Nontraumatic Assessment of Left Ventricular Wall Motion and Regional Stroke Volume After Myocardial Infarction

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The regional contraction patterns of the left ventricle, shortly after myocardial infarction, were assessed from computer-processed scintigraphic images and histograms of the first transit of an intravenously injected radionuclide bolus. Seventy-seven patients with documented myocardial infarction were injected with a compact bolus of ^{99m}Tc-pertechnetate which was coordinated with the ECG so that it arrived in the superior vena cava during diastole. Precordial activity during the initial passage was recorded in 50-msec intervals with a multicrystal scintillation camera interfaced to a dedicated minicomputer. Data frames of 4–7 cardiac cycles were summed into one representative cardiac cycle. In 73 of the 77 patients the images of the representative cycle, along with the corresponding time–activity curves, indicated wall-motion and stroke-volume anomalies corresponding with the electrocardiographic location of the infarct. This nontraumatic, essentially noninvasive technique permits serial examinations of the acutely ill patient for the spatial identification and estimation of suspected myocardial infarcts.

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After a myocardial infarction, there are three essential considerations that will determine the course of further treatment of the patient:

1. The amount of viable myocardium contained within the area of the infarct.
2. The severity of impairment of the function of this area, i.e., how well does it contract and expand?
3. The adequacy of the blood supply to this area through the corresponding vessels or collaterals.

An attempt has been made to look at the first two considerations noninvasively with nuclear medicine techniques: through the injection of a radionuclide that penetrates the myocardium, eventually being metabolized and washed out; the infarcted area then shows up as a "cold" or "hot" spot (*1–5*). The second is through the observation of the movement of a radioactively tagged blood pool in the heart (*4,6,7*), or through an analysis of the first transit of a radioactive bolus through the left heart (*8–12*). The ability of the latter method to visualize the wall motion and the regional stroke volume distribution of the left ventricle merely by means of an intravenous arm injection will be described. This method, in contrast with invasive angiography, can be performed with no danger to the patient during the first week after a myocardial infarction.

MATERIALS AND METHODS

Patients. A total of 90 examinations were performed in 77 patients, ranging in age from 27 to 75 yr. Each of the 77 patients was studied once, six were studied twice, two three times, and in three patients two subsequent studies were done in two different projections. All patients had been hospitalized, and myocardial infarction was established by

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electrocardiogram and serum enzyme tests. The location of the infarct, the time from infarction, and the number of reinfarctions are listed in Table 1.

Data collection. The following technical requirements must be fulfilled if one is to obtain sufficient activity over the left ventricle to permit a comprehensive evaluation of the data from an intravenous injection of 15–20 mCi of $^{99m}TcO_4$ into an antecubital vein.

1. A compact bolus must flow into the heart.
2. A scintillation camera with high temporal resolution and high sensitivity will be needed.
3. Simultaneous recording of the electrocardiogram, injection, and data frames are necessary.
4. Computer assistance is needed for data preparation and reduction.
5. The projection of the heart must be appropriate.

A compact bolus input in the heart is achieved by the following technique. After puncture of an antecubital vein, 15–30 mCi of $^{99m}TcO_4$ in 0.5 ml of saline are placed in a short Teflon tube (capacity, 0.5 ml) connected to the needle and a standard radiographic injector* (Fig. 1). Then, a rapid injection of the tracer bolus is carried out, with a saline flush of 16 ml at a flow rate of 8 ml/sec. The injection is timed so that the bolus arrives in the superior vena cava during diastole.

In dynamic scintillation imaging of rapid events, the resolution of a camera system consists of three components: temporal (the framing rate), statistical (the information density), and spatial (*9,13*). These

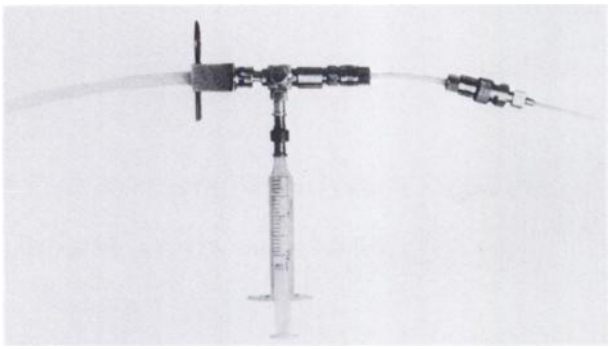

FIG. 1. Injection system consisting of a needle, short Teflon tube (capacity 0.5 ml), and three-way stopcock to which injector (flush volume 16 ml at 8 ml/sec) and syringe containing activity are attached.

three components are not mutually independent; rather, they must be optimized simultaneously. An attempt to improve the temporal resolution (by higher frame rates) without a concomitant increase in the statistical resolution (by increased count rate) may in fact decrease the total resolution, impairing the ability to detect a defect in the count distribution within the organ of interest.

