Thallium-201 Myocardial Imaging in Patients with Various Heart Diseases

Makoto Kondo

Department of Radiology, Keio University School of Medicine

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各種心疾患例におけるタリウム-201心筋イメージング

慶応義塾大学医学部放射線医学教室（主任：橋本省三教授）

近　藤　誠

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タリウム－201心筋シンチグラフィーを，245例の各種心疾患例に施行した．心筋イメージは左室壁の壊死部・虚血部の部位及び範囲を的確に捉えることができる．心筋梗塞における陽性率は，心電図のそれとほぼ等しい．心電図上異常Q波を認めない例でも欠損像を呈することがあり，他の検査で不明な心筋梗塞例も診断可能であった．

左室イメージの大きさは，左室負荷疾患では大きく，心房中隔欠損症では逆に小さかった．手術後の再検査で，大動脈弁閉鎖不全症と僧帽弁閉鎖不全症では左室イメージの著明な縮小を見，心房中隔欠損症では拡大を見た．イメージ上の左室壁厚は剖検時の壁厚よりも厚く，真の値を示していない．

173例の心臓カテーテル検査施行例において，右室壁描出度と血行動態との関連を調べた．82例に右室壁描出があり，これは右室負荷存在の判定にとって鋭敏性・特異性とも高い，良い指標となることが示された．また同時に左室負荷が存在すると，右室壁描出の可能性が減ることが明らかになった．

心筋梗塞例における欠損像の発見頻度，及び右室描出例における最良描出角度の観点から，本検査法における多方向撮像の必要性を論じた．その結果，3方向の左前斜位撮像を加えた5方向撮像の有用性が示された．

Myocardial imaging with radioactive tracers offers a noninvasive method for detecting myocardial infarction (MI) and transient myocardial ischemia.[1)-6)] Tracers such as thallium-201 (Tl-201), potassium-43, and rubidium-81 have similar biological properties that tracer uptake by myocardial cells is proportional to the regional myocardial blood flow.[7)] Tl-201, however, has more profitable physical properties for imaging than other tracers, and has also been used to evaluate various heart diseases other than coronary artery disease.[8)-10)] With the relatively limited experience with Tl-201 myocardial

imaging available, however, it would be desirable to assess its usefulness and limitations in clinical aspects on the basis of many of various heart diseases. The present study evaluates usefulmess of Tl-201 myocardial imaging in coronary artery disease and other heart diseases with left ventricular (LV), and/or right ventricular (RV) overloading. In addition, validity of multiple-projection studies is discussed to define a reasonable number of projections for myocardial imaging.

MATERIALS AND METHODS

Myocardial imaging was performed on 245 adult patients (Table 1). Diagnosis was made on the basis of the patient's clinical history, physical examination, laboratory findings, electrocardiography

Table 1. List of Patients

Coronary artery disease (CAD)		92
Myocardial infarction	63	
Angina pectoris	29	
Chest pain syndrome without CAD		14
Cardiomyopathy		28
Hypertrophic	23	
Congestive	5	
Valvular heart disease		57
Mitral stenosis (MS)	25	
Aortic regurgitation (AR)	9	
Mitral regurgitation (MR)	6	
Combined	17	
Congenital heart disease		36
Atrial septal defect (ASD)	18	
Ventricular septal defect (VSD)	7	
Tetralogy of Fallot	5	
Others	6	
Others		18
Total		245

(ECG), and cardiac catheterization. Cardiac catheterization was performed on 173 patients, including 33 patients with coronary artery disease (CAD), one patient with chest pain syndrome without CAD, and all of 139 patients with other disease. Cardiac catheterization was performed within 24 hr of myocardial imaging. Electrocardiography was performed on the day of the myocardial imaging in patients with CAD and in patients with chest pain syndrome without CAD, and was interpreted by two observers without clinical information. Myocardial infarction had once been documented in these patients on the basis of clinical history, typical evolution of ECG, and abnormal and evolving cardiac enzymes at the onset. Patients with chest pain syndrome showed no evidence of CAD in thorough clinical evaluation, and had other diseases causing chest pain.

Myocardial imaging was performed on patients at rest. Ten minutes after i.v. injection of 2 mCi of Tl-201 chloride in the supine position, myocardial imaging was begun and completed within 60 min. Images were obtained with a gamma camera, using a low-energy high-resolution parallel-hole collimator. The 30% energy window was set symmetrically over the mercury X-ray peak. Five

hundred thousand counts were recorded in the total image. A five-projection study was made with the detector placed in the anterior (ANT), left anterior oblique (LAO) 30°, 45°, and 60°, and left lateral (LAT) projections. In several cases of CAD or chest pain syndrome, right anterior oblique (RAO) 30° was also recorded. Analog images were recorded with Polaroid film and a microdot imager without additional contrast enhancement or computer processing.

