Phase and amplitude analysis of exercise digital left ventriculograms in patients with coronary disease

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SUMMARY Phase and amplitude analysis was applied to intravenous digital left ventriculograms to avoid the artefacts associated with image subtraction. Eight controls and 40 patients with known coronary artery disease underwent digital left ventriculography before and after a symptom limited supine bicycle exercise test. The resultant images were subjected to phase and amplitude analyses. In the control group there was no deterioration in left ventricular wall motion after exercise. In 30 of the 40 patients there was a deterioration in wall motion on exercise. This group contained all eight patients with three vessel disease and 12 of the 17 patients with two vessel disease. Ten patients showed no change in wall motion—five with one vessel disease and five with two vessel disease.

Phase and amplitude analysis of digital left ventriculograms is a method of detecting exercise induced myocardial ischaemia that may help in the assessment of patients with coronary artery disease.

Digital subtraction angiography has recently been applied to left ventricular imaging to detect abnormalities of wall motion.^{1,2} As in nuclear cardiology, exercise has been used to induce such abnormalities in regions rendered ischaemic by coronary stenoses, and results similar to those of nuclear gated blood pool scanning have been achieved.^{3,4}

When digital subtraction angiography is performed after exercise, however, the problems of motion subtraction artefact become more important, and the image quality can be considerably impaired. This has less effect on nuclear ventriculography, where the combination of the low resolution and lack of image degradation by bone and soft tissue structures enables satisfactory imaging despite considerable cardiac motion.

We showed that satisfactory digital images of the left ventricle after exercise can be achieved by the application of an analysis technique that is not dependent on image subtraction, namely phase and amplitude analysis.⁵ This has been widely used in nuclear cardiology^{6,7} and may be applied to any imaging technique that acquires data in a digital format. Therefore, we decided to evaluate this technique

in the assessment of patients with coronary artery disease.

Patients and methods

PATIENTS

We studied 48 patients who were investigated by coronary angiography for chest pain and suspected coronary artery disease. Eight of these patients were found to have normal coronary arteries or minor coronary disease (no coronary stenosis > 70%) and were used as a "control" group. Of these none had had a myocardial infarction and five were taking antianginal medication (β blockers in two).

The remaining 40 patients had angiographically significant coronary artery disease. Eighteen patients had suffered previous myocardial infarction and 36 were taking antianginal drugs (β blockers in 22).

METHODS

All patients remained on their antianginal medication and underwent intravenous digital left ventriculography at rest and after exercise.

The digital system used was the Siemens Digitron II connected on line to a standard Siemens Angioskop angiography suite. All digital data were stored on magnetic tape for subsequent analysis. We developed suitable software in our laboratory using

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the standard Fortran compiler available for this system.

A 5 French pigtail superflow catheter was inserted percutaneously or by cut down into the antecubital vein and advanced to the right atrium. A resting digital left ventriculogram was then performed. Images were obtained in the right anterior oblique projection after injection of 40 ml non-ionic contrast medium at a flow rate of 17–20 ml/s, after a delay of 5 s to allow for pulmonary transit. We used pulsed radiation at a frame rate of 12.5 frames/s and an image matrix size of 256×256 . All digital angiograms were performed in held inspiration to minimise motion artefact.

Immediately after the resting study, patients undertook a supine bicycle exercise test. This was in three stages of two minutes, with 12 lead electrocardiographic monitoring throughout. Exercise was terminated by angina, significant ST segment depression, fatigue, or by the end of the exercise protocol.

A second digital left ventriculogram was performed 10 s after peak exercise, as described above, in the same right anterior oblique projection.

Processing

We used mask-mode image subtraction to determine the sequence of image frames with optimal contrast opacification; from this we selected 3–5 cardiac cycles that started and ended with an end diastolic frame. We corrected the raw images for a changing background, which was principally caused by x ray scatter (see discussion). We used software developed in our laboratory to process the selected digital image sequences to produce phase and amplitude images by a method previously described.⁵

Image assessment

Phase and amplitude images were assessed both subjectively and objectively. Subjective assessment was performed by an independent observer blinded to the coronary anatomy. Phase and amplitude images obtained at rest and exercise were assessed for the presence of abnormalities as determined by reduced amplitude or delayed phase in three wall segments (anterior, apical, and inferior). The two digital studies were then compared for evidence of a deterioration in wall motion after exercise.

