

Exercise-induced ST-segment elevation possibly caused by coronary artery spasm

A case presentation and review

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Summary

A 36-year-old man with classic angina pectoris had marked ST-segment elevation (STE) in the inferior leads on stress-testing in the absence of chest pain. There was no evidence of previous myocardial infarction (MI). Selective coronary arteriography delineated severe obstructions in the right coronary artery (RCA) with additional left circumflex coronary artery (LCx) obstruction. Left ventricular cine-angiography established that there was normal contractility and confirmed the absence of past MI. Coronary artery bypass graft surgery to the RCA and LCx was unfortunately complicated by an acute transmural inferoposterolateral MI. Treadmill stress testing 6 weeks after surgery failed to demonstrate the pre-operative ST-segment change. The patient may have developed exercise-induced coronary artery spasm superimposed on the severe proximal RCA stenosis; this in turn may have caused the inferior STE. Exercise-induced STE is reviewed.

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Case presentation

A 36-year-old white man was initially admitted to the Cardiac Unit at Tygerberg Hospital on 25 January 1984 with a 1-week history of typical angina pectoris associated with nausea. In addition to these episodes he also complained of burning retrosternal discomfort at rest, which was relieved by sublingual nitrates. Apart from a long history of psoriasis and possible dyspepsia, he was healthy. Positive risk factors for ischaemic heart disease (IHD) were the fact that he smoked a pipe and had a brother who had died from an acute myocardial infarction (AMI) at 45 years of age. Clinical examination revealed a fit young man with cutaneous features of psoriasis. He was normotensive with no other abnormal signs. A 12-lead resting ECG demonstrated sinus rhythm at 68 beats/min, a P-R interval of 0,16 second, mean QRS axis of $+50^\circ$, incomplete right bundle-branch block and isolated asymmetrical T-wave inversion in standard lead III (Fig. 1). There were no signs of ischaemia or myocardial infarction (MI) (previous or

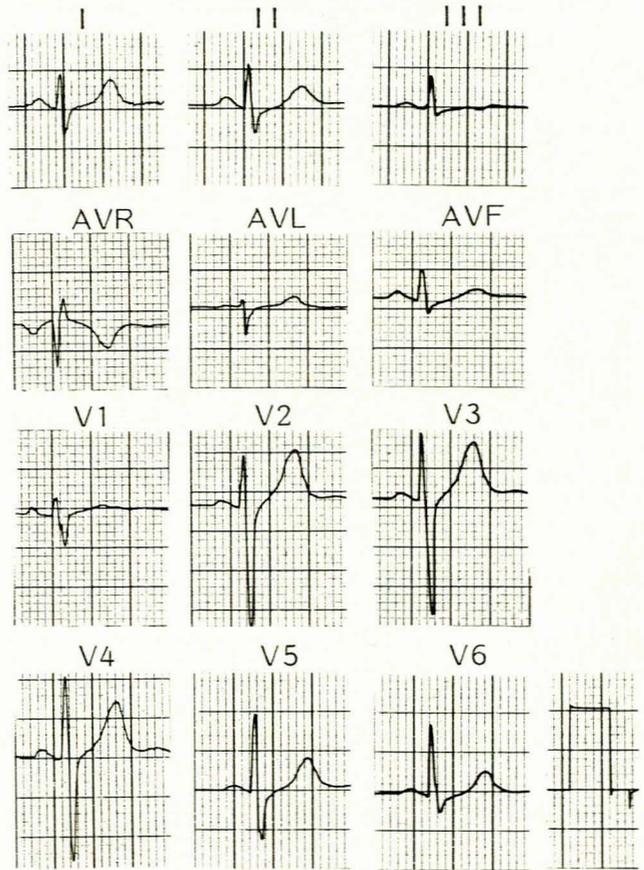


Fig. 1. Resting 12-lead ECG (full standardization) showing a P-R interval of 0,16 second, mean QRS axis of $+50^\circ$, sinus rhythm of 68 beats/min, incomplete right bundle-branch block and isolated asymmetrical T-wave inversion in standard lead III.

