

Acute myocardial infarction with a non-diagnostic electrocardiogram

Case presentation and overview

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Summary

The clinical presentation of a young hypertensive White man with acute high lateral non-transmural myocardial infarction (MI) is documented. This diagnosis was established on the grounds of a history of chest pain, elevated serial serum enzyme levels, technetium-99m pyrophosphate ('hot-spot') scintigraphy, exercise thallium-201 ('cold-spot') scanning, left ventricular cine angiography and selective coronary arteriography. Daily resting 12-lead ECGs failed to demonstrate unequivocal features of acute non-transmural subendocardial MI. The diagnostic difficulties facing the clinician in a case of acute MI associated with a non-diagnostic ECG are stressed, and the ECG features of acute subendocardial MI are reviewed.

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Case presentation

The patient was a 40-year-old White man, a heavy smoker who had been known to be hypertensive for the previous 4 years and was receiving propranolol 160 mg 3 times daily. An intravenous pyelogram and renal biopsy specimen were normal. Apart from this diagnosis he had been completely well until 25 March 1982 when he experienced a sudden crushing retrosternal pain radiating down the left forearm while sitting working at his desk. This pain lasted a few minutes and was accompanied by some

shortness of breath. He then went home where his wife, a nursing sister, found his blood pressure to be very high. The general practitioner was called and arranged for admission to a peripheral hospital. At this stage the hypertension was treated with repeated injections of dihydropyridine. A few hours later he again experienced a severe retrosternal pain and was transferred to the intensive coronary care unit (ICCU) of Tygerberg Hospital on 26 March 1982 with a diagnosis of unstable angina. On examination the only abnormal features were elevated blood pressure (190/120 mmHg), a loud fourth heart sound and grade 2 hypertensive retinopathy. A resting ECG delineated sinus rhythm of 80/min, a P-R interval of 0.16 second, a mean frontal QRS axis of -15° , and no evidence of ischaemia or myocardial infarction (MI) (Fig. 1a). The chest radiograph demonstrated a normal cardiac silhouette with clear lung fields. The results of urinalysis and a full blood count were within normal limits, as were the serum electrolyte, creatinine and urea values. On initial investigation the cardiac serum enzyme values were found to be moderately elevated (Table I). A clinical diagnosis of probable acute non-transmural MI with a normal ECG was made, and the patient was continued on β -blockade. In addition, he was given a continuous heparin infusion and the calcium-blocking drug nifedipine 20 mg 8-hourly as well as long-acting oral nitrates. Over the ensuing days serial enzyme estimations demonstrated the typical picture of an acute myocardial infarction (AMI) (Table I). However, serial resting 12-lead ECGs failed to show any definite features of an AMI (Figs 1-3). The hypertension settled and the patient no longer complained of chest pain.

In view of the non-diagnostic ECG it was decided to perform a technetium-99m pyrophosphate ('hot-spot') scan on 30 March 1982. This isotope investigation delineated a definite area of increased activity in the high lateral and slightly posterior aspects of the left ventricle, features in keeping with an AMI (Fig. 4). A diagnosis of a probable acute non-transmural posterolateral MI was entertained, but daily resting ECGs (including leads V7-V9) remained remarkably normal (Figs 1-3). The patient was mobilized and discharged on 10 April 1982 (2 weeks after admission to the ICCU) on maintenance therapy consisting of a combination of β -blockade, calcium blockade and long-acting nitrates.

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TABLE I. SERIAL SERUM ENZYME ESTIMATIONS

Enzyme	Date								Normal range
	26 Mar.	27 Mar.	28 Mar.	29 Mar.	30 Mar.	31 Mar.	1 Apr.	7 Apr.	
CK	218	218	88	37	16	10	6	4	0 - 50
AST	136	121	100	72	55	35	32	14	0 - 40
ALT	35	28	61	75	91	73	62	31	0 - 53
LD	386	581	570	502	499	373	335	316	100 - 350

All values given in U/l.

CK = creatine kinase; AST = aspartate transaminase; ALT = alanine transaminase; LD = lactate dehydrogenase.

