Site and severity of coronary narrowing and infarct size in man

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SUMMARY The relation between the site and severity of coronary artery lesion and infarct size was investigated in 59 patients with acute myocardial infarction. All patients had no prior myocardial infarction and had at least one significant coronary narrowing ($\geq 75\%$) in one of the major coronary arteries or in the first diagonal branch. Left ventriculography and selective coronary arteriography were performed on average 2.2 months after the onset of infarction to identify the site and severity of coronary narrowing and to assess the extent of the non-contracting segment (akinetic, dyskinetic, or aneurysmal). Thirty-four of 59 patients were studied enzymatically and total CK released was taken as an indication of infarct size.

Non-contracting segment and total CK released in group L-I (narrowing proximal to the first diagonal branch) were significantly larger than those in group L-II (a coronary lesion distal to the branch). The data also indicate that the perfusion area of the first diagonal branch is as large as that of the left anterior descending artery below the first diagonal branch. In contrast to left anterior descending artery disease, the involvement of the right ventricular branch did not significantly influence the infarct size. However, infarct size was significantly larger in eight patients with the left ventricular branch of the right coronary artery supplying the predominantly large area of posterior wall of the left ventricle than in nine patients with small left ventricular branches. It was also shown that the severity of coronary narrowing does not correlate with the infarct size in either left anterior descending or right coronary artery disease.

Recently many attempts have been made to reduce the infarct size in patients with acute myocardial infarction since infarct size determines very largely the cardiac function and prognosis.¹⁻³ One of the major factors related to infarct size and cardiac performance, however, is the coronary artery involved as well as the site of the coronary lesions, both of which have been thought to influence the ischaemic area directly.^{4,5} Though the relation between the site of the coronary occlusion and the infarct size has been studied experimentally,⁶ this relation in patients with acute myocardial infarction remains to be elucidated. Histologically assessed infarct size in postmortem studies does not reflect the infarct size in the acute phase; it may be modified during the postinfarction period, sometimes it may decrease during the healing process, and sometimes increase during

clinically undetectable extension of ischaemia. In this study the relation between infarct size, which was assessed from the total CK released or from the extent of non-contracting segment in the left ventriculograms, and the site and severity of the coronary lesions which were evaluated by selective coronary arteriography undertaken within six months (average 2.2 months) after the onset of infarction, was investigated in 59 patients with acute myocardial infarction.

Subjects and methods

Seventy-one patients (average age 51.6 years) with acute primary myocardial infarction, who had left ventriculography and selective coronary arteriography within six months of the attack, were studied. Four patients whose left ventriculograms were not suitable for quantitative evaluation of wall motion

Table 1 Involved coronary artery and infarct size in 59 patients with significant ($\geq 75\%$) coronary narrowing

With non-contracting segment		Without non-contracting segment	
Infarct site and involved vessel	No. of patients	Infarct site and involved vessel	No. of patients
Anterior myocardial infarction	30	Anterior myocardial infarction	
Left anterior descending group L-I	10	Left anterior descending	3
group L-IIa	9		
group L-IIb	11		
Inferior myocardial infarction	19	Inferior myocardial infarction	
Right coronary artery group R-L	8	Right coronary artery	2
group R-S	9		
Left circumflex	2	Left circumflex	0
Lateral myocardial infarction		Lateral myocardial infarction	
First diagonal branch	2	First diagonal branch	3
Total	51	Total	8

and eight patients who had insignificant coronary narrowing ($< 75\%$ in diameter) were excluded from this study (Table 2). The remaining 59 patients (52 men and seven women) with an average age of 52.9 years, consisted of 33 with anterior infarction, 21 with inferior infarction, and five with lateral infarction (Tables 1 and 3). In these 59 patients, 44 had single vessel disease. In 15 patients with multiple vessel disease, a coronary lesion whose location corresponded to the electrocardiographic changes in standard 12 leads, was studied in relation to the infarct size. Total CK released was obtained in 34 of 59 patients as an indication of infarct size by the method of Sobel *et al.*⁷ modified by Norris *et al.*⁸ Peripheral venous blood for CK determination was drawn every four hours for the first 24 hours and then at six-hourly intervals until serum CK activity returned to near normal levels.⁹ The total CK released which directly reflects the infarct size was calculated as follows:

$$\int_0^t f(t)dt = E(t) + K \int_0^t E(t)dt$$

where, $E(t)$ represents the instantaneous serum CK activity after subtracting the individual basal serum CK activity and $f(t)$ is the appearance rate of myocardial CK released from the injured myocardium into the circulation. The disappearance rate (K) was obtained individually from the terminal portion of the disappearance curve ($E(t)$) plotted semilogarithmically.

