

Coronary Heart Disease

Part 2: A review of the diagnosis of Myocardial Infarction in general practice

- E L Murray

Summary

The symptomatology, ECG and laboratory findings relevant to making the diagnosis of Myocardial Infarction in general practice are reviewed and discussed.

S Afr Fam Prac 1986; 7:244-9

Myocardial infarction implies death of heart muscle due to obstruction of a coronary artery.

It is usually a dramatic incident, with the crushing chest pain described as a tight band around the chest. The pain lasts longer than 20 minutes and is accompanied by pallor and sweating. The pain may be so severe as to cause nausea and vomiting.

Sometimes the pain is less severe and in one third of cases the infarct goes unsuspected by the patient and undiagnosed until a later ECG shows the characteristic Q-waves of an old infarct.

A scar suggestive of an old MI is found in 43% of sudden and unexpected deaths occurring without prior clinical history of heart disease, and an acute MI is noted in 11%.

While GPs are not always confident about treating all MIs, it is essential that all be fully conversant with the differential diagnosis of chest pain, vasovagal attacks, acute pulmonary oedema, etc., so that they can either make the diagnosis of MI when it occurs, or pass the patient on right away to someone who is competent to do so.

As two-thirds of premature deaths from MI occur before the patient is admitted to hospital (almost 100% due to preventable cardiac arrhythmias), the physician must also be expert in the emergency treatment of the MI and must also make the families of patients at risk aware of Cardio Pulmonary Resuscitation (CPR) and put them in touch with the local branch of National Heart Foundation, who will in turn arrange CPR instruction.

The GP must decide for himself on the best way of emergency handling for his patients. If he lives in a large metropolitan area with an efficient paramedical ambulance service, it might be quicker for such a service to get the patient to the hospital where the hospital staff would either treat or continue emergency treatment until the GP arrives. Other alternative approaches are available. In the Faculty

of GP Research Committee study in Cape Town, patients did far better than in any other study. Here the GP got to the patient at home and was able to institute the correct treatment for pain as well as the prevention of ventricular fibrillation (VF). The GP's presence as soon as possible after the onset of chest pain probably had a lot to do with their excellent results.^{1,2}

DIAGNOSIS OF MI

A MI must always be suspected in cases of chest pain, acute pulmonary oedema or syncopal attacks. Where the diagnosis cannot be confidently excluded, the patient should be admitted to hospital or confined to bed at home pending further investigation.

MIs are treated at home in some instances, but with the more modern approach to treatment most physicians feel uncomfortable in managing patients at home these days.

The definitive diagnosis is based on

- (1) the history
- (2) ECG changes
- (3) serial serum enzymes, or failing these
- (4) post mortem examination

1. History

The typical history is one of a chest pain with certain characteristics. The pain is as described for angina but of longer duration and is not relieved by sublingual nitrates.

The site is typically retrosternal but may radiate into the neck or jaw, to the shoulders, (more usually the left) or down the inside of the arm or the wrist.

The character of the pain is described as crushing or as a tight band around the chest. It is not a stabbing or burning pain.

The pain lasts for more than 20 minutes and is not totally relieved by nitrates but may be reduced by them.

The pain is usually more severe than anginal pain and may be associated by pallor, sweating and nausea.

Examination is necessary to record the patient's "vital signs" and to detect complication, but is usually non-contributory in making the diagnosis.

2. ECG

The ECG may show no changes at all if done early enough. It should, however, be recorded as soon as possible as the changes that occur make the diagnosis.

The sequence of changes in an acute infarction are:

- (i) within hours of the clinical onset of infarction there is ST segment elevation
- (ii) the R wave voltage then falls and abnormal Q waves appear
- (iii) the T waves invert and the ST segments gradually return to normal
- (iv) in time the ST segments and T waves return to normal, but the R wave amplitude might never return to normal, and the Q waves usually persist.

It follows that it is essential not to exclude an infarct because of a normal tracing, and that serial tracings are necessary for diagnosis.

The unequivocal ECG changes that occur in a MI are:

- (a) The development of a pathological Q wave
- (b) Current of injury lasting for more than 24 hours (level or convex upwards)
 - except
 - (i) In pericarditis the ST elevation has a different appearance, being concave upwards.
 - (ii) Digitalis effect is suggested by a mirror-image correction mark.
 - (iii) Strain pattern - depressed convex upward in leads V5 or V6 with left ventricular hypertrophy.

