Evaluation of the Left Ventricular Performance in Patients with Ischemic Heart Disease Using Radionuclide Angiography

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Radionuclide angiography was utilized for the measurement of left ventricular dynamics and the analysis of its segmental wall motion. Left ventricular performance was measured by the first pass method and gated equilibrium method in patients with ischemic heart disease. The left ventricular wall motion was also examined by the analysis of computer-drawn outlines of radioactivity counts of the left ventricular chamber. These measurements were well correlated with those obtained by invasive methods such as contrast cine-ventriculography and thermodilution method in the resting state.

The patients with effort angina often showed an almost normal left ventricular performance and wall motion in the resting state without ischemic episodes. However, at the time when anginal attack was provoked with exercise testing, an asynergy and a reduced performance of left ventricle were observed. The extent and localization of this asynergy well corresponded with the defect of myocardial scintigrams determined by 201-Tl stress myocardial imaging.

From above findings we conclude that the myocardial ischemia with asynergy is a cause of decreased left ventricular hemodynamics during anginal attack.

Although further evaluation is necessary to know limitations and to avoid inaccuracy, these techniques were shown to have a significant usefulness in evaluating ischemic heart disease.

Coronary artery disease is a well-known cause of regional myocardial ischemia and subsequently induced myocardial dysfunction. Nevertheless, some cases have shown apparently normal left ventriculogram despite the existence of severe coronary stenosis. These findings suggest that coronary artery disease has a wide spectrum of cardiac dysfunction depending on myocardial damage and its compensatory mechanism. Therefore, for the evaluation of coronary heart disease it is necessary to develop a new method which is sufficiently sensitive to detect the subtle decrease in left ventricular performance.

Radionuclide angiographic method has been recently introduced in determining the hemodynamic abnormalities of the left ventricle and

Key Words:
- Computer-drawn outlines of left ventricular chamber
- Edge detection
- Left ventricular performance
- Ejection fraction
- Ischemic heart disease

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Radionuclide Angiocardiography

Fig.1. Radionuclide angiograms.
A first pass technique and gated equilibrium technique were applied in this study. 99m-Tc DTPA first pass image obtained by RAO projection (upper), 99m-Tc HSA first pass image of LAO projection (bottom left) and gated equilibrium cardiac pool image of LAO view (bottom right) in the same normal subject were represented.

the regional wall motion abnormality with an advance of computer technology as noninvasive technique which makes the repetitive examinations relatively easy.

The purpose of this study was to determine the changes of the left ventricular hemodynamics and the left ventricular wall motion at rest and during anginal attack by exercise, utilizing qualitative and quantitative analysis of the radionuclide angiocardiography.

MATERIALS AND METHODS

The studies were performed in 28 patients with effort angina (age 43–65, mean 56.7 ± 9 years), 19 patients with old myocardial infarction (age 45–58, mean 54.6 ± 7 years) and 5 normal subjects (age 32–55, mean 48.5 ± 5 years).

The majority of these cases were examined by the left heart catheterization, X-ray cine-ventriculography and cine-coronary angiography using Angioskop system (Siemens).

Coronary stenosis of 50% or more in diameter on coronary angiography was defined as a significant coronary stenosis.

Normal subjects were selected from the patients with chest pain syndrome who were proved to have no significant coronary stenosis and no organic cardiac disease.

A) Procedures in the Resting State

The subjects were in supine position and Pho/Gamma camera LFOV (Searle) was placed close to the chest wall and directed to the heart.

The radionuclide ventriculograms were obtained following intravenous administration of radioactive tracers. All data were stored and

processed by the computer Scintipac-1200 (NOVA), for the analysis of the hemodynamic changes and a sequential analysis of the left ventricular wall motion.

i) Radionuclide angiography by the first pass technique and gated equilibrium technique

Fifteen mCi of 99m-Technetium Triamine Pentoacetic Acid (Tc-DTPA) was injected rapidly into the antecubital vein as a bolus. And the left ventriculogram was obtained in RAO projection (upper left in Fig. 1).

