Acute Heart Attack (predictable and preventable)

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Acute heart attacks, (AHA), include, acute myocardial infarction, (AMI), and "Sudden death" (SD), which is due, presumably to ventricular fibrillation or asystole.

The one-month mortality from AHA's has been consistently recorded at 40%. The majority (60 - 80%) of these deaths taking place soon after onset of symptoms prior to hospitalisation.

The first major attempt to reduce the death rate from AHA's was the introduction of the Intensive Coronary Care Unit (ICCU). This reduced the hospital death rate by 50% mainly by the prevention and reversal of major arrhythmias. However, as the majority of patients died before they reached hospital, this had virtually no effect on the community mortality rate.

Thus, it became evident that other strategies were needed to reduce mortality. The first of these was the mobile intensive coronary care unit (MICCU). The arch exponent of this method was Pantridge of Belfast, who successfully defibrillated patients in the early pre-hospital phase of AMI. He postulated, but never proved, that with adequate MICCU and ICCU facilities the overall community AHA mortality could be reduced to 25%.

The Cape General Practitioner Survey, where General Practitioners (GPs) responded promptly to patients calling with chest pain, and which effected a simple treatment, with the alleviation of pain and arrhythmia prophylaxis, showed an overall community mortality of 23% in patients under the age of 70 years. It appeared that GPs could effect a service highly comparable to that of any type of MICCU.

However, we are still left with a situation where 23% of patients who sustain an AHA die. If we look at the natural history study of the Cape GPs, we note, of the deaths recorded, half took place prior to hospital or any treatment, and the other half following hospitalisation.

Of the patients who died following hospitalisation, the majority succumbed from pump failure, ie. severe congestive cardiac failure or cardiogenic shock. Very little can be done for these patients, who usually have a large proportion of their myocardia damaged.

Treatments such as infarctectomy, Swartz-Gantz catheters and emergency bypass operations are often tried.

Of the patients who died prior to hospitalisation, over 90% died from recorded or probable arrhythmias. These arrhythmias, as we are well aware, have been shown to be preventable and reversible.

So again, in spite of these inroads mentioned, about half of the patients dying from acute heart attacks are still dying untreated and from probable preventable arrhythmias. Thus it appears to be possible to reduce the death rate by a further 10 - 15% if these "sudden deaths" can be prevented.

Hospital based medicine has developed, either directly or indirectly, two major strategies to deal with this problem. Firstly, the concept of "unstable" angina, "pre-infarction angina" or intermediate syndrome, crescendo angina, etc. has been identified in order to admit patients at risk into ICCU's and thereby hopefully preventing their AHA or allow it to occur in the "safe" environment of an ICCU.

The problems inherent in this approach are illustrated by the vast number of different syndromes which have evolved. Obviously hospital based doctors have to define "absolutes", for example, chest pain lasting ten minutes is equal to the pre-infarction angina.

It is interesting to note that different institutions have "liberal" criteria as to what constitutes a "pre-infarction syndrome" while others are far more stringent.

This must be borne out of necessity, such as the number of beds available and other resources. Thus an institution with several beds and resources can define the syndrome less stringently and vice versa.

The major difficulty in this approach is that is assumes that the natural history of coronary artery heart disease follows an orderly progression, which it does not. Coronary artery disease does not progress in all patients from mild angina which grows progressively worse until the patient infarcts or drops dead. Rather the acute heart attack occurs at any stage of the natural history, not necessarily heralded by a long progression of escalating events. This concept is discussed later in this article.

The second strategy used in the management of coronary artery heart disease is coronary artery surgery. While initially only alleviation of symptoms was claimed by this procedure, life expectancy is said to be prolonged in patients with certain clearly defined lesions of the coronary arteries. At no stage, however, has it been claimed that this procedure would prolong life in **all** patients with coronary artery heart disease. Moreover, if the dictum that "coronary angiography is the cornerstone of management" is followed, the financial and logistical sequelae would be mindboggling.

