not contrast enhancement was used, the possibility of detection of myocardial infarction and of evaluating its extent and the stage by this technique has drawn much interest.

To date there have been many reports of the CT findings in animals with experimentally produced infarction.2–16 Several studies reported that the infarcted myocardium appeared as an area of poor enhancement on the x-ray during the first few minutes of the intravenous administration of contrast material.4,8,10–16 Subsequently there was a greater degree of enhancement of the infarcted area compared with the normal myocardium. This phenomenon was called late enhancement of the myocardium and it was noted to be persistent for several minutes to ½ hr after the injection of contrast material.

In spite of these excellent results from fundamental studies, the clinical application of CT in the diagnosis of myocardial infarction has not become popular thus far. We have been studying the clinical value of CT in the evaluation of patients with myocardial infarction. In previous articles we reported the abnormality of regional wall movement in the infarcted heart detected by electrocardiographically gated CT17 and the increase in CT values in the congestive lung resulting from myocardial infarction.18 The purpose of this report is to describe other CT diagnostic findings in patients with myocardial infarction.

Methods

Subjects. One hundred and three patients with recent and "remote" transmural infarctions, including 86 men and 17 women with an age range of from 33 to 84 years, were studied. According to the electrocardiographic criteria for infarct location,19 they were classified into three groups; group A included 56 patients with anteroseptal or apical infarctions, group B 24 patients with extensive anterior infarctions, and group C 23 patients with inferior (diaphragmatic) or inferoposterior infarctions. Patients with multiple sites of infarction in different locations were excluded for simplification of the study. Those with reinfarction and subendocardial infarction were also excluded. In addition to this anatomic classification the patients were divided into a recent infarction group (infarction occurring less
than 1 month before study) and a remote infarction group (infarction occurring more than 1 month before study).

The diagnosis of myocardial infarction was based on the following criteria.

Recent myocardial infarction was diagnosed if the patient had (1) a history of typical prolonged chest pain, (2) electrocardiographic changes indicative of acute myocardial infarction, and (3) characteristic elevation of serum enzyme levels (creatine kinase, glutamic-oxaloacetic transaminase, lactic acid dehydrogenase).

Remote myocardial infarction was diagnosed if the patient had (1) a history of previous myocardial infarction, (2) diagnostic Q waves on the electrocardiogram, and (3) normal enzyme levels during the observation period.

All patients with recent myocardial infarction had been admitted to our service, and CT scan in those patients with acute myocardial infarction were performed 3 to 30 days after onset of the disease, when the patient was considered fit to be moved to the radiologic laboratory.

Procedures. In most cases a GE CT/T 8800 was used for the study, but a Siemens Somatom 2 was also sometimes used. Data were stored on magnetic tapes so that CT images could be displayed on a television monitor for investigation at any time. The record of CT images was obtained on x-ray films by a multiformat camera. The pixel size of both the GE CT/T and Siemens Somatom was 1 × 1 mm, and the CT value of each pixel was presented as a number ranging from +1000 to −1000 Hounsfield units (HU). All subjects were examined while they were in the supine position during a full inspiratory breath hold.

The routine CT examination was done in the following manner. First, conventional nonenhanced scans were done with 1 cm thick slices from the level of the aortic arch to the diaphragm with an exposure time of approximately 10 sec for each scan. These images were useful not only for the determination of the left ventricular slice levels, but also for the detection of pericardial effusion and calcification in the heart and vessels. Three or four early scans were then obtained at the various left ventricular levels immediately after each bolus intravenous administration of 15 to 20 ml of Renograin 76 (76% meglumine diatrizoate) in order to obtain enhanced CT images. The injection was made manually into the antecubital vein of each subject through an 18-gauge plastic cannula within several seconds.

In 23 selected subjects delayed scans at the levels of the left ventricle were performed 10 to 15 min after the bolus injection of the contrast material to evaluate late enhancement of the infarcted myocardium; in 12 subjects electrocardiographically gated CT scans with contrast enhancement were obtained to evaluate regional ventricular wall motion abnormalities.

