

Coronary heart disease – a review

– EL Murray



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Curriculum vitae

DR ELLIOT MURRAY was born in Durban in 1931. He obtained a degree in Civil Engineering from the University of Natal in 1954. He married in 1954 and moved to East London before going to Cape Town where he qualified in medicine in 1961. After a house job at Edendale hospital he went into general practice in East London where he has remained. He has been interested in post-graduate education for GPs and obtained the MFGP in 1976 and the MPraxMed, from Pretoria in 1977. At present he is a MASA Branch Councillor, a Rotarian, Chairman Border Branch of Heart Foundation and part time Medical Officer at Frere Hospital.

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Summary

This article deals with the role and responsibility of the general practitioner with coronary heart disease patients. It covers the following: anatomy of the coronary arteries, pathology, predisposing factors and prevention, syndromes of coronary heart disease, and treatment. This is the first of a three-part article.

Definition

Impairment of heart function due to inadequate blood flow to the heart muscle, usually caused by obstructive changes in the coronary arteries due to arteriosclerosis. Other rare causes include syphilis, embolism, rheumatism, collagen diseases, trauma, infection, etc., and are not dealt with.

Introduction

Coronary heart disease is a very important part of a primary care physician or general practitioner's (GP's) practice.

It is important because of the loss of life, morbidity and cost to the country affecting as it does the man in the most productive stage of his life. It is a very common disease which has a very dramatic impact on patients and their families. The mortality of CHD in white South Africans exceeds the combined mortality of malignant disease and road accidents.

Our responsibility lies in:

1. Prevention

This is fairly and squarely placed on the GP's shoulders as he alone sees the patient frequently and over such a wide age span. Prevention starts in screening first-degree relatives of all CHD patients and in educating the public at large.

2. Diagnosis

It is the responsibility of the GP to correctly diagnose chest pain so that we strike the correct balance between diagnosing CHD and not making non-sufferers cardiac cripples. Here we often need the help of our physician colleagues.

3. Treatment

Emergency treatment of Myocardial Infarctions (MI) is the GP's responsibility. The treatment of all CHD often has to be shared with our specialist colleagues, especially so with the greatly improved results achieved in the Intensive Coronary Care Unit (ICCU) in the treatment of MIs and the surgical treatment of angina by coronary artery bypass grafting (CABG), or percutaneous transluminal coronary angioplasty (PTCA), when necessary. Because of improved technology and different professional and financial interests in the various methods of treatment, the GP is essential to the patient, being able to orchestrate treatment and give unbiased advice.

4. Rehabilitation

Returning patients who have had MI or CABG to as effective and as full a life as their disability allows by restoring confidence more than anything else, is a skill that the GP can learn very easily, getting expert advice where necessary. Stress testing of the uncomplicated MI is a specialist skill, but the organisation and running of a rehabilitation programme can be very readily done by GPs.

5. Example

It is our responsibility to the community to set an example to others by reducing the risk factors in our own lives and by following a healthy life style.

This article will be dealing with the subject from a GP's point of involvement and will cover the following topics:

- (a) Anatomy of the coronary arteries.
- (b) Pathology of CHD.
- (c) Predisposing factors to arteriosclerosis and prevention strategies.
- (d) Syndromes of CHD.
- (e) Treatment.

a. Anatomy

There are three coronary arteries arising via two trunks from the aorta immediately above the aortic valve. (Fig 1.)

The right coronary artery arises from the right coronary sinus of aortic valve and passes down in the right atrioventricular groove. When it reaches the diaphragmatic surface it turns to run posteriorly where it eventually anastomoses with the circumflex branch of the left coronary artery. Branches of the right coronary artery supply the right ventricle and also run down the posterior interventricular groove to the apex, providing an important supply to inferior portion of septum and inferior aspect of left ventricle.

The left coronary artery mainstem branches into the left anterior descending artery and the circumflex artery. The left anterior descending artery

passes down the anterior interventricular groove until it reaches the apex. Branches of this vessel supply both ventricles.

The circumflex artery in the atrioventricular groove passes over to the back of the heart where branches supply the lateral wall of the left ventricle.

