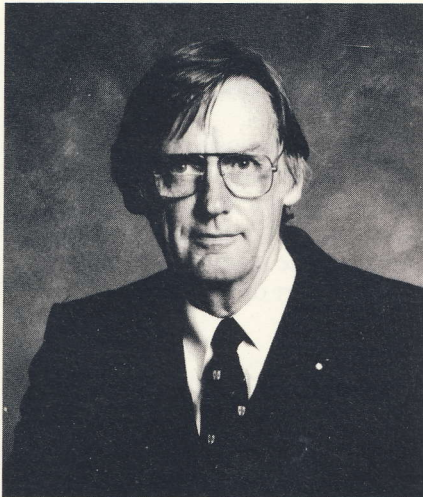


Angina Pectoris – A general practice review: Part I – EL Murray



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Curriculum vitae

Elliot Murray was born in Durban in 1931. He obtained a degree in Civil Engineering from the University of Natal in 1954. He got married in 1953, moved to East London for a while 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. Elliot 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 member of the Academy Council and Regional Director of Vocational Training in the East London/Ciskei area.

Summary

The author highlights certain aspects of angina pectoris after a series of workshops on this subject. The GP needs to diagnose carefully stable and unstable angina pectoris, two different conditions, and refer the latter. The different actions of the three calcium antagonists must be recognised as well as the limits of the drugs and their interactions. He discusses the perspective of risk factors and concludes with a section on silent myocardial ischaemia.

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KEYWORDS:

Angina Pectoris; Angina, unstable; Drug therapy; Ischaemia

This paper is the result of a series of workshops that the Academy of Family Practice/Primary Care has been conducting over the last year. The workshops were sponsored by Bayer Miles (Pty) Ltd to whom the Academy and I are very grateful.

The workshop format was used to discuss various points and I included my views as a handout and not a definitive text.

The points that I tried to highlight were:

1. stable and unstable angina pectoris are two different conditions; the former a general practitioner (GP) can usually adequately handle but the latter needing referral.
2. the three calcium antagonists that are presently available to us, have

different actions and we must recognise these differences.

3. to illustrate the limits of the drugs and their interactions and where the patient would benefit from revascularisation procedures.
4. to get the concept of risk factors into perspective, and
5. to give a brief summary of silent myocardial ischaemia.

Angina

Ischaemic heart disease is the result of an imbalance between the oxygen supply to and the oxygen demand by the myocardial muscle.

Angina is the discomfort which may or may not result from this, and this is the subject of this paper.

In 1768 Heberden described angina graphically as:

“There is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned at length. The seat of it and sense of strangling and anxiety with which it is attended, may make it not improperly be called angina pectoris. Those who are afflicted with it are seized while they are walking, and most particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast which seems as if it would take their life away, if it were to increase or continue; the moment they stand still all this uneasiness vanishes.” The only omission possibly being the sweating that may accompany the episode.

However, in one trial, of 77 patients who all fitted this picture perfectly,

... Angina Pectoris

the discomfort was found to have an oesophageal origin in one fifth of the patients.

On the other side of the coin, the perceived wisdom is that the majority of patients suffering a sudden death, had seen their doctors in the previous four to six weeks.

Stable angina pectoris and unstable angina pectoris are two different conditions

However, the Framingham study which admitted asymptomatic men aged 30 to 60 to the trial and then followed them through, found those who eventually had symptoms of ischaemic heart disease, the first manifestation of that was

Acute Myocardial Infarction	42%
Sudden death	13%
Unstable angina	7%
Stable angina	38%

Angina can vary from the classical symptoms as described by Heberden and the text books through to an atypical form of angina which might manifest only as a momentary attack of dyspnoea.

We as primary care physicians must not miss the diagnosis, but also must not make the wrong diagnosis and thereby create cardiac cripples.

Types of Angina

There are basically only two types of angina:

(a) *Stable Angina*

with a relatively good prognosis

(b) *Unstable Angina*

with a much worse prognosis.

(a) *Stable Angina Pectoris (SAP)*

Is due to a fixed narrowing in the epicardial arteries so that the same amount of exercise, emotion or external stimuli will cause the same degree of pain.

There is sometimes a slight variation in the degree of discomfort or the ease with which it starts due to the circadian rhythm* or due to the warming up phenomenon. Stable angina has an annual mortality of about 2-3%.

(b) *Unstable Angina Pectoris (UAP)*

Might manifest in one of the following forms:

- i) Significant angina of recent onset.
- ii) Angina at rest
- iii) Crescendo angina - pain worse or on lesser provocation.
- iv) Angina after recent myocardial infarction.

This form of angina has a much worse prognosis and in the first year 15% end up in death or a non fatal infarction.

Prinzmetal's Variant Angina

Only presents as angina at rest. It is never stimulated by effort and during an attack the ST segment on ECG is elevated. It is uncommon but glamorous, and I think it should be

*(Circadian rhythm: The body's metabolism is slowest in the morning. The patient will suffer more angina in the early morning and the majority of cardiac incidents occur in the first four hours of wakening)

included under unstable angina because it is associated with a high incidence of cardiac arrhythmias.

