Maximal exercise testing in patients with spontaneous angina pectoris associated with transient ST segment elevation

Risks and electrocardiographic findings

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Six patients with spontaneous angina associated with transient ST segment elevation had a multistage maximal exercise (bicycle) test. In 5 patients, typical electrocardiographic changes were recorded during exercise, namely ST segment elevation often accompanied by an increase in the voltage of the R wave and a widening of the QRS complex. Four of these patients developed severe rhythm disturbances: ventricular tachycardia (2 cases) and ventricular flutter (1 case) were the reason for early interruption of the test in 3 patients, while 1 patient had a short run of ventricular tachycardia after exercise. These rhythm disturbances which spontaneously regressed in all cases were consistently preceded by obvious ST elevation and in 2 patients were attended by slight chest discomfort.

Maximal exercise testing of patients suspected of variant angina provides important diagnostic information in many patients, but the risks of potentially lethal arrhythmias should be considered and resuscitation facilities should always be immediately available.

Transient ST segment elevation associated with spontaneous anginal pain is practically the only finding common to all cases reported as variant angina pectoris (Scherf and Cohen, 1974); simultaneously there is often an increase in the voltage of the R waves, the QRS tends to become wider, and ventricular arrhythmias or atrioventricular conduction abnormalities are frequent (Prinzmetal *et al.*, 1959, 1960; Lesbre *et al.*, 1968; Jouve *et al.*, 1969; Poggi *et al.*, 1971; MacAlpin, Kattus, and Alvaro, 1973). Apart from these well-established electrocardiographic changes occurring with spontaneous angina pectoris, it is almost impossible to separate clearly the variant form of angina pectoris from the classical one (Scherf and Cohen, 1974).

In the variant form of angina pectoris, exercise is classically well tolerated though many patients have occasional episodes of pain while exercising (Robinson, 1965; Amichot and Jouve, 1970; Cosby *et al.*, 1972; Cheng *et al.*, 1973; Cherrier *et al.*, 1973; Courtadon *et al.*, 1973; MacAlpin *et al.*, 1973).

It has also been reported that in the variant form of angina, the exercise electrocardiogram was either normal or exhibited depression of the ST segment (Prinzmetal *et al.*, 1959, 1960; Lesbre *et al.*, 1968; Amichot and Jouve, 1970); more recently, however, several authors have reported that the ST segment elevation commonly observed during the spontaneous episodes of anginal pain could also be precipitated by exercise testing, and at least 20 such cases have been published (Robinson, 1965; Raynaud *et al.*, 1969; Fortuin and Friesinger, 1970; Silverman and Flamm, 1971; Bobba *et al.*, 1972; Cosby *et al.*, 1972; Cheng *et al.*, 1973; Cherrier *et al.*, 1973; Courtadon *et al.*, 1973; MacAlpin *et al.*, 1973; Betriu, Solignac, and Bourassa, 1974; Bodenheimer *et al.*, 1974).

We have observed 6 patients with spontaneous angina pectoris as a major complaint. When the electrocardiogram was recorded during spontaneous anginal pain, it displayed ST segment elevation accompanied by typical QRS changes; 4 of these patients also had occasional episodes of exertional angina pectoris. Maximal exercise testing

TABLE Clinical data

Case No.	Age (yr)	Sex	Resting electrocardiogram	Electrocardiogram during spontaneous angina		Exercise test data				
				ST segment changes	QRS changes	Pain	Work-load (watts)	Maximum HR	ST segment changes	
1	58	M	Normal	3 mm rise II-III	Yes	No	160	130	2.5 mm depression V ₂ -V ₅	
2	47	M	Normal	4-10 mm rise V ₁ -V ₅ 3 mm depression aVF-III	Yes	Yes	120	124	3 mm rise V ₁ -V ₃ 2 mm depression II-III	
3	51	F	Normal	2 mm rise V ₁ -V ₃ 2 mm depression II-III	No	Slight	80	165	5 mm rise V ₁ -V ₃ 1 mm depression II-III	
4	34	M	Flat T waves II-III, V ₅ -V ₆	5 mm rise V ₁ -V ₄	Yes	Slight	160	150	4-6 mm rise V ₁ -V ₄	
5	45	M	Normal	3.5 mm rise V ₁ -V ₃	No	No	110	155	4-6 mm rise V ₄ 2 mm depression II-III	
6	34	M	Biphasic T waves V ₂ -V ₃	Not recorded		No	120	165	6 mm rise V ₁ -V ₃ 4 mm depression II-III	