Unprocessed photoscans were read by two observers without reference to the clinical data. For the purpose of describing perfusion defects on the myocardial images, the LV images were graded as positive (evident defect), questionable (area of diminished activity but no real defect) or negative (normal). When a perfusion defect was visible, the projection in which the perfusion defect occupied the largest part of the LV circumference compared with other projections in each individual was decided.

When Tl-201 activity was visible in the region of the RV, concentration of the tracer activity was graded according to Cohen's criteria[8]: 0 (activity in the region of the RV equal to that of the background), 1+ (RV activity less than that of the LV free wall), 2+ (RV activity equal to that of the LV free wall), 3+ (RV activity greater than that of the LV free wall). When the RV free wall was visible, measurement of the thickness of the visible RV free wall was made from the view in which the RV was best separated from the LV. Measurement of the thickness of the interventricular septum and LV free wall, and the transeverse diameter of the LV cavity was made from the LAO 45° view. The transverse diameter of the overall LV image was calculated by summing up those three values. Measurement of both LV and RV images was undergone at the mid-point of each ventricular wall with magnification corrections calculated from the data of a phantom study.

RESULTS

Coronary artery disease

Of 63 patients with myocardial infarction (MI), 28 were acute or recent cases (within 30 days from the onset), and 35 were old or silent cases. Of the 63 patients with MI, 12 had no significant Q waves on the ECGs on the day of the scanning. These 12 patients included three with subendocardial MI, five with insignificant Q waves, and single cases with complete right bundle branch block and a pace-maker rhythm. The other two patients having angina pectoris without clinical evidence of MI showed definite perfusion defects on myocardial imaging, and could be considered to be silent MI cases (Fig. 1). When new attacks occured in patients with old MI, these patients were classified as acute cases. The two silent MI cases were classified into one group with old MI cases.

Of the 28 patients with acute or recent MI, 23(82%) had perfusion defects. Of the patients with acute or recent MI, only five were scanned within 14 days after the onset. Of the 35 patients with old or silent MI, 26(74)% showed perfusion defects (Table 2). Of 14 patients with chest pain syndrome without clinical evidence of CAD, none had a perfusion defect. Of the 12 patients without significant Q waves, only five patients were shown to have positive scans (Table 3). These five patients included two with insignificant Q waves, one with a pace-maker rhythm, and two with silent MI. On the other hand, of 51 patients with significant Q waves, 44 had perfusion defects ($P<0.01$ by χ^2 test with Yates' continuous correction).

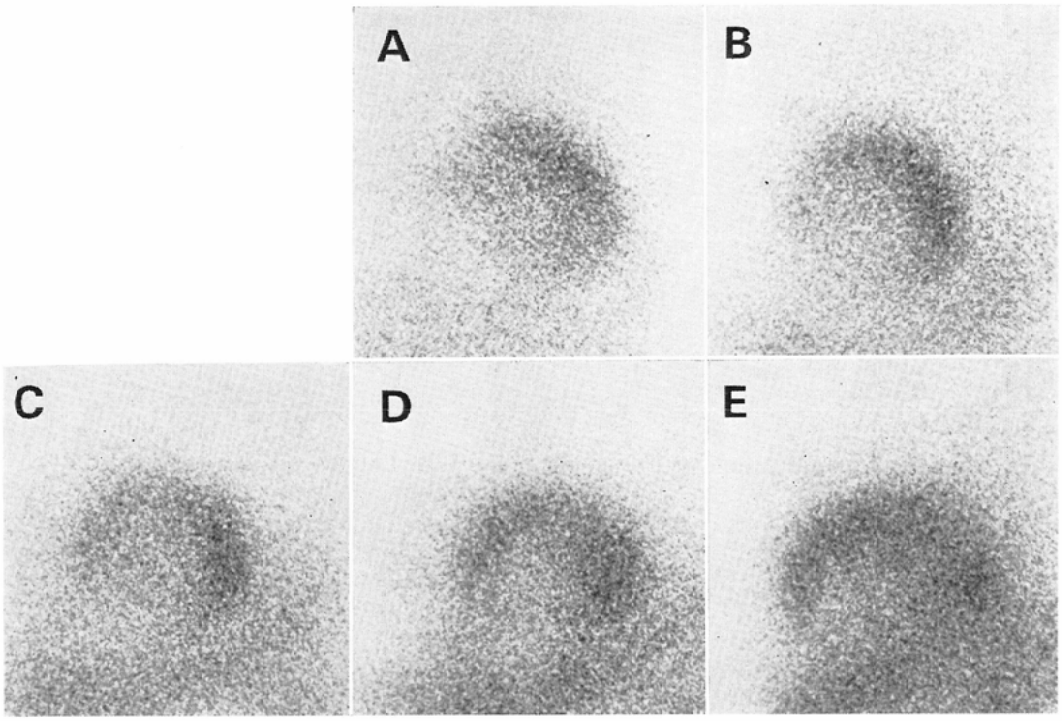

Fig. 1. A patient with angina pectoris having no evidence of myocardial infarction. Myocardial images, showing an extensive perfusion defect, indicate presence of silent myocardial infarction. A=ANT, B=LAO 30°, C=LAO 45°, D=LAO 60°, and E=LAT view.