Two-dimensional echocardiography, left ventriculography, and coronary angiography were carried out in most of the subjects within a week of the CT scans. Two-dimensional echocardiography was performed with a Toshiba electronic sector scanner (model 11A) and the parasternal long- and short-axis and apical four-chamber views were obtained at the standard transducer position and recorded on videotapes or 8 mm cine films. Left ventriculography and angiography were performed with a biplane Siemens image-intensifier system. Left ventriculograms were obtained in the 30 degree right anterior oblique and 60 degree left anterior oblique views. Both the ventriculograms and coronary angiograms were recorded on 35 mm film at a rate of 30 frames/sec.

CT images, two-dimensional echocardiograms, and left ventriculograms were interpreted independently by well-trained observers. The agreement of all observers was required for each determination and when agreement was not possible after discussion the finding was judged to be questionable.

Definitions. The CT, left ventriculographic, and two-dimensional echocardiographic terms used in this report were defined as follows:

CT terms. Filling defect was a region with a decreased x-ray attenuation coefficient compared with the surrounding normal myocardium within the first few minutes after intravenous administration of the contrast material.

Late enhancement was an increase in attenuation coefficient compared with that for the normal myocardium after 10 to 15 min of administration of contrast material.

Wall thinning was reduction of left ventricular wall thickness beyond normal values (9 ± 2 mm) on CT images.

A left ventricular aneurysm was a localized protrusion of pericardium and endocardium with wall thinning.

Left ventricular thrombi were filling defects between the left ventricular cavity and myocardium on enhanced CT images. Thrombi could be differentiated from filling defects because CT values in the former were not increased by contrast enhancement, but those in the latter were increased for from several minutes to ½ hr after administration of the contrast material. A small filling defect (less than 25 mm² in size) that was seen in only one slice of the CT scan was not considered a thrombus, because a low-density fleck owing to artifacts might have confounded the diagnosis.

Calcification was considered to be present at the site at which CT values were extremely high (>100 HU) on the conventional CT image.

Pericardial effusion was considered to be present in the zone surrounding the heart in which CT values ranged from 10 to 40 HU; this zone was not enhanced by contrast material.

Left ventriculographic terms. Left ventricular aneurysm was a localized, permanent, abnormal, blood-filled dilatation of the left ventricle and/or paradoxical movement of the left ventricular endocardium.20,21

Left ventricular thrombus was a filling defect attached to the left ventricular wall in the left ventricular cavity.22

Echocardiographic term. Left ventricular thrombus was a distinct mass of echos in the left ventricular cavity that was seen clearly throughout the cardiac cycle in at least two different echocardiographic views.23

Statistical analysis. All data are expressed as mean ± SD. Comparisons were made with a simple analysis of variance, and p < .05 was considered significant.

Results

Filling defect and late enhancement of the myocardium.

Filling defects and the late enhancement of the myocardium after administration of contrast material were documented frequently in patients with myocardial infarction. When CT scans were obtained immediately after the bolus injection of contrast material, the site of myocardial infarction was observed as an area for which CT values were decreased due to decreased staining of the myocardium (figures 1 and 2). The infarcted area gradually picked up the contrast material and the CT values for the area were increased beyond those of the normal myocardium; this lasted for up to ½ hr after the injection (figures 1 and 2).

The incidences of filling defect and late enhancement of the myocardium in the subjects who had myocardial infarctions at various times before the study are summarized in tables 1 and 2.
Filling defects and late enhancement of the myocardium were usually obvious in the patients with anteroseptal or apical infarctions, and their location coincided with the infarcted area estimated by electrocardiography, thallium-201 scintigraphy, two-dimensional echocardiography, left ventriculography, and angiography. However, these CT findings were rarely documented at the inferior or posterior wall, even in the cases in which obvious inferior or inferoposterior infarction was demonstrated by the other methods. It seemed that both filling defects and late enhancement of the myocardium indicated the area of infarction, but the mechanism of these phenomena appeared to be somewhat different. The area of the filling defect was always smaller than that of late enhancement, and the difference between them was increased with increasing amounts of time after onset of myocardial infarction.

