

'Pseudonormalisation' of the 'normal variant pattern' on the ECG of black subjects after intermittent acute myocardial ischaemia

A case report

J. Z. PRZYBOJEWSKI, P. H. BECKER

Summary

A middle-aged, urbanised black man with unstable angina pectoris showed unusual findings on serial resting ECGs recorded during episodes of chest pain, as well as during symptom-free intervals. The 'normal variant pattern', known to occur in blacks, was recorded in the absence of angina; in a white patient with chest pain it would have been considered as being due to a possible hyperacute myocardial infarction. During repeated episodes of severe angina, 'pseudonormalisation' of the 'normal variant pattern' was seen but the pointer to myocardial ischaemia was the simultaneous occurrence of ST-segment depression. Selective coronary arteriography delineated critical lesions in both the left anterior descending and dominant left circumflex coronary arteries. Coronary artery bypass surgery was successful. The importance of recognition of the 'normal variant pattern' in the black population in which the incidence of atherosclerotic coronary artery disease is rising, is emphasised.

S Afr Med J 1987; 71: 110-113.

Case report

Clinical presentation

A 56-year-old westernised black man, a pipe-smoker with diabetes mellitus (type 2) for approximately 3 years, had been treated with oral antidiabetic agents, and later by diet alone. Some 2 weeks before admission to the Intensive Coronary Care Unit at Tygerberg Hospital on 20 July 1985, he began to experience classic effort-induced angina pectoris. A few days before admission this assumed an unstable pattern and was frequently associated with *angor animi*.

On admission he was slightly overweight with no abnormal general signs, particularly not of hyperlipoproteinaemia. His pulse rate was 80/min and all peripheral pulses were easily palpable. The jugular venous pressure was not elevated and his blood

pressure was 150/90 mmHg. The fundi appeared normal. There was no evidence of cardiomegaly or cardiac failure. The heart sounds were normal and a loud 4th heart sound was audible. There were no cardiac murmurs. Side-room investigations were all normal, as was a chest radiograph.

A routine resting 12-lead ECG recorded 6 months earlier showed the following features: sinus rhythm 65/min, P-R interval 0,14 second, mean QRS axis $+60^\circ$, early ventricular repolarisation inferiorly and in all the precordial leads, and increased QRS voltage in the mid-precordial leads (Fig. 1a), but a second resting ECG recorded some 60 minutes later showed a higher heart rate of 78/min and persistent early ventricular repolarisation changes, with reduced-voltage T waves in the precordial leads (Fig. 1b). This was interpreted as a 'normal variant pattern' known to occur in this racial group, particularly since the patient had no chest pain. The resting ECG on admission, in the absence of chest pain, revealed the following: sinus rhythm 80/min, P-R interval 0,18 second, mean QRS axis $+50^\circ$ and even further reduced T-wave voltage in all the precordial leads (Fig. 1c). The early ventricular repolarisation was less pronounced than in the tracings recorded some 6 months earlier. A diagnosis of unstable angina pectoris associated with a 'normal variant pattern' was then made. The patient was given oral calcium antagonists, transdermal nitroglycerin and heparin infusion. Serum cardiac enzyme levels were normal.

The patient remained free of angina pectoris on this therapeutic regimen, but a resting ECG recorded without angina on the day after admission demonstrated biphasic T waves in the antero-septal leads with less pronounced early ventricular repolarisation and a heart rate of 66/min (Fig. 1d). The serum cardiac enzyme levels remained within the normal range. An ECG on the next day recorded a higher heart rate of 80/min with more obvious and extensive biphasic T waves in the precordial leads, as well as significantly greater QRS voltage in these same leads (Fig. 2a). Furthermore, the ST segments anterolaterally and antero-septally had an upward-coved configuration which would normally be accepted as a feature of hyperacute myocardial infarction. This finding was still considered to be a 'normal variant pattern'. On the following day, with the patient free of any chest pain, a resting ECG demonstrated a heart rate of 90/min, disappearance of the biphasic T-wave pattern and reduction of the QRS voltage in the precordial leads (Fig. 2b). The inferior leads appeared normal.