In view of the rapid movements of the heart, a time resolution of 50 msec (20 frames per second) is necessary to achieve a succinct and accurate framing of each phase of the cardiac cycle. For example, end-diastole is signalled electrocardiographically by the upslope of the R wave, which lasts approximately 50 msec. If a longer count interval is chosen, the frame may already include early hemodynamic elements of systole, thus blurring the temporal resolution. This blurring means that the ventricle would not necessarily be imaged in its state of maximum diastolic extension and a measurement derived from the image at "end-diastole" would include that element of error. Similarly, at gross framing rates, such as 5/sec, significant arrythmias (e.g., extrasystoles) might be completely masked by submersion in a normal beat.

Rapid framing rates will have minimal clinical utility, however, if the number of counts in each frame does not provide an adequate information density and signal-to-background ratio. Since the counts of scintillation imaging are governed by Poisson statistics, the "error" (standard deviation) of the information density is the square root of the information density. These considerations play a role during systole, when the change in counts over the ventricle should be proportional to the change in volume. But if the counts are so low that the change in count rate is 2–3 standard deviations of the end-diastolic count, this would appear to preclude accurate measurements based on such regional count

TABLE 1. COMPARISON OF ELECTROCARDIOGRAPHIC AND RADIONUCLIDE EVALUATION OF MYOCARDIAL INFARCTIONS

	Number of Cases	
	Electrocardiogram	Radionuclide angiogram
Patients (total)	77	77
Examinations (total)	77	90
Time from infarction:		
5 days to 5 weeks	33	—
more than 5 weeks	44	—
Reinfarctions	5	—
Correspondence between ECG and radionuclide location of infarct:	77	73 (95% of 77)
anterior	36 (47% of 77)	35 (97% of 36)
posterior	38 (49% of 77)	36 (95% of 38)
anterior and posterior	2	2
subendocardial	1	0

rate changes, e.g., the regional distribution of ejection fractions. Such factors become increasingly critical with lower and lower ejection fractions. Finally, the signal-to-background ratio must be sufficient for the definition of valve planes and the edge of the cardiac silhouette against the surrounding background. Without this, the accurate flagging of the ventricular region of interest, the assessment of ventricular wall motion, and the location of pure background regions for determining background corrections would be extremely difficult. With the properly timed summation of several heart cycles encompassing the first transit of the radionuclide bolus we achieved an average total count of over 30,000 counts in the uncorrected end-diastolic image, and this was usually adequate for the considerations being studied.

Spatial resolutions of 1 cm or less, which are nondegradable with increasing count rates, are adequate (*14*). Since the data are to be quantitatively analyzed, field uniformity and deadtime corrections should be applied to ensure that changes in counts are directly proportional to fluctuations in the amount of radioactivity within the camera's field of view.

A scintillation camera with a multicrystal matrix† was used in this study. Such a multicrystal camera offers frame durations down to 50 msec with count rates over 200,000 cps, and a spatial resolution of 1 cm in dynamic studies. The sensitivity of the 1.5-inch parallel-hole collimator gave 9,000–12,000 counts for each millicurie of the injected bolus within the field of view.

Simultaneous recording of the recorded frames and the patient's electrocardiogram is used for precise correlation of frames with the events of the cardiac cycle. This is particularly useful for determination of the end-diastolic frames during bolus transit through the left ventricle. The mechanical systole usually follows the R wave, so that the end-diastolic frame corresponds to the frame before the electrocardiographic R, although sometimes including it.

Basic computer programs must include capabilities to add, subtract, and divide sequences of frames and curves.

As in angiographic investigations, the right anterior oblique (RAO) projection was employed. The detector was rotated 5° and, in addition, the patient was turned somewhat to his left. In special cases (septal infarction or ventricular septal defect), the left anterior oblique projection was used, or a second injection and dynamic study was performed following the RAO study.

