Kawasaki Med. J. Vol. 4: No. 3, 143-154, 1978

THE CORRELATION OF REGIONAL LEFT VENTRICULAR ASYNERGY WITH LEFT VENTRICULAR FUNCTION IN ISCHEMIC HEART DISEASE

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Accepted for Publication on July 28, 1978

Abstract

Cine-ventriculography and selective coronary arteriography were performed in 59 cases with ischemic heart disease and correlation between the left ventricular function and left ventricular wall motion was examined.

Localized asynergy of the left ventricular wall was seen in 81% with myocardial infarction, but in only 19% with angina pectoris.

The value of ejection fraction was 0.75 ± 0.10 in normal wall motion, 0.60 ± 0.10 in asyneresis, 0.40 ± 0.19 in akinesis and 0.49 ± 0.11 in aneurysm.

A close correlation was recognized between the size of asynergy and ejection fraction and also between the left ventricular circumferential shortening ratio and ejection fraction.

Correlation between LVEDP and ejection fraction was $r = -0.53$ in angina pectoris, but $r = -0.30$ in myocardial infarction.

In order to evaluate the left ventricular function in ischemic heart disease, it is very important to find out the degree and the size of asynergy, the wall motion in non-infarcted myocardium and coronary arterial lesion by cine-ventriculography and coronary arteriography.

INTRODUCTION

The left ventricular disfunction in ischemic heart disease is usually based on localized asynergy in left ventricular wall, which differs from many other heart disease, and congestive heart failure may hardly manifest because of compensative adjustment by excessive contraction of remaining normal heart muscle and dilatation of the left ventricular cavity.

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Generally, ventricular wall asynergy in ischemic heart disease is caused by myocardial infarction and is irreversible, therefore the cardiac reserve decreases even if without congestive heart failure clinically.

In the present study we tried to define the significance of left ventricular abnormal wall motion in 59 cases with ischemic heart disease by correlation asynergic size and remaining healthy wall contraction with left ventricular function which was confirmed by cine-ventriculography.

SUBJECTS AND METHODS

Cine-ventriculography at RAO 30° and selective coronary arteriography were carried out in 59 cases with ischemic heart disease, 27 with angina pectoris and 32 with myocardial infarction.

The distribution of the number of significant coronary artery lesion with stenosis of 75% or more and the type of ischemic heart disease were shown in Table 1.

TABLE 1. Cases with ischemic heart disease

No. of involved vessels	angina pectoris	angina pectoris with myocardial infarction	myocardial infarction	tolal
None vessel disease	0	0	4	4
1 vessel disease	14	0	15	29
2 vessel disease	8	6	0	14
3 vessel disease	5	7	0	12
total	27	13	19	59

The left ventricular wall asynergy was classified as localized end-systolic bulge, asyneresis, akinesis and aneurysm.

End-diastolic and end-systolic volumes and ejection fraction were calculated by Chapman's method.[1] Length of asynergic region(A), end-diastolic circumference(A+B) and end-systolic circumference(A+C) were measured and size of asynergy $\left(\frac{A}{A+B}\right)$, circumferential shortening ratio $\left(\frac{B-C}{A+B}\right)$, long axis shortening ratio $\left(\frac{D-E}{D}\right)$ were calculated as shown in Fig. 1.

RESULTS

1. Left ventricular wall motion (Table 2)

In the group with angina pectoris, normal wall motion was seen in 13

A : length of asynergic region
A+B : end-diastolic circumferential length
A+C : end-systolic circumferential length
D : length of end-diastolic long axis
E : length of end-systolic region axis

1. Size of asynergic region : $\frac{A}{A+B}$
2. Circumferential shortening rate : $\frac{B-C}{A+B}$
3. Long axis shortening rate : $\frac{D-E}{D}$
4. Ejection fraction : $\frac{\text{stroke volume}}{\text{end-diastolic volume}}$

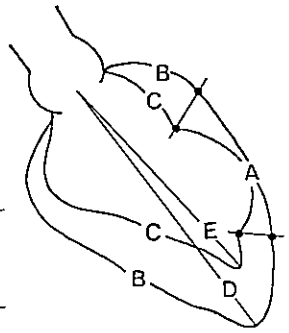

Fig. 1. Analysis of left ventriculogram (RAO 30°)