Among 16 subjects from group A and seven from group B, all of whom had recent myocardial infarctions, a filling defect was noted on the early scans of 14 (88%) and six subjects (85%), respectively. In the subjects with remote infarction a filling defect was documented in only six (15%) of 40 subjects from group A and three (18%) of 17 from group B. In group C filling defects were rarely seen in either those with recent or remote infarction, and only three questionable cases among the 23 subjects were found.

Late enhancement of the myocardium was detected in about half of the subjects from groups A and B, regardless the time course of infarction, but late enhancement of the myocardium was not documented in group C.

![Figure 1](image1.png)

**FIGURE 1.** Contrast-enhanced CT images from the middle level of the left ventricle of a 45-year-old man with anteroseptal myocardial infarction 9 days after the onset. *Top.* After a bolus injection of the contrast medium, a filling defect (arrows) appeared in the infarcted region. *Bottom.* Ten minutes after the injection, late enhancement of the myocardium appeared in the same region. RV = right ventricle; LV = left ventricle.

![Figure 2](image2.png)

**FIGURE 2.** Cardiac CT images from a 66-year-old man with anteroseptal myocardial infarction 3 weeks after the onset. *A.* A small filling defect was demonstrated in the anterior wall of the left ventricle immediately after a bolus injection of contrast material. *B.* Ten minutes after the injection, late enhancement of the anteroseptal wall appeared and the enhanced area was larger than that of the filling defect.
TABLE 1

Appearance of filling defects on contrast-enhanced CT scans in 103 patients with myocardial infarction

<table>
<thead>
<tr>
<th>Time from onset of disease</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>n</td>
<td>+</td>
<td>±</td>
</tr>
<tr>
<td>Within 1 week</td>
<td>7</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>1 week to 1 month</td>
<td>9</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>1 month to 3 months</td>
<td>13</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>3 months to 6 months</td>
<td>6</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>More than 6 months</td>
<td>21</td>
<td>0</td>
<td>21</td>
</tr>
<tr>
<td>Total</td>
<td>56</td>
<td>20</td>
<td>36</td>
</tr>
</tbody>
</table>

Furthermore, the filling defect was recognized only in patients with relatively recent infarctions (attack within 6 months), but late enhancement was seen in both recent and remote myocardial infarctions (figures 1 through 3).

Wall thinning. Wall thinning in the left ventricle was one of the indirect findings associated with the healing process after myocardial infarction. Figure 4 shows the left ventricular wall thicknesses from CT scans of normal subjects and the patients of each group. Wall thinning at the site of infarction was clearly noted in patients in groups A and B. However, it was not sufficiently recognized in group C because of the improper slice angle of CT scans.

In 41 subjects in groups A and B who underwent CT scans within 100 days after onset of myocardial infarction the relationship between the anterior wall thickness and the length of time from infarction to the CT study is demonstrated in figure 5. The wall thickness at the site of infarction gradually decreased over time. This was especially evident in certain patients (data represented by solid line in figure 5).

Left ventricular aneurysm (figure 6). Marked localized bulging in systole and akinesis or dyskinesis of particular segments were observed in all five patients with aneurysms that underwent electrocardiographically gated CT examination.

A left ventricular aneurysm was detected on the enhanced CT image in each of 16 subjects in group A, each of 20 in group B, and each of three in group C, and the detection rate of ventricular aneurysm in each group was 28%, 83%, and 13%, respectively. These results indicated that after myocardial infarction ventricular aneurysm usually develops in the anterior wall and sometimes extends to the apex.

Left ventriculography was performed in 78 subjects within a week of CT scanning for comparison of the findings obtained with both methods. The relationship between the results of CT and left ventriculography with regard to left ventricular aneurysms is summarized in table 3. Among the 78 subjects, 36 subjects, including 14 from group A, 19 from group B, and three from group C, were determined by CT to have left ventricular aneurysms. Among these 36 subjects, 25 were determined to have left ventricular aneurysms by left ventriculography. In the other 11 subjects, localized akinesis of the left ventricular wall at the infarcted site was evident, but there was no evidence of aneurysm. Among the 42 subjects in whom no ventricular aneurysms were found by CT, eight were found to have aneurysms by left ventriculography, and 34 were found not to have ventricular aneurysms; localized akinesis of the ventricular wall was found in 16 of the 34 subjects.