All patients had left ventriculography and selective coronary arteriography on average 2.2 months (one to six months) after the onset of infarction. Left ventriculography was performed after right and left cardiac catheterisation with premedication by intramuscular injection of hydroxyzine 25 mg. After the measurement of cardiac output by the

Table 2 Infarct site in eight patients without significant coronary narrowing

With non-contracting segment		Without non-contracting segment	
Infarct site	No. of patients	Infarct site	No. of patients
Anterior myocardial infarction	2	Anterior myocardial infarction	1
Inferior myocardial infarction	0	Inferior myocardial infarction	3
Lateral myocardial infarction	0	Lateral myocardial infarction	2
Total	2	Total	6

dye dilution method, a 30° right anterior oblique (RAO) projection of the left ventriculogram or both RAO and left anterior oblique (LAO) projections were obtained by injection of 40 to 50 ml sodium diatrizoate (Urografin 76), with exposure of 60 frames/s on 35 mm cine-film by Cardiodiagnost (Phillips Co).

Ventricular wall motion was evaluated and classified according to the criteria of the American Heart Association¹⁰: hypokinesis (reduced), akinesis (none), dyskinesis, and aneurysm of left ventricle. The segment of abnormal contraction was also expressed according to this classification: region 1 to 5 in RAO projection and region 6 to 7 in LAO projection. In order to identify the akinetic, dyskinetic, and aneurysmal segment, end-diastolic

Table 3 Number of involved coronary arteries in 59 patients with significant ($\geq 75\%$) narrowing

	Myocardial infarction		
	Anterior	Inferior	Lateral
Single vessel disease	29	13	2
Double vessel disease	4	6	3
Triple vessel disease	0	2	0
Total	33	21	5

and end-systolic silhouettes of the left ventricular chamber for a single beat were superimposed and the ratio of the extent of the non-contracting segment (akinetic, dyskinetic, or aneurysmal segment) of the ventricle to the total end-diastolic circumference was calculated as the non-contracting segment¹¹ (see Fig. 1). In patients who had left ventriculography in two projections (RAO and LAO), the non-contracting segment was calculated as a mean value on these two projections. Segment

Fig. 1 Measurement of the extent of the non-contracting segment.

of hypokinesis was not included in the non-contracting segment because the extent of this disorder of movement cannot be quantitatively assessed.

Ten patients who had a significant ($\geq 75\%$ in diameter) narrowing proximal to the first diagonal branch and also had a non-contracting segment in the anterior wall or apical portion of the left

ventricle were included in group L-I. Twenty patients who had the non-contracting segment in the anterior wall or apical portion but showed a significant narrowing distal to the first diagonal branch were included in group L-II. The latter group was further divided into two groups: group L-IIa (nine patients) with the coronary lesion between the origin of the first diagonal branch (DX_1) and that of the first septal branch (S_1), and group L-IIb (11 patients) with the lesion distal to the position of the first septal branch (see Table 1 and Fig. 2).

In right coronary artery disease nine patients with the narrowing proximal to the right ventricular branch (RV in Fig. 2) were included in group R-I, and eight patients with the coronary lesion in the distal portion below the right ventricular branch were classified as group R-II. Moreover, these 17 patients with right coronary artery disease were divided into two groups according to the relative size of area in the posterior wall of the left ventricle supplied by the right coronary artery. Group R-L included eight patients where the posterior wall of the left ventricle was predominantly (more than two-thirds of the area) supplied by the left ventricular branch of the right coronary artery and the posterolateral branch of the left circumflex artery was small or rudimentary. One patient with a large posterior descending branch distributed to the apical area of the left ventricle was also included in this group. Group R-S included nine patients with a small left ventricular branch which perfused less than two-thirds of the posterior wall.

In this series, 19 of 21 patients with inferior infarction had significant narrowing in the right coronary artery, all of them showing a predominant

Fig. 2 Location of the coronary lesion and classification of groups. Proximal is group L-I; and distal is group R-II.