TABLE 2. Left ventricular wall motion

Without myocardial infarction				
L V wall motion	1 vessel disease	2 vessel disease	3 vessel disease	total
normal	10	3	0	13
end-systolic bulge	3	3	3	9
asyneresis	1	2	2	5
total	14	8	5	27

With myocardial infarction					
L V wall motion	No vessel disease	1 vessel disease	2 vessel disease	3 vessel disease	total
normal	1	2	1	0	4
end-systolic bulge	0	0	2	0	2
asyneresis	1	6	1	1	9
akinesis	0	7	1	3	11
aneurysm	2	0	1	3	6
total	4	15	6	7	32

cases, 48%, end-systolic bulge in 9 cases, 33% and asyneresis in only 5, 19% of 27 cases.

Severe asynergy as akinesis or aneurysm was not seen in the cases without myocardial infarction. However, in myocardial infarction, normal wall

motion was observed in only 4, 15%, asyneresis in 28%, akinesis in 34% and aneurysm in 19% of 32 cases, especially in the cases with myocardial infarction and multiple vessel disease, severe asynergy as akinesis or aneurysm were often seen.

2. Ejection fraction and left ventricular wall motion (Fig. 2)

The values of ejection fraction were 0.75 ± 0.10 ($\bar{X}\pm SD$) with normal wall motion, 0.70 ± 0.09 with end-systolic bulge, 0.60 ± 0.10 with asyneresis, 0.49 ± 0.10 with akinesis and 0.49 ± 0.11 with aneurysm.

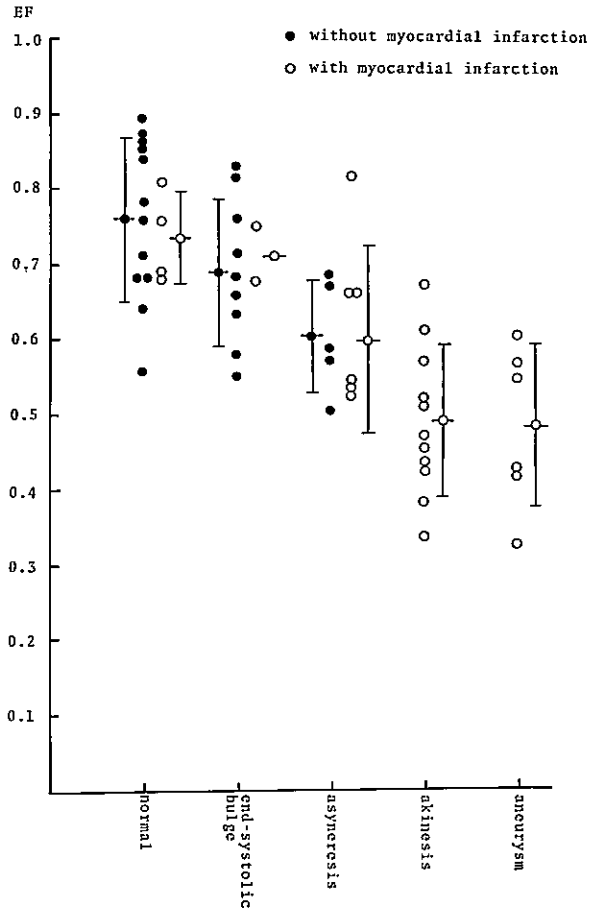

Fig. 2. Left ventricular wall motion and ejection fraction

Significant drop in ejection fraction was shown in the cases with severe asynergy as akinesis or aneurysm ($p=0.001$) but there was no significant dif-

ference in ejection fraction between angina pectoris and myocardial infarction in each asynergic group.

3. Ejection fraction and involving coronary vessels (Fig. 3)

Ejection fraction was relatively well preserved in spite of multiple vessel disease in the cases without myocardial infarction.