Data processing. The data reduction recognizes essentially three stages. First, the passage of the bolus through the various chambers on the right side (superior vena cava, right atrium, right ventricle, and pulmonary artery) is observed visually on the monitor and quantitatively recorded on histograms. Delays of transit and washout can be detected easily.

Then, a representative cardiac cycle (images of one diastole and one systole) of the bolus's transit through the left ventricle is formed. First, one defines from a serial display (consisting of 12 images) the frames with maximum count rates over the left heart. Then, on the electrocardiogram, one determines the end diastolic frame of the corresponding cardiac cycle, verifying by visual control that this frame presents maximum distension of the left ventricle and the next frame shows starting contraction. In practice, 4–7 sequential end-diastolic frames are selected (Fig. 2). A program in the system's computer is used to add the frame-content for the first cycle to that for the next one, etc., until all selected end-diastolic frames (ED), first systolic frames (S_1), second systolic frames (S_2), etc., are stored

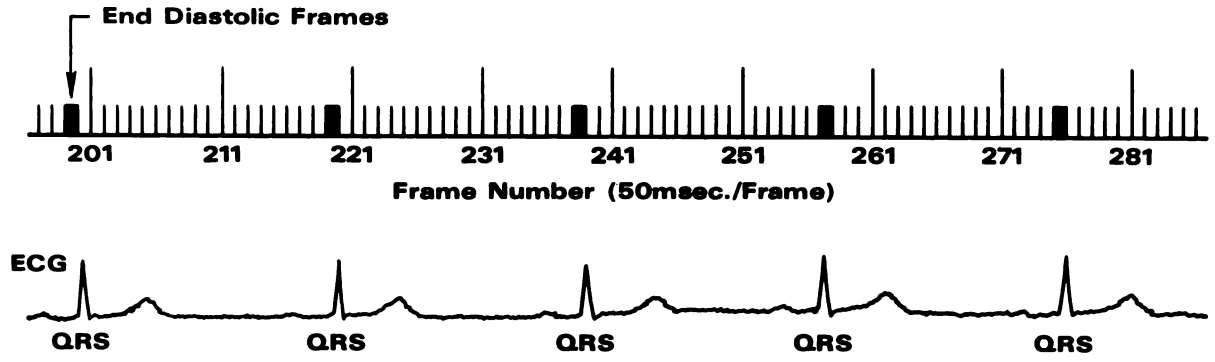

FIG. 2. Simultaneous recording of frames and patient's ECG. Accumulation intervals 50 msec. Black bars correspond to end-diastolic frames, where sequences of cardiac cycles start for summation. Time scale: 50 mm/sec.

as individual sums. The result is a representative cycle of the left ventricle starting at end-diastole and ending at the following end-diastole, showing left ventricular contraction and diastolic filling.

The images of the representative cycle facilitate the zoning of such regions as the left ventricle, aorta, and left atrium since the totals are statistically adequate for border definition. After recording the corresponding histograms, the same regions of interest are used for recording the histograms of the representative cycle. The left ventricular histogram is then corrected by background subtraction (*14*) so that the ejection fraction can be read directly from the curve. Delays of transit and washout can easily be shown, and (if present) peaks of reflux into the left atrium detected, indicating mitral insufficiency.

In the third step, the individual frames comprising the representative cycle are linearly interpolated from a data matrix of 14 × 21 (294 points) to one of 56 × 84 (4,704) points). The interpolated frames are stored for further analysis, and the background is eliminated without altering the data over the left heart. The color display of the system reproduces the activity distribution in sixteen shades of color from dark green (6.25%) to bright yellow (100%). The heart border is defined where the color contours lose irregularities and follow the shape of the heart.