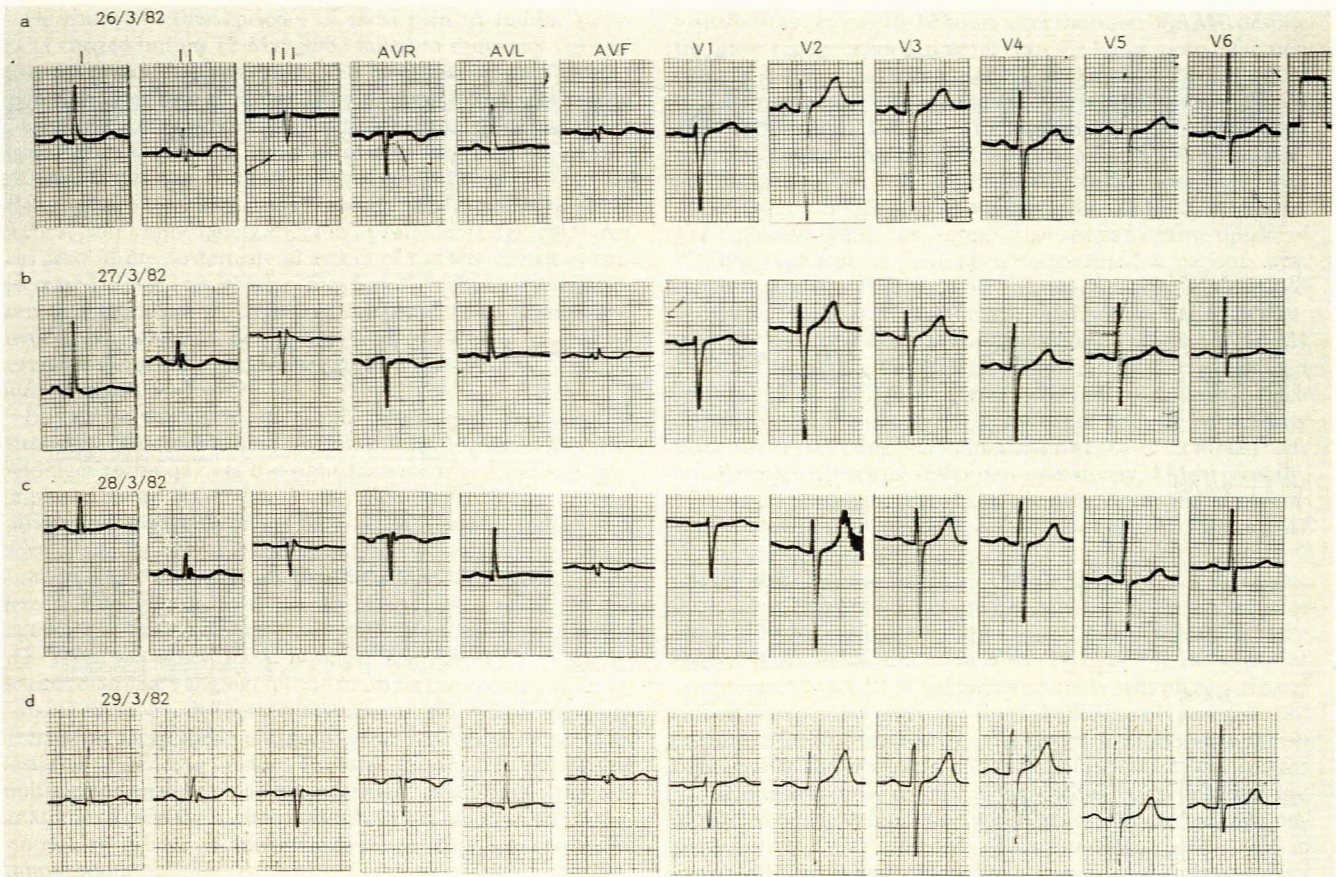


Fig. 1 (a - d). Serial 12-lead ECGs taken at rest. Minor left-axis deviation is evident. The only suggestion of possible myocardial ischaemia is the sharp ST-T-segment junction in the high lateral leads I and aVL (excluding the ECG of 26/3/82).

