Detection of Left Ventricular Asynergy in Myocardial Infarction by Means of Ultrasono-Cardiotomography and M-Mode Echocardiography

Hisaharu Hikichi, M.D. and Motonao Tanaka, M.D.

SUMMARY

The movements of the left ventricular wall in cases of myocardial infarction were studied by ultrasono-cardiotomography and M-mode echocardiography.

Ultrasono-cardiotomographic and echocardiographic data were collected from 15 consecutive patients with transmural myocardial infarction ranging in age from 28 to 60 years (4 with antero-septal infarction, 5 with postero-inferior infarction, and 6 with infarction of other areas) and also from healthy control subjects.

In myocardial infarction, a delay in timing and a decrement in amplitude of the excursion of the infarcted area were observed. As the result, compensatory movement occurred in the non-infarcted area. This compensatory movement was a major contributing factor for the maintenance of the function of the left ventricle.

In antero-septal infarction, the pump function was compensated for by the movement of the left ventricular posterior wall (LVPW) and by that of the non-infarcted area of the interventricular septum (IVS). On the other hand, in postero-inferior infarction, the pump function was mainly compensated for by an increment in movement of IVS.

This compensatory movement, which was very strong in the acute stage of myocardial infarction, decreased with time after infarction. On the other hand, in the hypokinetic infarcted area, there was a gradual increment of the amplitude of excursion with time after infarction.

These findings reflected the degree of the development of collateral circulation. It was found that the degree of asynergy could be determined by the evaluation of the delay in timing and amplitude of the excursion, and of mVCF at basal, middle, and apical portions of the left ventricle.

Additional Indexing Words:
Mean rate of circumferential fiber shortening (mVCF) Antero-septal infarction Postero-inferior infarction Paradoxic movement Wall excursion Ventricular aneurysm
NECROSIS and fibrosis which occur invariably in myocardial infarction induced by the obstruction of coronary arteries result in left ventricular dysfunction.

Depending on the severity of dysfunction, a spectrum of left ventricular failure develops ranging from a clinically unrecognized functional disturbance to cardiogenic shock.

Ultrasono-cardiotomographic and M-mode echocardiographic techniques are useful in evaluating the severity of myocardial damage. The non-invasive nature and the high sensitivity for the detection of left ventricular asynergy make this method an extremely attractive tool for the diagnosis of myocardial infarction. Previous studies\(^1\)-\(^10\) have shown that the movement of the IVS and that of the free wall were either decreased or paradoxical in myocardial infarction. This communication presents evidence for the usefulness of ultrasono-cardiotomography and M-mode echocardiography in the clinical study of left ventricular asynergy.

MATERIALS AND METHODS

Ultrasono-cardiotomographic and M-mode echocardiographic data were collected from 15 patients (the age ranging from 28 to 60 years) with transmural myocardial infarction in chronic stage except for 1 case.

Healthy control subjects were also examined by the same technique. Antero-septal infarction in 4 cases, postero-inferior infarction in 5 cases and myocardial infarction in 6 other cases of this study were diagnosed based on ECG findings. In all the patients of this study, the width of Q wave exceeded 0.04 sec.

Also typical chest pain associated with infarction and the time course of enzyme activity were taken into account for the establishment of the diagnosis. Two apparatus were used throughout this experiment. One was the same as that used in our previous report\(^11\),\(^12\).

The concave transducer of this apparatus had a resonance frequency of 2.25 MHz and a diameter of 30 mm. The other had an electronic sector scan device. Both of them were used together with an ALOKA SSD-110 echograph.

All the patients were examined in supine position. The transducer was placed at the standard position; at the left fourth intercostal space lateral to the left sternal margin. A long axis ultrasono-cardiotomogram was obtained by a scan along the long axis of the left ventricle. This echogram showed the aortic root, mitral valve, IVS, and LVPW.

All the echograms studied were of sufficient quality for interpretation. Quantitative estimation of the extent of asynergy on echogram was carried out using Q-Sc, Q-Sd, Q-Pc, Q-Pd intervals, Scd/Pcd ratio, and mVCF: The Q-Sc interval is the time interval from Q wave of ECG to the initial point of the excursion of the IVS. The Q-Sd interval is the time interval from Q wave of ECG to the point of the maximum excursion of the IVS. The Q-Pc interval is the time interval from Q wave of ECG to the initial point of the excursion of LVPW. The Q-Pd interval
Fig. 1. Echocardiograms of the left ventricle and schematic representation of the parameters to be measured. (See text for detail)

Fig. 2. Long axis ultrasono-cardiotomogram with antero-septal infarction

Left: an echogram at end-diastole and a kymogram.
Right: an echogram at end-systole and a schematic diagram which consists of superimposed end-diastolic and end-systolic silhouettes. The IVS is hypokinetic and the LVPW hyperkinetic.
is the time interval from Q wave of ECG to the point of the maximum excursion of LVPW.