acute). A chest radiograph confirmed a normal cardiac silhouette and clear lung fields. Results of urinalysis and side-room haematological tests were within normal limits. Biochemical parameters, including serial tests for serum cardiac enzymes, were all unremarkable. The patient was diagnosed as having unstable angina pectoris and started on oral nitrate therapy. However, despite this therapy he complained of frequent attacks of chest pain at rest unassociated with any ECG evidence of myocardial ischaemia. Oral nifedipine was therefore added to the nitrates with quite dramatic relief of his chest pain. The patient was then mobilized and subjected to a treadmill exercise test (Fig. 2). No abnormality could be seen on the resting trace, which was unchanged from that taken on admission. After completion of stage 4 of the Bruce protocol 2 mm ST-segment depression (STD) was visible in lead V5 but

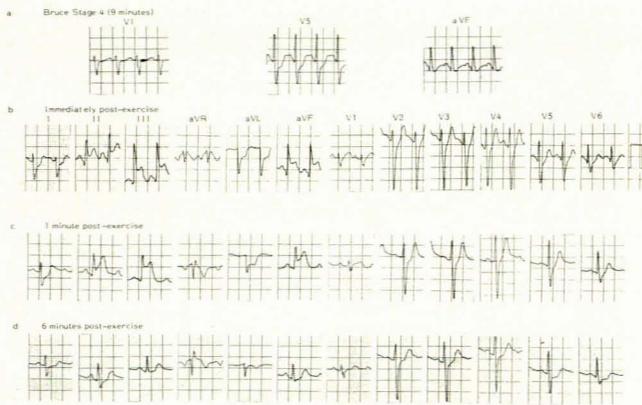


Fig. 2. Submaximal treadmill exercise test: (a) leads V1, V5 and aVF at completion of Bruce stage 4 (9 minutes) demonstrating 2 mm STD in lead V5 but no changes in the other two leads; (b) 12-lead ECG 40 seconds after Bruce stage 4 and immediately after stopping exercise — marked STE inferiorly (maximum of 6 mm in lead III) with reciprocal STD in leads I and aVL; (c) 12-lead ECG 1 minute after completing exercise showing less ST-segment deviation, but still abnormal; (d) 12-lead ECG 6 minutes after cessation of effort — the inferior and high lateral leads (I and aVL) are now normal but STD is seen in the lateral leads (maximum of 2 mm in lead V5).

not leads V1 and aVF (Fig. 2a). The patient continued exercising for a further 40 seconds, but then admitted to being too exhausted to continue. At this point his pulse rate was 161/min (predicted target heart rate 170/min) and systolic blood pressure 155 mmHg. The 12-lead ECG demonstrated ST-segment elevation (STE) in the inferior leads (maximum elevation of 6 mm in standard lead III) and reciprocal horizontal STD in the high lateral leads (maximum depression of 4 mm in standard lead aVL) without any chest pain (Fig. 2b). At 1 minute after cessation of exercise (Fig. 2c) the ST-segment changes were less marked than immediately afterwards. The ECG then gradually returned to normal; at 6 minutes the inferior and high lateral leads appeared normal but there was now horizontal STD in the lateral leads (maximum depression of 2 mm in lead V5) (Fig. 2d). Interpretation of the treadmill effort test proved somewhat difficult in view of the unusual response of significant STE. The two possibilities considered at this stage were severe inferior transmural myocardial ischaemia, either secondary to effort-induced coronary artery spasm or due to

ischaemia in the absence of such a pathophysiological mechanism. Lack of chest pain in this ECG setting of severe myocardial ischaemia was most surprising. After the test 12-lead ECGs and serial serum enzyme estimations were performed to exclude a possible AMI precipitated by exercise, but failed to demonstrate any AMI. The patient had no further episodes of chest pain. Since there was a real likelihood of significant IHD in this young man, cardiac catheterization was undertaken.

Cardiac catheterization

This procedure was carried out using the Seldinger technique from the right groin. The left-sided cardiac pressures were normal apart from a moderately raised left ventricular (LV) end-diastolic pressure of 18 mmHg (upper limit of normal 12–14 mmHg). LV cine angiography in the right anterior oblique projection demonstrated normal contractility of this chamber. Selective coronary angiography in multiple views was then performed using Judkins catheters. This confirmed the presence of significant double-vessel coronary artery disease, with 95% luminal obstruction of the proximal right coronary artery (RCA), a 70% lesion proximal to its graftable posterior descending branch (Fig. 3), and a 75% obstruction of the left circumflex coronary artery (LCx) just proximal to the origin of a graftable mid-lateral branch (Fig. 4).