HR, heart rate; PVC, premature ventricular contraction; VT, ventricular tachycardia; VF, ventricular flutter; LAD, left anterior descending; RCA, right coronary artery.

induced ST segment elevation in 5 patients, while 4 patients also developed severe ventricular rhythm disturbances.

Subjects and methods

Six patients with spontaneous angina pectoris and no previous myocardial infarction have been studied. The clinical history of these patients is briefly described (see case reports) and the most relevant data are summarized in the Table.

The methods for exercise testing were as previously described (Detry *et al.*, 1971; Rousseau, Brasseur, and Detry, 1973). Briefly, the subjects were asked to perform a multistage bicycle exercise test of maximally tolerated exercise with an initial workload of 50 watts and subsequent increments of 25 and 10 watts. Leads V₄, V₅, and V₆ were monitored throughout and recorded on paper every minute; a 12-lead electrocardiogram was recorded at rest, after every 5 minutes of exercise, or more frequently if needed, at maximal exercise, and during recovery. Besides the appearance of typical anginal pain, the only criterion for interrupting the exercise test was the occurrence of 3 or more consecutive premature ventricular beats. Coronary angiography was performed using the method of Bourassa in 5 of the 6 patients (Lespérance, Bourassa, and Saltiel, 1970). Venous bypass grafting was proposed to 4 patients and performed in 3.

Case reports

Case 1

This 58-year-old man had frequent spontaneous anginal

episodes during the day; his exercise tolerance was excellent and he played tennis regularly without any symptoms. During the spontaneous episodes, the electrocardiogram showed 3 mm ST elevation in II-III and a slight increase in the amplitude of the R wave in III (from 9 to 14 mm). The maximal exercise test was interrupted because of exhaustion and a 2.5 mm horizontal depression of the ST segment was recorded in V₂ to V₅ after exercise. Two months later the patient had an acute inferior myocardial infarction and he subsequently lost his angina.

Case 2

This 47-year-old man had spontaneous angina pectoris during the night for two months; each anginal episode was short but up to 10 attacks would follow one after another. Exercise, even heavy, was usually well tolerated though the patient mentioned some episodes of exertional symptoms. During spontaneous pain, the electrocardiogram showed a 4 to 10 mm ST segment elevation from V₁ to V₅; in the same leads, the QRS was slightly prolonged (0.10 s), and a moderate increase of the amplitude of the R wave was noted (from 5 to 10 mm in V₂). ST segment depression was present in the inferior leads.

The exercise test was interrupted for anginal pain and 3 mm ST segment elevation was observed in V₁ to V₃ where the voltage of the T waves was increased (20 mm); reciprocal ST segment depression was noted in the leads II-III. The mean pulmonary arterial pressure rose from 16 mmHg (2.1 kPa) at rest to 46 mmHg (6.1 kPa) during pain. Occasional

Angiographic data		
QRS changes	Rhythm disturbances	
No	No	Not performed
No	Occasional PVCs	LAD: 80 per cent proximal stenosis; RCA: $\leq$ 50 per cent stenosis; graft to LAD patent
Yes	VT	Normal arteries; apical dyskinesia
Yes	VT	LAD: 70 per cent proximal stenosis
Yes	Frequent PVCs and VF	LAD: 90 per cent proximal stenosis; anterior wall akinesia; graft to LAD patent
Yes	VT	LAD: 80 per cent proximal stenosis

ventricular premature beats were observed during and after exercise.

The coronary angiograms revealed an 80 per cent proximal stenosis of the left anterior descending artery and minimal lesions on the right coronary artery. This patient had a venous bypass graft and all symptoms disappeared. Three months after operation, the graft was patent; a maximal exercise test was interrupted because of exhaustion at 195 watts (HR: 165/min) and the exertional electrocardiogram was normal.

