Projection of electrocardiographic signs in praecordial maps after exercise in patients with ischaemic heart disease¹

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SUMMARY Praecordial surface maps of the electrocardiogram were recorded before and after exercise using 16 electrodes covering the left hemithorax. The ST segment and R and S wave changes were measured in the praecordial maps from 20 individuals with no detectable cardiovascular disease. These showed no significant alteration in ST segments or R/S. In contrast in 40 patients with angiographically proven coronary artery disease it was possible clearly to outline the distribution, severity, and time course of praecordial areas of ST segment depression (36 patients) and ST segment elevation (10 patients). In addition these praecordial areas of ST segment changes were accompanied by a regional and significant fall in the R/S.

The praecordial electrocardiogram with exercise complements the anatomical information obtained from the coronary arteriogram by clearly outlining electrocardiographic projections of regional myocardial ischaemia or cell death.

The exercise electrocardiogram has long been used in the diagnosis and evaluation of patients with coronary artery disease (Bruce and McDonough, 1969; Sheffield and Roitman, 1975; Blackburn, 1977). Interest has centred on ST segment changes on exercise (Roitman *et al.*, 1970; Redwood *et al.*, 1977) though in recent years changes in the QRS vector have been described (Kilpatrick, 1976). Though the praecordial electrocardiogram using 36 to 72 leads is being used to study the natural history of acute myocardial infarction and the effect of interventions (Maroko *et al.*, 1972; Selwyn *et al.*, 1977a; Selwyn and Shillingford, 1977), this technique has not been widely applied to the exercise electrocardiogram. This is the result of difficulties in recording the surface electrocardiogram before transient changes have regressed and in designing an adequate electrode system which is stable during severe exertion. In a recent study, a technique for recording the praecordial electrocardiogram, using conventional equipment available in most hospitals, has been described (Fox *et al.*, 1978). The purpose of the present study is to describe the variety of praecordial electrocardiographic abnormalities that occur on exercise.

Methods

Twenty normal male volunteers (aged 28 to 74, mean 52 years) and 40 consecutive male patients (aged 32 to 72, mean 54 years) with a typical history of angina pectoris, ST segment changes after exercise, and anatomical evidence of coronary artery disease shown by coronary arteriography were studied. Routine 12 lead electrocardiograms were recorded in all patients. All patients were in sinus rhythm at rest. Patients whose electrocardiograms showed abnormalities in the mean frontal axis (that is beyond -30° and $+120^\circ$) or QRS duration more than 110 ms before exercise were excluded from the study.

Praecordial exercise electrocardiograms were recorded using a direct writing ink jet Mingograf (Elema-Schonander) recording on four channels simultaneously. The gain employed was 10 mm for 1 mV and the paper speed was 25 mm per second. Sixteen points were marked on the chest using an indelible pen, distributed evenly over the left hemithorax as previously described (Fox *et al.*, 1978). Disposable press-stud electrodes were used and secured in position using adhesive tape applied sufficiently tightly to keep the electrodes stable in position but not so as to interfere with respiration. A specially designed switching box (four channels switching four ways) was used in which 16 com-

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mercially available screened electrode cables with press-stud ends were connected to the electrodes. The four unipolar leads of the Mingograf were then plugged into the switching box so that it was possible to record the 16 leads by recording four leads simultaneously and switching through four positions.

Exercise was performed on a bicycle ergometer (Lanooy and Bonjer, 1956). All recordings were made with the patient sitting comfortably, with the arms extended resting on the handlebars. A recording of the 16 leads was made before exercise, immediately after the end of exercise, and then at one minute, three minutes, five minutes, eight minutes, and 10 minutes after exercise. Recordings were then continued at three-minute intervals, as necessary, until the electrocardiogram had returned to normal. While respiratory swing was present in 12 of the 60 patients immediately after exercise, it was possible to overcome this by recording at least three beats during held end-tidal expiration. The electrodes remained in position in spite of maximal exercise. Patients were asked to exercise at a constant rate (50 rpm) and the work load was increased by 25 watts each minute and was continued until the patient was unable to continue because of fatigue, chest pain, or dyspnoea. The presence of multiple ventricular ectopic beats was also an indication to discontinue the test. Exercise was stopped in all the normal controls because of fatigue; the limiting factor in the patients with coronary artery disease was fatigue in six, chest

pain in 28, dyspnoea in five, and multiple ventricular ectopic beats in one. The electrocardiogram was monitored throughout the test and resuscitation equipment was available. Connecting up the patient took 10 to 15 minutes and the exercise test and post-exercise recordings 15 to 20 minutes. Each 16-lead praecordial map took about 10 seconds to record.