Table 2. Perfusion defects in patients with chest pain syndrome

		Perfusion defect			
		Positive	Questionable	Negative	Total
Myocardial infarction	Acute Recent	23	3	2	28
	Old Silent	26	5	4	35
Angina pectoris		0	5	24	29
Chest pain syndrome without CAD		0	1	13	14

Left-ventricular image

The results of measurement of the LV images are listed in Table 4. The patients with chest pain syndrome without CAD were used as a control group. The interventricular septi in patients with AR were thicker than those of the control group ($P<0.01$). The LV free walls in patients with AR or hypertrophic cardiomyopathy were thicker than those of the control group ($P<0.05$). The LV cavities in patients with MI, AR, MR, or congestive cardiomyopathy were wider than those in the control group ($P<0.001$). The LV cavities in patients with ASD were smaller than those of the control group ($P<0.001$). Transverse diameters of the overall LV images were greater in patients with

Table 3. A comparison of myocardial imaging with electrocardiography performed on the same day in patients with myocardial infarction

		Abnormal Q wave	Perfusion defect	
			Positive	Questionable Negative
Acute Recent	MI	+	22	3
		−	1	2
Old Silent	MI	+	22	4
		−	4	5
Total		+	44	7
		−	5	7

Table 4. Measurement of left ventricular images in the LAO 45° view in patients with various heart diseases

Clinical diagnosis	No.	Thickness of interventricular septum	Diameter of LV cavity	Thickness of LV free wall	Diameter of overall LV image
Control (chest pain syndrome without CAD)	14	1.90±0.28	2.52±0.38	1.96±0.31	6.36±0.62
Myocardial infarction	47[a)]	1.97±0.24	3.24±0.98*	2.08±0.25	7.35±0.93*
Angina pectoris	29	1.90±0.22	2.45±0.51	2.11±0.32	6.45±0.49
Atrial septal defect	18	1.87±0.20	2.01±0.24*	1.84±0.17	5.64±0.40**
Aortic regurgitation	9	2.31±0.40***	4.06±0.79*	2.31±0.50****	8.68±1.32*
Mitral stenosis	22[b)]	1.83±0.18	2.53±0.49	1.99±0.24	6.35±0.69
Mitral regurgitation	6	1.99±0.23	3.42±0.49*	2.13±0.20	7.53±0.34*
Hypertrophic cardiomyopathy	23	2.18±0.48	2.25±0.41	2.25±0.41****	6.66±0.68
Congestive cardiomyopathy	5	2.06±0.13	3.42±0.32*	2.02±0.42	7.50±0.58**

Mean ± s.d. (cm)

*$P<0.001$; **$P<0.005$; ***$P<0.01$; ****$P<0.05$, compared with mean values of control group

a) sixteen cases could not be measured because of perfusion defects

b) three cases could not be measured because of markedly increased background activity

MI, AR, MR, or congestive cardiomyopathy ($P<0.005$–$P<0.001$), and smaller in patients with ASD ($P<0.005$) than those of the control group.

In several patients with MR, AR, or ASD, myocardial imaging was repeated after radical operation. The transverse diameters of preoperative overall LV images were compared with those of postoperative overall LV images (Fig. 2). In patients with MR or AR, the postoperative myocardial images tended to decrease in size (Figs., 3 and 4). In patients with ASD, the postoperative overall myocardial images were rather larger than before, and the RV cavities became smaller (Fig. 5).

Autopsy was performed in seven patients, including four with MI, single cases of primary pulmonary hypertension, chronic cor pulmonale, and combined valvular heart disease. Thickness of both ventricular free walls measured at autopsy was compared with that measured on myocardial imaging (Table 5). Both ventricular free walls on myocardial images were thicker than those measured at autopsy. The RV free walls less than 5 mm thick were not visualized on myocardial imaging.

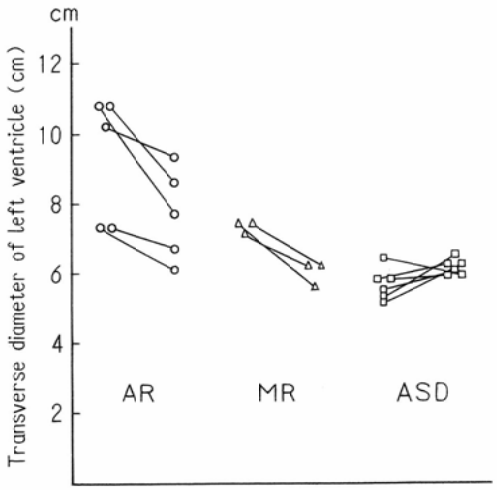

Fig. 2. Comparison of pre- and post-operative left ventricular diameters. Preoperative measurement is on the left side, and postoperative measurement on the right side. AR=aortic regurgitation, MR=mitral regurgitation, and ASD=atrial septal defect.