Case 3

This 51-year-old woman had exertional and mostly spontaneous angina pectoris for one month. Several episodes of spontaneous pain were observed in the coronary care unit: a 2 mm ST segment rise was consistently noted in V₁ to V₃ while there was 2 mm ST segment depression in the inferior leads.

During exertion, the QRS complex became wider (from 0.07 to 0.10 s in V₅) and a 5 mm elevation of the ST segment was noted in V₁ to V₃; 30 seconds later, the test was interrupted at 80 watts because of exhaustion (HR: 165/min) attended by slight chest discomfort. A few seconds later, a short episode of ventricular tachycardia unsuspected by the patient was recorded (Fig. 1); a 1 mm depression of the ST segment was observed at the third minute of the recovery in II, III, and V₄ to V₆. A second exercise test performed one month later caused the same electrocardiographic changes, including the ventricular tachycardia.

The coronary arteries were normal, while ventriculography demonstrated a small dyskinetic zone at the apex. During the 15-month follow-up, the anginal syndrome remained unchanged but the patient reported two syncopal episodes not preceded by chest pain; neurological examination was normal and we suspect that these episodes might be caused by transient severe ventricular arrhythmias.

Case 4

During the previous 6 months this 34-year-old man had frequent and disabling episodes of spontaneous nocturnal angina; his exercise tolerance was usually normal but he also reported a few episodes of exertional chest pain. During spontaneous angina pectoris the electrocardiogram showed a 5 mm ST segment rise from V₁ to V₄, with a widened QRS complex (from 0.07 to 0.10 s) and an increased R wave (from 10 to 21 mm); at that

FIG. 1 Case 3. Electrocardiogram recorded at rest, at maximal exercise, and during the first seconds of recovery.

FIG. 2 Case 5. Electrocardiogram recorded at rest, during submaximal exercise, and at the end of the test which was interrupted because of the onset of ventricular flutter; this rhythm disturbance with alternating ventricular complexes may be better described as 'torsade de pointes' (Desertenne, 1966).

time the mean pulmonary arterial pressure rose from 17 to 32 mmHg (2.3 to 4.3 kPa).

At the 5th minute of the exercise test an increase in R wave voltage (from 16 to 30 mm) and a slight widening of the QRS complex (from 0.08 to 0.10 s) were noted; they were followed by the appearance of a 4 to 6 mm rise in the ST segment from V1 to V4. The exercise test was interrupted one minute later at 160 watts because of the occurrence of two consecutive short runs of ventricular tachycardia; at that time the patient reported minor chest discomfort but no dizziness.

Coronary angiography revealed an isolated proximal 70 per cent stenosis of the left anterior descending artery and a venous bypass operation was performed; all spontaneous anginal crises disappeared, but 10 days after operation the patient died from acute and massive pericardial bleeding.

Case 5

This 45-year-old man reported frequent nocturnal spontaneous anginal episodes for one month; he also mentioned a few episodes of exertional angina pectoris. During spontaneous angina, a 3.5 mm rise in the ST segment was observed from V1 to V3.

At submaximal exercise level (75 watts; HR: 110/min) the electrocardiogram showed a 2 mm depression of the ST segment in V3 to V5. Leads V4 to V6 which were monitored during the last minute of the test first showed the appearance of frequent ventricular premature beats while the QRS complex became wider (from 0.06 to 0.11 s) and the R wave amplitude slightly increased (from 25 to 35 mm in V4). A 4 to 6 mm rise in the ST segment was then observed in V4, and 30 seconds later the test was interrupted at the onset of ventricular flutter which disappeared spontaneously after 5 seconds (Fig. 2); a second short episode of ventricular flutter occurred a few seconds later. The patient had no anginal pain and he remained unaware of his rhythm distur-

ance. At the fifth minute of recovery a 2 mm ST segment depression was noted in leads II, III, V5, and V6.