As a result of the stress ECG and coronary angiographic findings it was agreed that the patient would benefit from coronary artery bypass graft (CABG) surgery. On 5 March 1984 aortocoronary saphenous vein bypass grafts were inserted into the RCA and mid-lateral branch of the LCx. Within a few hours of the completion of the operation a resting ECG demonstrated features of a hyperacute transmural inferolateral MI with true posterior extension. This was confirmed by serum enzyme estimations (including CK-MB iso-enzymes), but the patient never had any chest pain. Six weeks later the patient underwent submaximal treadmill stress testing. The resting ECG demonstrated Q waves inferoposterolaterally, confirming the peri-operative MI (Fig. 5). He again attained a maximum heart rate of 161/min but this occurred earlier, at the end of stage 2 of the Bruce protocol, and without any chest pain. The pre-operative inferior STE was no longer in evidence. The patient continues to be free of angina on nitrates and nifedipine medication and is being followed up at the Cardiac Clinic.

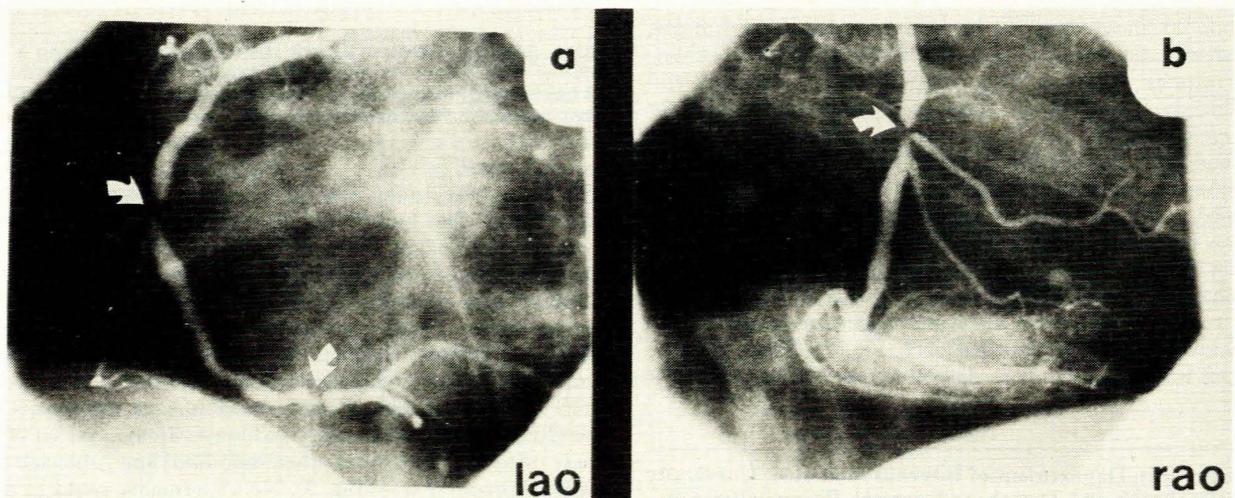


Fig. 3. Right coronary cine angiograms in (a) left anterior oblique (lao) and (b) right anterior oblique (rao) views. The vessel is dominant and 95% and 70% atherosclerotic lesions are visible (arrowed).