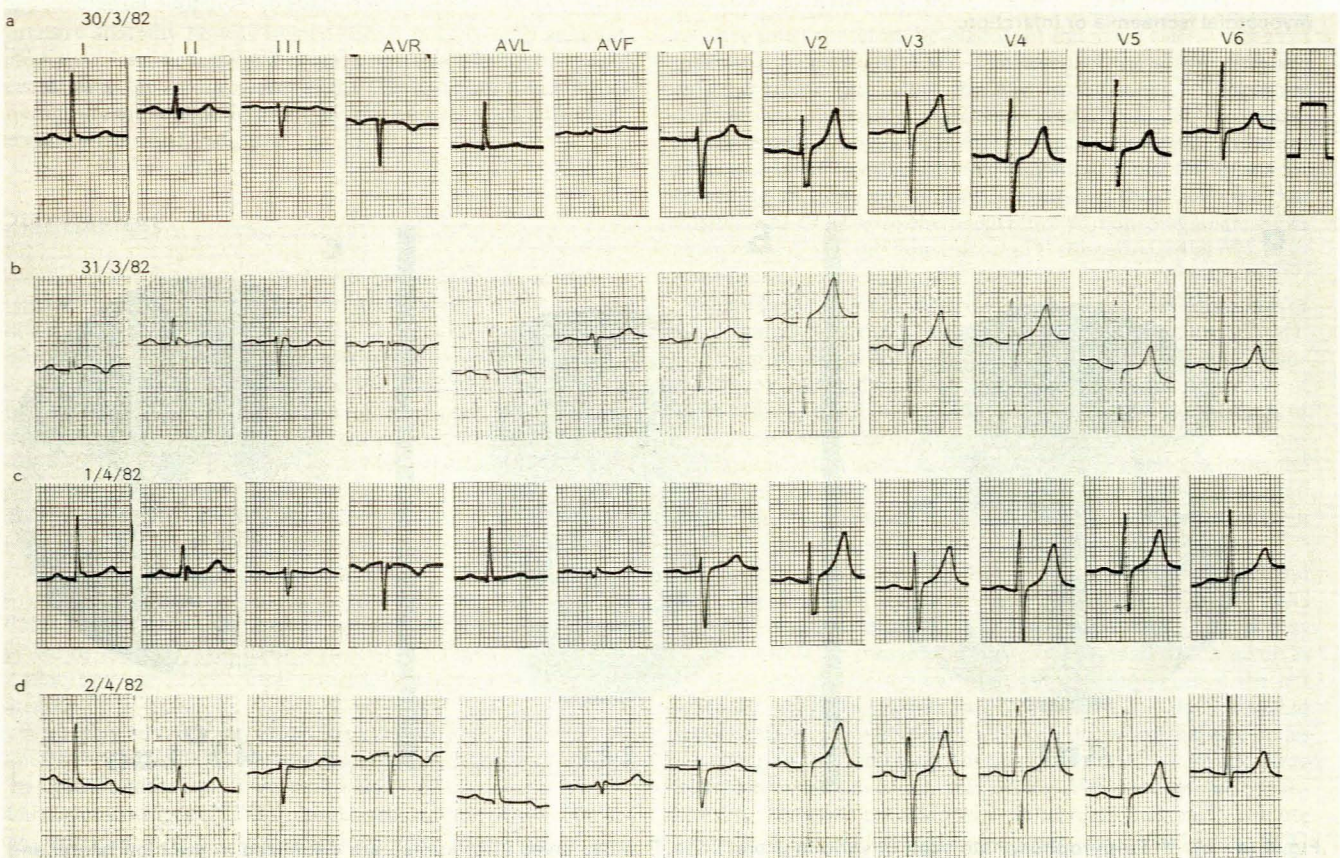


Fig. 2 (a - d). Serial 12-lead ECGs taken at rest. The only abnormality seen is slight left-axis deviation and asymmetrical T-wave inversion in standard lead I on 31/3/82.