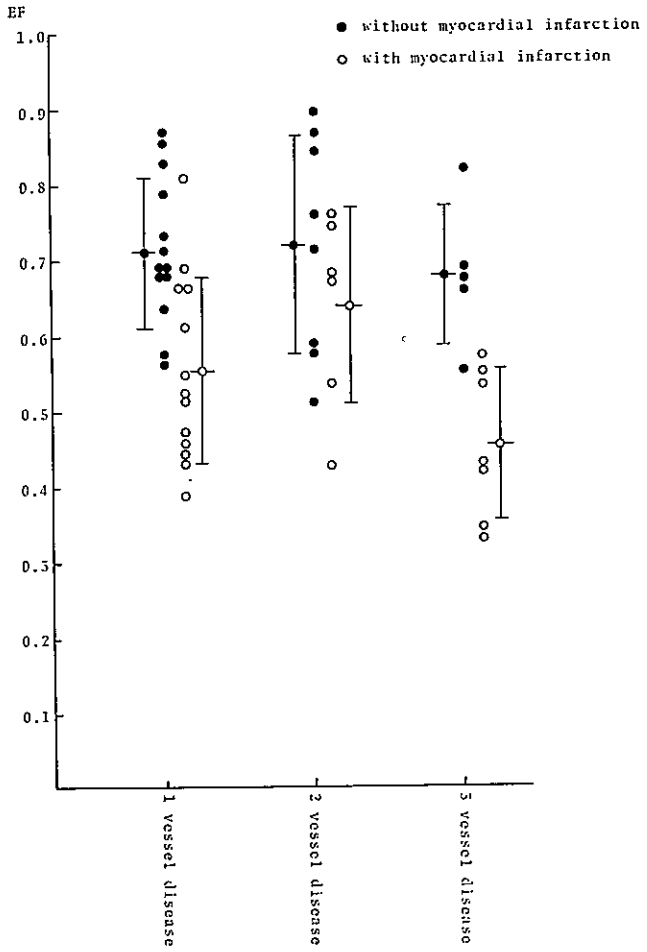

Fig. 3. Number of involved vessels and ejection fraction

4. Left ventricular end-diastolic pressure (LVEDP) and left ventricular wall motion (Fig. 4)

LVEDP was elevated according to the degree of asynergy in angina pectoris, but there was no significant influence on LVEDP by asynergy in myocardial infarction, especially LVEDP in angina pectoris with asyneresis was rather high than that in myocardial infarction; 11.9 ± 3.9 mmHg in myocardial infarction and 20.6 ± 8.6 mmHg in angina pectoris.

These findings suggest better developed compensative adjustment on the ventricular function in myocardial infarction than that in angina pectoris.