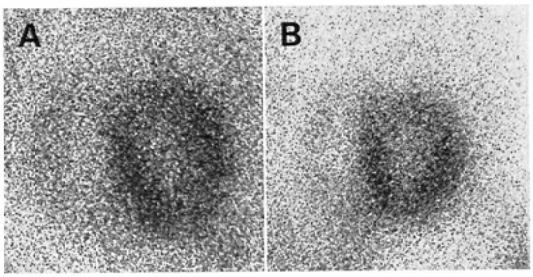

Fig. 3. A patient with mitral regurgitation. LAO 45° view, showing enlarged left ventricle and right ventricle preoperatively (A), and decreased left ventricular size and barely visible right ventricle postoperatively (B).

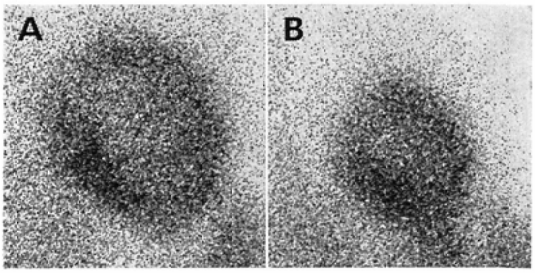

Fig. 4. A patient with aortic regurgitation. LAO 45° view, showing smaller left ventricular size in postoperative period (B) than preoperative period (A).

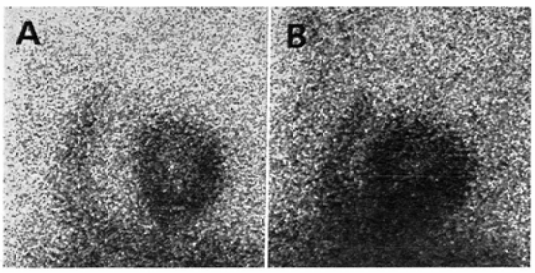

Fig. 5. A patient with atrial septal defect. (A) LAO 60° view, showing dilated right ventricle. (B) Postoperative LAO 60° view, showing shrinked rightventricular cavity and enlarged left ventricle.

Right-ventricular image

The RV free walls were visible in 82 patients, including 57 at grade 1+, 19 at grade 2+, and six at grade 3+. In Table 6, the data of cardiac catheterization are compared with the degree of RV visualization. The group with 1+ visualization had higher RV systolic pressures than the group with nonvisualization; the group with 2+ visualization had higher RV systolic pressures than the groups with 0 or 1+ visualization; and the group with 3+ visualization had higher RV systolic pressures than the groups with 0, 1+, or 2+ visualization ($P<0.01$–$P<0.001$). Several patients with nonvisralization, however, had abnormally high RV systolic pressures. Also, several patients with the visible RV free wall were shown to have normal RV systolic pressures. All patients with ASD

Table 5. Measurement of the both ventricular free walls on myocardial imaging and autopsy

Case No.	Diagnosis	LV free wall		RV free wall	
		Image	Autopsy	Image	Autopsy
1	Myocardial infarction	2.4	1.1	—	0.2
2	Myocardial infarction	2.4	1.0	—	0.4
3	Myocardial infarction	2.0	1.0	—	0.3
4	Myocardial infarction	2.0	1.0	1.8	0.5
5	Chronic cor pulmonale	2.3	1.8	+[a)]	0.6
6	Primary pulmonary hypertension	2.2	1.0	2.3	1.0
7	Combined valvular heart disease	2.0	1.4	2.2	1.2

in cm

a) Thickness could not be measured because of blurred margins

Table 6. A comparison of degree of right-ventricular visualization with findings of cardiac catheterization

	Degree of right-ventricular visualization				Significant differences
	0	1+	2+	3+	
Right-ventricular systolic pressure (mmHg)	24.4±6.5 (89)	36.3±10.7 (57)	59.5±31.5 (19)	120.0±27.7 (6)	$P<0.01$–$P<0.001$
Right-ventricular end-diastolic pressure (mmHg)	5.0±2.2* (86)	5.7±2.7 (56)	7.1±3.4* (19)	11.2±6.9 (6)	*$P<0.001$
Mean pulmonary arterial pressure (mmHg)	14.4±5.0*# (89)	22.0±8.2* (54)	32.0±18.0# (15)	70.0 (3)	*$P<0.001$, #$P<0.05$
Total pulmonary vascular resistance (dyne-sec/cm^5)	223.4±114.5* (85)	409.0±253.5* (47)	412.5±443.6 (13)	2117.0 (3)	*$P<0.001$
Stroke-work index of right ventricle (gram-meters/m^2)	13.7±6.1 (82)	20.±09.6 (49)	37.9±18.7 (13)	42.1 (4)	$P<0.01$–$P<0.001$
Stroke-work index of left ventricle (gram-meters/m^2)	77.2±25.7* (80)	71.0±55.5 (47)	55.5±24.2* (12)	69.5 (2)	*$P<0.01$