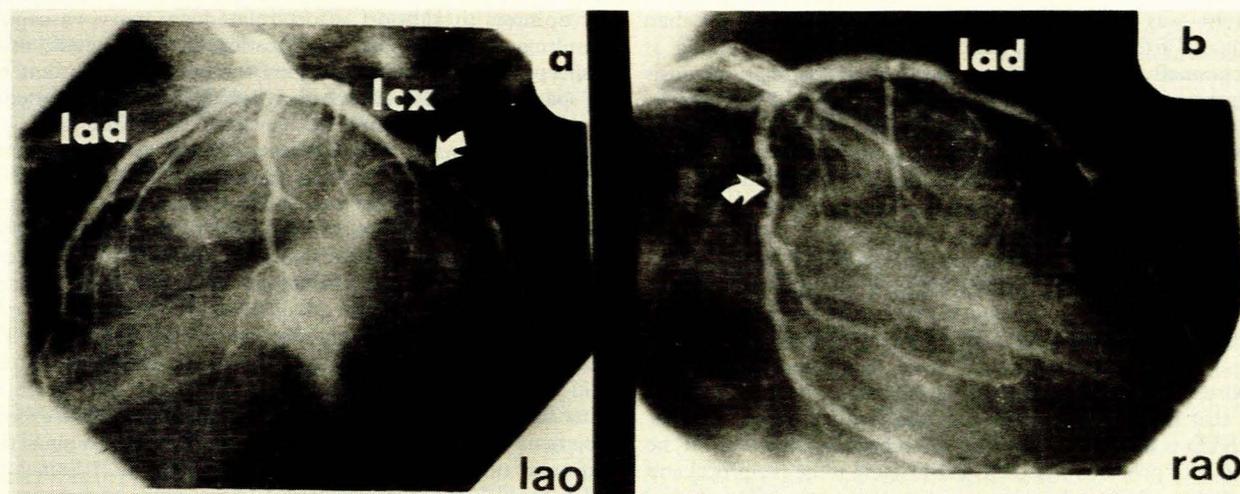


Fig. 4. Left coronary cine angiograms in (a) left anterior oblique (lao) and (b) right anterior oblique (rao) views, demonstrating a 75% obstructive lesion (arrowed) in the lcx just proximal to the origin of a graftable mid-lateral branch. The left anterior descending (lad) coronary branch has minor internal luminal irregularities.

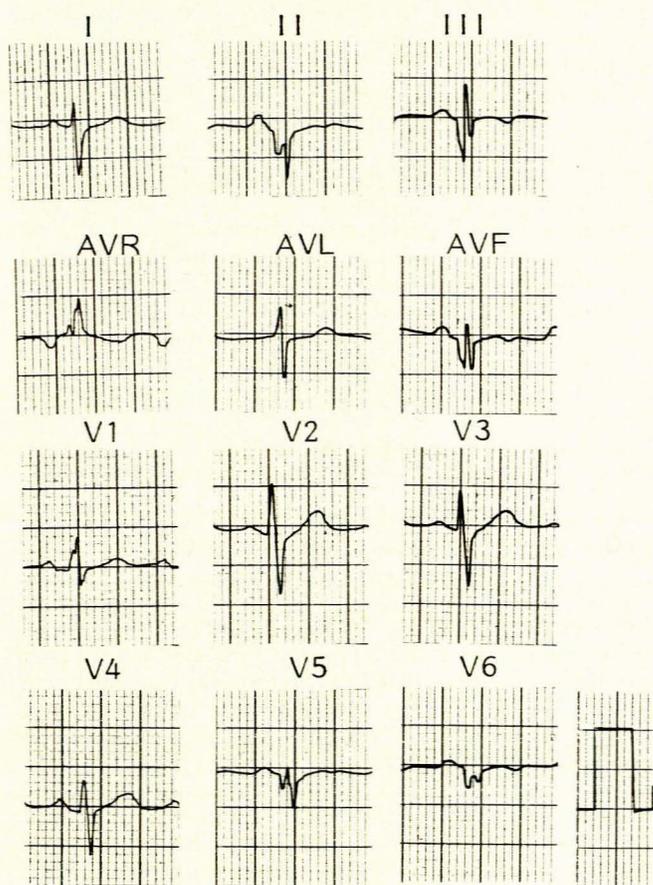


Fig. 5. Resting 12-lead ECG (full standardization) demonstrating characteristics of a previous transmural inferoposterolateral MI not present before CABG surgery.

Discussion

The submaximal effort test has been used routinely in clinical practice in order to demonstrate underlying IHD,¹ particularly in patients with angina pectoris, since its usefulness in asymptomatic subjects is somewhat controversial. A 'positive' test is usually interpreted as a 1 mm horizontal or downward-sloping

STD, as recommended by Sheffield and Reeves.² The most unusual finding of transient STE during exercise testing was first described in 1949 by Kroop *et al.*³ and was accepted as a sign of myocardial ischaemia. The purpose of this report is to re-emphasize the occurrence of effort-induced STE and to attempt to place this interesting feature in its true perspective. Broadly speaking, this ECG finding occurs in the following categories of patients: (i) asymptomatic individuals; (ii) patients after an acute transmural MI; (iii) patients with an LV aneurysm; (iv) patients with Prinzmetal's variant angina; and (v) symptomatic patients with severe underlying obstructive coronary artery disease. The frequency of effort-induced STE will be determined by such important factors as the incidence of IHD in the population being evaluated, the stress-test protocol, and the number of ECG leads employed.

STE on exercise in asymptomatic subjects

The only study in which this was documented was in the Seattle Heart Watch, by Bruce *et al.*⁴ This study involved 1 275 healthy men who underwent a maximal stress test with CB5 and XYZ lead systems. STE was detected in 6 of these men (0.5%). This incidence is the lowest documented in any of the five categories of patients.