The coronary angiograms revealed a localized 90 per cent stenosis of the left anterior descending artery, and

FIG. 3 Case 6. Electrocardiogram: 1) at rest; 2) during submaximal exercise; 3) at maximal exercise; 4) at the 5th minute of recovery.

this lesion was bypassed with a saphenous vein graft. All the symptoms have disappeared and a 3-month post-operative angiographic study demonstrated patency of the graft. A maximal exercise test caused a 1 mm horizontal depression of the ST segment in lead V₅.

Case 6

This 34-year-old man had frequent nocturnal spontaneous angina over a period of 8 months; he regularly undertook heavy exercise without symptoms. We never had a chance to record an electrocardiogram during a spontaneous episode of chest pain.

During the exercise test ST segment elevation was observed in leads V₁ to V₃, while the ST segment became depressed in the inferior leads (Fig. 3); the duration of the QRS complex increased from 0.06 to 0.10 s, and the amplitude of the R wave increased from 19 to 38 mm in V₄. The exercise test was interrupted 2 minutes later because of exhaustion at a workload of 120 watts; the QRS and the ST segment were then merged into a single giant wave. During early recovery, the patient had a short run of ventricular tachycardia (5 consecutive premature ventricular beats). These abnormalities slowly regressed and ST segment depression was still noted in V₅, II, and III five minutes after the end of the exercise test.

The coronary angiograms demonstrated an isolated proximal 80 per cent stenosis of the left anterior descending artery; the projected bypass procedure was refused by the patient. Two months later he had an anteroapical acute myocardial infarction and lost his anginal syndrome; he has now been symptomless for 22 months.

Discussion

All the patients described in the present paper were referred to the hospital for spontaneous angina pectoris and the electrocardiogram recorded during the pain showed QRST changes similar to those initially described by Prinzmetal *et al.* (1959, 1960). Since 4 patients also had occasional episodes of anginal pain with exertion, our group may not really be comparable to the cases previously reported as having the variant form of angina pectoris though the variability of this syndrome is recognized (Scherf and Cohen, 1974).

In 5 of the 6 patients, exercise testing caused QRST changes similar to those usually observed during spontaneous episodes of angina: in 4 of these patients (Cases 2, 3, 4, and 5), the exertional ST changes were observed in the same leads as during spontaneous pain and the reciprocal ST segment depression was also similar in 3 patients. Changes in the QRS complex were common during exercise (Cases 3, 4, 5, and 6) even when they had not been observed during spontaneous anginal episodes. The high incidence (5/6) of these typical electrocardiographic abnormalities with exercise contrasts

with previous reports claiming that in the variant form of angina pectoris, exercise tests were of little value, the exercise electrocardiogram being either normal or showing only ST segment depression; unfortunately the number of patients tested, the methods of testing, and the results of all exercise tests are not mentioned in these reports (Prinzmetal *et al.*, 1959, 1960; Lesbre *et al.*, 1968; Amichot and Jouve, 1970). Our data together with those previously reported indicate that ST segment elevation with exertion is a common finding in the variant form of angina pectoris (Robinson, 1965; Raynaud *et al.*, 1969; Fortuin and Friesinger, 1970; Silverman and Flamm, 1971; Bobba *et al.*, 1972; Cosby *et al.*, 1972; Cheng *et al.*, 1973; Cherrier *et al.*, 1973; Courtadon *et al.*, 1973; MacAlpin *et al.*, 1973; Betriu *et al.*, 1974; Bodenheimer *et al.*, 1974); the true incidence of these electrocardiographic abnormalities with exercise is, however, difficult to evaluate from previous reports, since many patients were observed only in coronary care units and had no exercise tests.

A second reason for the high incidence of ST segment elevation with exercise in our patients might be the recording of a 12-lead electrocardiogram during exercise. The ST segment abnormalities were always recorded in leads V₁ to V₄; these leads are sometimes neglected and ST segment elevation would have been overlooked if V₅ only had been used for monitoring the test. This methodological aspect could account for the lower incidence of ST changes recently reported by MacAlpin *et al.* (1973). A third likely explanation for our data is the use of a maximal exercise testing procedure which in the classical form of angina pectoris greatly enhances the diagnostic sensitivity of the test (Sheffield, Holt, and Reeves, 1965; Mason *et al.*, 1967; Detry, 1973).