The patient's progress continued to be most satisfactory with no evidence of cardiac arrhythmia or further angina pectoris. However, at 06h30 on 24 July, 4 days after admission, he experienced sudden severe precordial discomfort with sweating. An ECG revealed a heart rate of 100/min, P-R interval 0,14 second, mean QRS axis $+60^\circ$, 'normalisation' of the upward-coved T waves in the antero-septal leads and associated diminution of the R-wave voltage in these same leads (Fig. 2c). A most striking feature was the presence for the first time of 1-2 mm horizontal ST-segment depression in leads V4 and V5. A diagnosis of significant apico-lateral subendocardial ischaemia, possibly due to coronary artery spasm, was then made; additional sublingual nitrates and increased calcium antagonist dosage had a good effect. An ECG after disappearance of chest pain demonstrated a heart rate of 80/min, iso-electric ST segments in the precordial leads, and a return to a

Cardiac Unit, Department of Internal Medicine, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP
J. Z. PRZYBOJEWSKI, M.B. CH.B., F.C.P. (S.A.), F.I.C.A., F.A.C.C., F.C.C.P., F.A.C.P., F.S.C.A.
P. H. BECKER, M.D.

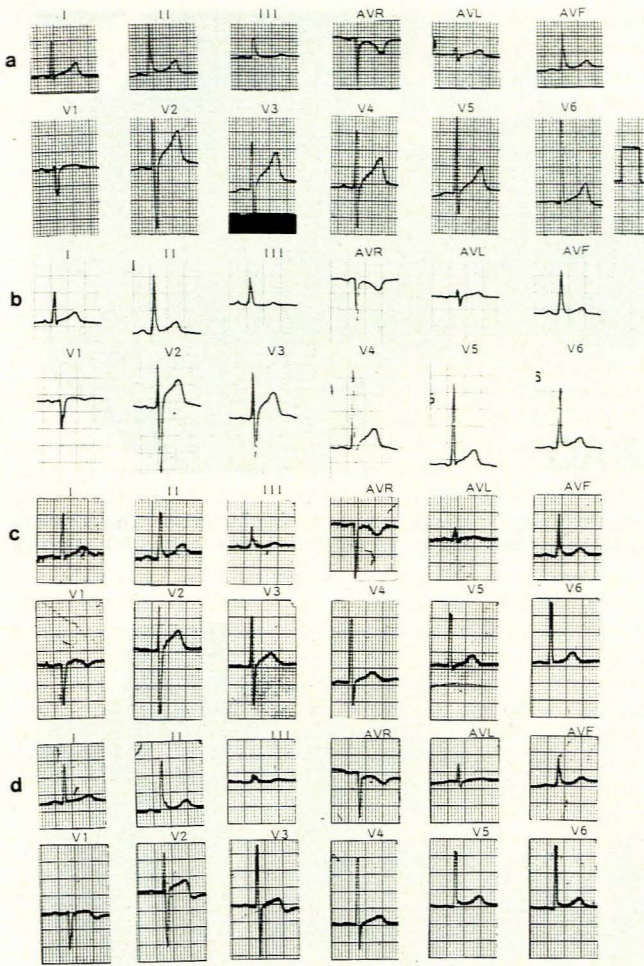


Fig. 1. Resting 12-lead ECGs on full standardisation. (a) Routine outpatient trace showing early ventricular repolarisation in the inferior and precordial leads in keeping with a 'normal variant pattern'. (b) Tracing 1 hour later: reduced T-wave voltage but persistent early ventricular repolarisation. (c) Recording on admission to the ICCU (no chest pain). Less pronounced early ventricular repolarisation and upward-coved flat T waves in the anteroapical leads. Trace suggestive of a 'normal variant pattern'. (d) In the absence of chest pain, biphasic T waves and less early ventricular repolarisation anteroapically. Trace possibly in keeping with an evolving acute myocardial infarction.