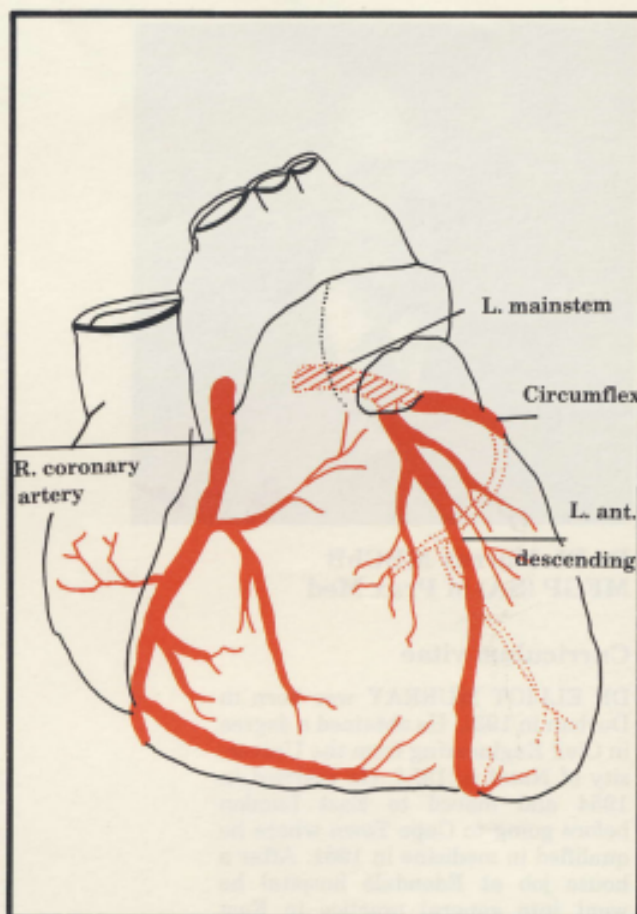


Figure 1

b. Pathology

The cause of CHD is, for practical purposes, due to atherosclerosis of the coronary arteries. There may be superimposed thrombosis, and arterial spasm also plays a part.

The vessels are usually 70% obstructed before symptoms develop. As the heart is an exercising muscle without rest, the oxygen extraction is far greater than for any other organ in the body.

c. Causes of atherosclerosis

The risk factors have been widely known for some time, and it is our duty as GPs to look for them in our patients in preventative medicine, and to correct them to the best of our ability. Steps taken to prevent or delay the formation of atherosclerosis should begin early in life, long before there is any suspicion of CHD.

South Africa leads the world in the incidence of CHD among the whites – a poor state of affairs. The death rate from CHD amongst white South African men is 2.5 times higher in the younger age group than that in the USA!

More white South Africans die of coronary heart disease than of carcinoma and road accidents combined

The factors to consider in prevention of premature atherosclerosis.

- (i) Elevated cholesterol
- (ii) Hypertension
- (iii) Cigarette smoking
- (iv) Diabetes mellitus
- (v) Physical inactivity
- (vi) Obesity
- (vii) Emotional stress
- (viii) Oral contraceptives.

(i) Cholesterol

This is the most important of our risk factors.

There are two types of cholesterol in the blood vessels.

- A high density lipoprotein (HDL) which makes up about 20-30 % of the total cholesterol (TC) and it is strongly and inversely related to individual risk of CHD.
- Low density lipoprotein (LDL), which is the main source of cholesterol in the atherosclerotic plaques.

Because of genetic differences, both favourable and unfavourable blood level of TC may not be related directly to the amount of cholesterol in the diet.

Statistically populations in whom CHD is common have a TC level which is relatively high (i.e. greater than 5,17 mmol/l) and conversely, if the TC level is low the incidence of CHD is also low.

The USA has managed to reduce its TC level in middle-aged men from 6,09 mmol/l in the 1950s and early 1960s to 5,44 as at 1975-76. Their incidence of CHD is still relatively high, but is being reduced by about 3 % per annum.

Populations with a level of 4,14 mmol/l manifest little or no CHD. To strike a level between good nutrition and good CHD risk rate we need to reduce our 'normals' to between these figures. It is felt that the laboratory normals are too high and that the following levels should be regarded as the maximum levels in mmol/litre.