Ordinarily one hour later, 15 mCi of 99m-Tc human serum albumin (Tc-HSA) was re-injected intravenously, and first pass angiosgraphy was re-imaged in LAO projection (bottom left in Fig.1), after subtraction of residual cardiac activities.

Thereafter time was allowed for equilibration of isotope (5 minutes later). Venous sampling and external counting for one minute were performed in order to calibrate the radioactivity curve.

After 10 minutes, cardiac pool imaging was performed by gated equilibrium method in LAO projection (bottom right in Fig. 1).

The accumulation of counts was synchronized in relation to the cardiac cycle using the R wave of ECG as a physiologic marker for 3 minutes.

ii) Calculation of hemodynamic parameters

Cardiac output (CO) and stroke volume (SV) were calculated from the radiocardiogram of the right ventricular chamber according to the method described by MacIntyre et al.

Ejection fraction (EF) was calculated after subtraction of background activity using a computer-derived time activity curve of the left ventricular chamber (Fig. 2) by both first pass and equilibrium method.

End-diastolic (EDV) and end-systolic volume (ESV) were calculated by the following formula;

\[ EDV = SV/EF \]
\[ ESV = EDV - SV \]

iii) Assessment of left ventricular wall motion

The left ventricular edge determination was made automatically by isocount method in cut-level of 50–60% of peak activity counts of the
They were superimposed sequentially through end-diastole to end-systole with a frame duration of 40–60 msec.

For the determination of localization and severity of asynergy in radionuclide left ventriculograms, computer-derived end-diastolic and end-systolic images were individually divided into 4 equal parts, as referred in Fig. 4.

Segmental shortening of each part of the left ventricle, were calculated on the assumption that the hypothetical segment of I-d shifted to I-s during ventricular systole.

These hypothetically considered percent shortenings of the left ventricular segments were calculated by I-d/I-s x 100, where I-d is diastolic hemiaxial length of the segment I and I-s is systolic hemiaxial length of the segment I. (Fig. 4).

B) Calculation of Hemodynamic Parameters during Exercise

Stroke volume during exercise (SVe) is calculated and calibrated as follows; SVe = DCr x SVr, where SVr is the stroke volume determined individually at rest by the dye-dilution principle using first pass image in LAO. DCr and DCe are the difference of count accumulation between end-diastolic and end-systolic in the left ventricular chamber at rest and during exercise. And the data was normalized by heart rate.

For the calculation of ejection fraction, gated equilibrium technique was applied both at rest and during exercise.

Each accumulation of the counts was corrected by subtraction of background activity.

201-Thallium stress myocardial imaging.

Myocardial perfusion imagings at rest and during bicycle ergometer exercise were performed in all of 28 patients with effort angina from 5 different views with a dose of 2 mCi of 201-Tl using Ohio-nuclear Sigma 410-S with high resolution collimator. Exercise was continued until the appearance of anginal attack or a manifestation of ST changes on ECG, when 201-Tl was injected and the same level of exercise was continued another 1–2 minutes.

The image defect delineated by the "stress-injected" 201-Tl myocardial imaging was evaluated by the method previously reported by us.

Ten patients who agreed to the further examination were selected from these 28 patients, and they were examined by the radionuclide
angiography within a week or two following the same exercise protocol individually.

The location of the image defect was individually compared with the localization of the segmental wall motion abnormality in the radionuclide left ventriculograms.

For the comparison, both left ventriculogram and myocardial scintigram obtained during exercise were divided into 3 segments; they were anterior, inferior and lateral area.