INTERPRETATION OF RECORDS

The usual criteria for the interpretation of exercise electrocardiograms were applied (Scandinavian Committee on Electrocardiographic Classification 1967). At each of the 16 electrode positions ST segment depression, ST segment elevation, the height of the R wave, and the depth of the S wave were measured in mm. ST segment depression and elevation were measured to the nearest 0.5 mm, using the TP segment as the isoelectric level or the PQ segment when there was no isoelectric TP segment because of tachycardia (Fox *et al.*, 1978). The ST segment was considered to be depressed if there was 1.00 mm or more of shift for at least 0.08 s (Mason and Likar, 1966). ST segment elevation was measured 0.06 s from the end of the QRS complex (Selwyn *et al.*, 1977b), and was ignored if less than 1 mm. None of the patients had ST segments which at rest lay above or below the isoelectric line. The area of ST segment change was defined as the number of praecordial positions that showed ST depression or elevation, and Σ ST the sum of the total ST segment depression in

Fig. 1 A typical example of the sequence of contour maps showing areas of ST segment depression after exercise. A fall in R/S was seen to involve the same areas of the praecordium.

Fig. 2 (a) The changes seen after exercise in the precordial area (mean $\pm$ standard error mean (SEM)) of ST segment depression (36 patients) and ST segment elevation (10 patients). All patients had angiographically proven coronary artery disease. (b) Mean Σ ST segment elevation and mean Σ ST segment depression in mm $\pm$ standard error of mean (SEM) after exercise in the same group of patients.

mm for all the 16 leads. The arithmetical sum of the height of the R wave and the depth of the S wave (R+S) in mm and the ratio of R to S (R/S) were calculated for each of the 16 positions. The 16 values of R+S and of R/S were added together to produce single values of each variable for each precordial map ((Σ R+S and Σ R/S). Contour maps of the area of ST segment depression and elevation were drawn for each recording. Q waves were identified using the criteria of the Minnesota Code.

Statistical analysis of the data was performed using Students t test for independent samples.

Results

Of the 40 patients with coronary artery disease, 36 developed ST segment depression alone after exercise. Four patients showed ST segment elevation only, but in six others there was both ST segment depression and ST segment elevation.

Fig. 1 shows the development of precordial ST segment depression in a patient with critical narrowing of the left anterior descending coronary artery. A fall in Σ R/S occurred immediately after exercise in this patient. The ST segment depression and fall in R/S both involved the same area of the precordium. Fig. 2a and 2b show the development of precordial ST segment elevation and ST segment depression that occurred after exercise in the 40 patients with coronary artery disease. These ST segment changes were not seen in the normal controls. Both ST segment elevation and depression followed a similar pattern, being present immediately after exercise and reaching a maximum in terms of both Σ ST and area of ST segment change within one minute of the end of exercise. ST segment elevation when present after exercise was found to be confined to the precordial electrode positions that showed either diminutive R waves (R/S < 1) or Q waves.

Fig. 3 shows the R/S ratio changes occurring after exercise in the two groups of patients. A fall in Σ R/S occurred immediately after exercise in the patients with coronary artery disease ($P < 0.01$) and

Fig. 3 The changes seen in the mean $\pm$ standard error of mean (SEM) of R/S after exercise in 40 patients with coronary artery disease (CAD) and 20 controls. A significant fall occurred immediately after exercise in the patients with CAD but not the controls.

Fig. 4 Further analysis of the changes in R/S after exercise in 40 patients with coronary artery disease showed that the fall was confined to the praecordial areas that also develop ST segment changes (ischaemic area). A mean R/S $\pm$ standard error of mean (SEM) is given for each area.

in the normal controls, though this fall was statistically significant only in the first group. The mean (± 1 standard deviation) $\Sigma R/S$ for the 20 normal controls was 65.75 ± 36.86 before and 55.70 ± 34.01 immediately after exercise and for the 40 patients with coronary artery disease was 50.35 ± 23.76 before and 37.03 ± 16.98 immediately after exercise. Further analysis of the changes in R/S ratio in the patients with coronary artery disease showed that the fall in R/S was greatest in the areas that showed ST segment changes ($P < 0.01$) (Fig. 4). The mean (± 1 standard deviation) R/S for the areas that showed ST segment change was 36.83 ± 22.32 before and 20.86 ± 12.62 immediately after exercise and for the remainder of the praecordial map was 19.50 ± 15.02 before and 17.78 ± 14.90 immediately after exercise. On the other hand there was no

Fig. 5 The changes seen in the mean $\pm$ standard error of mean (SEM) of the praecordial $\Sigma R+S$ in mm after exercise in 40 patients with coronary artery disease (CAD) and 20 controls. There were no significant alterations in either group.

significant alteration in either group in the height of the R wave plus the depth of the S wave ($\Sigma R+S$) on exercise (Fig. 5). The mean (± 1 standard deviation) $\Sigma R+S$ for the 20 normal controls was 204 ± 43.58 mm before and 210 ± 38.31 mm immediately after exercise and for the 40 patients with coronary artery disease was 177.2 ± 53.20 mm before and 171.9 ± 48.76 mm immediately after exercise. One patient with coronary artery disease developed an interventricular conduction defect which was present on all the praecordial leads for one minute after exercise.