Age	15-24	25-34	35-44	45-54	55-64
Male	4,10	5,00	5,52	5,68	5,75
Female	4,51	4,94	5,31	5,93	6,56

Dietary changes that are necessary to achieve a reduction are

- a reduction in saturated fat and dietary cholesterol
- an increase in complex carbohydrate consumption
- avoidance or correction of overmass
- a reduction of cholesterol intake to below 300 mg per day for an adult.

The value of exercise is illustrated by the Multiple Risk Factor Intervention Trial in the USA, which found that with a weight loss of as little as 3-4 kg a substantially greater reduction in TC is achieved and maintained, than that derived by change in dietary composition. The LDL cholesterol is reduced, the triglycerides fall and HDL cholesterol rises.

(ii) Blood Pressure (BP)

There is a correlation between elevated BP and CHD. There are enough other reasons to reduce the diastolic BP.

(iii) Smoking

The health hazards of cigarette smoking in adults and young people have been clearly documented.

There is no justification for smoking at all, even though the exact mechanism in increasing CHD risk is not known.

Smoking interacts with other CHD risk factors so that even light smoking may carry a significant risk.

It is disquieting to note how many young doctors and nurses are smoking cigarettes. It is our duty to stop smoking in our patients and our friends by banning smoking in our surgeries, hospitals and homes and thereby letting smokers know that they are undesirable people.

Most smokers will report that they smoke to relax. This is the result of good marketing by tobacco companies.

Inhalation of cigarette smoke causes tachycardia, hardly conducive to relaxation! Cigarette smoking also reduces HDL, constricts the coronary arteries together with other arteries and converts 5-10 % of haemoglobin to carboxy-haemoglobin, and nicotine elevates BP.

The good news for cigarette smokers is that if they give up their habit they will halve their risk factor in a year and this risk will be back to that of a non-smoker in 10 years.

(iv) *Diabetes mellitus*

There is at least a two-fold increase in the incidence of MI in diabetics compared to non-diabetics. The literature is unclear as to the effect of control of diabetes on the large vessel complications of diabetes, including CHD.

The prevention of non-insulin dependent diabetes requires the control and prevention of obesity.

(v) *Physical Activity*

Running does not make one immortal.

The effect of isotonic exercises (exercise associated with little physical effort but a large range of movement, as in running, walking, swimming, etc which increase pulse rate with little increase in systolic BP) appears to increase HDL, reduce body weight and reduce body lipids.

Hellerstein showed that physical exercise in a rehabilitation programme provided the following:

a) Improvement of subjective well-being and one

therefore takes better care of oneself viz. diet, smoking, etc., and decrease of psychological depression

- b) Marked improvement of aerobic power and cardiovascular haemodynamics (increased stroke volume, decreased tension time index and increased vigour of heart beat)
- c) Decreased lactate production for the same work load
- d) Reduction of a raised BP during exercise and of peripheral vascular resistance
- e) Diminution of heart rate at rest, during sleep and during exercise
- f) Quantitative lessening of the ST segment displacement during exercise
- g) Lowering of serum lipid levels
- h) Reduction of adipose tissue
- i) Enhanced oxygen extraction by the peripheral tissues
- j) Suggestive reduction in mortality
- k) Enhancement of inter coronary collaterals rarely demonstrated by coronary angiograms
- l) Partial or complete normalisation of pretraining pathological ballistograms

**BY DIE LANG-
TERMYN-BEHEER
EN STABILISERING
VAN ANGINA,**

**OM U VERTROUE
TE WEN MOES
ONS TWEE
DINGE DOEN...**

m) Enhancement of sexual activity.

It is reasonable to assume that most of the above would happen to normal people as well.

In order to obtain the optimum cardiovascular benefit from exercise, the heart rate should be raised to 180 minus the age in years, to get the beats per minute to which it should be increased for 20 minutes, 3 times a week.

In starting to exercise from an unfit state a suggestion is to get the person to measure out a 3 km circuit and to walk that distance at a leisurely pace. Provided there is no discomfort during the exercise, or the next day, the rate of walking is increased until the person is walking at a brisk rate. He can then start jogging. He jogs for 100 metres and then walks for some 400 metres and repeats this cycle. He then increases the running and reduces the walking until he can run the whole 3 km.

Increasing the amount of exercise is limited by not feeling uncomfortable during the exercise, being able to talk while jogging, not exceeding the pre-de-

termined pulse rate and having a pulse rate not higher than 100, ten minutes after the effort.