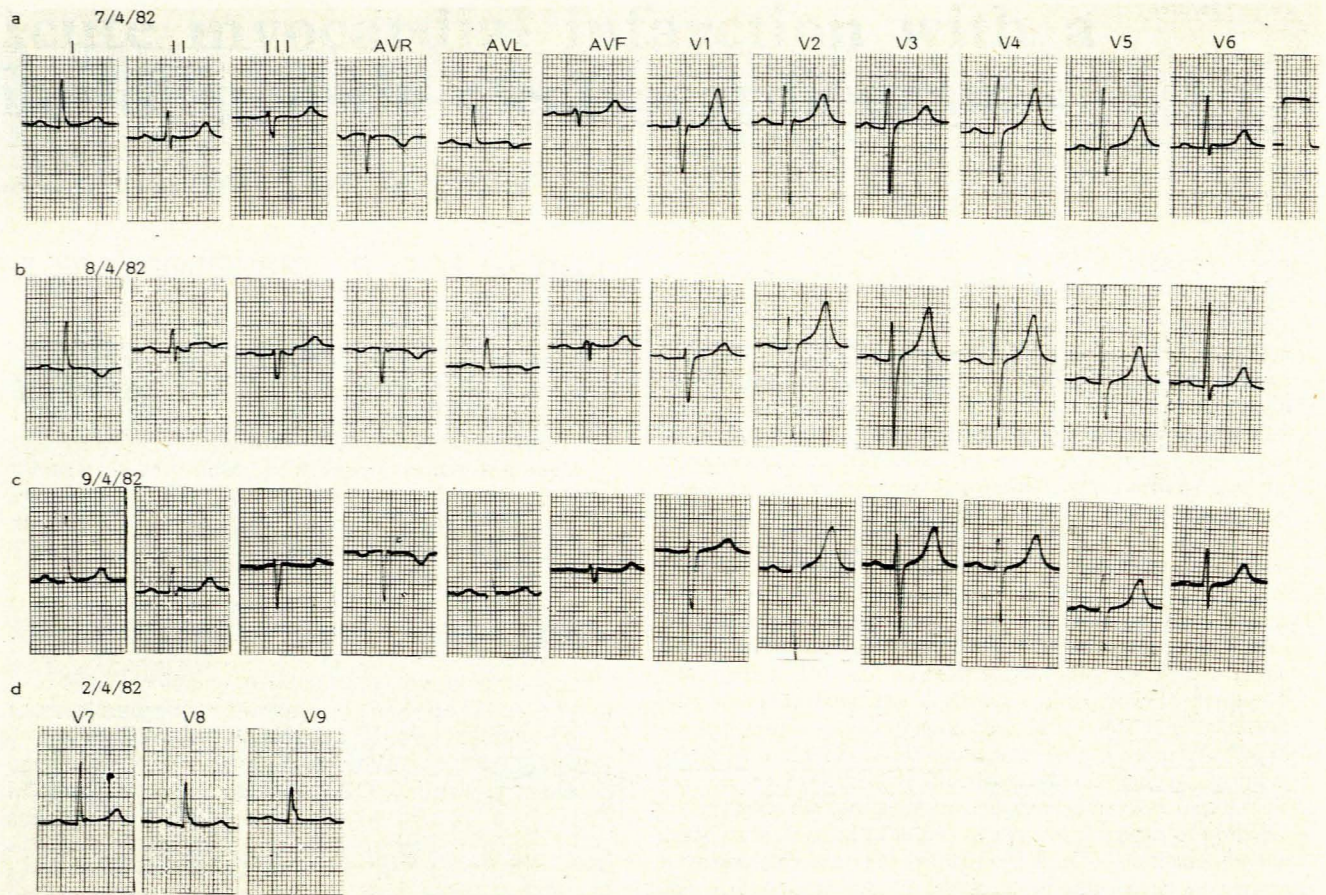


Fig. 3. Serial 12-lead ECGs taken at rest: a - c — isolated T-wave inversion in standard lead I (8/4/82) and slightly biphasic T waves in standard lead aVL is seen, together with persistent left-axis deviation; d — extended lateral chest leads demonstrate no evidence of myocardial ischaemia or infarction.