RESULTS

A) Results in the Resting State

1. Cardiac indices

Cardiac indices by the method of radionuclide angiography were $3.77 \pm 0.65 \text{ L/min/M}^2$ on an average (ranged from 2.9 to 6.8 L/min/M²) in normal subjects, $3.79 \pm 0.58 \text{ L/min/M}^2$ on an average (ranged from 2.0 to 5.8) in patients with effort angina and $2.71 \pm 0.48 \text{ L/min/M}^2$ on an average (ranged from 1.8 to 4.6) in patients with old myocardial infarction. A slight decrease in cardiac index was observed in the group of old myocardial infarction. However, no significant changes were obtained between these 3 groups.

The results were well correlated to those obtained by conventional invasive techniques of contrast ventriculography using area-length method ($r = 0.63, P < 0.01$) as well as those of thermodilution technique ($r = 0.73, P < 0.01$) as shown in Fig. 5.

2. Ejection fractions

Ejection fraction determined at rest by the gated equilibrium technique in LAO 30° projection was $0.57 \pm 0.06$ on an average (ranged from 0.51 to 0.68) in normal subjects, $0.62 \pm 0.10$ on an average (ranged from 0.46 to 0.64) in patients with effort angina, and $0.35 \pm 0.05$ on an average (ranged from 0.17 to 0.51) in patients with old myocardial infarction.

In the comparison between these three groups, ejection fraction was significantly decreased in the group with old myocardial infarction ($P < 0.05$).

Ejection fractions determined by the first pass technique both in LAO 30° and RAO 30° were as follows; in normal subjects, ejection fractions were $0.64 \pm 0.15$ on an average (ranged from 0.52 to 0.79) in LAO, and $0.64 \pm 0.17$ on an average (ranged from 0.41 to 0.78) in RAO. They agreed with each other ($P < 0.001$).

In patients with effort angina, ejection fraction in LAO was $0.57 \pm 0.06$ on an average (ranged from 0.52 to 0.61) and $0.52 \pm 0.10$ on an average (ranged from 0.46 to 0.62) in RAO. They were correlated significantly ($P < 0.005$). In patients with old myocardial infarction, ejection fraction was $0.32 \pm 0.11$ on an average (ranged from 0.17 to 0.45) in LAO and $0.35 \pm 0.09$ on an average (ranged from 0.21 to
Ejection fraction determined by the radionuclide angiographic method (from time-activity curve by gated equilibrium technique) showed also a good correlation with that determined by contrast ventriculography using area-length method ($r = 0.92, P < 0.005$) (Fig. 7).

3. Left ventricular volumes

End-diastolic volume index (EDVI) was $84.5 \pm 20.9$ ml/M$^2$ on an average (ranged from 52.1 to 108.8 ml/M$^2$) in normal subjects, 92.6 $\pm 23.8$ ml/M$^2$ on an average (ranged from 60 to 130.4) in patients with effort angina, and 130 $\pm 36.2$ ml/M$^2$ on an average (ranged from 112.2 to 137.2) in patients with old myocardial infarction ($P < 0.01$).

These measurements had a good correlation with those measured by contrast ventriculography both in end-systole and end-diastole (Fig. 8).

4. The quantitative evaluation of the localization and the extent of wall motion abnormalities

Using computer-drawn outlines of the left ventricular chamber, the sequential left ventricular contraction from end-diastole to end-systole, was shown in Fig. 9 by the percent shortening in each segment.

In normal subjects, the percent shortening was 40–50% in each segment except an apical area of 100%.

In cases with old myocardial infarction, the segmental abnormalities in left ventricular wall motion were reflected by the location and extent of involved areas assessed by ECG changes (by the appearance and localization of Q wave, and ST, T changes).

These results were compared as shown in Fig. 10, between 3 groups, normal subjects, patients with anterior myocardial infarction and patients with extensive myocardial infarction assessed by ECG changes.

A decreased percent shortening (24% or less) of the left ventricular wall corresponded to the segment and the extent of an infarcted area.