(a) Pathological Q Waves

To be pathological, the Q wave is

- (i) **Wide:** > 0.04 secs (One small block)
- (ii) **Deep:** Usually > 4 mm in depth
- (iii) Q wave $> 25\%$ of height of that R wave. (Fig 1)

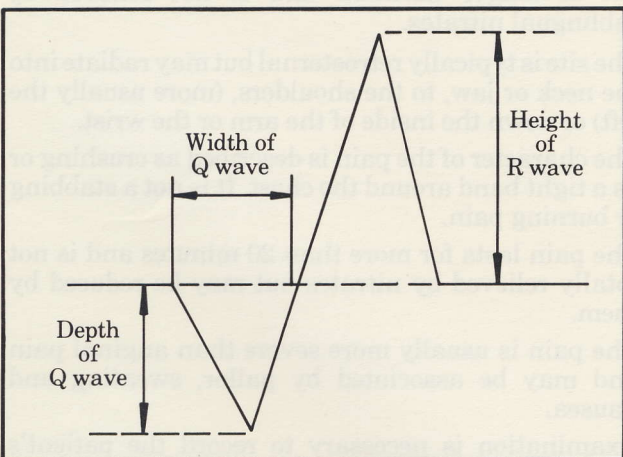


Figure 1

- (iv) not applicable in leads aVR and V1 and occasionally V2 where a QS complex is normally present
- (v) present in several leads. A Q wave may be present in aVL in a vertical heart as an isolated lead, and SIII may have a QS configuration in a horizontal heart. Repeating SIII in sustained inspiration will change this pattern if positional. Q waves may be absent in an infarction in the following cases:

- Subendocardial infarction
- A true posterior infarct
- When a left bundle branch block (LBBB) is present.

(b) Localisation of the infarction

For logical reasons MIs occur in the left ventricle (LV) only.

(i) Anterior wall infarction

The anterior surface of the LV faces the precordial leads and the anterolateral surface is reflected by leads aVL and S1.

- Anteroseptal infarction

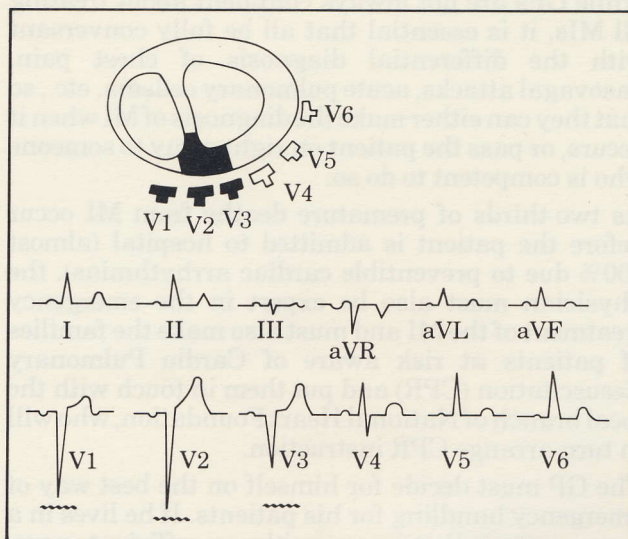


Figure 2 Anteroseptal infarct

Changes are noted in precordial leads V1, V2, V3 and maybe V4.

● Anterior infarction

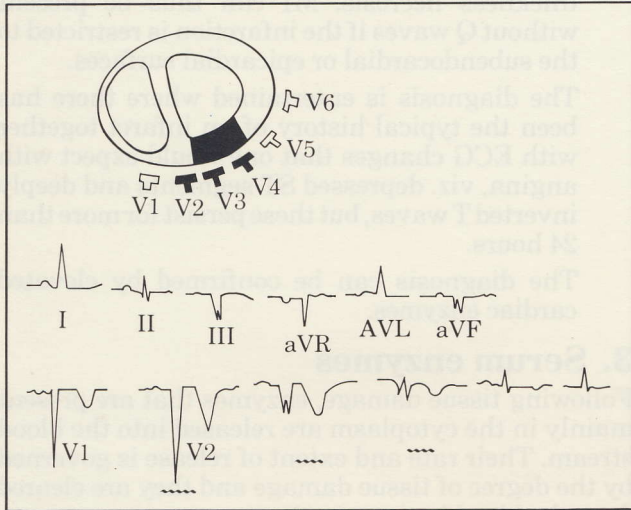


Figure 3 Anterior infarction

(Fig 3)
Would involve some of the more central precordial leads - possibly V2, V3, V4.