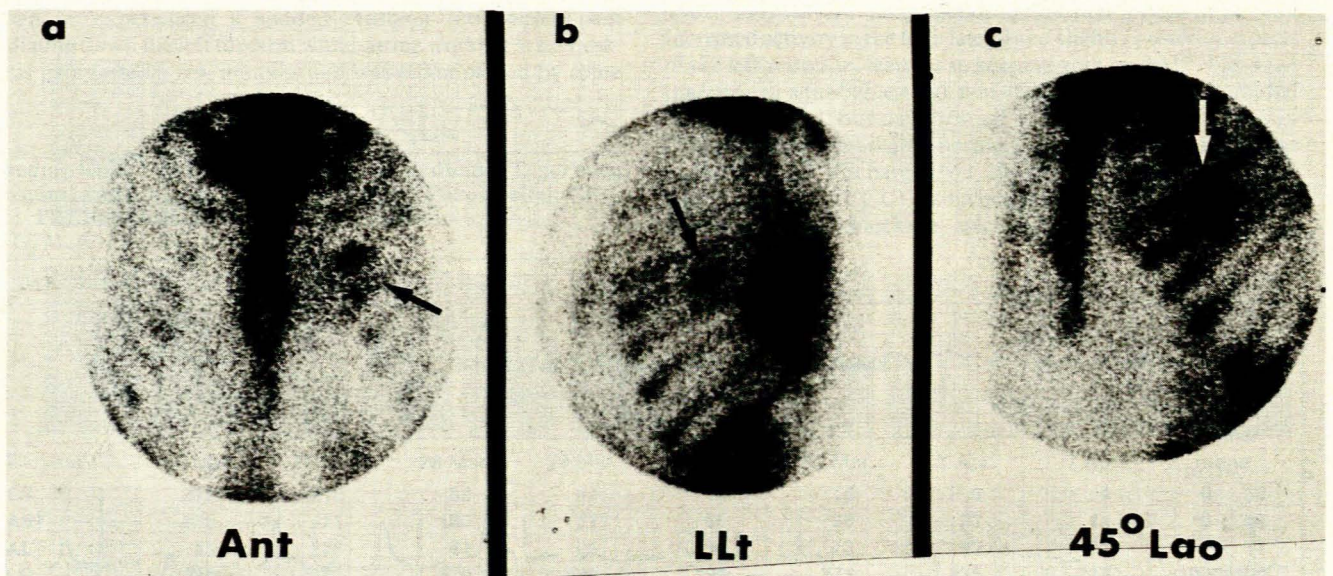


Fig. 4 (a - c). ^{99m}Tc -pyrophosphate scan in various projections. Positive areas ('hot-spots') are visualized in the high lateral and posterior region of the left ventricle (arrowed). (Ant = anterior; LLt = left lateral; 45° Lao = left anterior oblique).

He had no further episodes of chest pain. A routine stress ECG carried out on 12 May 1982 failed to show any signs of possible myocardial ischaemia. This investigation was followed soon thereafter by a thallium-201 stress test ('cold-spot scan'). During the latter procedure the patient reached a maximum heart rate of 135/min (instead of the target heart rate of 170/min); this was limited by tiredness, the patient having exercised for a total period of 16 minutes without the occurrence of chest pain. Immediately after this a prominent large 'cold' area was seen in the posterolateral aspect of the left ventricle; this decreased in size 4 hours later (Figs 5 and 6). The characteristics were therefore those of a large 'cold' area produced by irreversible myocardial ischaemia or previous MI as well as an area of reversible ischaemia secondary to significant obstructive coronary atherosclerosis.

In view of the above, cardiac catheterization and selective coronary angiography were undertaken on 13 May 1982. The Seldinger technique via the right femoral artery and vein was employed using 7F Goodale-Lubin and pigtail catheters. All the intracardiac pressures (apart from aortic pressure, which was elevated) and indices of cardiac contractility were normal. A left ventricular cine angiogram in the right anterior oblique projection demonstrated good general contractility, but there was prominent segmental inferior and anterobasal akinesia secondary to previous MI. There was no evidence of mitral insufficiency or prolapse. Selective coronary angiography in multiple projections, using 7F Judkins catheters, delineated a dominant ectatic right coronary artery with a significant obstructive lesion near the origin of its posterior descending branch. The left coronary artery displayed multiple internal luminal irregularities; in addition, the left circumflex coronary branch was totally occluded in the atrio-ventricular groove. Catheterization was completed without complication.