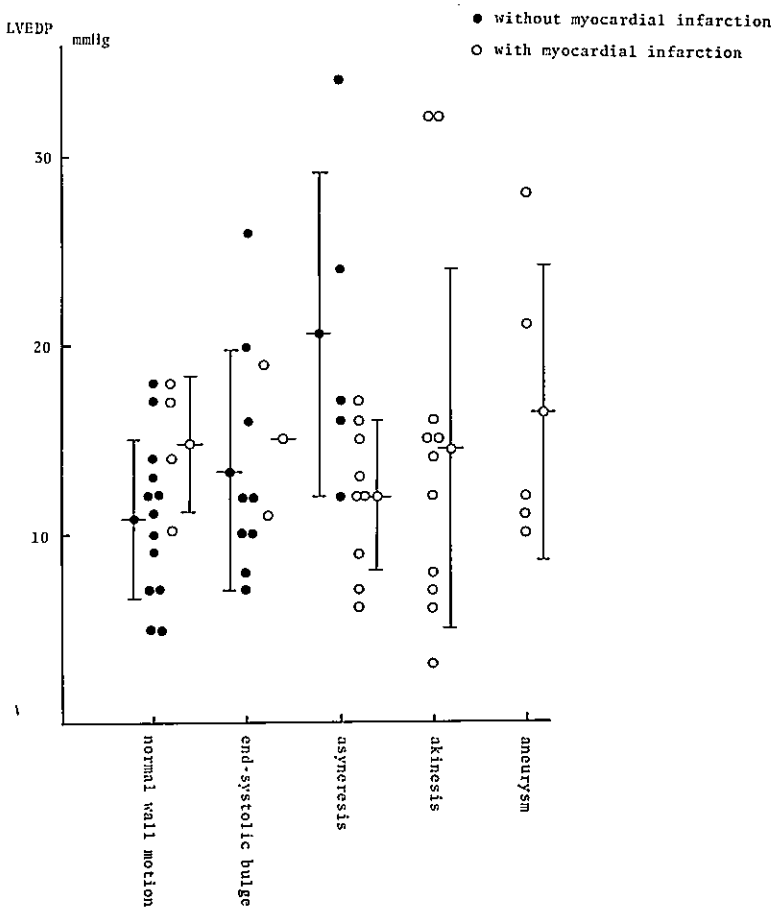

Fig. 4. Left ventricular wall motion and end-diastolic pressure

5. LVEDP and ejection fraction (Fig. 5)

A relatively good correlation ($r=-0.53$) was seen between LVEDP and ejection fraction without myocardial infarction but not in myocardial infarction ($r=-0.30$).

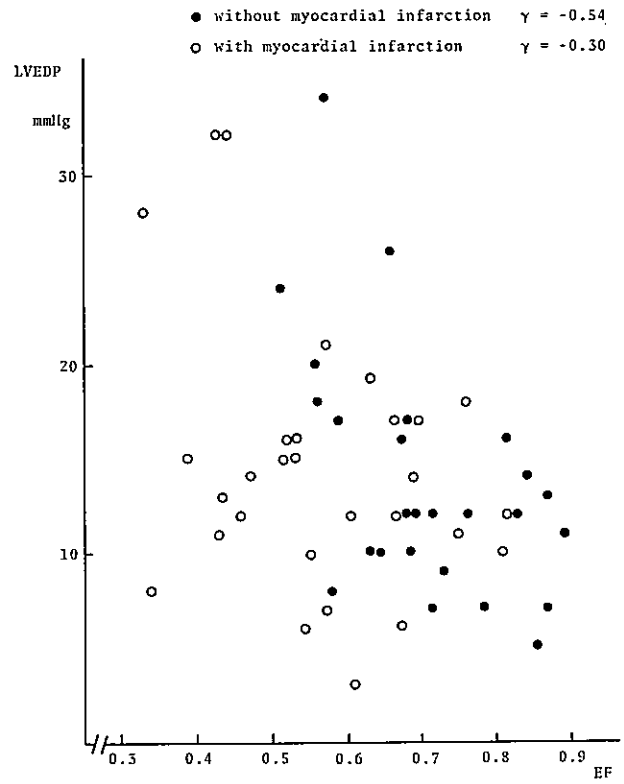

Fig. 5. Left ventricular end-diastolic pressure and ejection fraction

6. Size of asynergy and ejection fraction (Fig. 6)

There was a close correlation ($r=-0.64$) between the size of asynergy and ejection fraction, ejection fraction being preserved more than 0.6 when the size of asynergy was less than 25% of left ventricular end-diastolic circumference.