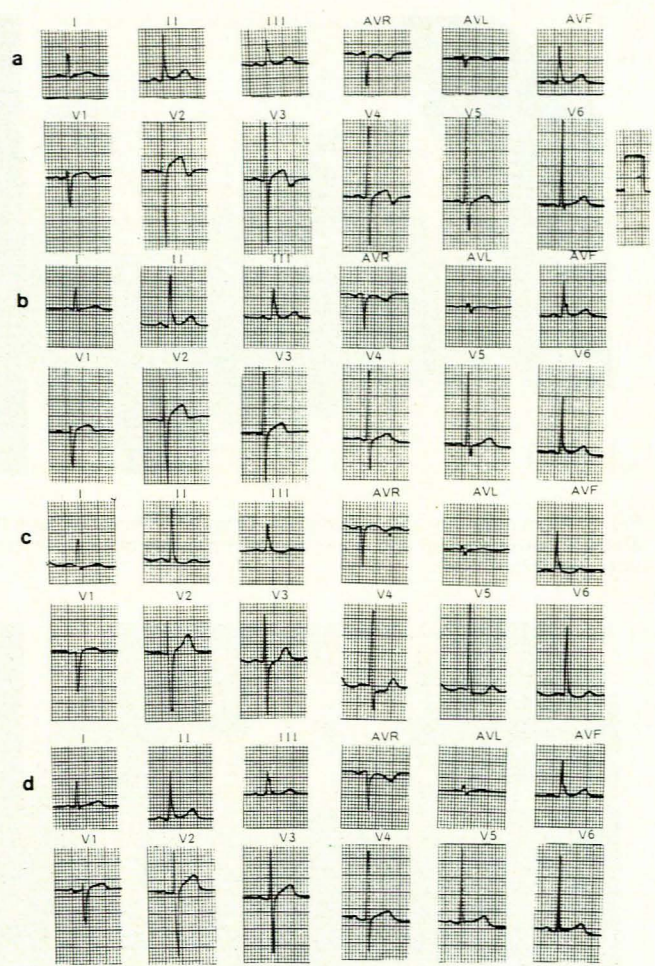


Fig. 2. Resting 12-lead ECGs recorded on full standardisation. (a) Tracing without chest pain. More extensive biphasic T waves and upward-coved ST-segments suggestive of a 'normal variant pattern' or an evolving acute myocardial infarction. (b) Absence of chest pain with now upright T waves and reduction of precordial QRS voltage. (c) Tracing during episode of severe precordial pain. 'Pseudonormalisation' of the upward-coved T waves with decreased R-wave voltage is seen anteroapically. Significant lateral ST-segment depression is visible for the first time. (d) After nitrates and nifedipine with relief of chest pain. Return of features suggestive of a 'normal variant pattern' and resolution of the previous lateral ST-segment depression.

pattern of upward-coved ST segments in these same leads suggestive of a 'normal variant pattern' (Fig. 2d). Serum enzyme levels remained normal. At 15h00 on the same day, an ECG during a further episode of severe precordial pain showed a sinus tachycardia of 108/min with 'normalisation' of the previous upward-coved ST segments in the anteroapical and high lateral leads. The patient was again given sublingual nitrates with slow relief. A continuous nitroglycerin infusion kept him angina-free.

Early on the following morning the patient again complained of very severe precordial pain. A resting ECG now recorded sinus rhythm of 96/min with upward-sloping ST-segment depression in the right precordial leads, the maximum depression being 1.5 mm in lead V4. The ST segments in the inferior leads were now horizontal but not depressed. The nitroglycerin infusion rate was increased with relief of the chest discomfort. An ECG recorded after total disappearance of chest pain showed upward-coved ST segments in the early precordial leads, and resolution of the ST-segment depression both precordially and in the inferior leads. The ECG again had the configuration of the 'normal variant pattern' seen in this racial group. Serum cardiac enzymes remained normal.

Very early on the morning of 26 July another episode of severe

chest pain occurred and resting ECG during this episode revealed a sinus tachycardia of 110/min, 'normalisation' of the precordial T waves, but upward-sloping ST-segment depression in the apico-lateral leads reaching a maximum of 1 mm in lead V4. The nitroglycerin infusion rate was increased and there was some relief of the chest discomfort. It was now clear that the patient was not being adequately controlled on drug therapy, and emergency cardiac catheterisation was undertaken to establish a definitive diagnosis with a view to possible emergency coronary artery bypass graft (CABG) surgery.

Cardiac catheterisation

This procedure was carried out on the morning of 26 July, 6 days after admission. The right coronary artery (RCA) was shown to be non-dominant and angiographically normal (Fig. 3). Quite extensive collateralisation to the left anterior descending (LAD) coronary artery was noted, signifying obstruction in that vessel. Injection of contrast into the left coronary artery (LCA) demonstrated a normal left mainstem, a long and shelf-like 90% diameter stenosis in the LAD coronary artery proximal to the first septal