Ventricular thrombus. Enhanced CT was found to be an excellent method for the detection of mural thrombi, which were recognized as filling defects in the ventricular cavity. Left ventricular thrombi were

TABLE 2

Appearance of late enhancement of the myocardium on delayed CT scans in the 23 patients with myocardial infarction

<table>
<thead>
<tr>
<th>Time from onset of disease</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>+</td>
<td>±</td>
</tr>
<tr>
<td>Within 1 week</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>1 week to 1 month</td>
<td>6</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>1 month to 3 months</td>
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<td>2</td>
<td>0</td>
</tr>
<tr>
<td>3 months to 6 months</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>More than 6 months</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>6</td>
<td>4</td>
</tr>
</tbody>
</table>
found on CT images from 23 of the 103 patients (22%), including 12 (21%) from group A and 11 from group B (46%). In all but one case the thrombi were found on the anterior wall or the apex; in the remaining patient the thrombus was on the high lateral wall. The thrombi in 18 subjects were detected in a left ventricular aneurysm (figure 3).

Seventy-eight patients were studied with CT and left ventriculography at approximately the same time. CT revealed left ventricular thrombi in 15, but thrombi were not detected in 62 subjects, and in one patient results were inconclusive. On the other hand, left ventriculography demonstrated thrombi in 13 patients, in 63 thrombi were not found, and in two patients results were inconclusive. The results obtained with the two methods agreed in 95% of the cases. Among the 15 subjects found to have evidence of thrombi with CT, thrombi were also detected by left ventriculography in 12, the results were questionable in one, and no thrombi were seen in two. On the other hand, in 12 of the 13 patients with thrombi detected by left ventriculography, thrombi were also detected by CT (table 4). Eighty-one patients underwent two-dimensional echo-

FIGURE 3. Cardiac CT images from a 52-year-old man with an extensive anterior infarction 4 months after the onset. A, CT image from the middle level of the left ventricle 15 minutes after the first bolus injection of contrast material. B, CT image from 10 mm lower than that in A. C, CT image from 10 mm lower than that in B. An area of late enhancement (arrow) of the infarcted myocardium and a mural thrombus (arrow head) in a left ventricular aneurysm were observed.
was also evident. Figure 8 illustrates an example of calcification at the site of old myocardial infarction (CT value approximately 200 HU).

Discussion

Initial studies by Adams et al.\(^1\) and Ter-Pogossian et al.\(^2\) demonstrated that the edematous myocardium of experimental infarction could be distinguished from normal myocardium on the CT image without contrast enhancement. However, in human patients infarcted and normal myocardium cannot be clearly distinguished on conventional CT images except by the calcification of the myocardium observed after infarction. The main reasons for the failure to demonstrate the reduction in CT values in the edematous myocardium in patients appear to be the distorted images obtained and the decreased contrast resolution due to the cardiac motion. Further studies for the evaluation of myocardial infarction by CT were carried out in living dogs and a few patients by Carlsson et al.\(^3, 4, 5\) They demonstrated that the infarcted myocardium was detected not only as a negative filling defect within the first few minutes after intravenous administration of contrast material, but also as a region of late enhancement of myocardium from several minutes to \(\frac{1}{2}\) hr after the administration of contrast material.