Exercise-induced STE after acute transmural MI

This clinical setting is the most common cause of STE (0.1 mV or greater) on stress testing, the incidence of this finding in several studies⁵⁻¹² varying between 18% and 41%. Some 16% of inferior and 30% of anterior MIs in these studies were followed by STE, often present at rest but increasing in magnitude with exercise. The ECG lead distribution of the ST-segment change was the same as that of Q waves. Atterhög *et al.*¹³ showed that stress testing 3 weeks after infarction is likely to show a higher frequency of STE than if done at 6-12 months thereafter. Several authors have correlated coronary arteriographic findings with STE on exercise in these patients. Chahine *et al.*⁹ in a study of 21 patients with this stress ECG feature after post-transmural anterior MI, found 'critical' stenosis of the proximal part of the left anterior descending coronary artery (LAD) in 19, of whom 18 had an apical LV aneurysm. Waters *et al.*¹⁴ reported on a similar experience and agreed with Chahine *et al.*⁶ that the ST-segment shift on

exercise was due to segmental LV dysfunction rather than coronary artery stenosis, since this stress ECG finding is exceptionally rare in patients with severe coronary stenosis with normal resting ventricular contractility. Furthermore, only 20% of these patients complain of effort-related angina pectoris, thus making it less likely that myocardial ischaemia is the cause of the STE. Nevertheless, our patient had a normal LV cine-angiogram, no previous transmural MI and no angina when he experienced STE on exercise.

The simultaneous occurrence of STE and STD in 'reciprocal' leads during exercise testing utilizing multiple ECG lead systems often poses a problem. Our patient had marked STE in the inferior leads with concomitant STD in the anterolateral leads. Some authors^{6,12} have demonstrated that this coexistence signifies multiple-vessel coronary artery disease and that the STE occurs in the leads with Q waves whereas the STD is due to new myocardial ischaemia. However, in the absence of previous MI the STD is likely to be reciprocal and not due to additional coronary artery obstruction. The studies of Waters *et al.*¹⁴ and Dunn *et al.*¹⁵ have shown that it is highly unlikely that the STE is reciprocal to the STD. Of great clinical importance is the fact that the occurrence of exercise-induced STE after a transmural MI signifies a less favourable prognosis.^{9,10,16} Thus, this finding might justify coronary angiography with a view to possible percutaneous transluminal coronary angioplasty or CABG surgery.

Exercise-induced STE in LV aneurysm

Gorlin *et al.*¹⁷ in 1967 were the first to document this finding in 16 of 24 patients with a proven LV aneurysm. Four of the 16 had no STE on their resting ECG, and this was only brought out by exercise testing. Rubenstein *et al.*¹⁸ noted that 8 of their 11 patients with exercise-induced STE had abnormal LV cine-angiograms, compatible with an LV aneurysm. Similar data were published soon afterwards by Manvi and Ellestad.¹⁹ Chahine *et al.*,⁶ in their analysis of 840 treadmill exercise tests, found 29 patients (3.5%) with effort-induced STE; 21 underwent angiography and 18 (86%) were shown to have an LV aneurysm, mostly with an associated severe lesion of the LAD. Chahine *et al.*⁶ concluded that 'exercise-induced ST elevation most commonly reflects severe coronary artery disease with associated left ventricular aneurysm', and that it appears to be a 'relatively sensitive indicator of left ventricular aneurysm'. They also made the important observation that 'ST elevation may relate more to the presence of abnormal wall motion than to myocardial ischaemia per se'. Weiner *et al.*¹² and Waters *et al.*¹⁴ echoed similar sentiments. Sriwatanakomen *et al.*,²⁰ in a series of 38 patients with exercise-provoked STE, noted that 25 had significant coronary atherosclerosis in addition to an LV aneurysm, and that the sites of STE correctly predicted the site of aneurysm formation in 91% of cases. These researchers²⁰ differed slightly from their predecessors in that they believed that 'ischemia and abnormal wall motion may independently or additively underlie the mechanism for S-T elevation during exercise'.