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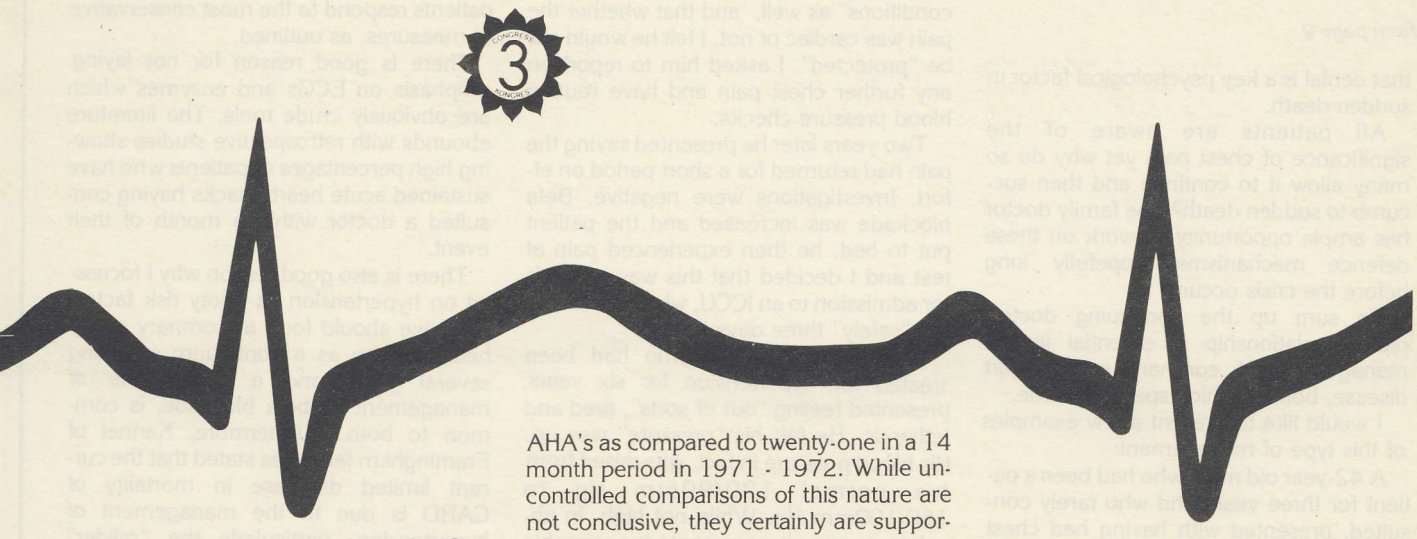
Kensley estimates that adopting the "correct approach", ie doing angiography on "appropriately" selected patients, 5 000 South Africans would require the operation annually at a cost of R60-million.

I doubt if any more than a few hundred are being done in South Africa at present and at no stage can I ever imagine this procedure being able to make a meaningful dent in the community mortality.

It would thus appear unrealistic to expect hospital based doctors to make any striking reduction in the community mortality for the reasons outlined.

In spite of what I have said, it seems unreasonable to accept that a catostropic event such as an acute heart attack comes without any warning. In a study



conducted, (following the Cape GP Coronary Care Project) into those patients who died, pre-hospital, medically unattended deaths ie. "sudden deaths", fifty of sixty-seven (75%) had "suggestive" stories of deficient coronary arterial supply.

While these ranged from increasing dyspnoea to frank retrosternal pain of varying intensity and duration, all these patients experienced deviations from their own norm. Furthermore, 75% of these patients had either diagnosed coronary artery heart disease and/or hypertension.

Thus taking all these factors into consideration, ie. a history of CAHD and/or hypertension and/or the occurance or worsening symptoms suggestive of CAHD, 96% of the patients that died of so called "sudden death" fell into this category.

On the basis of this data, I hypothesised that the patients at risk had hypertension or manifestations of coronary artery heart disease. Furthermore, I would vigorously treat hypertension and would regard any emergence or worsening of symptoms suggestive of coronary artery heart disease as "instability" which could lead to a sudden arrythmic death or an infarction.