The limiting factor to exercise is usually the muscles of the legs and these in particular should be stretched before and after the exercise as described in Tim Noakes' *Lore of Running*.

Provided the person runs "as a child at play and not as a man at work", he will be deriving cardiovascular protection plus a whole lot more.

The above recommendations are for normal people and not CHD patients.

(vi) *Obesity*

Obesity is associated with hypertension (HT), elevated blood lipids, increased incidence of diabetes and a sedentary life style. All of these factors are linked with a higher incidence of CHD.

(vii) *Emotional stress*

The Type-A behaviour pattern associated with competitive drive, hostility, and a sense of urgency or

SÊ WAT ONS BEDOEL

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[adalat[®] nw.-tasie suf], Die Bayer-woord vir die beheer en stabilisering van angina. Adalatasie word verkry deur die gebruik van Adalat as profilaktiese behandeling, òf alleen òf in kombinasie met ander vorms van hartmedikasie. a'damsappel. Beweeglike, uitstekende die strottehoof; keelknop; komha

Adalat is beskikbaar in 5 mg en 10 mg kapsules, wat die aanpasbaarheid van dosistitrasie bied. Aanbevole aanvangsdosis: 10 mg drie maal per dag.

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impatience is moderately associated with future CHD risk. Stress is a difficult component to measure, but we have all had the A type patient without other risk factors who has suffered the consequences of CHD.

Modification of this personality trait is being worked on and it will be interesting to see the results.

(viii) Oral contraceptives

Within high-risk populations, (women with hypertension, diabetes, hyperlipidaemia, etc.) oral contraceptive use is associated with increased risk of CHD, with high blood pressure and TC levels. The risk is compounded by cigarette smoking. In high-CHD societies the risk might be reduced by a more selective use of oral contraceptives, based on medical supervision and prescription.

I. Angina

Angina is the symptom that occurs during myocardial ischaemia and is invariably a manifestation of coronary artery disease.

The diagnosis of angina may be very easy when it is the characteristic crushing, substernal pain brought on by a fixed amount of exertion and relieved by rest in about 10 minutes and relieved faster by sublingual nitrates.

Angina may be brought on by mental stress and may come on at rest. The site of the pain experienced by the patient might be the arms, neck or jaw without any central chest pain. The pain might not be described as crushing, but burning, 'like indigestion'.

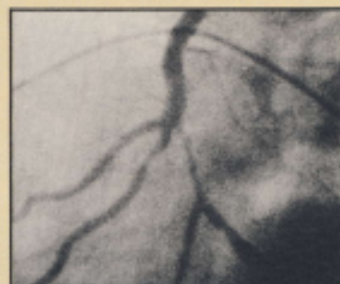
There are tests that can be performed to help with the diagnosis, but usually a typical history of classic angina is the best way of making a diagnosis.

NB: Normal resting ECG does not exclude angina. 'Believe the history not the ECG'.

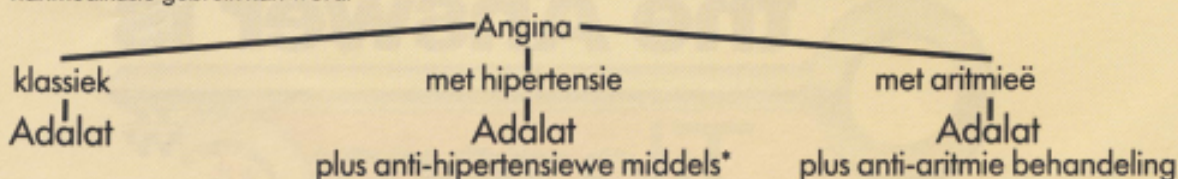
The myocardium extracts 70 % of available oxygen from the blood supply during normal circumstances. (cf. 30 % by skeletal muscle). The heart never rests, and is unable to function anaerobically, thus tolerates anoxia badly. Flow in the coronary arteries

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occurs mainly in diastole when the tension in the ventricles is reduced, so is affected by

- Coronary artery disease in the arteries
- Aortic diastolic pressure
- Length of diastole
- Transient impairment of blood flow due to spasm or plugging.