Other hemodynamic data by the radionuclide angiographic technique, such as increasing end-diastolic volume, decreasing ejection fraction, systolic max dV/dt and diastolic max dV/dt, were also correlated to the extent of myocardial infarction (Fig. 11).

The comparison between the localization of segmental wall motion abnormality (asynchrony)

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Segmental Percent Shortening

<table>
<thead>
<tr>
<th>LAO 30°</th>
<th>Septal wall</th>
<th>Apical</th>
<th>Posterolateral</th>
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<td>2</td>
<td>3</td>
</tr>
<tr>
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<td></td>
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<tr>
<td>Extensive MI</td>
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</table>

<table>
<thead>
<tr>
<th>RAO 30°</th>
<th>Anterobasal – Anterolateral</th>
<th>Apical</th>
<th>Diaphragmatic – Posterobasal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Segment No.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Normal</td>
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<td></td>
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<tr>
<td>Anterior MI</td>
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<td></td>
<td></td>
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<tr>
<td>Extensive MI</td>
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</tbody>
</table>

Fig. 9. Comparison of segmental wall motion. (upper, LAO projection); segments of 1–3 reflect septal wall motion and segments of 5–7 are assumed as a projection of posterolateral wall motion. (bottom, RAO projection); segments of 1–3 are appropriate in observing anteroseptal and anterolateral wall motion, segments of 5–7 reflect diaphragmatic and posterobasal area.
Segment 4 is utilized for observation of apical wall motion.

Segmental Percent Shortening

<table>
<thead>
<tr>
<th>RAO</th>
<th>LAO</th>
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</thead>
</table>
| ![Diagram](image)

- Normal (n = 5)
- Anterior MI (n = 12)
- Extensive MI (n = 7)

Fig. 10. Schematic representation of segmental percent shortening summarized in 3 groups; normal subjects, patients with proved prior anterior and extensive myocardial infarction.
Hypothetically calculated segmental wall shortening reflected well the localization and extent of myocardial damage.

Assessed by the radionuclide left ventriculograms and the contrast cine-ventriculograms was performed in patients with a proven old myocardial infarction.

As summarized in Table I, the site of asynergy was determined with a sufficient accuracy.

However, three cases in which apical asynergy was shown by contrast angiography, were not demonstrated asynergy in the radionuclide angiography.

B) Results in Exercise State

In patients with effort angina, although an abnormal wall motion was frequently not observed in the resting state without anginal attack, segmental asynergy appeared when the patients developed anginal pain during exercise testing.

In a case of effort angina, as represented in Fig. 12, the localized asynergy was delineated (arrowed parts) in accordance with an anginal attack induced by exercise, while this patient showed no abnormality of wall motion in the left ventriculogram at rest. This patient had proved triple vessel disease with 80% stenosis.
**P < 0.05  
* P < 0.005

![Graph showing hemodynamic parameters](image)

**Fig. 11.** Hemodynamic parameter. A decrease of ejection fraction and systolic max dV/dt, otherwise an increase in end-diastolic volume permit determination for decreased ventricular performance in accordance with the extent of myocardial damage.

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>LOCALIZATION OF ASYNERGY</th>
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<tr>
<td>Asynergy in RN Angio</td>
<td>Asynergy in Contrast Ventriculography</td>
</tr>
<tr>
<td>Anterior MI</td>
<td>Anterior MI</td>
</tr>
<tr>
<td>Apical MI</td>
<td>Inferior MI</td>
</tr>
<tr>
<td>Inferior MI</td>
<td>Asynergy(−)</td>
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<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
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The site of determined asynergy following radionuclide angiographic analyses are individually well corresponded with that of cine-ventriculography with sufficient clinical utility. Nineteen patients with old myocardial infarction were investigated, 7 patients showed no manifested asynergy and a patient had apical and inferior asynergy.

**Fig. 12.** Appearance of asynergy is shown during induced anginal attack with exercise test (arrowed).

The patient with effort angina, angiographically proved triple vessel disease, showed a normal left ventriculogram at rest without anginal attack (right).