A diagnosis of double-vessel atherosclerotic coronary artery disease with previous high lateral and posterior MI as well as systemic hypertension was thus established. In view of the patient's relative lack of symptoms and surgically unsuitable coronary anatomy he was treated conservatively with atenolol, nifedipine, long-acting nitrates and a diuretic. He has been seen several times as an outpatient at the Cardiac Clinic and denies any significant angina. In addition, the hypertension appears to be controlled satisfactorily.

Discussion

The clinician is quite often confronted with difficulty in making a definitive diagnosis of an AMI, particularly if this is non-transmural. Traditionally the triad of a history of significant chest pain, evolving ECG changes and elevation of 'cardiac' serum enzyme values have been accepted criteria for establishing such a diagnosis.¹ Resolution of this clinical problem was partly aided by the World Health Organization criteria, which are frequently quoted in publications.^{2,3} Nevertheless, these criteria were primarily established for use in epidemiological studies rather than for the clinician. In addition to the patient's history, ECG features and cardiac enzyme estimation results, the WHO criteria also considered postmortem findings. Three categories of 'definite', 'possible' and 'no' MI were delineated, and the WHO considered a diagnosis of definite MI if the history was typical or atypical with equivocal ECG changes and elevated cardiac enzyme levels. Our patient therefore falls into this category.

With the advance of technology more sophisticated and expensive techniques have evolved for the diagnosis of AMI.⁴ The most notable of these has been the use of the radio-pharmaceutical agent ^{99m}Tc-pyrophosphate in myocardial imaging ('hot-spot scan'),⁵⁻⁷ first described in 1974 by Bonte *et al.*⁸ This substance is taken up by the necrotic tissue and may be

visualized as early as 10-12 hours after the onset of AMI, despite the ideal recommended time of 48 - 72 hours post-infarction (usually synonymous with onset of chest pain).^{9,10} In our patient this scintiscan was performed about 7 days after onset of the MI and was positive (Fig. 4). This investigation has been most reliable, giving false-negative results only in 4% and false-positive scintiscans in 8-12% of cases.¹¹ A false-negative result usually occurs in the case of infarctions smaller than 3 g in size. False-positive scintiscans, often visualized as a diffuse uptake of ^{99m}Tc-pyrophosphate, have been documented in patients with congestive cardiac failure, stable angina,¹² unstable angina¹³ and ventricular aneurysms. Massie *et al.*¹² found this scan to be most insensitive as regards acute non-transmural subendocardial MI since diffuse uptake was seen (apart from in cases in which high creatine kinase MB iso-enzyme levels were present, which displayed discrete uptake). The 'hot-spot scan' is particularly valuable in detecting AMI in the presence of complete left bundle-branch block as well as peri-operatively.¹⁴ Most recently, ^{99m}Tc-pyrophosphate has been employed in single-photon emission computed tomography for the quantification of MI size, a parameter known to be crucial in determining prognosis.¹⁵

The other isotope commonly used in studies of ischaemic heart disease is ²⁰¹Th. This isotope, giving rise to a 'cold-spot scan', has been used in the diagnosis of AMI but is really reliable only if performed within the first 24 hours of the onset of symptoms.¹⁶ Since its initial high sensitivity falls off rapidly and since it is expensive and has a short shelf-life, it is generally not employed for this purpose. Furthermore, it cannot distinguish between old and new MIs, and is therefore mainly utilized for the demonstration of reversible myocardial ischaemia. The use of this isotope in conjunction with stress testing is well demonstrated in our case, since the constant 'cold spot' is due to previous MI whereas the decrease in the size of the isotope defect after 4 hours is due to the presence of reversible myocardial ischaemia (Figs 5 and 6).

Unfortunately, facilities for the above isotope investigations are not generally available outside university hospitals, and reliance must therefore be placed on the more classic means of making a diagnosis of AMI. The levels of the serum enzymes creatine kinase (including the MB iso-enzyme),^{17,18} aspartate transaminase, alanine transaminase and lactate dehydrogenase are commonly determined in most peripheral hospitals and are of great assistance in establishing a diagnosis. Since the means for recording an ECG are uniformly available, the discussion that follows will emphasize the occurrence of non-diagnostic ECG changes seen in acute non-transmural subendocardial MI, as in our patient.