The \text{Sd/Pcd} ratio is the excursion of IVS during ejection divided by that of LVPW (Fig. 1). The mean rate of circumferential shortening (mVCF) was calculated from the following equation: $\text{mVCF} = \frac{\text{EDD} - \text{ESD}}{\text{ET}}$. Ejection time was defined as the length of time from aortic valve opening to aortic valve closure.

These indices were measured at 3 different portions of the left ventricle.
1) The basal portion of the left ventricle was defined as the level where the echoes from both anterior and posterior mitral leaflets could be detected.
2) The middle portion was where the echoes from both mitral leaflets disappeared.
3) The apical portion was deep in the left ventricle, where the echoes from the posterior papillary muscle were detected.

\section*{Results}

\textbf{I. Left ventricular asynergy detected by ultrasono-cardiotomogram}

\underline{a) Antero-septal myocardial infarction}

The IVS was hypokinetic and the LVPW hyperkinetic (Fig. 2). Left

\begin{figure}
\centering
\includegraphics[width=\textwidth]{fig3.png}
\caption{Long axis ultrasono-cardiotomogram with postero-inferior infarction.}
\end{figure}

Left: an echogram at end-diastole and a kymogram.
Right: an echogram at end-systole and a schematic diagram made of superimposed end-diastolic and end-systolic silhouettes. The LVPW shows a paradoxical movement. The IVS is hyperkinetic.
Fig. 4. Long axis ultrasono-cardiotomogram with antero-lateral infarction. The antero-lateral wall is almost akinetic and the LVPW hyperkinetic.

Fig. 5. Q-Sc, Q-Pc interval at the basal, middle, and apical portions of the left ventricle in normal cases and in cases with myocardial infarction. 1 and 3 of "others" show cases of antero-lateral infarction. 2, 4, and 5 of "others" show cases with both postero-inferior and antero-lateral infarctions. Q-Sc interval in antero-septal infarction and Q-Pc interval in postero-inferior infarction are longest at the apical, intermediate at the middle and shortest at the basal portion. See text for detail.
ventricular asynergy was detected using a diagram which consisted of superimposed end-diastolic and end-systolic silhouettes.

b) Postero-inferior myocardial infarction.
The movement of the LVPW was paradoxical. On the other hand, the IVS was hyperkinetic (Fig. 3).
c) Antero-lateral myocardial infarction
Antero-lateral wall was almost akinetic and the LVPW hyperkinetic (Fig. 4).

II. Left ventricular asynergy detected by M-mode echocardiogram
1. Q-Sc, Q-Sd, Q-Pc, Q-Pd intervals in myocardial infarction
   a) Antero-septal myocardial infarction

   The Q-Sc interval was longest at the apical, intermediate at the middle and shortest at the basal portion. Namely the length of Q-Sc interval changed in a pattern opposite to that in normal cases.

Fig. 6. Q-Sd and Q-Pd intervals at 3 portions in normal cases and in cases with myocardial infarction. The length of Q-Sd interval at the 3 portions in antero-septal infarction shows a similar pattern as in normal cases except for 1 case, but the time difference in Q-Sd interval between the apical and the basal portions of IVS is smaller than that in normal cases except for 1 case. The value of Q-Pd interval in antero-septal infarction changes in a pattern opposite to that in normal cases. Also the value of Q-Pd interval in postero-inferior infarction changes in a pattern opposite to that in normal cases.
The values of Q-Sd interval determined at the 3 portions showed a similar pattern as in normal cases except for 1 case. But the time difference in Q-Sd interval between the apical and basal portions of the IVS was smaller than that in normal cases except for 1 case.

The values of Q-Pc interval determined at the 3 portions had a similar pattern as in normal cases.

The Q-Pd interval was longest at the apical, intermediate at the middle and shortest at the basal portion of the LVPW.

The value of Q-Pd interval changed in a pattern opposite to that in normal cases (Figs. 5, 6).

b) Postero-inferior myocardial infarction

Both Q-Pc interval and Q-Pd interval were longest at the apical, intermediate at the middle and shortest at the basal portion of the LVPW.