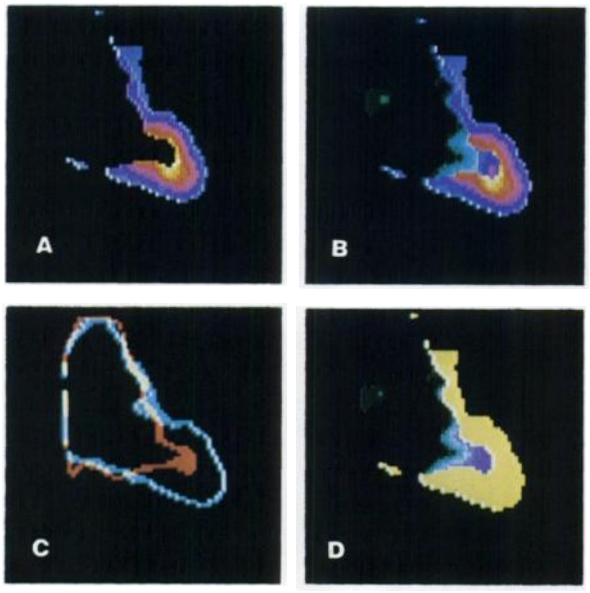

FIG. 3. Normal left ventricle. (A) Systolic wall motion: outer circumference = end-diastole; inner circumference = end-systole (removed); width of band gives a measure of wall motion. This is symmetrical between anterior and posterior walls. (B) Spatial stroke volume distribution: composed of the wall motion in plane of view and ejected volume displacement in other planes. Symmetrical distribution. (C) Perimter: outer perimeter (bright blue) = end-diastole; inner perimeter (red) = end-systole. Distance between perimeters = wall motion. End-systolic perimeter lies completely inside the end-diastolic; normal systolic centripetal motion of wall. (D) Spatial distribution of ejection fraction: bright yellow represents 100% ejection and corresponds to wall motion in plane of view. Symmetric distribution.

The image of left ventricular wall motion is produced by subtracting the shape of the heart at end-systole (the "mask" or normalized end-systole) from the image at end-diastole. The regional or spatial distribution of stroke volume is derived by subtracting the digital image at end-systole from that at end-diastole. By dividing the stroke volume frame by the end-diastolic frame, the spatial distribution of ejection fraction is obtained. Finally, the perimeters of left ventricular end-diastole and end-systole can be added to form the "perimeter image." Other images, also derived from the representative cycle and conveying additional information, are in the process of evaluation. New programs that automatically process some of the above images and curves, particularly the representative cycle, are now available and reduce processing time considerably.

Interpretation. After myocardial infarction, the evaluation of a dynamic study is particularly concerned with the following:

1. The status of left ventricular wall motion from end-diastole to end-systole.
2. The spatial distribution of the stroke volume in the left ventricle.
3. The spatial and total ejection fraction of the left ventricle.
4. The degree of congestion in the lung and involvement of the right heart.
5. Further complications.

The wall motion between end-diastole and end-systole is shown in Fig. 3A as a horseshoe, whose outer edge is the circumference of end-diastole and whose inner edge is the circumference of end-systole. One can determine immediately from the width of the "wall motion band" the extent of the wall motion.

Normal wall motion, as known from angiography, is approximately symmetric on the anterior and posterior walls of the heart (*15*). During systole, change in length of the transverse axis of the left-ventricular ellipsoid, assumed in angiographic measurements of left-ventricular volume, amounts on the average to 25% (*16*). After myocardial infarction, the wall motion of an affected area is frequently reduced, i.e., hypokinesia occurs, so that in the hypokinetic portion of the wall the width of the motion band will be narrowed compared with the mean width of the wall motion band (see Discussion). If, in a particular area, the wall-motion band is broken, one must decide whether this results from akinesia or dyskinesia (paradoxical motion). The image of the