There has been much controversy about the ECG diagnosis of acute 'subendocardial', 'subepicardial' and 'intramural' MI. These categories of MI have been collectively designated 'non-transmural', since the extent of necrosis should theoretically not extend from the subendocardial to the subepicardial surfaces of the myocardium. The distinguishing ECG feature of acute 'non-transmural' infarctions has been classically accepted as being the absence of the 'pathological' Q wave seen in acute 'transmural' infarctions. Nevertheless, these ECG definitions have often been disproved when correlated with autopsy findings. Horan *et al.*¹⁹ and Abbott and Scheinman²⁰ have documented both old and acute transmural MI in the absence of Q waves, the only ECG features being changes in the ST-T-wave segment. Q waves may be absent in acute transmural MI if this is small or if it involves the high lateral, lateral, and high anterior segments of the left ventricle. These Q waves may, however, be demonstrated occasionally by recording the ECG with the precordial electrodes placed one or two interspaces higher than usual.²¹⁻²³ To add more confusion to the issue, non-transmural MI (documented pathologically) has been reported with abnormal Q waves.²⁴⁻²⁶ Acute subendocardial MI has traditionally been diagnosed on the ECG by the presence of ST-segment depression with or without deep

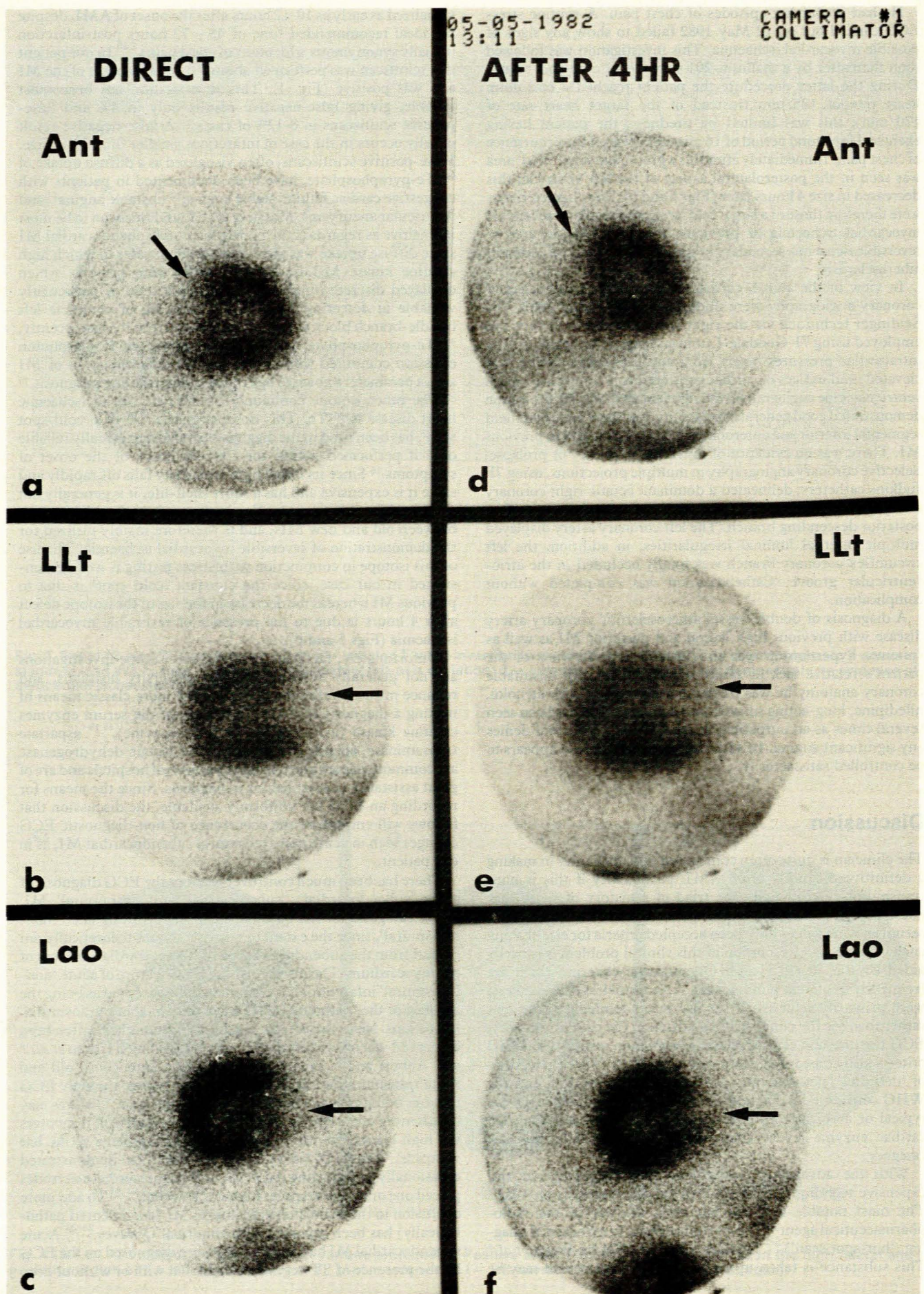


Fig. 5 (a - f). ^{201}Th scintiscan in various projections performed immediately after exercise testing (direct) as well as after 4 hours. A large 'cold' area (arrowed) is seen posterolaterally in the left ventricle directly after stress, this becoming smaller after 4 hours.

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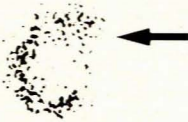
DIRECT
Ant



a

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LLt



b

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Lao

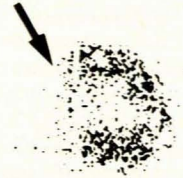


c

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AFTER 4HR
Ant



d

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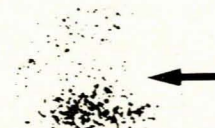
LLt



e

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Lao



f

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Fig. 6 (a - f). Extraction ^{201}Th scintiscans in various views showing features as described in Fig. 5.