Table 4 Values in six patients with single lesion in first diagonal branch (lateral infarction)

Case no.	Age (y)	Sex	Coronary arteriogram findings (%)	Abnormal motion of ventricular wall	Total CK (IU/ml)	Non-contracting segment (%)
1	54	M	Left anterior descending 50 First diagonal branch 50	Hypokinesis 2, 3	691	—
2	45	M	First diagonal branch 99	Hypokinesis 2, 3	—	—
3	55	F	First diagonal branch 75	Hypokinesis 2, 7	333	—
4	42	M	First diagonal branch 100	Hypokinesis 2	—	—
5	54	F	First diagonal branch 100	Dyskinesis 2, 3	771	19.2
6	62	M	First diagonal branch 100 Left circumflex 90	Dyskinesis 2, 3 Hypokinesis 7	956	17.7

right coronary artery. The remaining two patients had an occlusion in the left circumflex artery. Five patients had significant narrowing in the first diagonal branch and all of them had lateral infarction (Tables 1 and 4). One patient with less than 75 per cent coronary narrowing but with a hypokinetic segment in regions 2 and 3 is also included in the lateral infarction group (Table 4).

Results

(1) LOCATION AND SEVERITY OF CORONARY NARROWING IN LEFT CORONARY ARTERY AND INFARCT SIZE

All 33 patients with significant narrowing in the left anterior descending artery showed impaired wall motion in the anterior wall or apex of the left ventricle and 30 of these patients showed akinesis, dyskinesis, or aneurysm of the anterior wall. Mean non-contracting segment in groups L-I, L-IIa, and L-IIb were 35.7 ± 1.3 , 26.4 ± 3.0 , and 21.4 ± 2.7 per cent, respectively, indicating that the extent of infarction in group L-I is significantly larger than in groups L-IIa and L-IIb ($p < 0.01$, see Table 5). These groups showed insignificant differences in age and the incidence of collaterals, suggesting that the difference in the extent of the non-contracting segment is primarily the result of the difference in the site of the coronary lesion. The total CK released also showed that the infarct size in group L-I (total CK 1510 ± 190 IU/ml, $n=7$) was significantly larger ($p < 0.01$) than that in group

L-IIa (total CK 765 ± 50.4 IU/ml, $n=5$), or in group L-IIb (total CK 841 ± 49.5 IU/ml, $n=4$), though there was no difference in infarct size between groups L-IIa and L-IIb. These results suggest that a coronary lesion proximal to the first diagonal branch causes a large infarct and that involvement of the first septal branch may not primarily influence the infarct size. Fig. 3 and 4 show the difference in the extent of the non-contracting segment and the total CK released between groups L-I and L-II. These figures show the significant difference ($p < 0.01$) in infarct size between those with and those without involvement of the first diagonal branch; the mean total CK released in patients with a coronary lesion proximal to the first diagonal branch is approximately twice as great as that in patients with distal coronary lesions in the left anterior descending artery. This indicates that the size of the perfusion area of the first diagonal branch is almost similar to that of the left anterior descending artery distal to the first diagonal branch.

Six patients had narrowing only in the first diagonal branch (Table 4), including one patient (case 1) who was excluded from the statistical analysis because of the absence of significant coronary narrowing ($>75\%$). All had lateral infarction and four patients showed hypokinesis in region 2, while the remaining two had dyskinetic movement in the same region. The extent of the non-contracting segments in these two patients was 19.2 and 17.7 per cent and the mean total CK

Table 5 Location of coronary lesion in left anterior descending artery and infarct size (mean $\pm$ SE)

Group	No. of patients	Mean age (y)	Incidence of collateral (%)	Non-contracting (%)		Total CK (IU/ml)
				RAO	RAO + LAO	
L-I	10	53.8	40	35.7 ± 1.3	36.8 ± 2.2 ($n=7$)	1510 ± 190 ($n=7$)
L-IIa	9	53.0	56	26.4 ± 3.0	28.8 ± 3.5 ($n=8$)	765 ± 50.4 ($n=5$)
L-IIb	11	51.0	36	21.4 ± 2.7	23.8 ± 4.5 ($n=6$)	841 ± 49.5 ($n=4$)

* $p < 0.01$.

released obtained in four patients studied was 688 IU/ml, which was nearly equal to the difference (711 IU/ml) in the mean total CK released in group L-I and L-II; this confirms the result above that the size of the perfusion area of the first diagonal branch is as large as that of the distal left anterior descending artery.