Making the diagnosis of angina is not always easy. When the history is characteristic, the story is usually all that is required to make the correct diagnosis. The history is the most important part of making the diagnosis.

For reference the characteristics of the history are listed below:

Pain

Precipitating factors

Exercise

Effort which involves use of arms above head

Cold environment

Walking against the wind

Exercise after a large meal

Emotional factors

Fright, anger

Coitus

We need to recognise that the calcium antagonists available to us, have different actions

Quality

Sensation of pressure or heavy weight on chest

Burning sensation

Feeling of tightness

Short of breath with feeling or constriction above larynx or upper trachea

... Angina Pectoris

Visceral quality (deep, heavy, squeezing, aching)

Gradual increase in intensity followed by gradual fading away.

Location

Over sternum or near it

Anywhere between epigastrium and pharynx

Occasionally limited to L shoulder or L arm

Rarely limited to Rt arm

Limited to lower jaw

Lower cervical or upper thoracic spine

Left interscapular area

Radiation

Medial aspect of L arm

L shoulder

Jaw

Occasionally Rt arm

Duration

30 seconds to 30 minutes.

Most patients who suffered a sudden death had seen their doctors 4-6 weeks previously

Nitrate relief

Relief within 45 seconds to five minutes after taking sublingual nitrate.

In the examination, precipitating and complicating diseases should be

excluded. These are hypertension, hyperlipidaemia, anaemia, diabetes, peripheral vascular disease, aortic valvular disease, thyrotoxicosis, hypertrophic cardiomyopathy (HOCM), cardiac failure (CCF) and tachyarrhythmias.

... an imbalance between oxygen demand and oxygen supply by the myocardial muscle

The examination during an attack may reveal a non specific systolic murmur or a third or fourth heart sound. It is important to exclude other conditions, such as Barlows mitral valve prolapse and pericarditis.

Special examinations are done to confirm the diagnosis or used to help in unsure cases. The ECG is often normal at rest, but look for left ventricular hypertrophy or evidence of previous infarctions.

The Exercise Stress Test (EST) is used wherever possible to confirm the diagnosis or to judge the severity of the case, but is contraindicated in UAP. We all have a Masters staircase or a kitchen chair in our rooms for insurance purposes and we all resort to it in trying to confirm the diagnosis. The insurance physicians have great skill in interpreting the results to predict a prognosis, but I am having increasing difficulty in placing reliance on this test.

You cannot monitor what is happening to the heart during the exercise. We stop when the patient looks pale, probably due to a drop in BP, starts sweating or complains of

chest pain. It takes time to connect the patient up to the ECG at the end of the exercise. If the patient has ST depressions without chest pain, could this be silent myocardial ischaemia?

An EST done on a treadmill or a bicycle ergometer gives so much more information and the patient can be monitored for ST deviations, arrhythmias and for a blood pressure (BP) elevation during exercise. If the BP does not rise it may be an indication of left ventricular dysfunction. To eliminate interference requires a special ECG recorder and expensive electrodes, but you might try using a stationary bicycle in your rooms.

Listed below is a differential diagnosis of "anginal" pain:

Causes of pain above the diaphragm

- Myocardial Infarction (MI)
- Pericarditis
- Mitral valve prolapse
- Aortic dissection
- Pulmonary embolism
- Pleurisy
- Fibrositis
- Cervical arthritis
- Hyperventillation

Causes of pain below the diaphragm

- Oesophageal spasm
- Reflux oesophagitis
- Upper GIT pain
- Biliary system pain

NB Oesophageal spasm can be induced by exercise and that plus biliary and renal spasm can be relieved by sublingual nitrates.

... Angina Pectoris

Unstable Angina Pectoris (UAP)

This is a dangerous condition with a much higher mortality than Stable Angina Pectoris.

The aetiology was thought to be either due to rapid progression or to coronary vasoconstriction. It has now been shown to be due to episodic coronary thrombus formation following atherosclerotic plaque rupture. This rupture activates platelets which in turn releases thromboxane A₂. Thromboxane A₂ stimulates platelet aggregation and vasoconstriction.

A similar progression of events occur in the three acute ischaemic syndromes vis:

- i) Acute myocardial infarction (AMI)
- ii) UAP
- iii) Sudden death

Thromboxane A₂ metabolites can be detected in the urine of patients with angina refractory to medical treatment.

Diagnosis

The diagnosis of UAP at present is entirely clinical from a history of increasing angina not responding to medical treatment.

UAP must be distinguished from other causes of pain and particularly from AMI and especially a non Q wave or subendocardial infarction.

In UAP the clinical examination is also non specific.

The ECG changes are transient deviations of the ST segment often associated with T wave inversions. These changes clear completely or partially with relief of pain. Persistence of these changes for six to twelve hours suggests an infarction. Lack of changes in the ECG does not exclude the diagnosis if the history is suggestive.

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... Angina Pectoris

It must be emphasised that no EST is done until after stabilisation because of the danger of inducing an AMI.

Silent Myocardial Ischaemia (SMI)

There is a lot of research being conducted into this subject at present, so that a working knowledge of what is happening might be useful.