For the objective assessment of the phase and amplitude images we used an analysis technique developed in our laboratory (fig 1). We outlined the area representing the left ventricle by hand, starting at the superior border of the aortic valve plane and extending around the apex to the inferior border of the mitral valve plane. We used the phase image to determine the start and finish of the outline because there is an abrupt change in phase at these points, the

atrium and aorta being markedly out of phase with the ventricle. The outline was drawn around the amplitude image of the ventricle to avoid the effect of ventricular muscle movement, which causes the ventricular phase image to extend beyond the cavity.

Once the ventricle had been outlined it was subdivided into 25 sectors of equal angles. The set of segments defined by the outer 25% of these sectors, spanning the region most closely representing the wall motion of the anterior, apical, and inferior wall regions, was then used for automatic analysis. Specific segments were taken to represent anterior (3–10), apical (11–15), and inferior (16–23) wall motion.

Mean phase and amplitude were calculated for each of the 25 segments. These values were then plotted on a graph with the segment location from aortic valve to mitral valve on the x axis and the value of phase and amplitude on the y axis.

Because normal ventricular motion can give rise to systematic regional variations in both phase and amplitude, the data were fitted by regression lines—linear for phase and second order polynomial for amplitude—to highlight areas of abnormal phase and amplitude, and also to allow normalisation of the data so that data at rest and exercise could be compared. Once the data had been normalised in this way, a plot of the difference in phase and amplitude between the rest and exercise studies was produced. We used data for the control group to derive a normal range for change in phase and amplitude. This enabled us to analyse the data from the group with coronary artery disease to detect objectively a deterioration in left ventricular performance after exercise. An abnormality was deemed to be present if the amplitude fell below or the phase rose above the derived normal range in two or more consecutive segments or in two adjacent wall regions.

The results from the objective analysis were compared with those obtained by subjective assessment.

Cineangiograms were assessed subjectively by an independent observer. A coronary stenosis of $>70\%$ was regarded as significant, and assigned to one of the three main coronary arteries: left anterior descending, left circumflex, or right coronary arteries. The wall motion of each of three left ventricular wall segments (anterior, apical, and inferior) was subjectively assessed as normal or abnormal.

Results

CONTROL GROUP (TABLE 1)

The control group consisted of eight patients with normal coronary arteries or minor coronary disease. The left ventricle was reported to be abnormal on cineangiography in three of them.

Subjective reporting of the resting phase and

Fig 1 An example of objective analysis method. The phase image is divided into 25 equal sectors and a plot of phase in the outer 25% produced. After normalising the data, a difference plot is derived. In this case exercise induced a new phase abnormality in the anterior and apical segments.

amplitude images detected left ventricular wall motion abnormalities in the three patients with abnormalities on their cineventriculograms. The remaining five resting studies were reported to be normal.

All but one patient completed three stages of exercise. Two patients, one with normal coronaries, showed significant ST depression on the electrocar-

diogram at the end of the protocol. The mean heart rate achieved was 116 beats per minute.

After exercise in these eight patients the phase and amplitude images showed no deterioration in left ventricular function. In two of the three patients with an abnormal left ventricle at rest there was apparent improvement in left ventricular wall motion in the abnormal segments.

Table 1 Control group

No	LV	CAD	Stage*	HR†	P	E	Digital rest	Study exercise‡	Exercise abnormality§	
									Subjective	Objective
1	An, Ap	0	III	95	-	-	An, Ap	An	-	-
2	N	0	III	125	-	-	N	N	-	-
3	N	0	III	130	-	-	N	N	-	-
4	N	0	III	130	-	+	N	N	-	-
5	Ap	0	III	125	-	-	In	N	-	-
6	An	1	III	110	+	-	Ap	Ap	-	-
7	N	2	III	110	-	+	N	N	-	-
8	N	3	II	105	+	-	N	N	-	-

See footnote to table 2 for abbreviations.