● Anterolateral infarction

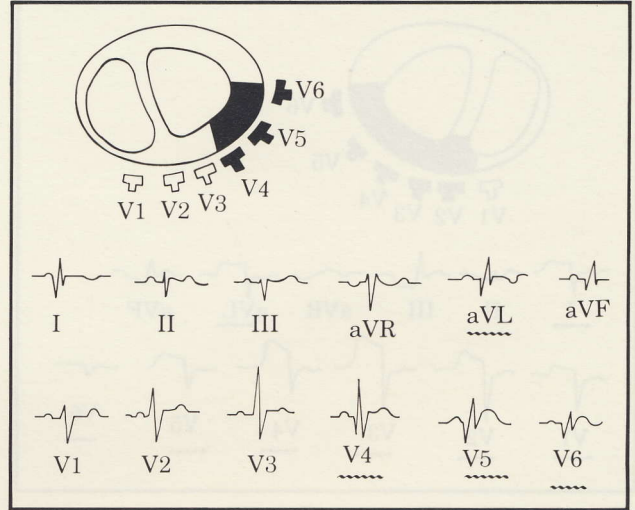


Figure 4 Anterolateral infarction

(Fig 4)
These changes are reflected in V4, V5, V6, SI, and aVL.

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● Extensive anterior infarction

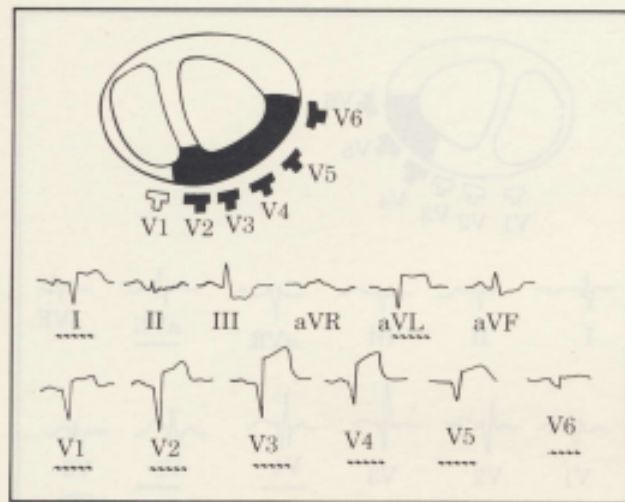


Figure 5 Extensive anterior infarction

Here SI and aVL are involved together with all the precordial leads V1 to V6.

(ii) Inferior infarction

This infarction is reflected in leads SII, SIII and aVF. There may be lateral or septal extension reflected by the appropriate precordial leads.

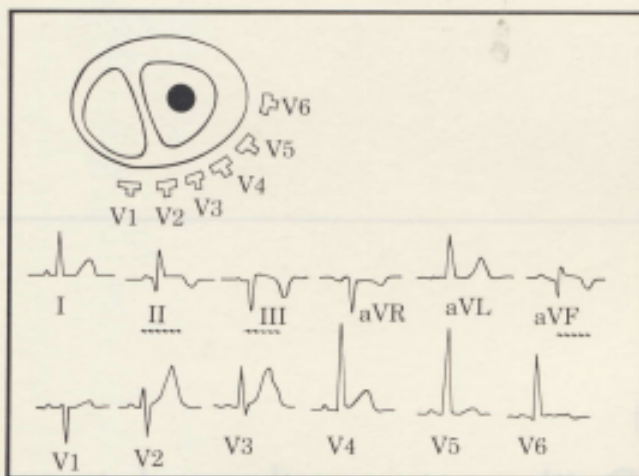


Figure 6 Inferior infarction

(iii) Posterior wall infarction

A true posterior infarct is very uncommon. As there are no leads directed to the posterior wall of the heart, the characteristic Q waves, ST elevation and inverted T waves are not present, but the diagnosis is made by reciprocal changes in precordial leads V1 and V2. Tall and slightly widened R waves are the reciprocal of the Q waves we would expect, ST depression is found in the acute stage and tall T waves are the equivalent of the inverted waves conventionally found.

(iv) Subendocardial infarction

Q waves are present where there has been full thickness necrosis. MI can thus be present without Q waves if the infarction is restricted to the subendocardial or epicardial surfaces.

The diagnosis is entertained where there has been the typical history of an infarct together with ECG changes that one would expect with angina, viz. depressed ST segments and deeply inverted T waves, but these persist for more than 24 hours.

The diagnosis can be confirmed by elevated cardiac enzymes.

3. Serum enzymes

Following tissue damage, enzymes that are present mainly in the cytoplasm are released into the blood stream. Their rate and extent of release is governed by the degree of tissue damage and they are cleared from the blood in time.

Enzymes are not released in angina as there is no tissue damage.

The measurement of enzymes is not always essential for the diagnosis of MI if the history is confirmed by specific ECG changes, but is very useful in cases of equivocal ECG changes, subendocardial infarctions, LBBB, for documenting extensions of an infarct and in case of a previous MI.