Fig. 3 Location of the coronary lesion in the left anterior descending artery and the extent of the non-contracting segment (NCS). Group L-I includes the patients with a significant ($\geq 75\%$) narrowing proximal to the first diagonal branch and group L-II includes the patients with a coronary lesion ($\geq 75\%$) distal to this branch.

Fig. 4 Location of the coronary lesion in the left anterior descending artery and the total CK released (Σ CK). (Groups L-I and L-II are the same as those in Fig. 3.)

In two patients with a significant lesion in the left circumflex artery, one had the balanced type of coronary distribution and the other had a predominant left coronary artery. In the former patient the extent of the non-contracting segment was 30.2 per cent and total CK was 976 IU/ml, and the latter had hypokinesis in region 2 with a total CK of 884 IU/ml.

Fig 5 depicts the relation between the severity of coronary narrowing and the extent of the non-contracting segment in each of the groups L-I and L-II. It shows no significant difference in the extent of infarction resulting from the difference in the severity of narrowing.

(2) LOCATION AND SEVERITY OF NARROWING IN RIGHT CORONARY ARTERY AND INFARCT SIZE

The non-contracting segment (akinesis, dyskinesis, or aneurysm) was observed in 17 of 19 patients with significant narrowing in the right coronary artery. The mean non-contracting segments in group R-I (proximal) and R-II (distal) were 20.4 ± 2.1 per cent and 23.7 ± 3.1 per cent, respectively (Fig. 6). The mean total CK in these two groups also showed no significant difference: 1238 ± 59.3 IU/ml in group R-I and 1070 ± 104 IU/ml in group R-II (Table 6). These results suggest

Fig. 5 Severity of coronary narrowing and extent of the non-contracting segment (NCS). (Groups L-I and L-II are the same as in Fig. 3.)

Table 6 Location of coronary lesion in right coronary artery and infarct size (mean $\pm$ SE)

Group	No. of patients	Mean age (y)	Incidence of collateral (%)	Non-contracting segment (%)		Total CK (IU/ml)
				RAO	RAO+LAO	
R-I	9	51.8	78	20.4 $\pm$ 2.1	18.9 $\pm$ 2.1 (n=7)	1238 $\pm$ 59.3 (n=4)
R-II	8	56.9	75	23.7 $\pm$ 3.1	23.8 $\pm$ 4.6 (n=5)	1070 $\pm$ 104 (n=7)

that infarct size is not influenced by involvement of the right ventricular branch which is the main branch of the right coronary artery. The mean non-contracting segments and total CK in group R-L (where the left ventricular branch predominantly perfuses the posterior wall of the left ventricle), however, were 27.6 per cent and 1297 IU/ml, respectively, which are significantly larger than those in group R-S (where only a minor part of the posterior wall is perfused by the left ventricular branch of the right coronary artery): 18.2 per cent for the mean non-contracting segment and 920 IU/ml for the mean total CK (Fig. 7). This indicates that the size of the perfusion area of the right coronary artery in the left ventricle mainly determines the infarct size in patients with right coronary artery disease.

Fig. 6 Location of the lesion in the right coronary artery and the extent of the non-contracting segment (NCS). In group R-I the narrowing of the right coronary artery is proximal to the right ventricular branch and in group R-II distal to the right ventricular branch.

Fig. 7 Distribution area of the right coronary artery and infarct size (NCS and Σ CK). The patients in group R-L had the large left ventricular branch of the right coronary artery supplying the predominantly large area of the posterior wall of the left ventricle. The patients in group R-S had a minor left ventricular branch which supplied less than one-third of the posterior area of the left ventricle.

Fig. 8 shows the relation between the severity of coronary narrowing and the extent of the non-contracting segment. There was no significant difference in the mean non-contracting segment between the patients with subtotal or complete occlusion in the right coronary artery. As shown in the case of the left anterior descending artery, the severity of narrowing in the right coronary artery did not influence the infarct size.