Based on their observations, in our study the initial filling defect and late enhancement after administration of contrast material were considered direct evidence of infarction. It is obvious that the initial filling defect is due to decreased perfusion in the myocardium. However, the mechanism by which contrast material accumulates in damaged myocardium is unclear. Microvascular damage at the infarcted area may allow leakage of contrast to the extravascular space, and the damaged cell membrane may allow the entry of con-

![FIGURE 6. A left ventricular aneurysm (An) is shown on the contrast-enhanced CT image from the middle level of the left ventricle in a 66-year-old man with remote extensive anterior infarction.](image)

<table>
<thead>
<tr>
<th>TABLE 3</th>
<th>CT and left ventriculographic (LVG) findings with regard to left ventricular (LV) aneurysm</th>
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<tbody>
<tr>
<td>CT findings</td>
<td>LV aneurysm</td>
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<tr>
<td>Group A (n = 43)</td>
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<tr>
<td>LV aneurysm (n = 19)</td>
<td>12</td>
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<tr>
<td>No aneurysm (n = 24)</td>
<td>4</td>
</tr>
<tr>
<td>Group B (n = 17)</td>
<td></td>
</tr>
<tr>
<td>LV aneurysm (n = 14)</td>
<td>11</td>
</tr>
<tr>
<td>No aneurysm (n = 3)</td>
<td>1</td>
</tr>
<tr>
<td>Group C (n = 18)</td>
<td></td>
</tr>
<tr>
<td>LV aneurysm (n = 3)</td>
<td>2</td>
</tr>
<tr>
<td>No aneurysm (n = 15)</td>
<td>3</td>
</tr>
<tr>
<td>Total (n = 78)</td>
<td></td>
</tr>
<tr>
<td>LV aneurysm (n = 36)</td>
<td>25</td>
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<tr>
<td>No aneurysm (n = 42)</td>
<td>8</td>
</tr>
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</table>

<table>
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<tr>
<th>TABLE 4</th>
<th>Detection rate of the mural thrombi by CT, left ventriculography (LVG), and two-dimensional echocardiography (2-DE)</th>
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<tbody>
<tr>
<td>CT</td>
<td>+</td>
</tr>
<tr>
<td>LVG</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>12</td>
</tr>
<tr>
<td>±</td>
<td>1</td>
</tr>
<tr>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
</tr>
<tr>
<td>2-DE</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>13</td>
</tr>
<tr>
<td>±</td>
<td>2</td>
</tr>
<tr>
<td>-</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
</tr>
</tbody>
</table>
DIAGNOSTIC METHODS–COMPUTER-ASSISTED TOMOGRAPHY

FIGURE 7. Cardiac CT images from a 79-year-old man with inferior infarction 10 days after the onset. Pericardial effusion (PE) separated from the myocardium by subepicardial fat (F) is seen around the left ventricle (LV) and right ventricle (RV). Calcifications of the coronary arteries are also shown. AA = Ascending aorta; RVOT = right ventricular outflow tract; RAU = right auriculum; LCA = left coronary artery; LAD = left anterior descending artery; LCF = left circumflex artery; RCA = right coronary artery; RA = right atrium.

The late enhancement of the myocardium in dogs with recent and remote infarctions is usually visualized well on delayed CT scans. However, late enhancement of the myocardium is visualized in only about half of patients with myocardial infarction. This discrepancy may be explained by the injected dose of contrast material per kilogram of body weight, which in dogs is three to four times that in patients, and by the fact that experimental infarction in dogs differs from the infarction caused by coronary sclerosis in man in that in the former there is much formation of collateral vessels in the infarcted wall.

One of the indirect signs of myocardial infarction detected by CT is ventricular wall thinning. Two mechanisms for wall thinning in myocardial infarction have been postulated. One is wall stretching soon after the occurrence of myocardial infarction, and the other is the elimination of necrotic infarcted tissue in the course of healing.

From our observations of wall thinning, usually a week after the onset of infarction, the latter appears to be more likely. The site of wall thinning, as detected by CT, was limited in most cases to the anterior or apical wall; thinning of the inferior or posterior wall was rarely observed. This was assumed to be due to the slice angle of the CT examination, which was unsuitable for detecting inferior wall abnormalities.
Edwards defined a ventricular aneurysm pathologically as a protrusion of a localized portion of the external aspect of the left ventricle beyond the remainder of the cardiac surface, with simultaneous protrusion of the cavity. This definition can be applied in enhanced cardiac CT. There have been only a few studies of ventricular aneurysm by CT. Lipton et al. reported a case of a ventricular aneurysm after anterior infarction and Lackner and Thurn reported a case of ventricular aneurysm after posterior wall infarction. We observed 39 patients with ventricular aneurysms out of 103 patients with myocardial infarction. The results of our study indicated that the ventricular aneurysm that develops after myocardial infarction usually develops in the anterior wall or the apex, but rarely occurs in the inferoposterior wall.