The values of Q-Pd interval determined at the 3 portions changed in a similar pattern as that in anteroseptal myocardial infarction.

The value of Q-Sc interval and that of Q-Sd interval changed in a similar pattern as in normal cases respectively (Figs. 5, 6).

c) In cases with both antero-lateral and postero-inferior infarctions:

![Fig. 7. The initial point of excursion of the antero-lateral wall at the apical portion appears later than that in normal cases. The antero-lateral wall is akinetic.](image)
namely, in cases 2, 4, and 5 (referred to as “others” in Figs. 5 and 6)

The values of Q-Pc interval and Q-Pd interval at the 3 portions changed in a similar pattern as that in postero-inferior myocardial infarction.

d) In cases with antero-lateral infarction: namely, in cases 1 and 3 (referred to as “others” in Figs. 5 and 6)

The values of Q-Pc and Q-Sc intervals at the 3 portions changed in a similar pattern as that in normal cases. The value of Q-Pd interval in case 1 changed in a similar pattern as in postero-inferior infarction. In another case of antero-lateral infarction, the initial point of excursion of the antero-lateral wall appeared later than in normal cases. Of the 3 portions, it was the latest at the apical portion (Fig. 7).

M-mode echocardiograms with abnormal patterns are shown in Figs. 8 (antero-septal infarction), 9 (postero-inferior infarction), and 10 (antero-septal aneurysm).

2. Scd/Pcd ratio in myocardial infarction

a) Antero-septal myocardial infarction

Fig. 8. Q-Sc, Q-Sd, Q-Pd interval at the 3 portions in antero-septal infarction. Q-Sc interval and Q-Pd interval are longest at the apical, intermediate at the middle and shortest at the basal portion. The values of Q-Sd interval determined at the 3 portions shows a similar pattern as in normal cases.
b) Postero-inferior myocardial infarction

The Scd/Pcd ratio was more than 1.0 at the middle and apical portions of the left ventricle.

c) In a case with 2 areas of infarction, namely antero-lateral and postero-inferior infarction (referred to as “others” in Figs. 5 and 6)

The value of Scd/Pcd ratio at the 3 portions changed in a similar pattern as that in postero-inferior infarction.

In a case of antero-lateral infarction (case 1), the Scd/Pcd ratio changed in a similar pattern as that in postero-inferior infarction (Fig. 11).


The Scd/EDD-ESD ratio at the apical portion decreased with time after infarction. Finally the above ratios at the 3 portions approached to the nor-
Fig. 10. Q-Sc interval in antero-septal aneurysm. Q-Sc interval is longest at the apical, intermediate at the middle and shortest at the basal portion.

Fig. 11. Scd/Pcd ratio at the 3 portions in normal cases and in cases with myocardial infarction. In antero-septal infarction, Scd/Pcd ratio is less than about 1.0 at the middle and apical portions. It is highest at the basal and lowest at the apical portion of the left ventricle. In postero-inferior infarction, the Scd/Pcd ratio is more than 1.0 at the middle and the apical portions of the left ventricle.

It was thus found that the Scd/Pcd ratio at the 3 portions decreased gradually with time after infarction. On the other hand, the Pcd/EDD-ESD at the apical portion increased gradually with time after infarction.
Fig. 12. The Scd/EDD-ESD, Pcd/EDD-ESD, and Scd/Pcd ratio in 1 case of postero-inferior infarction with time after infarction. The Scd/EDD-ESD ratio at the apical portion decreased with time after infarction. Finally the above ratios at the 3 portions approached the normal value. On the other hand, the Pcd/EDD-ESD at the apical portion increased gradually. Therefore, the Scd/Pcd ratio at the 3 portions decreased gradually with time. Measurement along line 1 was made within 1 week after infarction, that along line 2 after 2 weeks, that along line 3 after 1 month and that along line 4 after 2 months.

Fig. 13. The values of mVCF in both normal cases and myocardial infarction. See text for detail.

4. mVCF in myocardial infarction

The mVCF at the apical portion in postero-inferior infarction was lower than that at the middle portion.

The value of mVCF at the apical portion was less than about 1.0. The values of mVCF at the 3 portions in antero-septal infarction changed in a pattern opposite to that in normal cases except for 1 case (Fig. 13).

DISCUSSION

The left ventricular contraction begins at the apical portion in both IVS and LVPW and the wave of contraction travels towards the basal portion of the left ventricle. It is by this mechanism of contraction that the blood is
effectively pumped into systemic circulation.