(3) LOCATION OF CORONARY LESION AND CARDIAC FUNCTION

Table 7 shows the pertinent haemodynamic indices in patients with significant narrowing in the left anterior descending artery (group L-I and L-II) and the right coronary artery (groups R-L and R-S). In 30 patients with left anterior descend-

Fig. 8 Severity of coronary narrowing in the right coronary artery and the extent of the non-contracting segment (NCS).

ing artery disease, the mean ejection fractions in group L-I (proximal) and L-II (middle and distal) were 32.1 ± 3.1 and 46.8 ± 2.1 per cent, respectively. Though the cardiac index showed no significant difference between the two groups, the left ventricular end-diastolic volume was significantly larger in group L-I, suggesting that the cardiac index was compensated by the Starling mechanism because the heart rate in these groups did not show a significant difference (group L-I: 78.8 ± 4.9 beats/min; group L-II: 72.0 ± 4.4 beats/min). This haemodynamic compensation by the Starling mechanism was also seen in groups R-L and R-S with right coronary artery disease (see also Table 7). Only left ventricular end-diastolic volume showed a significant ($p < 0.01$) difference between groups R-L and R-S, while the ejection fraction, cardiac index, and heart rate showed no significant difference between these two groups.

Discussion

The present study shows that the site of the coronary lesion in the left anterior descending artery is the major determinant of infarct size, and that involvement of the first diagonal branch results in a large infarction. In contrast to the left anterior descending artery, in right coronary artery disease the variation of the perfusion area of the right coronary artery in the posterior wall of the left ventricle was shown to be a major determining factor of infarct size, and the involvement of the right ventricular branch did not significantly influence the extent of infarction. It was also shown that the severity of coronary narrowing assessed by coronary arteriography does not correlate with the infarct size both in left anterior descending artery and right coronary artery disease.

Although the close relation of the involved major coronary artery to the site of the myocardial lesion has been well established,^{12, 13} the size of the perfusion area of its branches remains to be elucidated. Fulton,¹⁴ and Mitchell and Schwartz¹⁵ described the pathological features of coronary arteries associated with large or small infarcts. Some previous investigators also reported the relative size of the myocardial mass perfused by each of the major coronary arteries in the heart at necropsy.¹⁶⁻¹⁸ Infarct size, however, may be modified by many factors such as haemodynamic conditions, clinical interventions, presence of collaterals before myocardial infarction, and the tolerance of the myocardium to ischaemia.³ Therefore, the relation of infarct size and the site of the coronary lesion should preferably be investigated clinically. We considered the significant ($>75\%$) coronary narrowing, which corresponded to the infarct site judged from the electrocardiographic changes in standard 12-leads, as the cause of the infarction. This assumption is acceptable because narrowing of more than 75 per cent was observed in coronary arteriograms of almost all patients with acute myocardial infarction^{13, 19} and the site of the

Table 7 Cardiac function in patients with left anterior descending artery disease and right coronary artery disease (mean $\pm$ SE)

Group	Ejection fraction (%)	Cardiac index (l/min per m ²)	Left ventricular end-diastolic volume (ml/m ²)	Heart rate/min
L-I	32.1 ± 3.1	2.8 ± 0.3	143 ± 11.8	78.8 ± 4.9
L-II	46.8 ± 2.1	2.7 ± 0.2	113 ± 5.3	72.0 ± 4.4
R-L	52.1 ± 3.8	2.6 ± 0.2	130 ± 9	72.4 ± 11.2
R-S	53.7 ± 2.0	2.4 ± 0.3	88 ± 11	65.5 ± 3.1

* $p < 0.01$.

maximal stenosis or occlusion of the coronary artery had also been reported to correspond to the infarct site.²⁰

In this study the figures for mean total CK released in patients with proximal left anterior descending artery disease, left circumflex disease, and right coronary artery disease were 1510, 930, and 1131 IU/ml, and their relative percentages were 42.2, 26.0, and 31.7, respectively. Kalbfleisch and Hort¹⁶ reported that the relative perfusion areas of these three coronary arteries in the human heart were 41.5 per cent left anterior descending, 22.3 per cent left circumflex, and 36.2 per cent right coronary artery. These values are similar to those of the present study described above. The infarct size in right coronary artery disease may be smaller, however, than the perfusion area of the right coronary artery because right ventricular infarction is much less frequent than left ventricular infarction by right coronary artery involvement. This may account for the smaller value (31.7%) of relative total CK in right coronary artery disease in this study than the 36.2 per cent of Kalbfleisch's data. Ahmed *et al.*¹⁷ also reported that the perfusion area of the left coronary artery was almost equal to that of the right coronary artery, confirming our results obtained in clinical study.