On the study of the natural history of CAHD this appeared to be valid, since, as mentioned before, the vast majority of patients had some deviation of their norm before succumbing.

While the majority of these patients would rarely have met the absolute criteria to be admitted to an ICCU or be candidates for bypass surgery, their change in their normal pattern was enough to cause the catastrophic consequences. On patho-physiological grounds there is ample support for this hypothesis – that small areas of necrosis or even ischaemia can cause sudden arrythmic death.

Thus, for a two year period in 1976 - 1978, I vigorously treated hypertension as well as instituting a regimen of management for patients who had worsening symptoms indicative of CAHD. In this two year period I had a total of four AHA's as compared to twenty-one in a 14 month period in 1971 - 1972. While uncontrolled comparisons of this nature are not conclusive, they certainly are supportive of my hypothesis.

Furthermore, three of the four AHA's were "predicted and one was a new patient. While I have discussed this fully in another communication, I would like to elaborate further on my findings since that date.

The key principle involved in the management is that any "worsening" of a patient's condition puts him at risk. For example, a patient who is able to walk only 20 metres before getting angina is at less risk than someone who can normally jog three kilometres but is now getting pain after two and three-quarter kilometres. The former patient is stable while there is a **relative** change in the latter that makes him "unstable" and at risk.

This not only applies to angina but sudden rises in blood pressure relative to the patient's normal blood pressure as well as dyspnoea and profuse sweating, etc.

The next key principle involves seeking a cause for the change in the patient's status. Is it an infection? Is it a marital dispute? Obviously, as well as treating the worsening position one should try and treat the cause. Depression is commonly associated with a worsening of a patient's condition and a cause must be sought.

Not much reliance can be placed on the objective parameters such as electrocardiograms or cardiac enzymes in this situation as they rarely change. However, there might be slight deviation from the patient's existing ECG's which may be significant.

The suggested management:

Suggested management

1. Any patient with crescendo angina, which I define as any deviation from the patient's norm, should be put to bed if possible, for one to seven days.
2. A possible precipitating cause should always be sought; raised blood pressure, infection, or emotional upset, all of which should be treated.
3. Patient should be treated with beta-blocking drugs in low doses. If already on beta blockade this may be increased. Contra-indications to beta blockade are atrioventricular block, bronchospasm, and cardiac failure. Beta blockade has only recently been used in pre-infarction situation. The danger is that should the patient infarct he will have lost his sympathetic drive and will go into cardiac failure and even cardiogenic shock. Thus where there is a fear of incipient cardiac failure the patient may be digitalised before being put onto beta blockers.

   Beta blockade is considered the cornerstone of management in this situation as well as in hypertensives. I look forward to the evaluation of calcium antagonists in the community situation, in the management of hypertension and angina, as I am sure as the importance of coronary spasm becomes more highlighted, the greater the place for these drugs will become.
4. A long-acting nitrate, such as isosorbide dinitrate 5 - 10mg, should be given six-hourly.
5. The patient should be sedated with one of the benodiazepine group, such as diazepam 5mg tds.

On the above regime the majority of patients improve and the crisis appears to be averted. If they do not improve or get worse, this is usually a criterion for hospital admission.

It is obvious that the only way to manage patients in this matter is by continuing personal care by a readily accessible doctor, the General Practitioner/Family Physician.

Moreover, the family doctor is in the best position to identify non-compliant patients – patients who are "defended", patients who resent control and sometimes ironically get their only respite from their whirlwind or conflict-stricken world with an acute heart attack. There is no doubt

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that denial is a key psychological factor in sudden death.

All patients are aware of the significance of chest pain yet why do so many allow it to continue and then succumb to sudden death? The family doctor has ample opportunity to work on these defence mechanisms, hopefully long before the crisis occurs.

To sum up the continuing doctor-patient relationship is essential in the management of coronary artery heart disease, both of which span a lifetime.

I would like to present a few examples of this type of management:

A 42-year old man, who had been a patient for three years and who rarely consulted, presented with having had chest pain radiating down his left arm for thirty minutes the day before. He was a manager at a factory and had only come at his wife's insistence. He couldn't really afford the time.(At the appropriate time, I discussed his attitude to illness and what it meant to him).