1, 2, 3, minor disease in one, two, or three vessels.

We derived a normal range for the objective analysis from this group.

CORONARY ARTERY DISEASE GROUP (TABLE 2)

Forty patients had significant coronary artery disease, 15 with one vessel disease (cases 1–15), 17 with two vessel disease (cases 16–32), and eight with three vessel disease (cases 33–40). Left ventricular abnormalities were reported on cineventriculography in 30.

The phase and amplitude images of the resting digital left ventriculograms were reported to be abnormal in 37 patients. But phase and amplitude images were normal despite abnormalities on cine-

ventriculography in three cases, and they detected abnormalities in 10 patients with normal cineventriculograms.

Thirty one patients completed three stages of exercise and nine patients stopped at an earlier stage because of angina. The main heart rate achieved was 104 beats/min. Twenty six patients developed angina and 23 developed significant (1 mm) ST depression on the electrocardiogram. There was ST depression in six patients with one vessel disease, 10 with two vessel disease, and seven with three vessel disease.

SUBJECTIVE ANALYSIS

After exercise a deterioration in wall motion was

Table 2 Patients with coronary artery disease (grouped according to coronary anatomy and result of exercise study)

No	LV	CAD	Stage*	HR†	P	E	Digital rest	Study exercise‡	Exercise abnormality§	
									Subjective	Objective
1	An, Ap	Ad	III	100	+	–	An, Ap	An, Ap	–	–
2	In	R	III	95	+	+	In, Ap	In, Ap	–	–
3	In, Ap	R	III	105	–	–	N	N	–	–
4	N	Cx	III	100	+	–	An, Ap	An, Ap	–	–
5	An, Ap	Ad	III	120	–	–	An, Ap	An, Ap	–	In, Ap
6	An, Ap	Ad	III	85	+	–	An	An	An	–
7	An	Ad	II	80	+	–	In	In, Ap	In, Ap	–
8	N	Cx	III	140	–	–	In, Ap	In, Ap	Ap	–
9	In	Cx	III	95	–	–	In, Ap	In, Ap	In, Ap	–
10	N	Ad	III	80	+	+	An, Ap	An, Ap	An, Ap	An, Ap
11	Ap	Ad	III	105	–	–	An, Ap	An, Ap	An, Ap	Ap
12	An, Ap	Ad	III	150	+	+	An, Ap	An, Ap	An, Ap	3
13	An, Ap	Ad	III	100	+	+	Ap	Ap	Ap	An, Ap
14	An	Ad	III	95	+	+	An, Ap	An, Ap	An, Ap	Ap
15	An, Ap	Ad	II	110	+	+	An	An	An	An, Ap
16	N	Ad, Cx	III	85	+	–	In, Ap	In, Ap	–	–
17	N	R, Cx	III	95	+	–	In, Ap	In, Ap	–	–
18	3	Ad, R	III	100	–	–	An	An	–	In
19	Ap	Ad, R	III	150	–	–	N	N	–	An
20	In	R, Cx	III	100	–	–	N	N	–	In
21	N	Ad, Cx	III	90	+	+	Ap	In, Ap	In	–
22	In	Ad, R	II	120	+	+	In, Ap	3	3	In, Ap
23	3	Ad, R	II	110	+	+	An, Ap	An, Ap	Ap	An, Ap
24	In	Ad, R	III	85	+	–	An	An, In	In	An
25	N	Ad, R	III	90	–	+	Ap	Ap	Ap	In, Ap
26	N	Ad, R	II	105	+	+	Ap	An, Ap	An, Ap	An, Ap
27	N	Ad, Cx	III	105	+	+	In, Ap	In, Ap	In, Ap	In, Ap
28	An, Ap	Ad, Cx	III	130	–	–	Ap	Ap	Ap	3
29	An, Ap	Ad, Cx	I	100	+	+	An, Ap	An, Ap	An, Ap	In, Ap
30	In	R, Cx	II	80	+	+	Ap	In, Ap	In	In
31	In	R, Cx	III	115	+	+	In	In, Ap	In, Ap	In
32	3	R, Cx	III	95	–	+	An	An, Ap	Ap	In, Ap
33	N	3	III	80	–	+	3	3	In	–
34	In	3	II	130	+	+	In, Ap	3	3	An, Ap
35	Ap	3	III	90	+	+	Ap	An, Ap	An, Ap	An, Ap
36	In, Ap	3	III	135	–	+	In, Ap	In, Ap	Ap	3
37	In, Ap	3	I	90	+	+	In, Ap	In, Ap	Ap	Ap
38	3	3	III	140	–	–	An, Ap	An, Ap	An, Ap	3
39	3	3	III	110	+	+	In, Ap	In, Ap	Ap	In, Ap
40	Ap	3	III	70	+	+	Ap	In, Ap	In, Ap	In, Ap