In the cases with myocardial damage (necrosis or fibrosis) after myocardial infarction, this mechanism fails to function properly and results in abnormality of the left ventricular contraction and in left ventricular asynergy. Then blood pressure may become lower and the output of the left ventricle may become smaller depending on the severity of dysfunction. However it is not until the final stage that the abnormality in hemodynamics occurs. The pump function is usually compensated for by hyperkinetic movements in the non-infarcted myocardium, namely by the compensatory movement. Therefore, the detailed investigation of asynergy in cases of myocardial infarction is very important for the evaluation of the severity of dysfunction of the left ventricle.

In both antero-septal and postero-inferior infarctions, the contraction of IVS and LVPW started at the basal portion and the wave of contraction traveled towards the apical portion of the left ventricle. When the normal myocardial tissue at the apical portion of the IVS or LVPW was replaced with fibrotic tissue, the appearance of the initial excursion of the IVS or LVPW was delayed. It is thought that marked delay of initial contraction in infarcted area may produce ventricular aneurysm in that area. In antero-septal infarction, the presence of the myocardial damage at the apical portion of the IVS was responsible for the prolonged Q-Sc interval at the apical portion. But the values of Q-Sd measured at the 3 portions of the left ventricle changed in a similar pattern as that in normal cases except for 1 case.

The time difference in Q-Sd interval between the apical and basal portions of the IVS was smaller than that in normal cases. Namely the Sc-Sd interval was found to be decreased at the apical portion. Such change is an unfavorable factor, as far as the maintenance of the cardiac output is concerned. Probably the prolonged Q-Pd interval at the apical portion was a compensation for shorter Sc-Sd interval.

In postero-inferior infarction, both Q-Pc interval and Q-Pd interval were longest at the apical portion. It is likely that the prolonged Q-Pc interval at the apical portion was caused by the presence of myocardial damage at the apical portion of the LVPW. The prolonged Q-Pd interval at the apical portion resulted from the increment of Q-Pc interval. Similar results were obtained also in experimental chronic myocardial infarction.

The time delay and the decrement of the excursion of the infarcted area are unfavorable for the maintenance of cardiac output. Therefore, congestive heart failure may develop in the cases in which a compensatory mechanism fails to function.

In postero-inferior infarction, the excursion of the apical portion of the
LVPW decreased and the Scd/Pcd ratio was more than 1.0 at the middle and the apical portions of the left ventricle. Thus it is likely that the compensatory movement of the IVS is a major contributing factor for the maintenance of the function of the left ventricle.

In antero-septal infarction, the Scd/Pcd ratio was less than about 1.0 at middle and apical portions of the left ventricle. It was highest at the basal and smallest at the apical portion of the left ventricle. The Scd/Pcd ratio at the apical portion was smaller than that in normal cases, because the excursion of the IVS at the apical portion decreased in comparison with that of the normal cases. But the excursion of the IVS at the basal portion was of larger amplitude than in normal cases because of the compensatory movement. The amplitude of excursion was approximately either of the same magnitude as that of the LVPW, or larger than that of the LVPW at the basal portion. It was concluded from these facts that the decreased excursion was compensated for by the hyperkinetic movement of the non-infarcted area.

Therefore, in antero-septal infarction, the pump function was compensated for by the movements of LVPW and of the non-infarcted area of the IVS (the basal portion of the IVS). On the other hand, in postero-inferior infarction, the pump function was compensated for mainly by an increment in movement of the IVS.

These compensatory movements are very strong in acute stage of myocardial infarction and contribute to the maintenance at a normal level of the pump function.

In the hypokinetic infarcted area, there was a gradual increment of the amplitude of excursion with time after infarction, which might reflect the degree of the development of collateral circulation. The characteristics of asynergy was evaluated in detail based on the values of the Scd/Pcd ratio determined at the 3 portions.

It was concluded from the high value of Scd/Pcd ratio and the relative delay of the initial point of excursion of the LVPW at the apical portion that myocardial damage extended not only to the lateral wall, but also to the posterior wall of LVPW in case 1 with antero-lateral infarction (Figs. 5 and 6). This case suggested that the Scd/Pcd ratio and time delay of excursion obtained by M-mode echocardiography in this experiment may be used as a useful index, in the case whose ECG diagnosis is difficult.

In myocardial infarction the mVCF at the apical portion was lower than that at the middle portion, presumably due to the presence of myocardial damage in the apical portion of the left ventricle. The values of mVCF measured at the 3 portions of the left ventricle can also be used for the diagnosis of myocardial infarction with asynergy.
REFERENCES


