Prompt return to normal of depressed right ventricular ejection fraction in acute inferior infarction¹,²

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A relatively simple, bedside, radionuclide technique has been developed to measure right ventricular ejection fraction. This technique uses a collimated scintillation probe (5 x 5 cm sodium iodide crystal) and ¹¹³mIndium injected into the superior vena cava to record a right ventricular time-activity curve. The radionuclide method was validated in 34 men (14 normals, 20 with coronary artery disease) with biplane right ventriculography (r=0.82). Using this radionuclide method right ventricular ejection fraction was measured in 26 men (average age 51 years) with an acute transmural myocardial infarction. Right ventricular ejection fraction was initially depressed (0.40±0.02; mean±SEM; normal 0.57±0.01) in all of 11 men with an acute inferior infarction but returned to normal by the third day in 10 of them (0.58±0.01; NS). In the 15 men with an acute anterior infarction the average right ventricular ejection fraction was normal initially (0.54±0.01; NS) and individually 10 of 15 had a normal ejection fraction. Left ventricular ejection fraction was initially depressed in all patients and only 4 of 34 (12%) had returned to normal at the third day. These results suggest that right ventricular ejection fraction is regularly depressed in patients with an acute inferior infarction but normal in those with an anterior infarction. Right ventricular performance rapidly improves after inferior infarction; whereas less improvement occurs in left ventricular ejection fraction.

Left ventricular ejection fraction has been found to be regularly depressed in acute myocardial infarction both early (Kostuk et al., 1973) and late after infarction (Rackley et al., 1970; Hamilton et al., 1972; Stewart et al., 1974). Kostuk and associates (1973) have shown that a relation exists between left ventricular ejection fraction and myocardial infarct size, as measured with serial determinations of serum creatine kinase activity. Though a disparity between right and left ventricular filling pressures in acute myocardial infarction has been appreciated (Forrester et al., 1971) and infarction of the right ventricle has been recognised both clinically (Cohn et al., 1974; Rotman et al., 1974) and pathologically (Wartman and Hellerstein, 1948; Wade, 1959; Erhardt, 1974), relatively little attention has been paid to right ventricular performance in either chronic coronary artery disease or acute myocardial infarction.

We had previously developed a radionuclide method for estimating left ventricular ejection fraction which uses a portable scintillation probe (Steele et al., 1974). The purpose of the present study was to develop a radionuclide method for estimating right ventricular ejection fraction and to apply this technique to the serial study of patients with acute myocardial infarction.

Subjects and methods

Right ventricular ejection fraction was measured from radionuclide cardiograms obtained using a collimated scintillation probe by a modification of a radionuclide method for estimating left ventricular ejection fraction (Steele et al., 1974). To measure right ventricular ejection fraction about 1 millicurie of ¹¹³mIndium was injected into a catheter placed in the superior vena cava and flushed with saline. Using a scintillation probe (5 x 5 cm sodium iodide crystal) placed over the midpoint of
the right ventricle in the anteroposterior projection, with the patient supine, a time-activity curve was recorded (Fig.). This curve was obtained with a ring collimator (3.5 cm port) as previously described (Steele et al., 1974). After recording the right ventricular time-activity curve, 1/8 of the initial dose of $^{113m}$Inium was injected into the catheter and flushed with saline. A second curve was obtained and then a third curve was recorded with the shield collimation using half the initial dose of radionuclide. In these studies left ventricular ejection fractions were measured immediately after the right.

Accurate positioning of the probe was necessary to obtain a satisfactory record. The midpoints of the right and left ventricle were estimated from a supine anteroposterior chest film obtained just before the radionuclide study. Confirmation of correct probe position was obtained from the time-activity curve as each measurement of ejection fraction (beat to beat variation) should not differ by more than 10 per cent.