Mean±s.d. (number of cases)

having visible RV free walls had significant RV volume overload caused by left-to-right shunt. However, these patients were shown to have relatively lower RV systolic pressures than other patients in each visualization group. Therefore, presence of RV overloading was diagnosed by documentation of at least one of the following criteria[11]: (1) RV systolic pressure was 36 mmHg or more, (2) total pulmonary vascular resistance of 310 dyne-sec/cm^5 or more, and (3) presence of significant RV volume overload. Then, false positive cases (namely, having RV visualization without RV overload) were

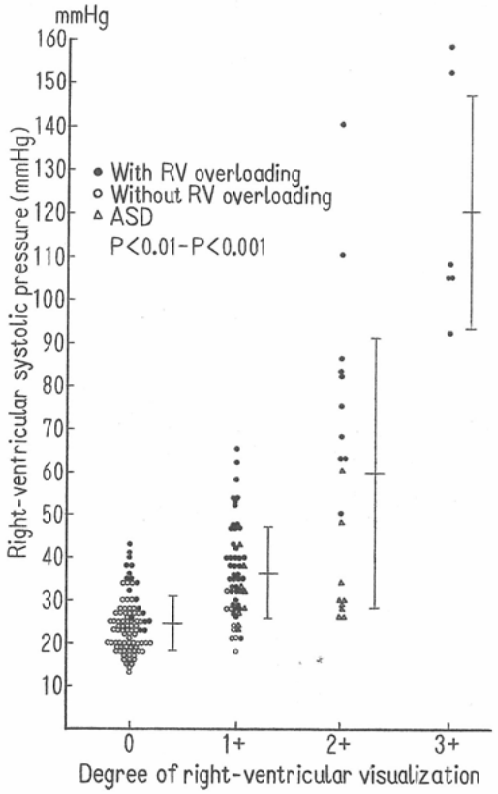

Fig. 6. Relationship between degree of right-ventricular visualization and right-ventricular systolic pressure. Presence of right-ventriclar overloading is judged by strict criteria. All cases of atrial septal defect have right-ventricular volume overload.

eight, and false negative cases were 18. The sensitivity to detect RV overloading by visualization of the RV free wall was 80%, and the specificity was 90% (Fig. 6). These criteria for RV overload were rather strict, because these values are over 95% confidence levels of normal subsjects. The sensitivity and specificity would vary depending on the criteria for RV overload used. When the presence of RV overload was diagnosed by documentation of RV systolic pressure of 30 mmHg or more, and/or total pulmonary vascular resistance of 310 dyne-sec/cm^5 or more, and/or significant RV volume overload, the sensitivity was 90%, and the specificity was 90%. When the presence of RV overload was diagnosed by documentation of RV systolic pressure of 30 mmHg or more, and/or significant RV volume overload, the sensitivity was 80%, and the specificity was 84%.

These 173 patients with cardiac catheterization were divided into two groups; one with a disease causing significant LV overloading such as AR, MR, etc. and one without LV overloading. Then the sensitivities for detecting RV overloading in these two groups with or without LV overloading were 48% and 90%, respectively; and the specificities were 85% and 92%, respectively, when the strict criteria for RV overloading were used (Table 7).

Table 7. A comparison of right-ventricular visualization with vight-ventricular overloading in patients with or without left-ventricular overloading

LV overloading	RV visualization	RV overloading	
		+	−
+	+	11	3
	−	12	17
−	+	63	5
	−	6	56
Total	+	74	8
	−	18	73

Table 8. Incidence of perfusion defects in five projections in patients with myocardial infarction

Location of MI on ECG	No.	No. with defect	ANT	LAO 30°	LAO 45°	LAO60°	LAT
Anteroseptal	10	8	5	5(2)	8(1)	8(4)	5(1)
Strictly anterior	3	1	1(1)	1	1	1	0
Anterolateral	7	6	5(2)	4(2)	2(1)	2	3(1)
Extensive anterior	8	8	5	4(2)	5(1)	5(2)	4(3)
Inferior	12	12	3(1)	5(1)	8(2)	7(3)	6(5)
Inferolateral	4	3	1	1	2	2(1)	3(2)
Posterolateral	1	1	1	1	1(1)	1	0
Combined*	6	5	2	2(1)	3(1)	2(2)	2(1)
Negative on ECG	12	5	1	2(1)	3(1)	4(3)	2
Total	63	49	24(4)	25(9)	33(8)	32(15)	25(13)

( ): Number of cases showing largest perfusion defect in that projection
*Include four with inferior and anteroseptal MIs, and two with inferior and anterolatral MIs