The incidence and location of left ventricular aneurysm after myocardial infarction based on CT scanning results were in agreement with the pathologic results reported by Edwards. However, the correlation between CT and left ventriculographic results in terms of demonstrating ventricular aneurysm in this study was somewhat poor. There are two explanations for this. First, biplane right and left anterior oblique ventriculograms may not be sufficient to visualize all parts of the left ventricle. Second, the diagnostic criteria for the left ventricular aneurysm on CT scans were based on the anatomic definition, which differs from the angiographic criteria. The former does not include wall movement disturbance, while the latter includes both anatomic protrusion of the left ventricle and functional disturbance of the wall movement, but does not consider localized wall thinning of the aneurysm. Further studies using electrocardiographically gated CT could possibly solve this problem.

Left ventricular thrombosis is one of the most significant complications of myocardial infarction. Two-dimensional echocardiography has been validated as a reliable noninvasive method for the detection of thrombi. However, there has been a significant number of reports of failure to detect left ventricular thrombi by two-dimensional echocardiography. With left ventriculography the opacification of the left ventricular cavity determines the surface area of thrombi precisely, but the border between thrombi and endocardium of the left ventricle cannot be identified sufficiently. The contrast resolution of CT is much superior to that of left ventriculography, so that very subtle differences in attenuation of the x-ray between cardiac structures can be identified. Godwin et al. reported that enhanced CT scans distinguished mural thrombi very clearly in three patients with recent myocardial infarction and that CT offered some advantages over left ventriculography and echocardiography in detecting thrombosis. Nair et al. studied 16 patients suspected of having left ventricular thrombi and found that CT and two-dimensional echocardiography predicted thrombi in 10 and eight of the 16 patients, respectively. Both CT and left ventriculography correctly predicted the presence of thrombi in two patients and their absence in three patients in whom the diagnosis was confirmed surgically. In the same patient group, however, two-dimensional echocardiography failed to predict the presence of a thrombus in one patient. Our results also support the superiority of CT in the detection of ventricular thrombi. Since the search for ventricular thrombi is usually done by two-dimensional echocardiography only, it is presumed that thrombi could well be much more common in patients with myocardial infarction than has been previously reported.

Although calcification of myocardium and pericardial effusion are easily detected on CT images, these CT findings have been never reported in patients with myocardial infarction. Since calcification and effusion are very significant complications, CT would be very useful in the clinical evaluation of patients with myocardial infarction. In terms of the other CT findings that were regarded as indirect evidence of myocardial infarction, we previously reported the disturbance of wall motion at the infarcted area and the increase in CT values in the lung due to pulmonary congestion.

As far as the possible risk involved in CT studies is concerned, all our patients with recent myocardial infarction underwent CT scanning with intravenous drip infusion and close electrocardiographic monitoring. Total time required for one examination was, on the average, about 30 min. Although rapid bolus injections of contrast provide enhanced CT images of better quality than do drip infusions, injections could produce more undesirable side effects than infusions. The amount of contrast material used for the study should be minimized. We injected 15 to 20 ml of 76% meglumine diatrizoate for each CT scan until the total dose was 60 to 120 ml. Using this method we observed no significant side effects and the minor effects observed in a few patients were those such as nausea, urticaria, and pain at injection site.

Thus, CT scanning is considered to be a safe noninvasive method to evaluate patients with myocardial infarction, even in the acute phase, and it can provide information that is useful for the diagnosis of myocardial infarction and for the evaluation of the hemodynamic status of the patient. Although there are still a
few technical problems, such as the rather limited capability to evaluate the status of the inferior wall, which could hopefully be solved in the future with the development of more efficient equipment, CT scanning should be more widely used in the evaluation of patients with myocardial infarction.

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