symmetrical T-wave inversion.^{27,28} These changes have often been accompanied by ST-segment elevation in lead aVR,^{29,30} but some workers could not demonstrate this finding.^{31,32} More subtle changes such as a slight diminution in R-wave amplitude have sometimes been encountered in acute subendocardial infarction;^{26,29,32} if these are seen in the lateral precordial leads some workers²⁵ believe that they have the same significance as Q waves in these specific leads. The accuracy of ECG localization of AMI by using postmortem findings has been considered somewhat unreliable, especially as regards subendocardial MI.³³ Yasuda *et al.*³⁴ used ^{99m}Tc-pyrophosphate scintigraphy and found that ECG localization was accurate in 85,9% of 34 patients studied. Furthermore, the sensitivity of the ECG in detecting high lateral MI was 80%, whereas the specificity was 87,5%.

The nonspecificity of the ECG in acute subendocardial MI has been partly attributed to 'electrocardiographically silent' myocardium.³⁵⁻³⁷ Thus, alterations of the QRS complex may not be seen and reliance may have to be placed on changes in the ST segment.³⁸ Some authors,^{39,40} however, dispute the existence of silent areas and claim that subendocardial infarction can be detected by leads orientated to the epicardial surface overlying the infarcted region. The appearance of Q waves in subendocardial MI was further supported by the findings of Boineau *et al.*⁴¹ Other explanations, such as the 'boundary interface effect', have been proposed for slight ST-segment shift in subendocardial MI.³⁸ The duration and degree of ECG injury manifestation is also known to be more marked in subepicardial injury when compared with subendocardial damage.

Schamroth³⁸ describes the occurrence of two different and distinct ECG 'syndromes' of acute subendocardial MI, 'hyper-acute subendocardial injury' (no Q waves) and 'subendocardial necrosis' (presence of Q waves). Dunn *et al.*⁴² came to the rather elegant conclusion that 'infarcts of the lateral wall of the left ventricle, particularly in their acute phases, are not electrocardiographically 'silent'; their voice is, however, one of weak and mingled tones'. Nevertheless, no matter how quiet this 'voice' might be, the ECG *per se* is not totally adequate in establishing the diagnosis of AMI. This fact is aptly demonstrated in this case presentation, which illustrates the importance of performing more sophisticated tests as exemplified by isotope studies and coronary arteriography.

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