The only abnormal finding was a persistent raised blood pressure 160/110mm Hg. The ECG and repeated ECG's were normal as were the enzymes. A subsequent effort ECG was also normal.

I explained to him that I was still not sure of the cause of his pain but his blood pressure should be treated; that the management of his blood pressure would be with a drug which was used in "heart conditions" as well, and that whether the pain was cardiac or not, I felt he would still be "protected". I asked him to report on any further chest pain and have routine blood pressure checks.

Two years later he presented saying the pain had returned for a short period on effort. Investigations were negative. Beta blockade was increased and the patient put to bed. he then experienced pain at rest and I decided that this was grounds for admission to an ICCU, where he infarcted "safely" three days later.

A 52-year old male who had been treated for hypertension for six years, presented feeling "out of sorts", tired and lethargic. He felt his "pressure" was up. His blood pressure in fact, was raised from his normal 120/90mm Hg to 160/100mm Hg. While not high, in absolute terms, it represented a sizeable percentage increase in this patient.

He also spoke of pain in the chest when he was anxious. This became more and more frequent as his hyperactive son was getting more difficult, and he disagreed with his wife on his management. He failed to respond to management and he was admitted to an ICCU where he sustained some more pain at rest. This was accompanied by slight raising of enzymes and ECG changes which reverted to normal. By definition this patient did not infarct. He settled and is back at work, symptomless.

Although I have presented these two patients, I must stress that the majority of patients respond to the most conservative of measures, as outlined.

There is good reason for not laying emphasis on ECGs and enzymes which are obviously crude tools. The literature abounds with retrospective studies showing high percentages of patients who have sustained acute heart attacks having consulted a doctor within a month of their event.

There is also good reason why I focussed on hypertension as a key risk factor, since we should look at coronary artery heart disease as a continuum spanning several years and a cornerstone of management – beta blockade, is common to both. Furthermore, Kannel of Framingham fame has stated that the current limited decrease in mortality of CAHD is due to the management of hypertension, particularly the "milder" types.

This does not mean that the other risk factors such as cigarette smoking, diabetes, hypercholesterolaemia etc. are any less important and must not be addressed.

Conclusion

In conclusion, I believe that acute heart attacks do not appear in a vacuum, that patients do exhibit warning signs which are significant to them, and that the only way to manage this situation is by ongoing holistic processes and in so doing it is hoped that a major reduction in mortality from CAHD can be realised. □

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preting ECG's and making decisions based on this mis-information are painfully obvious.

Simple tests

Finally, let us look at some of the simple tests which we perform in our daily work. Some are traditional procedures which have stood the test of time. Others illustrate the appropriate application of technology to the needs of general practice.

The E.S.R. and the haemoglobinometer.

The numerous paper dipsticks for testing urine.

The tuberculin tine test, much simpler if less precise than some of the other tests for tuberculin sensitivity.

The paper tests for blood glucose and the glucometer so useful in the management of diabetes.

The haemoccult test for the diagnosis of occult blood in stools.

Anglutination tests of which the pregnancy test is the most widely used.

The slipslide test which has been a major contribution to the diagnosis and management of urinary tract infection in General Practice.

The microscope with its many uses, not least in the diagnosis of ringworm.

The mini-Wright peak flow meter so simple to use, so accurate in measuring pulmonary function.

(A recent B.M.J. editorial[11] suggests that its use be as routine as recording blood pressure).

The audiotester is an audiometer adapted to the needs of the General Practitioner. It tests hearing in three frequencies and results can be simply recorded.

The use of the blood analyzer in urban practice is debatable but it certainly is of value to the rural practitioner.

Conclusion

In conclusion, let me stress that it is not my purpose to denigrate or undervalue the benefits that modern technology has brought to medicine and to the General Practitioner in particular.

I have only sought to show how a better understanding of the content and the process of our work helps us to use special investigation appropriately and to the benefit of our patients. □

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