It is important to know when the cardiac enzymes are released and when they return to normal, otherwise money is wasted on unnecessary tests. It is also important to know what other reasons there might be for an elevation of the enzymes in interpreting the results.

Normal ranges vary with individual laboratories and so levels will be expressed here as times increased above normal. Normal levels should be included on the report from your laboratory. The degree of increase reflects the size of the infarct.

a) Creatine kinase (CK) (formerly Creatine phosphokinase) (CPK)

Increases in about 95% of MI, this is the enzyme that is released earliest, and the amplitude of the increase is greater than other enzymes. The disadvantages of this enzyme are:

- (i) return to normal levels early
- (ii) moderately increased levels may be due to
 - muscle injury (IM injections)
 - after surgery
 - physical exertion or defibrillation
 - hypothyroidism
- (iii) enzyme is unstable and specimens should be tested as soon as possible.

An isoenzyme CK, MB which is specific for myocardial muscle can be measured, but the test is expensive and is not done by smaller laboratories.

b) Aspartate transaminase (AST) (formerly SGOT)

This enzyme is present in many tissues, particularly

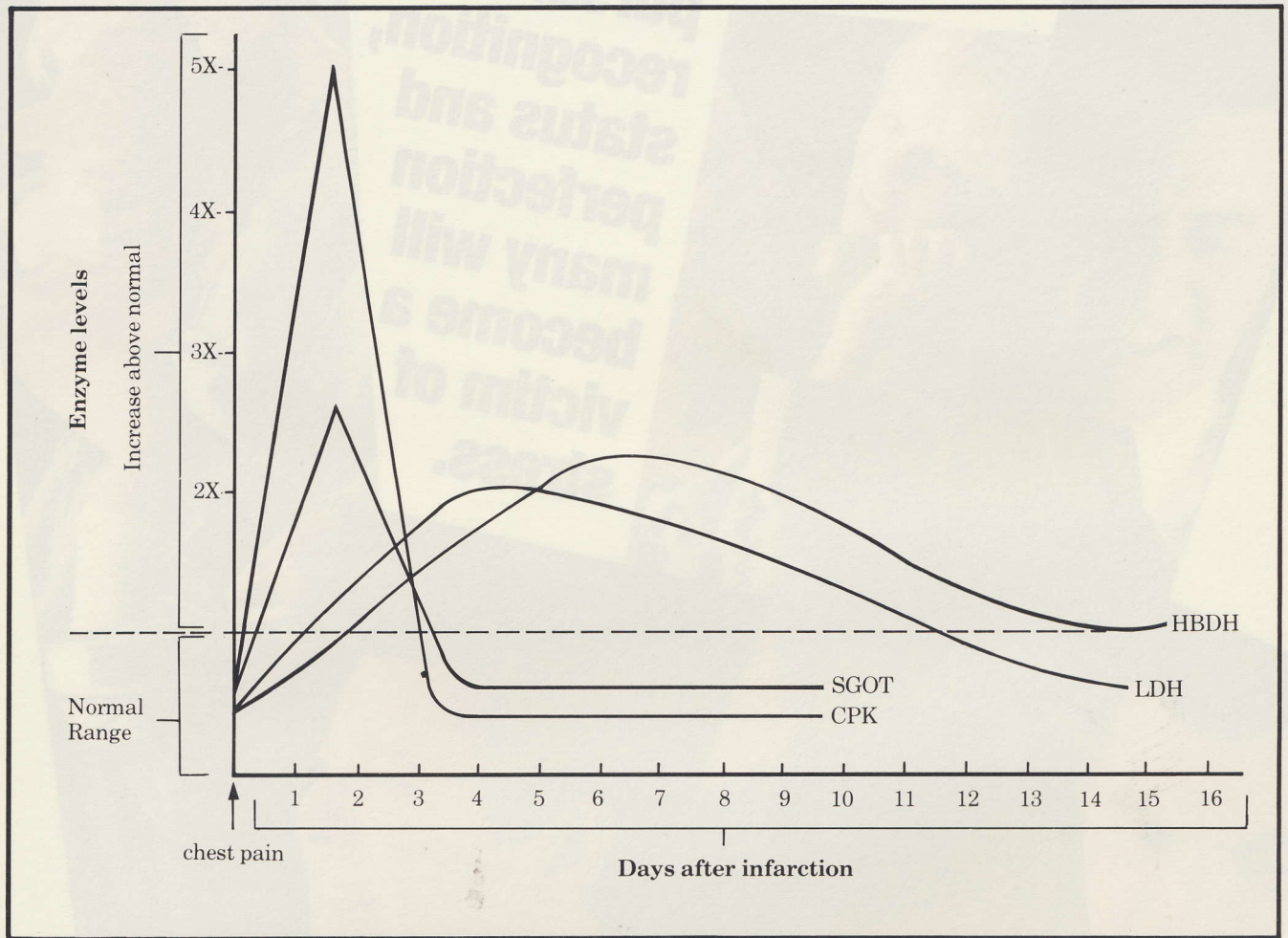


Figure 7 How serum enzyme levels change in myocardial infarction

liver, but less so in lung and skeletal muscle, so that it is less affected by IM injections. The time of release is similar to CK.