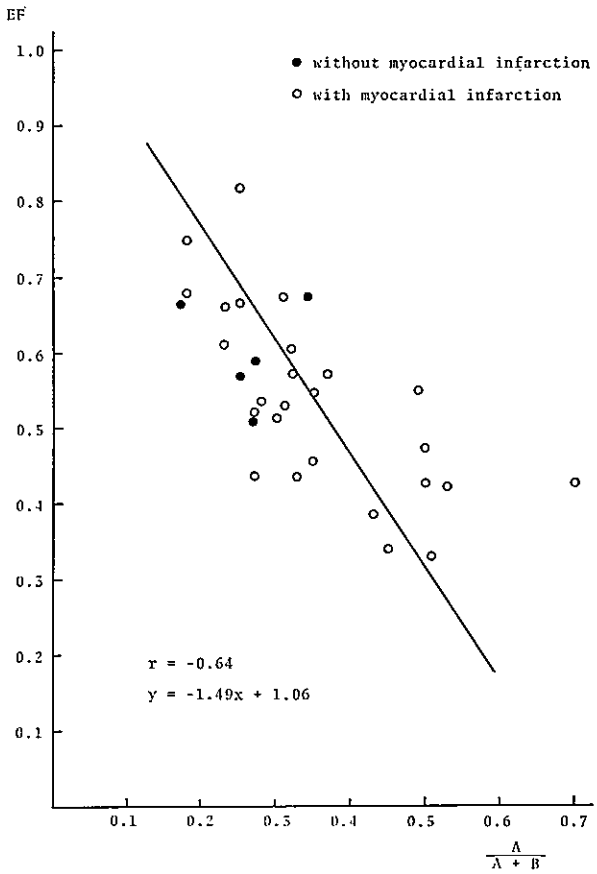

Fig. 6. Size of asynergy ($\frac{A}{A+B}$) and ejection fraction

7. Left ventricular circumferential shortening ratio and ejection fraction (Fig. 7)

Generally, the smaller is the size of asynergy and the more excessive is the remaining left ventricular wall contraction, the better preserved is the left ventricular function. Circumferential shortening ratio in normal contraction is 0.32 ± 0.07 and the value of ejection fraction is 0.75 ± 0.10. There is a good correlation ($r = 0.78$) between the left ventricular circumferential shortening ratio and ejection fraction in the cases with asynergy, and ejection fraction is kept more than 0.6, when the value of circumferential shortening ratio is more than 0.25.

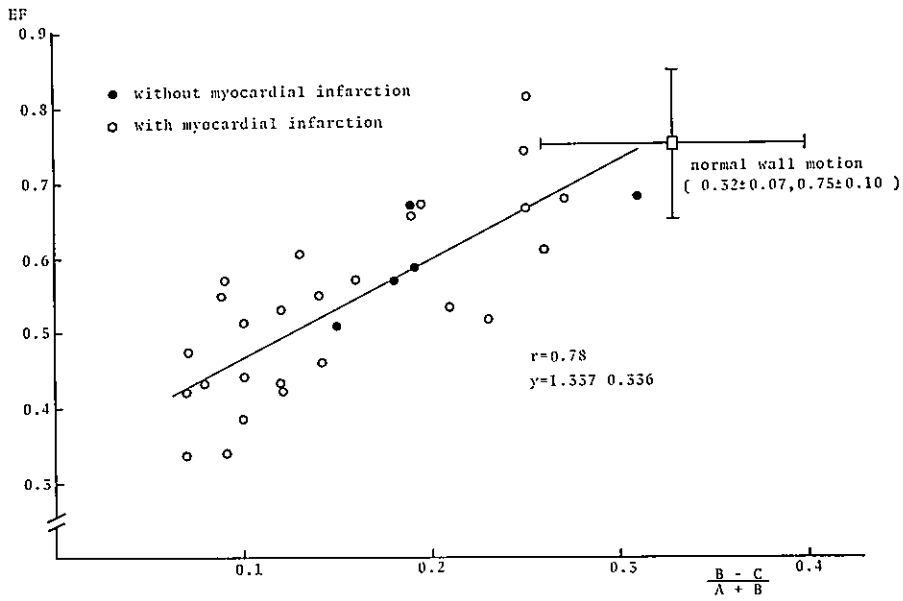

Fig. 7. Left ventricular circumferential shortening ratio $(\frac{B-C}{A+B})$ and ejection fraction

8. Left ventricular wall motion and left ventricular long axis shortening ratio (Fig. 8)

Long axis shortening ratio of left ventricle is 30.2±8.5% in normal wall motion group without myocardial infarction, and 27.0±8.9% with myocardial infarction. However, it markedly decreases according to the degree of asynergy; 12.1±7.3% in akinesis, and 6.7±2.6% in aneurysm.

These findings show that abnormality of the left ventricular contraction first affects its long axis shortening, irrespective of the location of ischemia.