The exercise test induced severe ventricular rhythm disturbances in 4 of the 6 patients. These ventricular arrhythmias were always preceded by obvious ST elevation for 30 seconds to 2 minutes and were accompanied by a slight chest discomfort in 2 patients; the ventricular arrhythmias did not cause dizziness or syncope and they were all spontaneously reversible. It is noticeable that the ventricular arrhythmias occurred only in those patients who reached a heart rate of 150/min or more. Four similar cases have been previously reported: in 3, the ventricular tachycardia was preceded or followed by exertional ST segment rises (Raynaud *et al.*, 1969; Cherrier *et al.*, 1973) while in 1 case, the ventricular tachycardia was preceded by ST segment depression (Bodenheimer *et al.*, 1974).

This incidence of severe ventricular rhythm

disturbances precipitated by exercise is in striking contrast to their rarity in the presence of classical coronary heart disease (Ellestad *et al.*, 1969; Rochmis and Blackburn, 1971) even when exertion caused ST segment rises (Fortuin and Friesinger, 1970). Such ventricular arrhythmias are, however, not uncommon in variant angina pectoris since during the episodes of spontaneous angina, 40 per cent of the patients develop atrioventricular conduction abnormalities or arrhythmias (Lesbre *et al.*, 1968; Poggi *et al.*, 1971; MacAlpin *et al.*, 1973); these are most often noted when the pain is intense and/or the ST segment rise is striking. Our results are possibly related to the fact that the exercise tests were continued until exhaustion, angina pectoris, or onset of a ventricular arrhythmia, despite the appearance of ST segment rises which gradually increased; such ST segment elevation reflects a severe degree of myocardial ischaemia (Ekmekci *et al.*, 1961) and is often considered as a criterion for early interruption of a diagnostic exercise test (Rochmis and Blackburn, 1971).

The varying QRS axes during the ventricular rhythm disturbance (Fig. 2) strongly suggest that it is a 'torsade de pointes'¹ rather than a ventricular flutter (Desertenne, 1966; Motte *et al.*, 1970; Krikler, 1974); this observation suggests a new cause for 'torsade de pointes', namely an exercise-induced subepicardial injury current.

The mechanism responsible for the electrocardiographic changes precipitated by exercise in our patients with the variant form of angina are not known, but their similarity to the electrocardiographic abnormalities observed during spontaneous attacks implies a similar mechanism; Prinzmetal *et al.* (1959, 1960) suggested that transient coronary arterial spasm could account for most characteristics of the syndrome, and such spasm has now been demonstrated angiographically both in patients with coronary lesions (Dhurandhar *et al.*, 1972; Gonin *et al.*, 1973; MacAlpin *et al.*, 1973) and in patients with normal coronary arteries (Cheng *et al.*, 1973; Froment, Normand, and Amiel, 1973; Oliva, Potts, and Pluss, 1973; Betriu *et al.*, 1974; Kerin and Macleod, 1974). The true role of spasm in the pathophysiology of variant angina pectoris remains to be firmly established and it is not known whether or not it can account for the electrocardiographic abnormality during exercise. If one postulates that spasm is involved in the response to exercise, then myocardial hypoxia could itself

be a triggering factor, since ST segment rises similar to those reported here with exercise have also been precipitated by atrial pacing (Cheng *et al.*, 1973; Gonin *et al.*, 1973; Betriu *et al.*, 1974) and by arterial hypoxia (Bekaert, Afschrift, and De Tollenaere, 1971; Cherrier *et al.*, 1973).

We conclude that maximal exercise testing of patients suspected of variant angina pectoris often provides important diagnostic information. The risks of testing these patients are much greater than usually believed since severe ventricular arrhythmias frequently occur.

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¹A form of ventricular tachycardia characterized by twisting alteration in the QRS morphology; it is usually induced by a ventricular extrasystole occurring late in the cycle, in patients who have underlying bradycardia or QT prolongation; it may cause syncope and lead to ventricular fibrillation.

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