LV, Left ventricle from cineventriculogram; CAD, coronary artery disease; P, presence (+) or absence (–) of chest pain during exercise; E, presence (+) or absence (–) of significant ST depression during exercise; N, normal left ventricle; An, abnormality of anterior segment; Ap, abnormality of apical segment; In, abnormality of inferior segment.

3, three abnormal segments or three vessel disease; Ad, stenosis of left anterior descending artery; R, stenosis of right coronary artery; Cx, stenosis of left circumflex artery.

*Stage of exercise reached. †HR, heart rate achieved during exercise. ‡Results of phase and amplitude digital study of the left ventricle at rest and during exercise. §Segment with either deterioration or new abnormality after exercise.

Fig 2 The resting digital study (phase (a) and amplitude (b)) in patient 28 showed a reduction of apical amplitude. After exercise, a new abnormality of apical phase was seen (c) with a further pronounced reduction of apical amplitude (d).

detected by subjective analysis in 30 patients. This group included all eight patients with three vessel disease (cases 33–40), 10 of the 15 patients with one vessel disease (cases 6–15), and 12 of the 17 patients with two vessel disease (cases 21–32). In 10 patients there was no change in wall motion: five patients had one vessel disease (cases 1–5) and five had two vessel disease (cases 16–20).

In all but one of the 10 patients with one vessel disease who showed a deterioration in wall motion, deterioration was confined to a segment that was abnormal at rest. In the remaining patient with an abnormality of the inferior wall at rest, there was deterioration in this segment and a new abnormality in the adjacent apical segment.

In contrast, an abnormality of a previously normal segment was detected in seven of the 12 patients with two vessel disease in whom there was a deterioration in wall motion. In all cases this new abnormality was

compatible with the underlying coronary disease.

Of the eight patients with three vessel disease, six had an abnormality of two or more segments at rest. In three cases an abnormality developed in a previously normal segment and in five, previously abnormal segments deteriorated.

In the 10 patients in whom no deterioration was detected after exercise, abnormalities of wall motion were detected at rest in seven and these persisted after exercise.

OBJECTIVE ANALYSIS

When the rest and exercise studies were compared by objective means, a deterioration in wall motion was detected in seven patients with one vessel disease, 14 with two vessel disease, and seven of the eight patients with three vessel disease. In the group with one vessel disease this method did not detect four patients found to be abnormal by subjective analysis

Fig 3 The resting digital study (phase (a) and amplitude (b)) in patient 35 showed a reduction of apical amplitude. After exercise a pronounced reduction in anterior and apical amplitude was seen (d) associated with a phase abnormality in the same segments (c).

and detected one further patient. In the group with two vessel disease the objective method missed one patient found to be abnormal subjectively, but detected three further patients. This method failed to detect one patient with three vessel disease.

Discussion

There is now evidence from recent trials that coronary surgery can improve the prognosis in certain groups of patients with ischaemic heart disease. Those with most to gain have moderate symptoms and three vessel coronary disease.^{8,9} There is, therefore, much emphasis on non-invasive methods to detect the population that should proceed to the more invasive technique of coronary angiography.