Discussion

A non-invasive and physiological means for detecting and assessing myocardial ischaemia may help to rationalise diagnosis and treatment in patients with coronary artery disease. Ideally the investigation should be simple, easily reproducible, and have both a low morbidity and mortality. The exercise electrocardiogram is now an important non-invasive investigation and is used routinely in the diagnosis of chest pain. Initially only single electrocardiographic leads were monitored but in recent years most centres have favoured three leads or even a full 12-lead electrocardiogram (Frank, 1956; Mason *et al.*, 1967).

The praecordial exercise electrocardiogram adds an extra dimension to these tests since it allows a measurement of the area, severity, and time course of electrocardiographic abnormalities that occur after exercise. The semiquantitative assessment which can be made in each patient may help in evaluating medical and surgical treatments. Before praecordial mapping of the exercise electrocardiogram can be used in the diagnosis of chest pain and in the evaluation of medical and surgical treatment, it is necessary to investigate the praecordial electrocardiographic abnormalities that occur in normal subjects and in patients with coronary artery disease. In this study we have investigated the changes that occur on exercise in the ST segment, R/S ratio, and the pattern of the QRS complex ($R+S$) in the two groups of patients.

Both ST elevation and ST depression occurred after exercise in patients with coronary artery disease. In six patients both were present in the same study. The time course of these electrocardiographic signs after exercise was similar. These changes were not seen in the normal controls.

A small (though not statistically significant) decrease in $\Sigma R/S$ occurred immediately after exercise in the normal controls. This has been noted previously (Simoons and Hugenoltz, 1975) and, though the cause is not known, it has been

suggested that alterations in the haematocrit, changes in the intracardiac blood volume, or temporary increases in the serum potassium level may be responsible. The wide standard deviation found in both groups makes it difficult to apply the information obtained from $\Sigma R/S$ changes after exercise to individual patients in the diagnosis of coronary artery disease. However, in patients with coronary artery disease a regional decrease in R/S ratio occurred in the areas affected by ST segment change. Since the height of the R wave plus the depth of the S wave did not alter in either group, these alterations in R/S ratio must be the result of a decrease in R wave amplitude plus an equal increase in S wave amplitude rather than a fall in R wave voltage alone. The cause of the regional decrease in R/S ratio is unclear though a localised fall in the isoelectric line, regional dyskinesia, electrolyte or metabolic disturbances may be responsible (Simoons *et al.*, 1977). However, this regional alteration in R/S may provide information complementary to that obtained from alterations in the ST segment in identifying patients with coronary artery disease. The $\Sigma R/S$ at rest was lower in the patients with coronary artery disease than in the normal controls; this is to be expected since eight patients with coronary artery disease had had a previous myocardial infarction.

ST segment elevation was seen after exercise in 25 per cent of the patients with coronary artery disease. This figure is higher than that previously reported using standard exercise tests (Fortuin and Freisinger, 1970), but it is likely that by recording from over the whole of the left hemithorax ST segment elevation on exercise will be seen more often. The underlying pathophysiology responsible for ST segment elevation is unclear. In those patients with both ST segment elevation and depression it is uncertain if these electrocardiographic manifestations are the consequence of reciprocal changes or represent two separate areas of ischaemia (Kleber *et al.*, 1978). Prinzmetal and his colleagues have suggested that ST segment elevation represents localised transmural ischaemia, while ST segment depression occurs as a consequence of patchy ischaemia (Prinzmetal *et al.*, 1961). The conclusions were, however, based on animal experiments and the interpretation of the meaning of these electrocardiographic signs in patients is still controversial.

In conclusion, this study has defined the praecordial changes that occur in the ST segment, and in the R and S waves on exercise. Praecordial surface mapping of the exercise electrocardiogram should, by measuring the changes in the area, time course, and severity of these electrocardiographic

signs, help in the diagnosis of chest pain and in the non-invasive assessment of medical and surgical treatment for ischaemic heart disease.

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