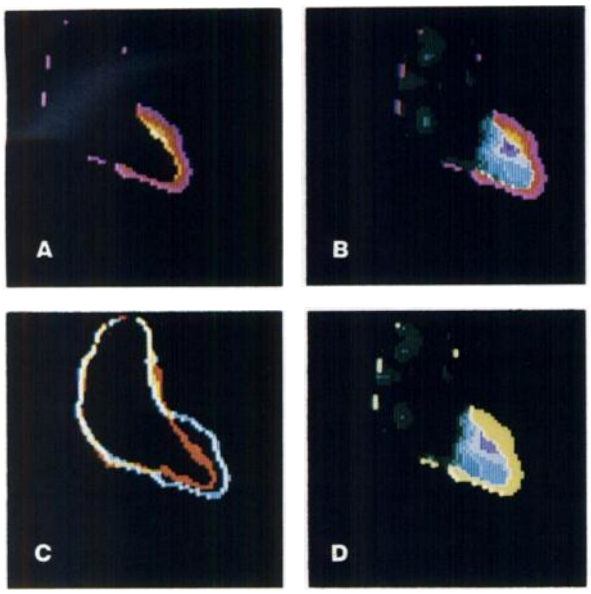

FIG. 4. Left ventricle with 25-day-old posterior infarct, RAO view. (A) Systolic wall motion: hypokinesia predominant on posterior wall, extending down to apex of left ventricle with normal dimensions. (B) Spatial stroke volume distribution diminished in same region of reduced wall motion (posterior wall to apex). (C) End-systolic perimeter (red) and end-diastolic perimeter (bright blue) show hypokinesia on posterior wall and to lesser degree at apex. (D) Spatial ejection fraction distribution is asymmetric, with maximum reduction at posterior wall.

heart in end-systole with the end-diastolic perimeter superimposed, or the added end-systolic and end-diastolic perimeters, permit one to differentiate between akinesia or dyskinesia. Normally the outer edge of end-systole lies within the end-diastolic perimeter and more or less uniformly separated from it (Fig. 3C). If, however, end-systole at any point lies outside the end-diastolic perimeter, dyskinesia is indicated. Akinesia is shown when the end-systolic perimeter overlaps the end diastolic one (Fig. 6C).

The image of the spatial distribution of the stroke volume is a two-dimensional representation of the three-dimensional blood volume displacements arising from wall motion parallel with and perpendicular to the plane of the detector. Therefore, information about the spatial origins of the stroke volume is contained in this image.

Under normal function the stroke volume inferred from anterior and posterior wall displacements is fairly symmetric (Fig. 3B). Decreasing stroke volume originates from the space of residual ventricular volume below the aortic valve. A region of diminished wall motion usually involves to a reduction of the underlying space of stroke volume. The greater this reduction, the larger the deficient area; hence, the image of the spatial distribution of stroke volume offers a basis for the evaluation of wall motion (contraction) perpendicular to the detector.

The spatial distribution of the ejection fraction seen in the plane of the detector is obtained by dividing the spatial stroke volume distribution by the end-diastolic image. In the area of wall motion there is 100% ejection; in the outflow tract below the aortic valve, ejection decreases rapidly as does stroke volume (Fig. 3D).

Total ejection fraction, a weighted average of the spatial fractions, can be read directly from the histogram of the representative cycle after background subtraction. It also can be calculated from an average of the greatest three maxima (end-diastoles) and minima (end-systoles) of the left ventricle histogram after the representative background has been subtracted out (*14*).

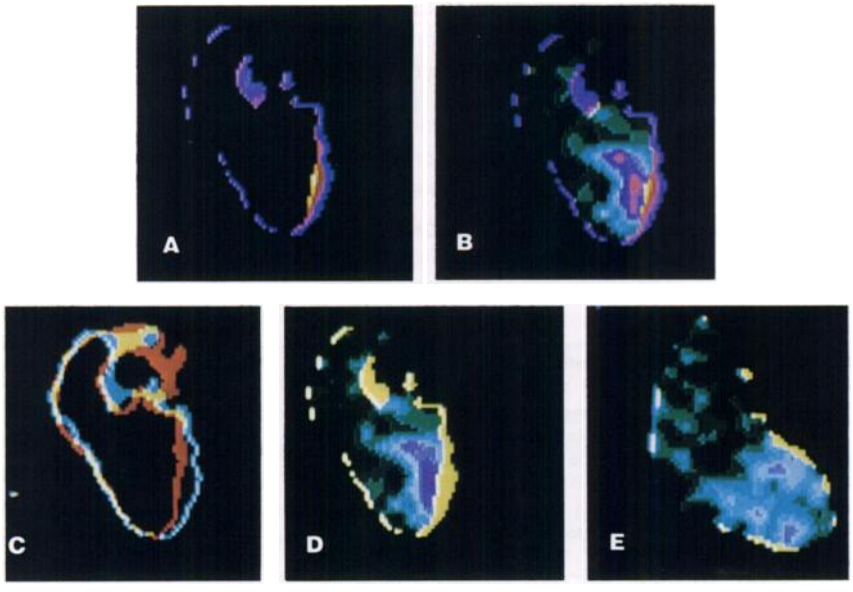

FIG. 5. Left ventricle with anteroseptal infarct (2 months after infarction). (A) Systolic wall motion (LAO) reduced, particularly along septum. (B) Spatial stroke volume distribution (LAO) with deficient areas (distribution defects) from septum to apex. (C) LAO view of end-systolic perimeter (red) and end-diastolic perimeter (bright blue) show severe hypokinesia and akinesia (yellow) along septum. (D) Spatial ejection fraction distribution (LAO) shows ventricular ejection coming primarily from lateroposterior wall. (E) Spatial ejection fraction distribution (RAO) shows additional akinetic zones on posterior wall, with small distribution defects.