TYPES OF ANGINA

1. Chronic stable angina
2. Unstable angina or Crescendo angina.
 - a) Recent onset angina
 - b) Progressive angina
 - c) Angina decubitus
 - d) Acute coronary insufficiency
 - e) Post infarct angina
 - f) Variant (Prinzmetal) angina.

1. Chronic stable angina

Here the angina has been present for some time and is precipitated by the same factors and is predictable.

The quality of the pain is usually described as the tight band, etc., which usually develops in the chest, but might be a pain or discomfort that radiates up into the neck or jaw, down the medial aspects of the arms (usually the left). It might manifest as a pain in the elbow or jaw without the chest component. The pain is relieved by rest or nitrates within a few minutes.

2. Unstable angina

Unstable angina refers to a variety of presentations which place the patient at increased risk of potential infarction or sudden death.

a) *Recent onset angina.* Any angina that has occurred of recent onset (arbitrarily 2 weeks) is regarded

Prevention starts in screening, first degree relatives of all coronary heart disease patients

as being unstable because it may progress rapidly. Everyone has to have recent onset angina at some stage, so we limit it to recent onset and severe angina.

b) *Progressive angina.* Describes angina which was stable but is now increasing in severity and is brought on by less exertion than usual and lasts longer; may even come on at rest.

c) *Angina decubitus.* Occurs when the patient lies down and is resting. May even wake him from sleep.

d) *Acute coronary insufficiency.* (Acute Medical

Emergency). Also known as pre-infarction angina or the intermediate coronary syndrome. The episodes of pain last for longer than the usual 10 minutes, but there is no ECG or enzyme evidence of infarction. Often ECG evidence of ischaemia is indistinguishable from sub-endocardial or non-transmural infarction until enzymes are available.

e) *Post infarction angina.* Angina which persists or recurs within 3 to 6 weeks after documented infarction, is also potentially dangerous and considered as unstable.

f) *Variant angina.* Prinzmetal angina has the characteristic feature of ST elevation during the episode of pain which may come on at rest and even when the patient is sleeping. This is due to coronary artery spasm causing transmural ischaemia instead of the usual sub-endocardial ischaemia which produces the ST depression, characteristic of angina.

The above classification is for the splitters. It is probably better to regard them all as simply unstable angina as the advocated management is the same at present.

Distinction is semantic: unstable agina =
DANGER, therefore DO SOMETHING!

DIAGNOSIS OF ANGINA

The *history is the most important part* of making the diagnosis of angina. It is, however, reassuring to have some documented evidence to support the diagnosis.

Prevention of coronary heart disease is primarily the task of the GP

The physical examination is not usually of help in making the diagnosis, but more in looking for causes of the angina, viz. hypertension, cardiac failure, valvular lesions, anaemia, thyroid abnormalities, obesity, smoking and hypercholesterolaemia (arcus, xanthomata, xanthelasma).

SPECIAL TESTS

Electrocardiographic interpretation is an empirical process. An 'abnormality' is defined as such because it is not generally found in normal healthy subjects. Each abnormal criterion will have a greater or lesser degree of *sensitivity* and *specificity*.

Sensitivity is defined as the number of true positive detections divided by the number of positives in the group tested.

A 100 % sensitive test would pick up every case of the abnormality tested for in a community, i.e. it is an index of the capability of a test to detect an abnormality.

Specificity is defined as the number of true normals detected, divided by the total number of normals in the group tested. A 100 % specific test would guarantee that only normals are detected.

In practice no ECG criterion is 100 % specific or 100 % sensitive and the greater the number of criteria fulfilled the greater the confidence of the diagnosis.

ECG changes

If an ECG is done while the patient is having an ischaemic attack changes may be seen in the ST-segment and the T-wave.

ST-segment

The ST-segment may change in

- (a) Shape
- (b) Depression

South Africa leads the world in the incidence of coronary heart disease among whites

Shape – The normal ST-segment merges imperceptibly with the upstroke of the T-wave. In myocardial ischaemia, one of the earliest signs is an alteration in the shape of this junction giving a sharp angled ST-T-junction which gives the appearance of a more horizontal ST-segment.