Ejection fraction was calculated as the fractional fall in count-rate from end-diastole to end-systole divided by the end-diastolic count rate. The correct baseline (corrected for scattered radiation) for measuring the end-diastolic count rate was obtained by matching the troughs of the 2 time-activity curves, port and shield, and drawing the shield record on the port recording (Fig.). Ejection fraction was calculated for as many beats as possible and averaged. This always included at least 2 beats for the right ventricle and 3 for the left ventricle.

In 34 men who underwent cardiac catheterisation and coronary arteriography for evaluation of chest pain, right ventricular ejection fraction was measured with the radionuclide method and from biplane (anteroposterior and steep left anterior oblique) contrast cineventriculograms using the area measurement method of Arcilla and associates (1971). Twenty men were found to have coronary artery disease and 14 had normal coronary arteriograms.

To derive the right ventricular ejection fraction from the ventriculograms, the outline of the right ventricle was traced from the projected end-diastolic (largest) and end-systolic (smallest) frames in both the anteroposterior (AP) and left anterior oblique (LAO) projections. The right ventricular volume ($V$) was calculated, using the planimetered areas of the anteroposterior and left anterior oblique projections with the maximum apex base dimension ($L$) in the LAO projection, as:

$$V = \pi LM_{LAO} \cdot M_{AP} / 6$$

(1)

where $M_a$ the minor axis, was calculated from the planimetered areas ($A$) using the ellipse formula as:

$$M_{LAO} = 4 A_{LAO} / \pi L$$

(2)

and

$$M_{AP} = 4 A_{AP} / \pi L$$

(3)

Using this method, Arcilla et al. (1971) noted good
correlation between contrast ventriculography and right ventricular casts.

In another group of 21 men without cardiac disease and with normal coronary arteriograms, left ventricular ejection fraction was measured with the scintillation probe and from single plane (right anterior oblique) contrast left cineventriculograms. The contrast ventriculograms were analysed using area length concepts (Dodge et al, 1960; Kennedy et al, 1970). The left ventricular silhouette was traced at end-diastole and end-systole and ejection fraction was computed as the difference between the end-diastolic and end-systolic volume divided by the end-diastolic volume, with volume (V) calculated as:

$$V = \pi LM^2/6$$

where M was the minor axis in the right anterior oblique projection.

Serial radiograms were obtained from 26 men with an acute transmural infarction. These men ranged in age from 39 to 66 years (average 51 years) and had clinical and electrocardiographic evidence (Q waves in at least 3 leads) of acute anterior or inferior infarction of less than 24 hours duration. No patient had clinical or electrocardiographic evidence of previous myocardial infarction. All patients were studied at their bedside in the cardiac care unit as soon as possible after admission and initial stabilisation.

Radiocardiograms were recorded on the first, second, third, and twelfth day after infarction. All patients were in normal sinus rhythm and none had clinical evidence of mitral regurgitation. All patients were in Killip class I, II, or III (Killip, 1968) and none was in shock. Diuretics, digitalis, nitrates, or catecholamines were not administered at any time during their stay in hospital. All patients gave their informed consent for performance of these studies.

Eleven men had acute inferior infarction and 15 had acute anterior infarction. None had previous infarction and there were no differences between the two groups in age, Killip class, history of chronic obstructive airways disease, or systemic hypertension (Table). All patients survived to the twelfth day, with 2 subsequent deaths in men with anterior infarction.

### Results

In the 14 normal men, right ventricular ejection fraction, estimated with the scintillation probe, averaged 0.57 ± 0.01 (±SEM) with a range of 0.53 to 0.62. Using two standard deviations about the mean, the normal range was computed as 0.52 to 0.62. Right ventricular ejection fractions obtained in these 14 men from their ventriculograms had values with an average of 0.58 ± 0.06. In the 34 men, right ventricular ejection fraction was measured with the scintillation probe correlated (r = 0.82) with the right ventricular ejection fraction measured from contrast cineventriculography.