When anginal attack developed with exercise, the ventriculograms showed a manifestation of asynergy. Quantitatively, the site and extent of asynergy were determined by comparing with the values of percent shortening at rest and during anginal attack (bottom).

of LAD, 50% stenosis of RCA and 90% stenosis of Lcx branch.

Quantitatively, this patient showed a decreased percent shortening in segment 2, 3, 4, and 5 reflecting involved anteroseptal, and apical area (Fig. 3, 12).

In 9 of the patients with effort angina hemodynamic parameters could be examined repetitively when the patients developed anginal attack during the same level of exercise testing (Fig. 13).

Ejection fraction at rest decreased from $0.62 \pm 0.09$ to $0.46 \pm 0.11$ during developed

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angina with exercise. A slight decrease in systolic max $dV/dt$ from $232 \pm 81\%$ ml/sec to $227 \pm 61\%$ ml/sec and diastolic max $dV/dt$ from $225 \pm 50\%$ ml/sec to $202 \pm 49\%$ ml/sec were also observed during an evoked angina, while end-diastolic volume index at rest increased from $92.6 \pm 23.8$ ml/M² to $116.7 \pm 46.8$ ml during angina.

The significant (P < 0.01) decrease in ejection fraction was obtained, although an increase in end-diastolic volume and a slight decrease in systolic and diastolic max $dV/dt$ were not significant, the results suggested a tendency of transient reduction of left ventricular performance during angina.

Moreover, in patients with effort angina the relation between the site of the segmental wall motion abnormalities and the localization of defects in myocardial imaging, was suggestive of myocardial ischemia (Table II).

The site of wall motion abnormalities which appeared during an exercise-induced angina, agreed with the location of 201-Tl defects in “stress-injected” myocardial imaging.

Eighteen out of 30 segments determined from 10 patients with effort angina showed manifest image defects in myocardial scintigrams.

Asynergy was demonstrated in 14/30 segments, 12/18 segments agreed, and 4/18 segments disagreed with these defects in myocardial stress-scintigrams.

In anterior segment, an appearance of asynergy and perfusion defect agreed with each other in 6 cases, and not correlated in 2 cases.

In inferior segment, the location of the asynergy and the defect agreed with each other in 2 cases and disagreed in 1 case. And in latero-posterior segment, the site of the asynergy and the defect agreed with each other in 4 cases and disagreed in 3 cases.

**DISCUSSION**

For the evaluation of left ventricular performance⁸⁻¹² radionuclide angiography has been shown to be useful with the first pass technique and the gated equilibrium technique.

However, each of these techniques has intrinsic advantages and disadvantages. With the first pass method, the data collection is completed within the first transit of circulation, usually less than 30 seconds for most patients. The sequential arrival of radionuclide in the right ventricle, lungs and left ventricle permits the resultant temporal separation of each chamber which avoids an anatomic overlap by selecting optimal projection.

However, this technique has a limitation of insufficient imaging because of a short acquisition time. Moreover, in the first pass method, imaging is limited to a single view. The right anterior oblique view permitted a visualization of anterior, apical and posteroinferior areas of the left ventricle, while the left anterior oblique view provided optimal direction for the delineation of the septal and posterolateral areas. In the studies, 99m-Tc DTPA was used at first in RAO projection followed by 99m-Tc HSA in LAO projection after 1 hour to obtain images in a different view.