Both exercise testing and nuclear thallium scanning are often used for this purpose. Both are limited, however, by false positive and false negative results.¹⁰

Nuclear, gated blood pool scanning of the left ventricle comes closer to the requirements for the ideal test. It is also limited, however, by low spatial resolution, and the fact that, because it is an equilibrium study, images can only be acquired in the left oblique projection to avoid counts from the overlying right ventricle. First pass techniques with multicrystal cameras and short half life isotopes may overcome this latter problem, but such equipment is not readily available in most centres.

Intravenous digital subtraction left ventriculography has been used to detect exercise induced wall motion abnormalities in the same way as gated blood pool scanning. This is a first pass technique and can therefore be performed in the standard right anterior oblique projection, and also has the advantage of high spatial resolution. The technique compared well with nuclear studies, but was limited considerably by the increased subtraction artefact introduced by

motion. For this reason, images must be acquired after peak exercise, but even then subjective interpretation of the images is impaired by subtraction artefact.¹¹

For this reason we decided to apply the technique of phase and amplitude analysis to intravenous digital left ventriculography. This has been widely used in nuclear cardiology and allows an objective assessment of left ventricular function. Furthermore, because the analysis is based on data derived from several cardiac cycles and is not dependent on image subtraction, the artefact introduced by minor degrees of cardiac motion is minimised.

PHASE AND AMPLITUDE ANALYSIS

Phase and amplitude processing replaces the time-density variation at each image pixel by a sinusoid of best fit (calculated by standard mathematical techniques).¹² The sinusoidal variation is described completely by two parameters: its amplitude or degree of cyclic density change and its phase or relative timing of these changes. Thus the dynamic image sequence is summarised by two parametric images—variations in grey scale intensity represent local changes of the relative timing and relative magnitude of opacified dimension changes and phase changes are described in terms of fractions of a cycle, measured on a scale of 0° to 360°.

A normal ventricular phase image shows the region of the ventricular outline as a relatively constant low phase angle (light), which is sharply distinct from the atria which have a constant but different phase (darker, by about 180°). The corresponding amplitude image shows large values (dark) in regions of maximum motion inside the ventricular boundaries and shows localised variations caused for example by the inclusion of papillary muscle or by regional gradations.

Because there is little change in dimension or density in regions outside the heart they show very low amplitude and random phase. Organs such as the lung and diaphragm, which may overlie the heart and move with respiratory cycle, create severe artefact on normal digital subtraction angiography but have much reduced effects on phase and amplitude images.

The phase image of abnormal ventricles will be affected by any change in the pattern of myocardial contraction. Myocardial ischaemia induces a delay in the timing of contraction of the abnormal wall segment and this therefore produces a shift towards higher values of phase. This appears as a dark region of the ventricle on the phase image. Nuclear studies have shown that the degree of phase delay is related to the severity of the abnormality.¹³

Abnormalities of wall motion can also be detected

by the demonstration of reduced amplitude, because ischaemia affects not only the timing of contraction but also the degree of fractional shortening of the affected segment. This appears as a light region of the ventricle on the amplitude image.

Since this analysis fits only the first harmonic it has enhanced sensitivity to changes occurring at the cardiac frequency but is relatively insensitive to finer details of the motion. The phase angle will be affected by any change in the pattern of myocardial contraction, and changes in timing can be detected that are quite small compared with the actual framing rate. Thus a delay in the timing of the onset of contraction, peak contraction, or relaxation of part of the myocardium will all lead to an increase in phase angle of the affected wall segment. In theory, a delay in contraction associated with early relaxation could, however, lead to no change in phase angle.

In the normal ventricle the timing of contraction of each wall segment occurs within a narrow window, and thus the normal ventricle will show a fairly uniform phase. We found this in our control group. In this group, however, we noticed a tendency for phase either to increase or decrease progressively from aortic valve to apex to mitral valve. We believe this may be related to sideways movement of the ventricle throughout the cardiac cycle, and may possibly explain the wide confidence intervals for phase found in nuclear studies.¹³

CHANGING BACKGROUND

When phase and amplitude analysis are applied to raw digital angiographic data, the resultant images are distorted. Static image regions have a significant amplitude; and a ring of zero amplitude appears inside the left ventricular boundary, which contains signals of generally lower amplitude. This results from a modulated background, which is principally the result of *x* ray scatter.