Left ventricular ejection fraction radiographically measured in 21 normal men averaged 0.66 with a range of 0.57 to 0.77. Using two standard deviations the range of normal left ventricular ejection fraction was 0.56 to 0.76. The cineangiographic left ventricular ejection fraction in these 21 men averaged 0.66 and the range was 0.55 to 0.76. The left ventricular ejection fraction measured with the radionuclide method correlated with the left ventricular ejection fraction obtained from single plane cineangiography (M = 0.91; N = 48).

Serial radiocardiographic determinations of right and left ventricular ejection fraction (1–3 repeat studies in a 1–2 hour period) were undertaken in 12 men. Variation of average was always less than 10 per cent. Thus, the radionuclide method for estimating right and left ventricular ejection fractions seems to be accurate as measured against cineventriculography and to be reproducible.

The right ventricular ejection fraction was depressed (< 0.52) on the first day in all the men with acute inferior infarction and the average value ±SEM was 0.50 ± 0.02; P < 0.001. By the second day the average right ventricular ejection fraction was normal (0.53 ± 0.01) and 10 of the 11 patients had an increase in right ventricular ejection fraction of 0.05 or more. Some further increase was noted on the third day (0.58 ± 0.01) but there was no further change from day 3 to 12 (0.58 ± 0.01). At the third day only 1 patient had an abnormal right ventricular ejection fraction and this was only barely abnormal at 0.51. On the twelfth day all patients had a normal right ventricular ejection fraction.

In the men with acute anterior infarction the average right ventricular ejection fraction on the first day was normal (0.54 ± 0.01) but in 5 of the 15 it was depressed. By the second day it averaged 0.56 ± 0.01 and in 2 of the 5 where it was initially

### Table: Clinical comparison of men with inferior and anterior infarction

<table>
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<tr>
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<th>Inferior infarction (N = 11)</th>
<th>Anterior infarction (N = 15)</th>
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<tbody>
<tr>
<td>Average patient age (y)</td>
<td>49</td>
<td>52</td>
</tr>
<tr>
<td>Killip Class III</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Clinical evidence of chronic obstructive airways disease</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>History or clinical evidence of systemic arterial hypertension</td>
<td>3</td>
<td>2</td>
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depressed it was now normal. On the third day 2 of
the 15 had a depressed right ventricular ejection
fraction and the average was 0.56 ± 0.01 which
persisted to the twelfth day (0.56 ± 0.01). Thus,
right ventricular ejection fraction was regularly
depressed early after acute inferior infarction and
promptly improved, while in acute anterior in-
farction the ejection fraction was less often de-
creased and failed to return to normal in some
patients.

The average left ventricular ejection fraction for
the 11 men with acute inferior infarction was
depressed (0.44 ± 0.02 days; P < 0.001) and in-
dividually all had a decreased left ventricular
ejection fraction. Some improvement was noted
with time, averaging 0.50 ± 0.01 on the second,
0.51 ± 0.01 on the third, and 0.52 ± 0.01 on the
twelfth day after infarction. Only 2 men had a
return to normal of the left ventricular ejection
fraction (>0.55) on the twelfth day and only 1 of
these on the third day.

After acute anterior infarction the left ventricular
ejection fraction was depressed in all patients and
averaged 0.39 ± 0.02 (P < 0.001) on day 1. Some
increase in average ejection fraction was noted on
day 2 (0.43 ± 0.02), day 3 (0.45 ± 0.02), and day 12
(0.46 ± 0.02), but only 2 patients had a normal left
ventricular ejection fraction on day 12 and these 2
were also normal on the third day. Thus, left
ventricular ejection fraction did not appear to have
the same potential for prompt increase as right
ventricular ejection fraction after acute myocardial
infarction. The 2 men who subsequently died had
distinct depression of their left ventricular ejection
fraction (0.28; 0.33) and had no improvement during
the 12 days that they were studied.