Following the obstruction of a cardiac artery, the normal sequence of events is:

- i) Wall motion changes as noted by echocardiograph
- ii) ECG changes
- iii) Pain

In SMI there are coronary artery lesions, and ECG changes noted either on Holter monitoring or on routine Exercise Stress Testing (EST), but no symptoms of ischaemia are noted by the patient.

Silent myocardial ischaemia is defined as objective evidence of myocardial ischaemia without chest pain or other anginal equivalents.

EST should only be done after stabilization to avoid AMI

Various types have been described.

Type 1: Person with ischaemia, but no symptoms and no history of myocardial infarction or angina. This has been estimated to be 2-5% of the population or 1-2 million men in United States aged 35-60.

Type 2: Persons who are asymptomatic after myocardial infarction, but still show active (but painless) ischaemia. 18% of this population or 50,000 a year in United States fall into this category.

Type 3: Patients with both angina and silent ischaemia. In 655 patients with angina with positive EST, 236 were painless (44%). There are about three million cases in the United States.

Taking the history is the most important part of making the diagnosis

SMI is not a false positive EST as the patients do have coronary artery obstruction.

The reason for patients not having pain has not been fully or adequately explained. Theories which have been put forward include:

- diabetes mellitus, because of the neurological deficit that may occur;
- that the effort involved when the ECG changes are recorded is just not sufficient to cause the pain.

The only hard evidence so far is that those patients who have SMI have a higher pain threshold than patients who experience angina.

There is a 5 to 15 times increased chance of a patient with SMI having an acute myocardial infarction or sudden death in 5 to 15 years, but we

have no way of knowing which of the patients with SMI are those at risk.

Some authorities recommend treating all patients with SMI, the treatment ranges from Aspirin to calcium antagonists or beta blockers, but this could be very expensive.

Most cardiac incidents occur in the first 4 hours of waking

Possibly the best approach would be to select patients at high risk, ie those with a bad family history or those with bad risk factors, and subject them to an EST. If the test is positive at a low amount of effort, he needs an angiogram. Those with a positive EST at a higher level of effort need to be monitored with a Holter. Those with more than a certain time of ischaemia in 24 hours, need to have angiography.

These cut off points will become defined with the vast amount of research that is being conducted at present.

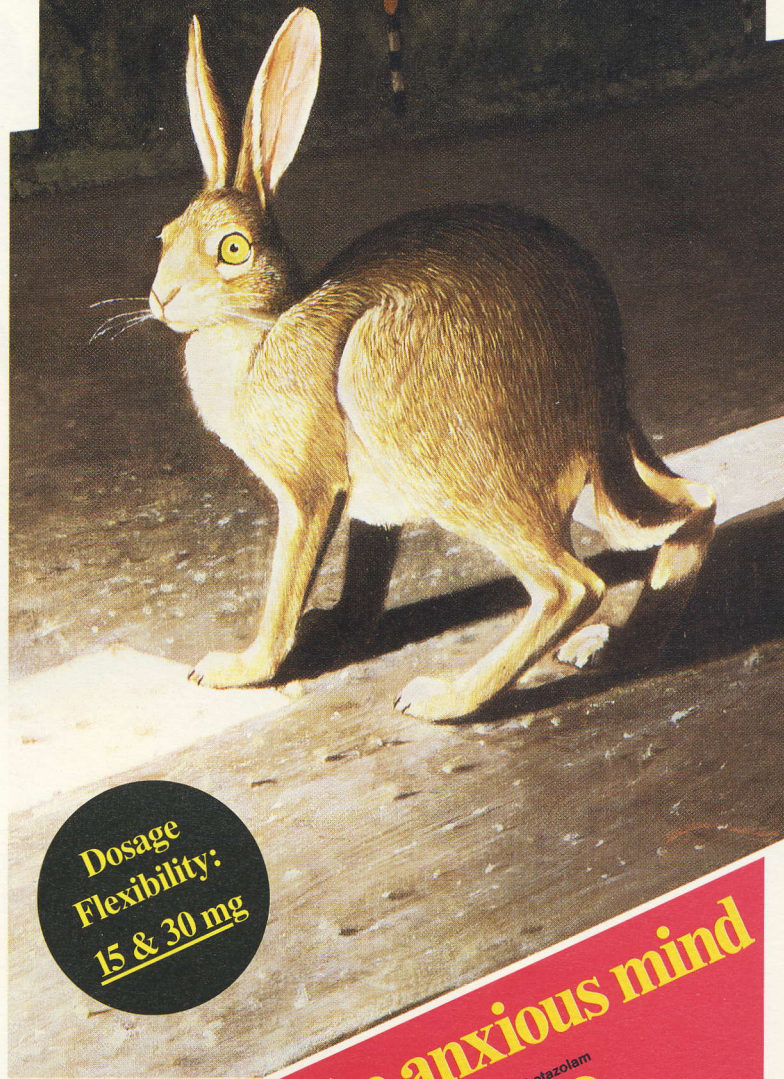
Further treatment would be dictated by the results of the angiogram.

Part II of this article will be published in the next issue of SA Family Practice.

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