At the *x* ray energies used during digital angiography *x* ray scatter makes a larger contribution to *x* ray attenuation than *x* ray absorption. This effect can be seen around the edges of the image where the image density outside the coned field appears to be less than expected. Because the degree of scatter is proportional to the density of the structure under investigation, there will be a greater amount of scatter at end diastole when there is more contrast medium in the left ventricle than at end systole. Thus the *x* ray scatter will vary with the cardiac cycle, producing a variation in image density that has its own amplitude and is out of phase with the left ventricle. It is important to correct for this variation in *x* ray scatter when phase and amplitude images of the left ventricle are produced. Because scatter is isotropic, we have assumed this effect to be uniform throughout the

image. An area of the image, distant from the left ventricle, was therefore selected and the phase and amplitude of the x ray scatter was calculated. Each image was then corrected for scatter by subtracting the calculated amplitude of scatter from the raw image data.

ANALYSIS METHODS

We used subjective and objective methods to analyse the phase and amplitude images. The character of the phase and amplitude images is such that there are advantages in both visual and quantitative analysis. On one hand the images can have a complex structure, representing complex details of anatomical motion, best assessed by expert clinical judgement that cannot yet be reproduced by automatic methods. Against this, the eye is not a reliable means of assessing relatively precise numerical information presented visually; and with automatic methods interobserver variations and the need for wide observer experience are removed. Ideally, in practice the two methods would be used to complement each other. We compared the methods because we are still seeking to validate the techniques.

The subjective interpretation was performed by one person with considerable experience in interpretation of nuclear phase and amplitude images. This method enables direct comparison of the rest and exercise images, making no assumption about left ventricular geometry, and taking into account the appearance of the entire left ventricular images. On the other hand, objective methods, although simple to perform, offer greater precision over a very much narrower analytical domain. The present method is a relatively uncomplicated process. It relies on strict geometric definition of the regions analysed, so that anatomical localisation of abnormalities may be in error—and only the outer section of the ventricle, where most significant abnormalities appear, is used. Despite these problems there is a generally good agreement between the two analysis techniques, and we believe that further refinement of the objective method would produce even closer agreement. For these reasons we attached more importance to the subjective reporting.

PATIENT DATA

The reference standard for the normal left ventricle is healthy people shown at angiography to have normal coronary arteries. Because we used invasive techniques we could only study patients with chest pain and suspected coronary artery disease who were shown angiographically to have normal or insignificantly stenosed coronary arteries. Our control group consisted of eight people, five who had normal coronary arteries and three who had only minor

coronary disease. Three of these had abnormalities of the left ventricle at rest. These cannot, therefore, be regarded as healthy controls but it is in just this population that a reliable non-invasive test would avoid the need for coronary angiography. We therefore believe this to be a valid control group for this study.

The mean heart rate achieved during the supine bicycle exercise was rather low in comparison with conventional exercise testing. Supine exercise, however, is known to be associated with a lower heart rate than upright exercise.¹⁴ Furthermore, 22 of the patients with coronary artery disease were receiving β blocking drugs. The fact that 26 of the 40 patients with coronary disease developed angina, 23 showed significant ST depression, and 30 developed new wall motion abnormalities despite relatively low heart rates suggests that the exercise was sufficiently severe.

We found phase and amplitude analysis of digital left ventriculograms to be a very satisfactory method of assessing left ventricular function both at rest and immediately after exercise. The images produced were simple to interpret subjectively, with a little experience, and this assessment of phase and amplitude agreed quite well with that obtained by a simple method of objective analysis.