Value of multiple-projection study

The next study was undertaken to evaluate the value of multiple-projection studies on myocardial imaging for detecting perfusion defects or the visible RV free walls. All patients with MI were divided into ten groups according to the locations on the basis of ECGs' findings.[12] In 49 patients with a perfusion defect, overall incidences of perfusion defects appearing in the ANT, LAO 30°, 45°, 60°, and LAT views were 24(49%), 25(51%), 33(67%), 32(65%), and 25(51%), respectively (Table 8). Incidences of the largest perfusion defect in each individual appearing in the ANT, LAO 30°, 45°, 60°, and LAT views were four (8%), nine (18%), eight (16%), 15(31%), and 13(27%), respectively. In eight patients, a perfusion defect could be detected in only one projection, including single cases of inferior MI and anterolateral MI in the ANT view, single cases of inferior MI and anterolateral MI in the LAO 45° view, one with inferior MI in the LAO 60° view, and single cases of extensive anterior MI, anterolateral MI, and inferolateral MI in the LAT view. In one patient with anterior MI having negative or questionable scintiphotos in the five projection study, a definite perfusion defect was revealed in the RAO 30° view (Fig. 7).

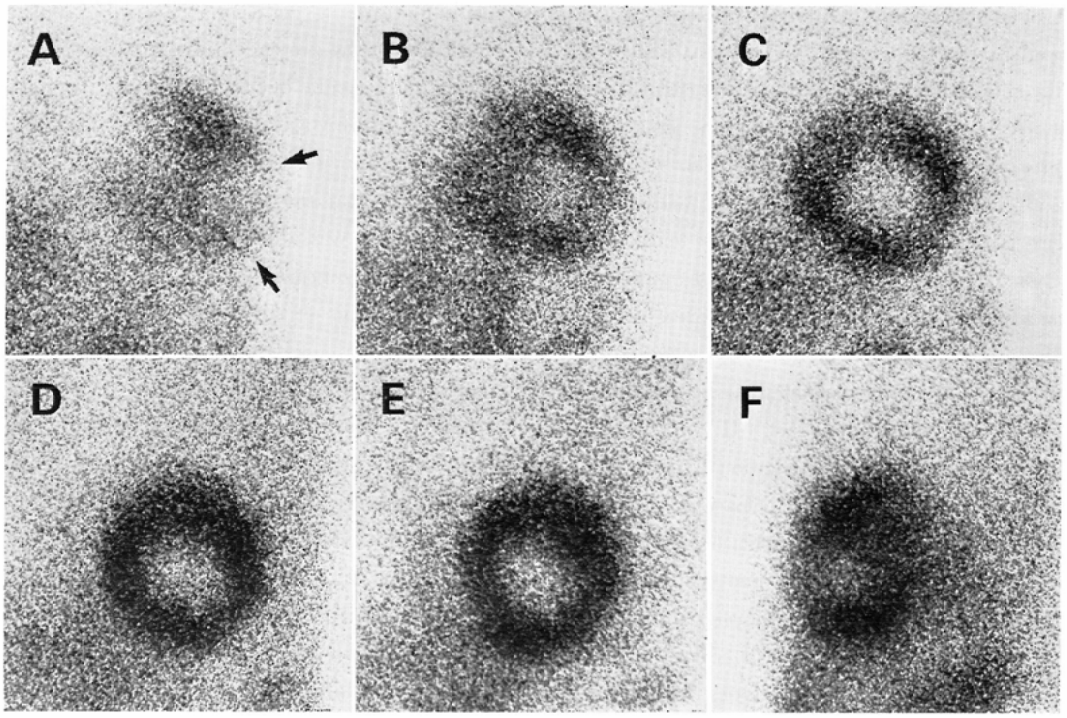

Fig. 7. A patient with anterior myocardial infarction. A=RAO 30°, B=ANT, C=LAO 30°, D=LAO 45°, E=LAO 60°, and F=LAT view. A perfusion defect is clearly visible in RAO 30° view (arrows), and negative or questionable in other projections.

Table 9. Incidence of the best visibility of the right-ventricular free wall in one of five projections in each patient

RV visualization	ANT	LAO 30°	LAO 45°	LAO 60°	LAT
1+	0	3(2)	16(6)	38(12)	0
2+	0	0	5	12	2
3+	0	1	1	2	2
Total	0	4(2)	22(6)	52(12)	4

( ) number of cases showing the RV free wall only in that projection

In each individual studied, only one projection of the five views showed the best visibility of the RV free wall (Table 9). The RV free wall was best visible in the LAO 30°, 45°, 60°, and LAT view, in four (5%) 22(27%), 52(63%), and four (5%) cases, respectively. In 20 cases showing grade 1+, the RV free wall was visible in only one projection; two, six, and 12 cases, in the LAO 30°, 45°, 60° view, respectively. In patients with grade 2+ or 3+, the RV free wall was visible in at least two projections.