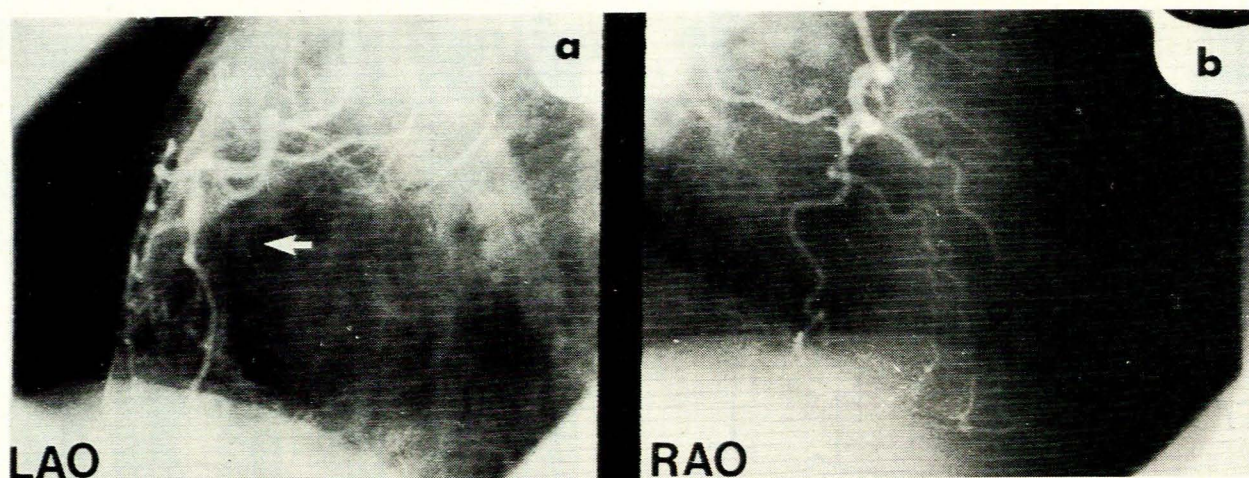


Fig. 3. Right coronary cine angiograms in (a) left anterior oblique (LAO) and (b) right anterior oblique (RAO) projections. The coronary artery is non-dominant and is angiographically free of atherosclerosis. There is some right-to-left collateralisation to the LAD coronary artery (lad) (arrowed).

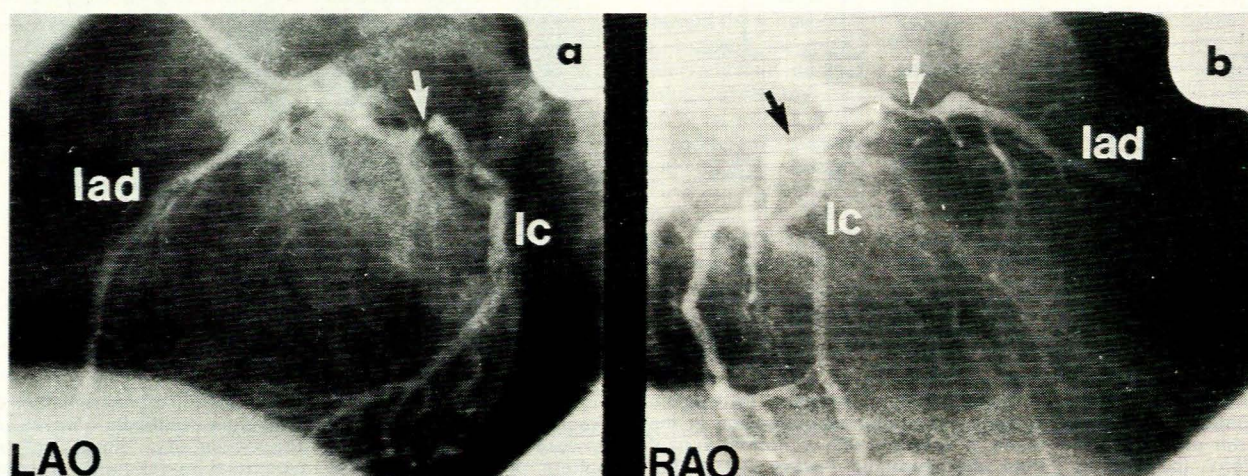


Fig. 4. Left coronary cine angiograms in (a) LAO and (b) RAO views. A long, 90% diameter stenosis (arrowed) is seen in the LAD coronary artery (lad). There is also a short subtotal obstruction (arrowed) in the dominant lcx coronary artery (lc) just distal to the mid-lateral branch. The vessels are suitable for bypass surgery.

perforator artery, and a short subtotal obstruction in the dominant left circumflex (lcx) coronary artery just distal to the mid-lateral branch (Fig. 4). No coronary calcification or left-to-left collateral flow could be seen. The patient experienced several episodes of angina pectoris during this procedure.

He underwent emergency CABG surgery the following morning. A right internal mammary artery bypass graft was inserted into the LAD coronary artery distal to the stenosis, and a saphenous vein was used to graft the lcx coronary artery proximal to a large posterolateral branch. The operation was successful.