This technique detected some wall motion abnormality at rest in three of the controls and 37 of the group with coronary artery disease. In 10 cases the resting digital study detected abnormalities not reported on the cineangiograms, whereas it failed to detect abnormalities seen on the cineventriculogram in three. Apical abnormalities were detected far more frequently than anterior or inferior ones, and the apex was the abnormal segment in nine of the 17 people (controls and patients) with only one abnormal segment. It is possible, therefore, that the technique of phase and amplitude analysis is overdiagnosing abnormality of the apical segment. After exercise, however, apical abnormalities were detected in 34 people, of whom 24 had shown a deterioration compared with resting function. If this analysis is overdiagnosing apical abnormalities, it seems unlikely that this effect would be emphasised in the exercise studies. It therefore seems more likely that the rest images were detecting minor apical abnormalities that became more apparent during ischaemia. It also seems from our data that the ventricular apex is the wall segment most vulnerable to myocardial ischaemia, regardless of the distribution of coronary stenoses.

The next question we must answer is whether this technique can detect the patients with multivessel disease who need further investigation, and whether it is possible to select out the patients with minor and

single vessel disease who need only relief of symptoms. The standard technique at present for studying exercise induced myocardial ischaemia is exercise stress testing, and in particular the development of exercise induced ST segment depression on the electrocardiogram. In this study 25 people (controls and patients) developed significant ST depression and this group included 17 of the 25 patients with multivessel disease. One patient with three vessel disease was missed, however, and two patients with either normal coronary arteries or minor disease were falsely identified. We therefore looked to see whether our digital technique was more selective.

Any technique that relies on the localisation of myocardial ischaemia to predict the underlying coronary anatomy is limited. In the first place it is necessary for ischaemia to occur. A patient can have severe coronary artery disease and not show myocardial ischaemia on exercise, and such patients cannot be detected. Secondly, when ischaemia does occur, even in the presence of multivessel disease, exercise will be limited by symptoms resulting from the area of myocardium that becomes ischaemic first. Therefore the wall motion abnormality that is shown may be confined to one wall segment. Furthermore, a stenosis of a single coronary artery may produce abnormalities in two adjacent segments (anterior and apical) or (inferior and apical) if the artery is large enough. Thus the number of abnormal segments alone cannot determine the coronary anatomy.

In our control group, no patient developed a deterioration in left ventricular function on exercise. Two of the three patients with an abnormal ventricle at rest showed an improvement with exercise.

Subjective analysis showed no deterioration in wall motion on exercise in five of the patients with one vessel disease. Of the remaining 10 patients, exercise induced deterioration in wall motion was confined to previously abnormal segments in nine, and in the last case an inferior abnormality extended to the apex. Thus in this group, exercise was rarely associated with extension of the ischaemic area, and in all cases the abnormality was consistent with a single coronary lesion.

In the group with two vessel disease, however, a previously normal segment was more often affected (seven cases). In five cases our technique failed to detect exercise induced abnormalities, and these patients could not therefore be distinguished from the group with one vessel disease. In the remaining five patients a deterioration was detected that affected previously abnormal segments, including the apical segment in all five.

A deterioration in wall motion after exercise was detected in all eight of the patients with three vessel disease. New abnormalities were detected in three. This group contained only two patients with a single

abnormal wall segment at rest, and after exercise all patients had two or three abnormal segments.

Thus this method detected 20 of the 25 patients with multivessel disease, and none with normal coronary arteries or minor disease. It did, however, identify more patients with significant one vessel disease than the electrocardiographic changes alone.

Neither digital left ventriculography nor ST segment analysis was totally specific for multivessel coronary disease. We believe that our technique may help when the results of exercise testing are misleading and in particular in the management of patients with a false positive exercise test. Furthermore, the combination of ST depression and an exercise induced abnormality in wall motion is more specific for multivessel disease than either marker alone, although some sensitivity is lost.

We have developed a new method of detection of exercise induced myocardial ischaemia, namely phase and amplitude analysis of exercise intravenous digital left ventriculograms. This technique detected left ventricular wall motion abnormalities both at rest and after exercise and provided some insight into the underlying coronary artery disease. In our series we subjectively identified all the patients with three vessel disease, and in general the extent of the induced ischaemia was dependent on the number of coronary stenoses. Phase and amplitude analysis of exercise digital left ventriculograms may therefore help in the selection of those patients for whom further investigation by coronary angiography is indicated.

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