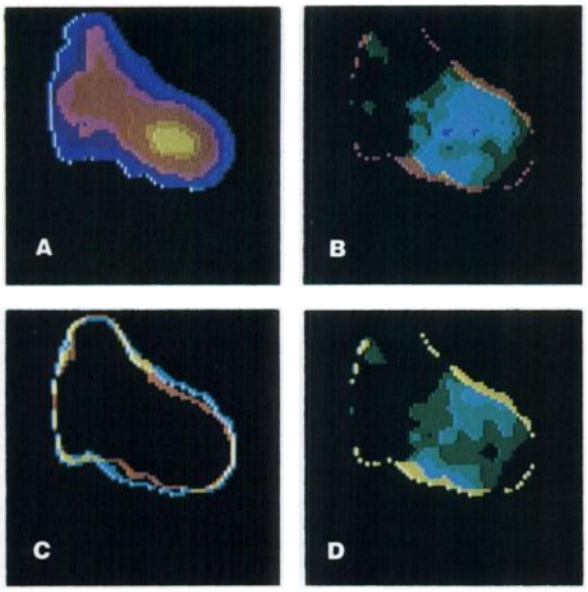

FIG. 6. Left ventricle with 2-year-old anterior infarct (RAO); patient currently suffering increasing angina and decreasing exercise tolerance. (A) End-systolic high activity pool in ventricle, characteristic of left ventricular aneurysm. (B) Spatial stroke volume distribution is severely diminished in dilated left ventricle (left heart failure) and has apical distribution defects. (C) End-systolic perimeter (red) and end-diastolic perimeter (bright blue) show severe hypokinesia and akinesia (yellow) around apex. (D) Spatial ejection fraction distribution clearly shows severe reduction in region corresponding to aneurysm and has central distribution defect typically seen in aneurysms.

RESULTS

The frequency of the observed anterior and posterior wall infarcts is reported in Table 1. In 73 of 77 patients the location of the major anomalies of wall motion and stroke volume corresponded to the electrocardiographic location of the infarct. Three of the exceptions consisted of a patient with a subendocardial infarct and two patients with 3-week-old posterior infarctions. In these patients end-systole showed a symmetric contraction of the free and diaphragmatic walls. The final exception was a patient where only the left anterior oblique (LAO) projection was recorded; hence it was not possible to determine precisely the site and extent of motion anomaly.

All the other patients exhibited wall-motion disorders, as is summarized in Table 2. In 31 patients the involved portion of the wall recovered so well that only hypokinesia of the infarcted area remained. In 12 of these patients hypokinesia was combined with short-lasting dyskinesia. On the other hand, 42 patients had areas of akinesia, mostly in conjunction with hypokinetic segments (31) or dyskinesia (11) (Figs. 4 and 5).

Three patients presented aneurysms of the left ventricle (Fig. 6). One of these developed the aneurysm between the time of the first radionuclide study (2 weeks after infarction) and the second study (3 weeks later). Three patients had ventricular septal defects, and 43 exhibited generally slight to moderate mitral reflux. This can appear either as a result of generalized ventricular dilation or of a posterior-wall infarct, probably with papillary muscle dysfunction.

Forty-four patients showed diminished wall motion in areas other than those associated with the major wall motion disorder in the infarcted region. In some cases this could be attributed to earlier infarcts or multiple infarcts.