DISCUSSION

Thallium-201 myocardial imaging permits diagnosis of MI by revealing a perfusion defect in the

LV circumference. Although transient ischemic attack can cause a perfusion defect, myocardial imaging showing a perfusion defect at rest would be an indicator of the presence of necrotic masses.[3)-6)] The sensitivities for detecting perfusion defects in patients with acute or old MI were somewhat identical, probably because present cases were not submitted to myocardial imaging during very acute phases of MI. Wackers et al.[3)] observed that myocardial imaging showed a perfusion defect in all the patients with acute MI within 6 hr after onset, and that the incidence of positive scan decreased thereafter. However, the sensitivity of the present study was almost identical with that of ECGs on the day of the scanning. In patients with MI having no significant Q waves on the day of the scanning, myocardial imaging tended to be invaluable. Hamilton et al.[6)] also reported similar correlation between diagnostic Q waves and image defects in patients with suspected CAD. On the other hand, myocardial imaging a definite and extensive perfusion defect in patients with angina pectoris and no evidence of MI on clinical basis. These patients probably were silent MI cases, although histological confirmation has not been available. Myocardial imaging also will be extremely helpful in diagnosing MI in patients with abnormal conduction patterns.[3)] Moreover, myocardial imaging has potentials to evaluate size and location of necrotic myocardium with more accuracy than ECG.[5)] These potentials come from the fact that myocardial imaging permits the extent of a perfusion defect visible in multiple projections and enables three-dimentional assessment.

In patients with volume overloading such as AR or MR, and in patients with MI or congestive cardiomyopathy, the LV cavities and overall LV images were larger than those in the control group. The reason why the LV images were larger in patients with MI was probably that this group might include patients with coronary cardiomyopathy, congestive heart failure, or longstanding history of hypertension. In patients with AR or hypertrophic cardiomyopathy, the LV free walls were thicker than those of control group. These findings were probably due to contractile wall motion and/or LV hypertrophy. On the contrary, in patients with ASD, the LV cavities and overall LV images were smaller than those in the control group. On comparison of myocardial images and autopsies, measurement of images of both ventricular free walls did not represent true values, probably because of the contractile movement of the walls, and the limited analytical capability of the equipment in defining thickness in millimeters. On the other hand, in patients with AR or MR, the enlarged LV images decreased in size after valvular replacement. In patients with ASD, the LV cavities tended to increase in size after radical operations, and the RV cavities decreased in size. These results indicated that myocardial imaging would be useful for evaluation of LV overloading or unloading, as well as perfusion defects, although further studies are needed.

Although the right ventricle is usually not well visualized during myocardial imaging, Cohen er al.[8)] and Kondo et al.[9)] reported that this technique was a sensitive and specific method for detecting RV overload judged by the presence of the visible RV free wall. It was more useful than the electrocardiographic criteria for the determination of RVH,[8)13)14)] and than the echocardiography and chest X-ray.[13)] Also myocardial imaging provides distinguishing characteristics between RV pressure and volume overloading.[9)] Among many of the cases with RV pressure overloading, the septum appeared straight. There were also occasional cases showing tilting of the straightened septum, which may be due to RV hypertophy and/or dilatation. In cases of RV volume overloading due to

ASD, the RV cavity appeared dilated, and the septum displayed a convexity into the right ventricle.

In the present study treating a larger number of cases than before, similar results were obtained. The sensitivity and specificity for detection of RV overloading varied depending on the criteria of hemodynamics for the determination of RV overloading. The sensitivity was between 80% and 90%, and the specificity was between 84% and 90%. Rabinovitch et al.[15] predicted that in case of biventricular hypertrophy, LV hypertrophy might obscure concurrent RV hypertrophy. Indeed, it was revealed that in the presence of LV overloading, the sensitivity for detecting RV overloading was significantly smaller than in the absence of LV overloading. Because LV overloading would make the tracer more densely taken up by the LV myocardium than usual, and the preset counts were fixed at 500,000. Therefore, when relatively increased amount of the tracer accumulated into the LV myocardium, myocardial imaging might cease when insufficient counts were gained to visualize the RV free wall.

Rabinovitch et al.[15] tested the sensitivity of Tl-201 to detect and quantitate RVH as a function of diminishing LV/RV ratio in hypoxic rats, and observed that Tl-201 activity ratios correlated well with those obtained by ventricular weights. In the present study, higher the degree of RV visualization, higher were the RV systolic pressure and work of the right ventricle. Longstanding RV overloading would cause RVH. Although the RV free wall visualization might occur in the presence of RV overloading without an accompanying increase in RV mass, each group with the visible RV free wall probably had more RV masses than the lower grade groups. Among the seven patients autopsied, four RV free walls 0.5 cm or more thick were visualized in vivo which is the usual crietria for RVH.[16]

In myocardial imaging, a multiple-projection study is necessary to avoid underdiagnosis. In patients with a perfusion defect, the view in which the perfusion defect was shown to be largest than in other projections differed from patient to patient. Therefore, when a small number of projections were used for myocardial imaging, the same perfusion defect could be undetectable or be underestimated according to their extent. In addition, when the RV free walls were visible, 24% of these cases were seen in only one of the LAO projections in each individual. The LAO 60° view had the largest incidence of the visible RV free wall as well as the largest incidence of visualization of the RV free wall in only one projection. Again, for detecting perfusion defects, the RAO 30° view would have some value. Therefore, it would be desirable to perform myocardial imaging in as many projections as possible.