The gated equilibrium technique⁹ with its single projection, permits repeat studies to
TABLE II  LOCALIZATION OF ASYNERGY VS 201-T1 MYOCARDIAL IMAGE DEFECT

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Procedure</th>
<th>Anterior</th>
<th>Inferior</th>
<th>Latero-posterior</th>
<th>CAG</th>
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<td>1</td>
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<td>201-T1 myocardial scintigram</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>LAD 99%</td>
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<tr>
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<td>46 y.o.</td>
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<td>2</td>
<td>male</td>
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<td>+</td>
<td>-</td>
<td>WNL</td>
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<td>4</td>
<td>male</td>
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<tr>
<td></td>
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<td>RI angiogram</td>
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<td>+</td>
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<td>LCx 90%</td>
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<tr>
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<td>RCA 90%</td>
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<tr>
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<td>male</td>
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<tr>
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<td>-</td>
<td>LCx 75%</td>
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<td>-</td>
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<td>(−)</td>
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<td>+</td>
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<td>+</td>
<td>-</td>
<td>+</td>
<td>LAD 100%</td>
</tr>
<tr>
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<tr>
<td>8</td>
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<td>(−)</td>
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<td>10</td>
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<tr>
<td></td>
<td>70 y.o.</td>
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The site and extent determined by the radionuclide angiographic analyses agreed individually well with those of image defect determined by 201-T1 imaging study. Both T1 myocardial scintigrams and RI angiograms were obtained during exercise. The presence of defect or asynergy is shown by +. CAG; coronary angiographic findings. WNL; within normal limit. (−); coronary angiography is not performed.

observe the effects of intervention such as exercise-testing. Assessment of wall motion abnormalities at rest and during exercise was made by LAO view of the gated equilibrium technique.

By this technique an adequate acquisition of counts makes it possible to analyse the hemodynamic changes during exercise.

For the calculation of hemodynamic parameters, the count-based method is commonly used with a computer by which the changes in counts from end-diastole to end-systole against time are processed.

The advantage of this technique is the relative independence of the time-activity curve from geometric assumptions inherent in the area-length technique. However, care could be taken in assigning the appropriate end-diastolic region of interests.

The major limitation of the count-based calculation involves appropriate correction for background activity.

Back ground activity in a first pass or a gated equilibrium study results primarily from count arising outside of the left ventricular chamber. Furthermore, overlapping of the structures, such as the left atrium, pulmonary vasculature and aorta make difficult to accurate edge delineation of the left ventricular end-diastolic and end-systolic silhouette. In this studies,
angled LAO (inclined 10°–15° to the head) was applied in order to avoid inaccurate detection of the left ventricular chamber from the anatomical overlap.

To assess an asynergy of the left ventricle, several approaches have been used. These include qualitative observations by radionuclide cine-ventriculograms and more quantitative approaches using computer-drawn outlines of the left ventricular chamber. Although it has been emphasized that determination of edge of the left ventricular chamber is fundamental to any assessment of either wall motion or hemodynamics, the quantitative analysis of wall motion has been provided by the iso-count method we described previously, for serial studies of left ventricular contraction using the computer-drawn outlines of the radionuclide activity in the left ventricle.

However, an optimal cut-level selection was necessary in a case either with an enlarged chamber or an inadequate accumulation of counts such as observed in congestive heart failure.

In this study, 3 cases in which apical asynergy was observed by the contrast ventriculography were not demonstrated in the radionuclide angiography. Therefore, care must be taken in diagnosing asynergy in apical area.

Until recent years, invasive procedures such as contrast ventriculography have been the only method by which the sequence of the left ventricular contraction could be assessed.

Since Blumgart first used a radioactive tracer to evaluate velocity of blood flow in man, remarkable improvements in hemodynamic studies have been made by imaging devices and computer techniques.

From our results in resting state it was observed that left ventricular wall motion analysis, as well as computer processed hemodynamic data, such as contrast cine-ventriculography and thermodilution technique.

These non-invasive studies were also of particular use for assessing left ventricular performance in exercise state.

In patients with effort angina, it was frequently observed that both hemodynamic changes and an appearance of asynergy were seen only when anginal attack was provoked with exercise testing.

Consequently, these techniques should be applied for evaluating ischemic heart disease and permit determination of left ventricular performance with sufficient clinical usefulness.

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