TABLE 2. RADIOTRACER-DIAGNOSED ABNORMALITIES

	Number of cases
Wall motion anomalies (total):	73
hypokinesia	19
hypokinesia and dyskinesia	12
akinesia and hypokinesia	31
akinesia and hypokinesia + dyskinesia	11
Left ventricular aneurysm	3
Ventricular septal defect	3
Mitral insufficiency (slight to moderate)	43

DISCUSSION

The diagnosis of myocardial infarction is based on elevated serum enzymes and on typical electrocardiographic patterns (ST-segment elevation, T-wave changes, abnormal Q waves, reduced R waves), the latter being well-established indicators of the location of the infarcted area in the acutely ill patient. Patients without positive findings and nonhospitalized patients were excluded from the study. Comparison of the major disturbances of wall motion and of the spatial distribution defects of stroke volume and ejection fraction showed correspondence with the electrocardiographic site of the infarct in 73 of 77 patients, thus indicating the functional impairment of the infarcted area.

Anomalies of wall motion are as much a result of ischemia of the myocardium as of necrosis and scar formation, eventually even of disturbances of condition. Thus, an area of reduced or absent wall motion along with an imaged defect in the spatial stroke volume distribution or ejection fraction cannot answer questions as to the extent of the remaining viable myocardium, the size of the scar, and the degree of perfusion in the involved region. These are reserved for other nuclear medicine techniques. Nonetheless, the dynamic study of the wall motion and spatial stroke volume distribution allows one to establish the degree of impaired function of the left ventricle as a result of both diminished perfusion

and scar formation. Furthermore, areas of reduced motion at the opposite wall may be due to a previous infarction or to ischemia through a second significantly narrowed coronary vessel, and thus can provide an indication for further diagnostic procedures. One can also assume that collateral flow to the major area of motion disturbance may be poor or absent.

This information can be obtained within the first weeks after an infarct, as in 33 of our patients, namely, during a period in which the course of the disease is determined and eventual complications may set in. Thus this important information can be obtained early, with no risk to the patient and with much less radiation exposure than that accompanying invasive procedures.

It is our policy to study patients early after infarction if no clinical improvement in the intensive care unit can be achieved. Thus, complications such as ventricular aneurysm, mitral insufficiency, and ventricular septal defect, which would eventually require further procedures, can be detected early. In all other cases, the dynamic study is done before the patient is discharged from the hospital, so that his left ventricle is functionally evaluated before rehabilitative treatment or further diagnostic procedures takes place.

A strong correlation between radionuclide ejection-fraction measurements and angiographic measurements has been proven in several series (*11–14*) in which the computation of the ejection fraction used the integral of the end-diastolic and end-systolic spatial activity distribution over the left ventricle. Hence, accuracy and proportionality to blood volumes can be presumed for these frames, which are also used in this study for elaboration of the images of wall motion and stroke volume distribution. Furthermore, these frames are taken from the representative cycle (ED_1 and ES) derived from the heart cycles on the downslope of the left ventricular histogram curve. This guarantees optimal mixing and the averaging of slight differences in volume displacement from beat to beat. The subsequent subtraction procedures eliminate the remaining background in front of and behind the left ventricle. Indeed, wall motion represented on the wall-motion frame does not differ significantly from that resulting from the stroke volume image.

Ventricular wall motion is considered as normal if: (A) it is symmetrical on the anterior and posterior walls; and (B) systolic shortening amounts to an average of 25% of the transverse axis. Nevertheless, there still may be questionable cases of hypokinesia. In such instances, the image of the regional stroke volume distribution is useful in deciding whether hypokinesia is indeed present. In significant hypokinesia, the narrowed wall motion band is always accompanied by a reduced activity in adjacent areas of the regional stroke volume distribution.

Difficulties in the evaluation of the wall motion may also arise when the left ventricle is in failure, in which case the motion of the entire wall is significantly decreased, so that differences between the wall segments may vanish. In these instances the images of the spatial stroke volume and ejection fraction are useful, since they still show "defects" in the distribution of the activities corresponding to the most severely affected wall areas. If these cannot be detected, the region of major disturbance may be located at the septum following septal infarction or strictly lateral. Consequently, a second study in the LAO projection may be indicated.

With the gated blood pool method, the LAO projection is preferred because overlapping of the right and left sides of the heart is avoided. In all other projections, the overlapping increases as the projection is rotated from the straight anterior to the RAO view. When a true lateral view of the left ventricle is reached, i.e., without foreshortening of the long aorta-to-apex axis, maximum overlapping of the left and right ventricle occurs, making studies of the spatial distribution of activities within the left ventricular cavity very difficult. Therefore, we prefer to study the first passage of the tracer using: (A) the aforementioned special bolus technique in which there is no overlap of the right and left heart because of their *temporal* separation, combined with (B) the RAO projection, which views the maximum spatial extension of the activity distribution within the cavity.