STE with exercise in Prinzmetal's variant angina

It is now generally accepted that coronary artery spasm is the pathogenetic mechanism in Prinzmetal's variant angina and has in fact become virtually synonymous with this condition, but it is important to appreciate that Prinzmetal *et al.*²¹ emphatically stated in their original classic article that variant angina (and thus coronary artery spasm) could not be induced by exercise. This concept was then further supported by McAlpin *et al.*²² Since then there has been a substantial body

of opinion to support effort-related coronary vasospasm resulting in STE. This modification of Prinzmetal's initial concept has therefore underlined the possibly important role of spasm in classic angina pectoris, which is usually represented by STD on the stress ECG. Nevertheless, Yasue *et al.*,²³ as well as Maseri *et al.*²⁴ have demonstrated that coronary artery spasm can give rise to STD on exercise testing in patients both with Prinzmetal's variant angina and Heberden's classic angina pectoris, supporting Maseri's concept of 'mixed forms'. These ECG findings make it difficult to assess the incidence of coronary artery spasm in the spectrum of IHD. More recently, Shimokawa *et al.*,²⁵ utilizing thallium-201 exercise scintigraphy, demonstrated most elegantly that transient effort-induced STE gave rise to a more extensive reversible perfusion defect than did effort-induced STD in the same patient; they were the first to show these features in a patient with variable exercise tolerance and a history of Prinzmetal's variant angina, thus substantiating the previous work of Maseri *et al.*²⁴ Ekmekci *et al.*²⁶ demonstrated that 'primary' STD (as opposed to reciprocal STD) was a manifestation of a lesser degree of myocardial ischaemia when compared with 'primary' STE. This differentiation is made more difficult by the fact that ST-segment changes detected by unipolar epicardial electrodes (as carried out by the standard ECG) are a less reliable indicator of myocardial ischaemia than recordings with unipolar intramyocardial electrodes.^{27,28}

The exact prevalence of exercise-provoked STE in variant angina is not known. Results in three large series of patients have been published.^{22,24,29} MacAlpin *et al.*²² studied 20 patients, 3 of whom developed exercise-related STE. Maseri *et al.*²⁴ noted this ECG change in 17 of their 96 patients who were stressed, and Waters *et al.*²⁹ documented STE in 25 of 82 patients. Results in smaller series were published by Detry *et al.*,³⁰ Higgins *et al.*,³¹ Gottlieb *et al.*,³² and Specchia *et al.*,³³ and most of these studies showed that the ECG lead distribution of STE induced by stress testing was the same as that occurring during spontaneous rest attacks of chest pain, highlighting the almost certain role played by coronary artery spasm. The prevalence of exercise-induced STE in patients with variant angina is thus in the range of 10-30%, in sharp contrast to the 3.0-6.5% in patients with classic angina pectoris.³⁴

A variation of the response of STE to exercise with the change during the recovery phase rather than during the exercise period was documented by Weiner *et al.*³⁵ These workers studied 4 patients with Prinzmetal's variant angina with STE during recovery; 3 of them had STD during exercise and of these 2 developed STE only 2 minutes or later after completion of effort. This ECG feature was associated with hypotension in 3 patients and ventricular arrhythmias in 1 patient. Weiner *et al.*³⁵ suggested that this response was associated with a poor prognosis, and isolated cases of a similar ST-segment response were reported by Kemp,³⁶ Sweet and Sheffield,³⁷ and McLaughlin *et al.*³⁸ Lahiri *et al.*,³⁹ in their description of 5 such cases, also noted that STD during exercise preceded STE during the recovery phase. Their patients also had a worse prognosis when compared with Prinzmetal's angina patients with STE during exercise. Nevertheless, Chaitman *et al.*³⁴ could not substantiate a worse prognosis in this subgroup and attributed this difference to the wider use of calcium antagonist drugs in their patients, as well as a possibly less severe degree of underlying coronary atherosclerosis. In our patient, STE in the inferior leads only occurred immediately after peak exercise and was not preceded by STD in those leads, although STD was noted in the lateral chest leads. STD was documented in the high lateral leads at the time of the inferior STE, and this change was considered to be reciprocal. It is of interest that our patient still had STD in the lateral chest leads as late as 9 minutes into the recovery

period, and that none of these ST-segment changes were accompanied by any angina.