Depression – Sub-endocardial ischaemia results in a depression of the ST-segment. This depression should be horizontal, sagging or down-sloping. Junctional ST-depression may be significant and depression is measured at the point 0,08 secs., beyond the J-point (two small blocks away). (Fig 2)

Other causes of ST-depression must be borne in mind, viz. ventricular hypertrophy, intraventricular conduction problems, including Wolff-Parkinson-White syndrome (WPW), cardiomyopathy, myocarditis, digitalis, hyperkalaemia and pericarditis.

As noted earlier, variant angina produces transient (up to 20 minutes) elevated ST-segments, together with tall and widened T-waves and increased ventricular activation time. These are all features of the acute phase of a myocardial infarction (MI), but in

this case the condition lasts for hours or even days. ST-segt elevation = transmural ischaemia while ST depression = sub-endocardial ischaemia.

The T-wave

The T-wave is the most unstable component of the ECG recording, changes occurring with hyperventilation, heavy metals, anxiety, smoking, drinking iced water, changes in body position and decrease in BP; there are approximately 40 causes of T-wave changes.

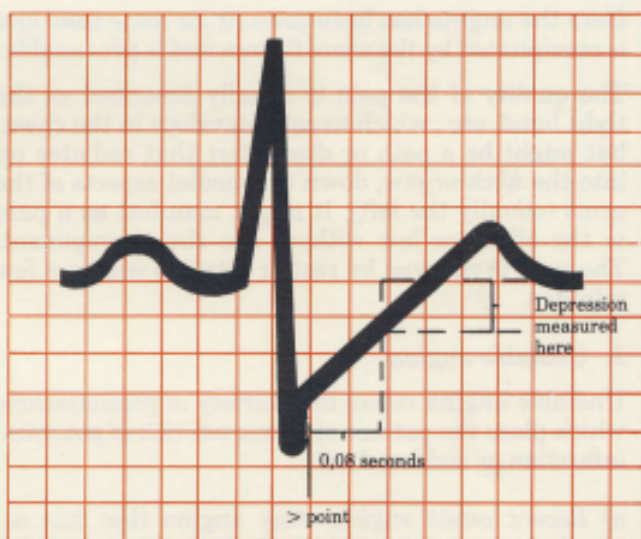


Figure 2

Despite this certain changes are suggestive of ischaemia, but only symmetrical T-waves – upright or inverted are hard indications (The normal T-wave is asymmetrical.)

T-waves that are chronically inverted in a patient with CHD occasionally become upright or 'normalised' during a further ischaemic episode so this 'normalisation' is almost always indicative of myocardial ischaemia.

Emergency treatment of MI is the responsibility of the GP

The mean frontal plane QRS-axis and the mean frontal plane T-wave axis are usually similarly directed. In the presence of myocardial ischaemia

the T-wave axis tends to deviate whereas QRS-axis usually remains normally directed or may even deviate in the opposite direction. If this angle exceeds 45° it is usually a sign of myocardial disease.

Exercise testing

As stated earlier a good history is the best way of diagnosing angina, but the pain may be atypical and the history doubtful. In these cases objective evidence is useful and an ECG is recorded with the myocardium stressed.

There are 2 methods available. The one is using a Master's Step Test in our rooms or the other is to have a formal graded exercise test using a treadmill or bicycle ergometer.

(i) Master's step test

In this test the patient makes a specific number of trips over a standard apparatus according to sex, weight and age table in 1½ minutes after having had a normal resting ECG. The test is symptom limited in that the patient stops the test immediately he feels any discomfort or giddiness. The ECG is then recorded immediately the test is stopped and at specified intervals. The protocol is available from any life assurance office.

The disadvantage of this test as performed in our rooms are:

1. We cannot watch the ECG changes while the patient is exercising, thus potentially dangerous ST-segment changes or arrhythmias may be missed.

Vessels must be 50 % obstructed before any symptoms develop

2. There is a delay in getting the patient connected up to the ECG machine before recording the stat tracing.
3. The heart rate response is usually inadequate for the test to be sufficiently sensitive.

(ii) Graded exercise test

We use a treadmill (as everyone knows how to walk) and the Bruce Protocol where the speed and elevation of the treadmill are increased at 3-minute intervals. The testing is done with a doctor present and with resuscitation drugs and a defibrillator ready. The patient should not be on digitalis or anti-anginal drugs if a diagnosis of ischaemia is suspected.