With the gated blood pool technique, one usually accumulates from 200,000 to 500,000 counts over several hundred cardiac cycles to produce images at end-diastole and end-systole. Significant information, however, lies in the volume changes during ventricular contraction and dilation, as is well known from the pressure–volume curves of the ventricles. Thus, recording of the entire cardiac cycle is important.

To increase count rates during first tracer passage through the left ventricle, a compact bolus, a highly sensitive and fast detector system, and a summing procedure must all be combined. In preliminary tests (*17*) we have shown that, with the described injection technique using flow rates of 8 ml/sec and flushing volumes of 16 ml saline, a significantly shorter and more compact bolus could be achieved than with the usual Oldendorf technique (range of bolus rise time: 0.6–1.0 sec vs. 0.8–2.3 sec). Flow rates greater than 8 ml/sec may fragment the bolus;

lower rates may not be as efficient and approach hand-injection velocities. Flushing volumes in excess of 16 ml may dilate the venous system, producing a premature diffusion of the bolus, while lower flushing volumes may not be sufficient to displace the blood column up to the superior vena cava.

With this injection technique, the bolus is accelerated and, in adults, appears in the superior vena cava after about 0.8 sec (0.74 ± 0.03; $n = 109$). Timing of the injection is done with the trigger unit of the injector, so that bolus arrival occurs during diastole, usually with an effective arrival in the right atrium during systole. Since the tricuspid valve is closed during systole, the bolus accumulates in the right atrium and produces a maximum count in the right ventricle immediately during the diastole that follows. Diastolic arrival in the right atrium would lead to a division of the bolus activity between the right atrium and ventricle, with lengthening of the bolus.

Particular attention is required in the determination of the end-diastolic frames of the representative cycle. Experience in the analysis of left ventriculograms has shown that the time of the R wave corresponds best to the end-diastolic cine frame. With a frame rate of 20 per second in radioactive studies, the end-diastolic frame is the last accumulation interval before the R wave, frequently comprising the peak of the R wave. If the selected accumulation interval comprises, besides the peak of the R wave, a larger segment of the descending limb of the deflection, it may already include the first systolic frame; therefore, the frames are initially determined with the aid of the ECG and then are visually displayed. Because the summing up of end-diastolic and first-systolic frames would blur information, visual control of the frames with maximum left ventricular distension, and of the following frames, which should represent initial contraction, is recommended. Temporal resolution better than 50 msec would certainly reduce the error that is possible if the end-diastolic frames are selected only by means of the electrocardiographic R wave and without visual control.

In significant arrhythmias, cycles of approximately the same length are selected for summation. Fortunately, minor changes in cycle length affect more the diastolic phase than the systolic phase, so that the error may increase towards the diastolic end of the representative cycle, but is minimal during the early systolic portion. Indeed, for preparation of the stroke volume images, spatial, and total ejection fraction, the first end-diastolic frame is always used. If repeated ventricular premature beats occur, a bolus of lidocain precedes the tracer injection. There has not been a single case in which a representative cycle could not be developed.

In 70 consecutive cases (comprising coronary and valvular disease as well as myocardial infarction and normals), an average of 7.4 (± 0.18) end-diastolic frames could be selected for summation (in 87% of the cases: 6–9 frames). The uncorrected end-diastolic image, ED_1, reached an average total count of 32,920 (± 170) (in 90% of the cases: 20,000 to 50,000 counts). Within the zone of the left ventricle, the average total count was 9,646 (± 208) on the uncorrected end diastolic image (in 87% of the cases: 3,000 to 13,000 counts). These counts are essential for adequate border definition, as the time resolution of 50 msec is needed to obtain adequate temporal definition and to avoid blurring of the information. A highly sensitive detector system with short deadtimes along with adequate computer assistance are the prerequisites for a dynamic heart study on the wall motion and spatial stroke volume distribution of the left ventricle after myocardial infarction.

FOOTNOTES

* Contrac 3E, Siemens, Erlangen, West Germany.
† System-Seventy, Baird-Atomic, Bedford, Mass.

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