The fact that coronary artery spasm is the cause of exercise-induced STE was elegantly demonstrated by Specchia *et al.*,^{33,40} as well as by Yasue *et al.*,²³ who performed coronary arteriography during stress testing. The above workers, as well as Waters *et al.*,⁴¹ also showed that exercise-induced STE was reproducible, particularly during the morning when parasympathetic activity was high. Several workers also noted STE with stress testing in patients with Prinzmetal's variant angina.⁴²⁻⁵⁰ Therapy with calcium antagonists negated this ST-segment response to exercise further substantiating the role of coronary vasospasm.^{23,41,51}

Exercise-induced STE in symptomatic patients with severe underlying obstructive coronary atherosclerosis

It is widely acknowledged that 'ST-segment elevation during or after exercise is rare in the absence of active variant angina or previous myocardial infarction'.³⁴ Chaitman *et al.*⁵² found 2 such cases in a series of 200 male patients, and commented that both these men had typical angina pectoris and multivessel coronary artery disease. Others^{6,16,53} have reported incidences of exercise-induced STE in 0.2%, 1% and 1.7% of cases respectively. The great majority of these had haemodynamically significant coronary lesions in the distribution relating to the exercise ECG. Most workers would agree that effort-induced myocardial ischaemia gives rise to STD in patients with severe fixed coronary lesions in the absence of superimposed coronary vasospasm.^{54,55} This appears to be the category in which our patient belongs. Nevertheless, the possibility of additional coronary artery spasm aggravating the severe RCA obstruction cannot be excluded entirely. If the myocardial ischaemia was entirely due to a fixed coronary artery obstruction, worsened by increased myocardial oxygen consumption necessitated by the exercise test, then STD should have preceded STE in the same ECG leads as non-transmural ischaemia deteriorated to transmural ischaemia. The absence of such a sequence of events in our patient makes the likelihood of superimposed coronary vasospasm, precipitated by exercise, more likely. Moreover, he did not experience angina pectoris during the phase of STE possibly caused by superadded coronary vasospasm. Przybojewski and Ellis⁵⁶ recently demonstrated ergometrine maleate-provoked angiographic coronary vasospasm without angina or ECG ischaemia in a young man with a previous transmural MI and near-normal coronary arteries on angiography. Furthermore, other workers have demonstrated the occurrence of angina pectoris on ergometrine maleate provocation in the absence of ECG changes and vice versa.^{57,58} Thus, there exist several permutations and combinations in the profile of coronary artery spasm, highlighting the need for further clinical research in this fascinating field.

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Ependymal cyst of the ovary

A case report

A. J. TILTMAN

Summary

An unusual morphological variant of a mature cystic teratoma was found at caesarean section in a 23-year-old patient; it appeared as a thin-walled cyst containing clear fluid. Microscopy showed the cyst to be lined by ependyma and the wall to be composed of glial tissue with a small protuberance containing squamous epithelium, adipose tissue and bone.

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Mature cystic teratomas of the ovary are frequently referred to as dermoid cysts because they are lined by squamous epithelium with adnexal glands and hair follicles and are filled with sebum and hair recognizable even at body temperature. Thin-walled translucent cysts are at the time of surgery usually thought to be of follicular origin or cystic 'epithelial' neoplasms. On rare occasions the dominant cyst of a mature cystic teratoma is lined by epithelium other than epidermoid and can present in this manner. This report concerns such a case, in which the cyst was lined by ependyma.

Case report

A 23-year-old woman, para 5, gravida 2, was admitted to hospital near term for an elective caesarean section. The two previous deliveries had also been by caesarean section for cephalopelvic disproportion 2 and 4 years previously. The two other pregnancies had ended in abortion at 12 weeks. At operation a thin-walled cyst was found in the left ovary. The right ovary was normal. A caesarean section, left ovarian cystectomy and bilateral tubal ligation were performed. The patient was discharged on the 7th postoperative day.

Laboratory investigation

The specimen was a thin-walled cyst measuring 6 cm in its greatest diameter and containing clear straw-coloured, watery fluid. The inner lining was smooth and glistening. In one area there was a thickening of the wall by lobulated solid yellow tissue resembling a corpus luteum. In another focus there was a cluster of three separate plaques, together measuring approximately 1 cm in the greatest diameter, lying within the wall of the cyst and protruding into its lumen. When these were incised a yellow pultaceous material containing loose hairs was found.

Microscopically, the three small cystic structures were lined by squamous epithelium with underlying sebaceous glands and hair follicles extending down into a thin rim of adipose tissue. A microscopic focus of woven bone was present. The dominant cyst was lined in all sections examined by a single or in areas pseudostratified layer of columnar, cuboidal or flattened cells. In some areas distinct cilia could be seen. These cells

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