The patient is questioned and examined to ensure that he is not in congestive cardiac failure (CCF), is not excessively hypertensive, had a MI in the

preceding 9 weeks or that he has unstable angina, aortic stenosis or HOCM.

After a normal resting ECG the patient is exercised on the treadmill. V5 is monitored on a screen throughout the test as this is the lead most likely to show ischaemic changes. At three minute intervals (or whenever required) the BP and heart rate are recorded before increasing the speed of the treadmill and a full 12-channel ECG recorded.

The test is terminated if the patient develops symptoms, at his request, if the doctor is unhappy about the patient's performance or appearance, the BP fails to increase with increasing work load, the patient develops a worrying arrhythmia, achieves the target heart rate, or has 2 mm or more of ST deviation.

Run as a child at play and not as a man at work

The full 12-channel ECG is again recorded immediately the test is terminated and repeated as soon as he lies down and again at 3 minutes and 6 minutes, or until it reverts to normal.

The target heart rate is 90 % of the maximal heart rate. Maximum heart rate is taken from tables (roughly 220 minus age of patient). By using this high heart rate we increase the specificity of the test.

Positive test

- (a) Any ST-deviation associated with anginal chest pain (not a typical chest pain)
- (b) ST deviations ≥ 2 mm as measured at J-point plus 0,08 secs.

In 10 % of cases the positive changes are seen only while the patient is exercising and disappears as soon as exercise is terminated. In order to record this expensive recording equipment is needed.

In about 5 % of cases the ST-changes only occur after the termination of the test, reinforcing the need for recurring ECG after the exercise.

ST deviation is normally (98 %) depression, but elevation is important as it usually indicates severe stenosis in major proximal branches of the coronary arteries and transmural ischaemia and not sub-endocardial ischaemia.

Isolated T-wave alterations in direction or morphology during or after exercise are considered to be of no diagnostic importance and most exercise laboratories pay no attention to alterations in T-waves for the diagnosis of coronary artery disease.

Coronary heart disease – Part 1

CORONARY ANGIOGRAPHY

It is not practical to undertake coronary angiography in every patient judged to have a high probability of coronary artery disease merely to confirm the diagnosis. Angiography is essential for the pre-operative evaluation of patients being considered for bypass surgery.

Patients with severe symptoms not relieved by antianginal drugs need angiography, as do those with triple vessel and left main artery disease. These will usually be detected by the presence of strongly positive (2 mm ST dep.) effort test. All patients with such effort stress tests should undergo angiography.

A poor exercise tolerance, exaggerated ST-segment depression and a failure of systolic BP to rise with increasing work load are indicative of poor myocardial function, usually the result of extensive coronary artery disease, and predict a poor prognosis if untreated.

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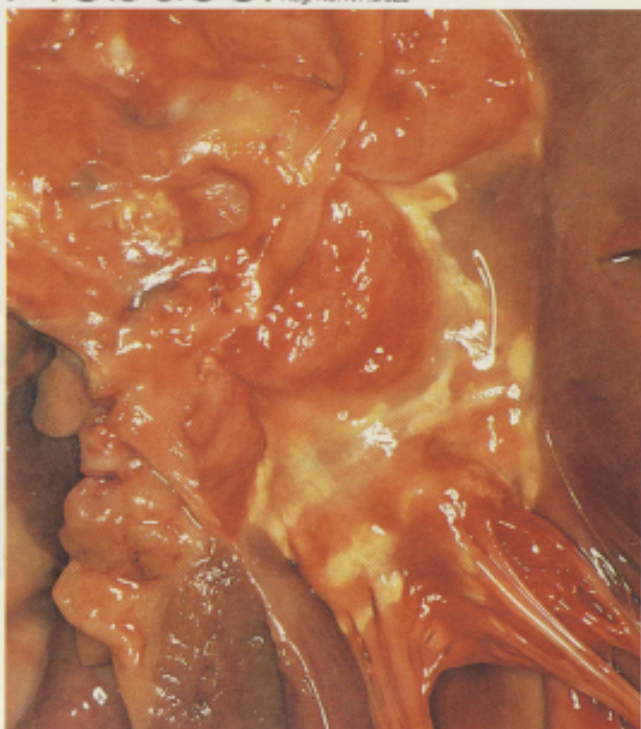
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