Discussion

This case demonstrates the importance of being aware of the 'normal variant pattern' known to occur in the black population of this country, and highlights the difficulty which may be experienced in assessing suspected acute myocardial ischaemia or hyperacute myocardial infarction in this setting.

The 'normal variant pattern' in black adults in the RSA was first documented by Brink.^{1,2} This was followed by attempts at classification of the types of ECG changes by Grusin,³ who suggested three 'patterns' as determined by changes affecting the QRS and ST-T-wave segments. These were as follows:

pattern I (the most common), characterised by ST-segment depression and asymmetric deeply inverted T waves in the right ventricular precordial leads (V1 - V3); pattern II (the next most common), verified by prominent ST-segment elevation associated with tall peaked T waves and large amplitude R waves, seen mainly in the left precordial leads (V4 - V6); pattern III (the least common and most nondescript), in which 'flat' or rounded T waves occur in all the precordial leads (V1 - V6). Grusin³ documented this 'normal variant pattern' in as many as 63% of blacks in 'good health', the latter statement being severely criticised by Woods and Laurie.⁴ Przybojewski *et al.*,⁵ in a study involving 1 605 healthy black males, reported a much lower frequency of 15,3% of the 'normal variant pattern', the most common being the type II pattern, followed by the type III pattern and only then the type I pattern. Przybojewski *et al.*⁵ also noted that in both the type I and type II patients the patterns were not only confined to the right or left ventricular precordial leads respectively, but they were seen in most of the precordial leads. A type IV pattern, i.e. a combination of Grusin's three different patterns, was first described by Przybojewski *et al.*⁵ in 3,3% of 1 605 subjects. These researchers also confirmed the findings of previous workers⁶⁻⁹ documenting the intermittent change from

one pattern to another. Similar ECG patterns have been noted in American blacks as well as in the younger white population. Various descriptive terms have been applied to these findings: 'premature repolarisation';¹⁰ 'early repolarisation variant';¹¹ 'normal RS-T elevation variant';¹² 'unusual RT segment deviation';¹³ 'early repolarisation syndrome';¹⁴ and 'juvenile pattern of adult Negro males'.¹⁵

The resting ECG, in the absence of angina pectoris, of our patient was a combination of Grusin type II and type III patterns. If these same findings had been encountered in a white patient in the presence of acute precordial pain a diagnosis of possible hyperacute anteroseptal and anterolateral myocardial infarction would have been seriously considered. Of great interest was the 'pseudonormalisation' of the 'normal variant pattern' during episodes of severe angina pectoris and acute myocardial ischaemia, ECG features which would be considered normal in whites even in the presence of angina pectoris. The clue to acute myocardial ischaemia in this black man was the concomitant reversible ST-segment depression, an ECG feature accepted in any population group. Selective coronary cine angiography established the presence of significant double-vessel atheromatous coronary artery disease in the anatomical distribution which correlated with the intermittent acute myocardial ischaemia documented on repeated resting ECGs. It is impossible to be certain whether episodes of coronary vasospasm ('functional stenosis') were superimposed on the 'fixed' stenoses, although this mechanism is possible. The 'normal variant pattern' has also been documented by Przybojewski and Heyns^{16,17} as causing great difficulty in the diagnosis of both acute coronary artery spasm and acute myocardial infarction secondary to industrial nitroglycerin withdrawal.

Atherosclerotic coronary artery disease is now being encountered more frequently in westernised or urbanised rather than rural blacks.¹⁸⁻²³ Assessment of the black patient with chest pain, although this might appear atypical for angina pectoris, must take into account the 'normal variant pattern' on ECG, since this is a most useful and frequently used tool in the diagnosis of the spectrum of ischaemic heart disease. It must be remembered, however, that this 'normal variant pattern' can be confused with such conditions as acute pericarditis,²⁴⁻²⁶ chronic constrictive pericarditis,²⁷ acute or previous non-transmural myocardial infarction,²⁸ hypertrophic cardiomyopathy,^{29,30} dilated (congestive or idiopathic) cardiomyopathy,³¹ hyperventilation syndrome,³² and hyperkalaemia.³³

The authors wish sincerely to thank Miss Hettie Weymar of the Cardiac Unit, Tygerberg Hospital, for preparing the ECG illustrations. Thanks are also due to Mrs I. Bickel, Head of the Photographic Unit, Bureau for Medical and Dental Education at the University of Stellenbosch, for preparing the cine angiograms. We would also like to express our gratitude towards Dr J. P. van der Westhuyzen, formerly Chief Medical Superintendent, Tygerberg Hospital, for permission to publish.

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