In this study also the involvement of the first diagonal branch was found to influence directly the extent of the non-contracting segment. Since the extent of the non-contracting segment represents the ratio of the segment length of the non-contracting portion to the circumference of the left ventricle, the relative area of the non-contracting portion should be represented by the squared value of the non-contracting segment. The ratio of the non-contracting area (squared non-contracting segment) in group L-I (with the lesion proximal to the first diagonal branch) to that in group L-II (with the lesion distal to the first diagonal branch) was 1.0:0.46. This indicates that the perfusion area of the first diagonal branch is as large as that of the distal portion of the left anterior descending artery below the first diagonal branch. On the other hand, the mean total CK released in patients with only left circumflex artery disease was 688 ± 113 IU/ml, which is also as large as the mean total CK (799 ± 38 IU/ml) in group L-II with distal left anterior descending lesion. These results strongly suggest that the first diagonal branch has a substantially large perfusion area in the left ventricle. Mullen *et al.*²¹ also emphasised the great contribution of the first diagonal branch to the formation of ventricular aneurysm which often accompanied a large infarction. In contrast to the first diagonal branch, the contribution of the first septal branch

to the infarct size was thought to be small. It has been believed that the septal branch supplies blood only to the anterior half of the interventricular septum, and, therefore, a single lesion in the left anterior descending artery with complete necrosis of the septal wall is rare.²² In this study most of the patients with the coronary lesion proximal to the septal branch have no other coronary lesions, and this may account for the lower contribution of the first septal branch to infarct size.

In contrast to left anterior descending disease, the infarct size in right coronary artery disease was not directly dependent upon whether the major branch, that is the right ventricular branch, was involved or not, but it was greatly influenced by the distribution balance of the right coronary artery and left circumflex artery in the posterior wall of the left ventricle. Kalbfleisch and Hort¹⁶ also pointed out the great variation in size of the area perfused by the right coronary artery, ranging from 16.4 to 46.4 per cent of the total area, depending upon the predominance of vascular distribution, that is left coronary or right coronary artery type. In this series the patients in group R-L with a large left ventricular branch of the right coronary artery had the larger infarct (non-contracting segment: 27.6%, total CK: 1297 IU/ml) than those in group R-S with a large posterolateral branch of the left circumflex (non-contracting segment: 18.2%, total CK 920 IU/ml). Contrary to this, the site of the coronary lesion in the right coronary artery did not influence infarct size and nor did involvement of the right ventricular branch; this indicates indirectly that right ventricular infarction is much less frequent than left ventricular infarction. Though the relation between the site of the coronary lesion and infarct size was elucidated in this study, it should be noted that the perfusion area may be different from the infarct size. Schaper *et al.*²³ described the perfusion area as being usually larger than the infarct area because of the collateral flow supplying the surrounding area of the infarction. In our series, however, it seems likely that the collaterals do not influence the infarct size because there were no significant differences in the incidence of collaterals between groups L-I and L-II, and also between groups R-L and R-S.

As one can estimate from the significant difference in infarct size between groups L-I and L-II, the ejection fraction was significantly smaller in group L-I than in group L-II. Cardiac index, however, was not reduced in group L-I; it may be a result of the compensatory mechanism of the Starling effect since left ventricular end-diastolic volume was larger in group L-I than in group L-II, while heart rate was similar in these two groups

(Table 7). On the other hand, in right coronary artery disease the ejection fraction in groups R-L and R-S showed no significant difference though infarct size in group R-L was significantly larger than that in group R-S. This paradoxical finding is probably a result of the smaller contribution of the inferior wall than of the anterior wall to the left ventricular pump function.¹¹

There was no correlation between the severity of coronary narrowing and infarct size. This result is compatible with the previously reported results in patients with acute myocardial infarction¹³ though the cross-sectioned area of the coronary artery without any coronary lesions closely correlates with the perfusion area.¹⁸⁻²⁴ Taking the secondary thrombosis after the infarction and the recanalisation of coronary occlusion into account, there may be no correlation between the severity of coronary narrowing and infarct size. The relation between the results on the site of the coronary lesion and infarct size shown in this study, however, should be of clinical importance in evaluating the therapeutic interventions and also in estimating prognosis for the patients.

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