Discussion

The radionuclide technique described in this report
seems to measure right ventricular ejection fraction
accurately. Time-activity curves of the right ven-
tricle should accurately reflect changes in volume if
the radionuclide is properly mixed within the
chamber, as the volume estimated from the count
rate does not depend on the geometry of the
chamber. Injection of radionuclide into the superior
vena cava should provide adequate mixing as another
chamber, the right atrium, is traversed before entry
of tracer into right ventricle. A number of contrast
angiographic methods have been developed for
estimating right ventricular volume which use
several geometric models for the right ventricle
(Arcilla et al., 1971; Graham et al., 1973; Ferlinz
et al., 1975). All have been shown to be relatively
accurate when compared with casts of the right
ventricle. The advantages of the radionuclide

Right ventricular time-activity curves must be
(corrected for the effect of scattered radiation if they
are to be used to estimate ejection fraction. Radia-
tion scatter in tissues occurs and consequently the
time-activity curve recorded from the right ven-
tricle contains counts from tissues surrounding this
chamber. Therefore, a second time-activity curve is
recorded with the right ventricle shielded. This
recording reflects counts from tissues which sur-
round the right ventricle.

Using the scintillation probe method for measur-
ing left ventricular ejection fraction, with injection
of radionuclide into the superior vena cava such
that it traverses the pulmonary circulation, one-half
of the initial dose of 113mIndium was used with the
second injection (shield or eclipse tracing) to correct
for scattered radiation. In the case of the right
ventricle, one-eighth of the initial dose was found to
provide the best estimate for calculation of ejection
fraction (Fig.). The apparent lessened contribution
of radiation scatter to the right ventricular time-
activity curve as compared with the left ventricular
curve probably reflects the absence of radionuclide
within the pulmonary vasculature.

In our patients with acute inferior infarction all
had an early decrease in right ventricular ejection
fraction with a prompt improvement by the second
day after infarction in 10 of 11 men. By the third
day after infarction the right ventricular ejection
fraction was normal in all but one patient. In another
group of men with chronic coronary artery disease we
found right ventricular ejection fraction to be
normal in 22 of 29 (76%) patients with a history of
inferior infarction, and 4 of the 7 with depressed
right ventricular ejection fraction had triple vessel
coronary artery disease on coronary arteriography
(Steele et al., 1976). In men with acute anterior
infarction, right ventricular performance was
generally preserved.

The blood supply of the right ventricle is usually
derived from the right coronary artery. It is, there-
fore, not surprising that right ventricular perfor-
mance is compromised in acute inferior infarction.
It is of interest, however, how rapidly right ventri-
cular ejection fraction improves. It is not certain
which of several possible factors are responsible for
this prompt improvement in performance. The
right ventricular myocardium receives blood both
during systole and diastole, whereas the left ventri-
cular myocardium receives flow only during diastole
(Wearn, 1941; Marshall and Shepherd, 1968). In
addition the right ventricle can receive flow directly
through the thebesian vessels and collateral coronary blood flow is more extensively developed for the right than the left ventricle (Zaus and Kearsn, 1952).

Rigo and associates (1975) found increases of right ventricular end-diastolic volume early after infarction in patients with acute inferior infarction and a normal right ventricular end-diastolic volume in patients with an acute anterior infarction. Of 14 patients with inferior infarction, 6 had an increased end-diastolic volume. These investigators used an electrocardiographically gated radionuclide technique.

Of additional interest, though of no particular surprise, is the fact that left ventricular ejection was decreased in all of our patients with acute infarction. Some early improvement in left ventricular ejection fraction was noted, but not nearly as strikingly as for the right ventricle. Kostuk and associates have performed serial measurement of left ventricular ejection fraction in 55 patients with acute infarction and noted improvement in ejection fraction in 30 (Kostuk et al., 1973).

As discussed by Cohn et al. (1974) and Rigo et al. (1975) appreciation of the fact that right ventricular dysfunction might contribute to hypotension and shock after acute infarction is an important consideration in the management of these patients. The opportunity to assess right ventricular function at the bedside of these patients using the relatively simple radionuclide technique of Rigo et al. (1975) or the scintillation probe method discussed in this report should usefully complement their haemodynamic assessment.

References


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