Conclusively, although ECG gated myocardial imaging might be desirable to assess both ventricles, ungated myocardial imaging would have a great value for evaluating CAD, assessing LV and/or RV overloading, and following process of the patients with or without operation.

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Requests for reprints should be addressed to:
Makoto Kondo, M.D.
Department of Radiology
Keio University School of Medicine
35 Shinanomachi, Shinjuku-ku, Tokyo 160, Japan

REFERENCES

1) Lebowitz, E., Greene, M.W., Fairchild, R., Bradley-Moore, P.R., Atkins, H.L., Ansari, A.N., Richards, P. and Belgrave, E.: Thallium-201 for medical use. 1. J. Nucl. Med., 16: 151—155, 1975
2) Bradley-Moore, P.R., Lebowitz, E., Greene, M.W., Atkins, H.L. and Ansari, A.N.: Thallium-201 for medical use. II: Biologic behavior. J. Nucl. Med., 16: 156—160, 1975
3) Wackers, F.J., Sokole, E.B., Samson, G., Schoot, J.B., Lie, K.I., Liem, K.L. and Wellens, H.J.: Value and limitations of thallium-201 scintigraphy in the acute phase of myocardial infarction. N. Engl. J. Med., 295: 1—5, 1976
4) Bailey, I.K., Griffith, L.S., Rouleau, J., Strauss, H.W. and Pitt, B.: Thallium-201 myocardial perfusion imaging at rest and during exercise. Comparative sensitivity to electrocardiography in coronary artery disease. Circulation, 55: 79—87, 1977
5) Wackers, F.J., Becker, A.E., Samson, G., Sokole, E.B., Shoot, J.B., Vet, A.J., Lie, K.I., Durrer, D. and Wellens, H.: Location and size of acute transmural myocardial infarction estimated from thallium-201 scintiscans. A clinicopathological study. Circulation, 56: 72—78, 1977
6) Hamilton, G.W., Trobaugh, G.B., Ritchie, J.L., Weaver, W.D., and Gould, K.L.: Myocardial imaging with intravenously injected thallium-201 in patients with suspected coronary artery disease. Analysis of technique and correlation with electrocardiographic, coronary anatomic and ventriculographic findings. Am. J. Cardiol., 39: 347—354, 1977
7) Ritchie, J.L.: A review of myocardial imaging. In Ritchie, J.L., Hamilton, G.W. and Wackers, F.J., ed.: "Thallium-201 Myocardial Imaging," pp. 1—8, 1978, Raven Press, New York
8) Cohen, H.A., Baird, M.G., Rouleau, J.R., Fuhrmann, C.F., Bailey, I.K., Summer, W.R., Strauss, H.W. and Pitt, B.: Thallium 201 myocardial imaging in patients with pulmonary hypertension. Circulation, 54: 790—795, 1976
9) Kondo, M., Kubo, A., Yamazaki, H., Ohsuzu, F., Handa, S., Tsugu, T., Masaki, H., Kinoshita, F. and Hashimoto, S.: Thallium-201 myocardial imaging for evaluation of right-ventricular overloading J. Nucl. Med., 19: 1197—1203, 1978
10) Bulkley, B.H., Rouleau, J., Strauss, H.W. and Pitt, B.: Idiopathic hypertrophic subaortic stenosis: Detection by thallium 201 myocardial perfusion imaging. N. Engl. J. Med., 293: 1113—1116, 1975
11) Barratt-Boyes, B.G. and Wood, E.H.: Cardiac output and related measurements and pressure values in the right heart and associated vessels, together with an analysis of the hemodynamic response to the inhalation of high oxygen mixtures in healthy subjects. J. Lab. Clin. Med., 51: 72—90, 1958
12) Lipman, B.S., Massie, E. and Kleiger, R.E.: "Clinical Scalar Electrocardiography," 6th Ed, pp. 218—225, 1972, Year Book Medical Publishers, Chicago
13) Khaja, F., Alam, M., Goldstein, S., Anbe, D.T. and Marks, D.S.: Diagnostic value of visualization of the right ventricle using thallium-201 myocardial imaging. Circulation, 59: 182—188, 1979
14) Ohsuzu, F., Handa, S., Kondo, M., Yamazaki, H., Tsugu, T., Kubo, A., Takagi, Y. and Nakamura, Y.: Thallium-201 myocardial imaging for evaluation of right ventricular overloading: A comparative study with EKG and hemodynamics. (Unpublished)
15) Rabinovitch, M., Fisher, K., Gamble, W., Reid, L. and Treves, S.: Thallium-201: Quantitation of right ventricular hypertrophy in chronically hypoxic rats. Radiology, 130: 223—225, 1979
16) World Health Organization: Some present practices concerning anatomical criteria for right ventricular hypertrophy and for emphysema. In Chronic Cor Pulmonale. Report of an Expert Committee. WHO Tech. Rep. Ser. No, 213, pp. 34—35, World Health Organization, Geneva, 1961