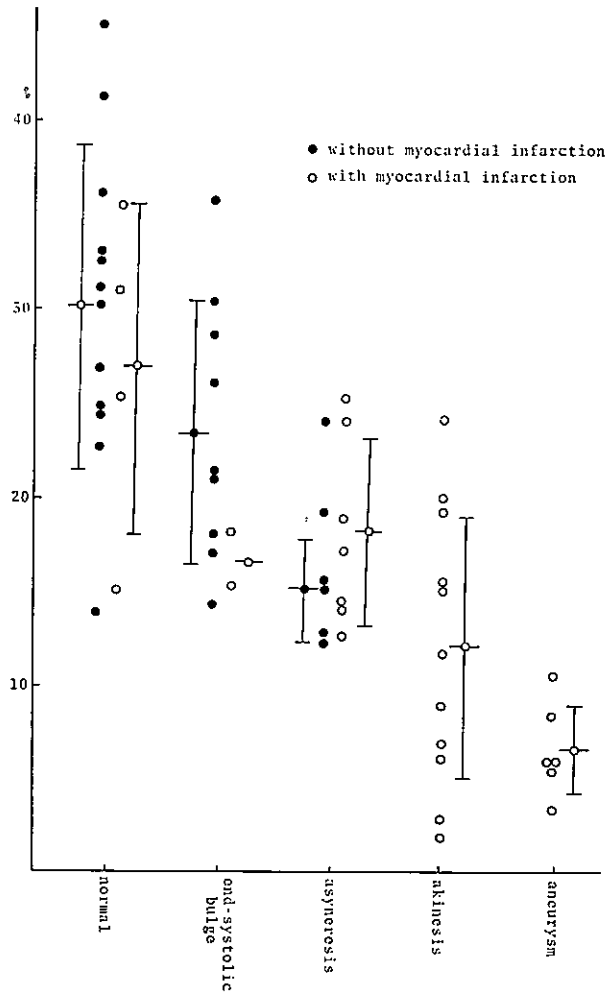

Fig. 8. Left ventricular wall motion and long axis shortening ratio

DISCUSSION

The left ventricular disfunction in ischemic heart disease is mainly based on localized asynergy in the ventricular wall involved coronary artery lesion[2], and it is distinctive and differs from many other heart diseases that the left ventricular disfunction in ischemic heart disease is compensated by dilatation of the left ventricular cavity and an excessive shortening of remaining heart muscle.

Various types of parameters given by ECG, MCG, UCG, scanning of heart muscle and cardiac catheterization cannot indicate ventricular function independently and sufficiently in ischemic heart disease. On the other hand, cine-ventriculography is the most reliable method in evaluating left ventricular function in ischemic heart disease from the synthetical point of view.

The wall motion in cine-ventriculography at RAO 30° was classified into normal wall motion, end-systolic bulge, asyneresis, akinesis and aneurysm. End-systolic bulge shows a slightly localized dilatation at the end-systolic phase, that is seen in the region involving coronary artery lesion. It is unknown whether the mechanism of end-systolic bulge is due to localized hypocontraction or localized shortening of the systolic period.

Localized asynergy is seen in 81% of myocardial infarction but only in 15% of non-myocardial infarction.

Usually the left ventricular function is relatively well preserved in the cases of severe multiple vessel disease without myocardial infarction[3].

In our study, a decrease of the ejection fraction was recognized when the size of asynergy was more than 25% of end-diastolic circumference and the left ventricular circumferential shortening ratio was less than 25%.

Feild *et al.*[4] and Hori *et al.*[5] reported a close correlation between the size of asynergy and ejection fraction.

Hood[6] also reported that the dilatation of left ventricle or an increase of fiber shortening in non-infarcted myocardium was necessary to maintain the same stroke volume as preinfarction, in myocardial infarction with asynergy.

Klein *et al.*[7] and Herman *et al.*[2] showed the decrease of stroke volume, the dilatation of left ventricle and the elevation of end-diastolic pressure appear when the area of akinesis or aneurysm becomes more than 20% of left ventricular surface area. The reason is that the increase of fiber shortening in non-infarcted muscle reaches to its limit.

The increased fiber shortening in non-infarcted myocardium makes up for the decrease in the ejection fraction and the left ventricular dilatation keeps up the stroke volume as compensative adjustment, while the decrease in the stroke volume and the elevation of LVEDP follow clinically congestive heart failure.

In non-infarcted cases, a close correlation was recognized between the ejection fraction as the pump function and LVEDP as the left ventricular compliance. The decrease in the ejection fraction accompanied by the elevation of LVEDP. However, there was no correlation between them in myocardial infarction.

Baxley *et al.*[3] and Hamilton *et al.*[8] stated that congestive heart failure is seen clinically when ejection fraction drops lower than 40%.

In order to evaluate the left ventricular function in ischemic heart disease, it is very important to find out the degree and the size of asynergy, the wall motion in non-infarcted myocardium and coronary artery lesion by cine-ventriculography and coronary arteriography.

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