c) Lactic dehydrogenase (LDH)

Level of LDH is late to increase, but lasts for a long time in the blood stream, so is of value in detecting infarctions that occurred some days previously.

This enzyme is found in red blood cells, liver, and skeletal muscle as well as in the myocardial muscle so the test is less specific and influenced by haemolysis of the test sample. Pulmonary infarction also increases the enzyme level.

d) Hydroxybutyric dehydrogenase (HBD)

This enzyme parallels LDH but persists for a longer time in the blood. More specific than LDH.

Unless looking for a base line, the enzyme tests should be selected in relation to the time that has elapsed, since the possible MI. CK or AST is selected after 6-12 hours and up to 3 days. Later than that LDH or HBD is requested.

Rising levels of the enzymes are significant and tests should be asked for 24 hours apart.

Significant increases of two or three times normal are early indications of a moderate infarct. Slight increases might fall within the limits of laboratory error but possibly a 20% increase might be considered significant.

Enzyme	Time after infarction		
	Starts to rise (hours)	Peak elevation (hours)	Duration of rise (days)
CK	3 - 12	24 - 48	2 - 4
AST	6 - 24	24 - 48	3 - 6
LDH	12 - 36	48 - 96	7 - 12
HBD	12 - 36	48 - 96	12 - 15

Table 1

SUDDEN DEATH

Definition: Death occurring within 15 minutes as against "instantaneous" death which occurs within 30 seconds.

Myocardial infarction

Studies have indicated that instantaneous death is probably a primary electrical event, but that sudden death results from an acutely evolving arterial obstruction superimposed on a chronic stenotic plaque. The resulting myocardial ischaemia results in electro-physical changes leading to ventricular fibrillation (VF).

Resuscitation studies have shown that in 90% of cases of "sudden death" the arrhythmia is VF. Resuscitation of cases of VF, both inside and outside hospital, commonly lead to long-term survival.

Accent has to be directed towards training the public and paramedics in CPR and ensuring that all doctors, whatever their line of work is, are expert at CPR and defibrillation.

References

1. Levenstein JH The role of the General Practitioner in the early management of Acute Myocardial Infarction. S Afr Med J 1971; 45:1103-xx
2. Levenstein JH Emergency Management of Acute Myocardial Infarction by the General Practitioner. S Afr Med J 1976; 50: 531-xx

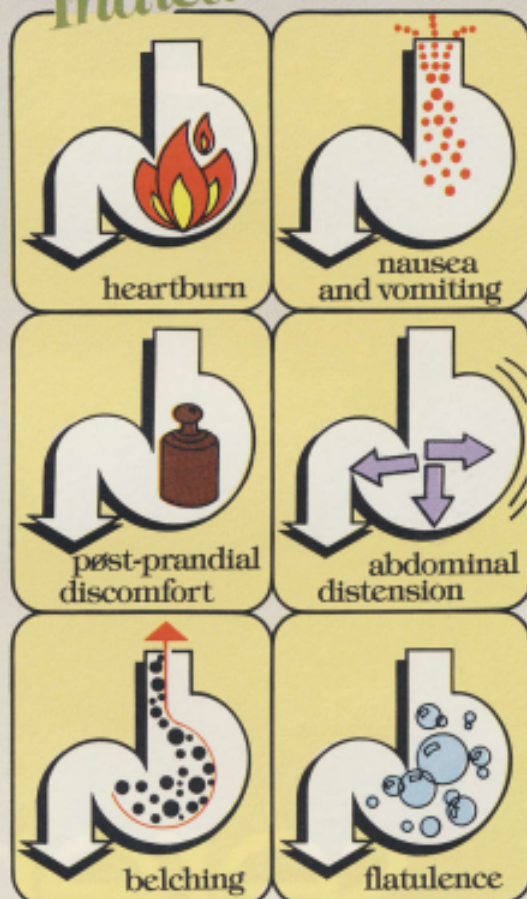
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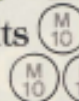
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Dosage

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children



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