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The SINGAPORE FAMILY PHYSICIAN

CARDIOLOGY IN PRACTICE

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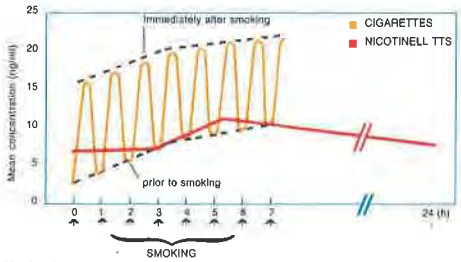

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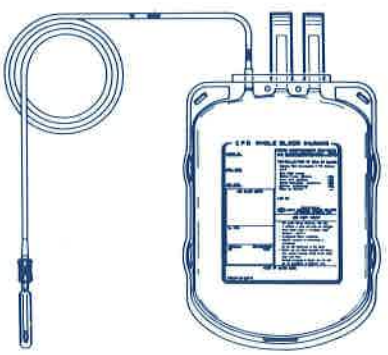

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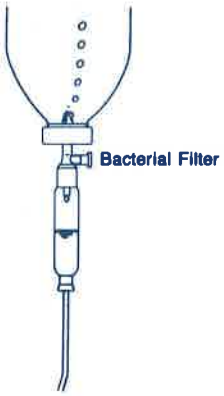

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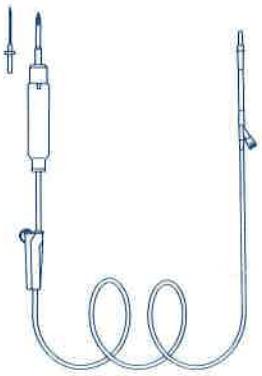

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EDITORIAL

THE FAMILY LIFE CYCLE, FAMILY DYNAMICS AND THE FAMILY PHYSICIAN

At this point in the history of the College, with the considerations to change its name from College of General Practitioners Singapore to College of Family Physicians Singapore, it is appropriate to take another look at the whole meaning of Family Medicine and the work and role of the family doctor. This would help add credence to the claim that **Family Medicine** is a distinct and unique discipline or specialty, as opposed to **General Practice**, which has carried with it connotations of mediocrity, both in quality and role or place.

The main reason why healthcare is targeted at the family instead of at the individual person is that it allows healthcare to be applied to the smallest biological and social unit which allows at the same time optimal results to be obtained. It has long been recognised that individual health is inseparable from family health and that many individual illnesses, from communicable disease to behavioural problems, are illnesses of the family as well. James Dennis[1] has written in 1969: "It is in the family milieu, and very early in life, that we find the genesis of social or antisocial human behaviour, mental health or illness, many communicable diseases and the nutritional or other factors that ultimately lead to many of the chronic degenerative and disability disorders of later life. It is not possible to separate poor mental and physicial health, ignorance and poverty from the pathology of the family".

The structure of the conventional basic family unit of two parents and their children has evolved over the decades with changes in our society, and at least thirteen different types of households have been described. However all family types remain one special kind of group. While its members share a long history, which carries with it varied genetic, economic, social, religious and cultural influences, the family unit itself serves to help integrate each of its members into the community and the wider society, while at the same time addressing itself to the material, as well as the emotional, cultural, social and sexual or physical, needs of its members. The family offers each member nurturance which permits psychosocial growth and development, creates a sense of historical perspective and provides a base from which the process of social definition begins[2].

Family Medicine is a co-ordinated multi-disciplined approach to comprehensive healthcare of the family unit, encompassing healthcare for all the major events of the family — marriage, birth, infancy, childhood, adolescence, adulthood, aging, trauma, illness and death.

The Phases in the Cycle of Family Development

Duvall, an American sociologist, has described eight definable phases or stages in the family life cycle — a description which is applicable to the Asian family as well:

Stage 1 : Newly married
2 : Birth of first child (till he is 30 months old)
3 : With preschool children (the oldest child aged 30 months to 6 years)
4 : With children in school (oldest child aged 6 to 13 years)
5 : With teenagers (oldest child aged 13 to 20 years)
6 : Launching years (from when the first child leaves home to last child leaving home)
7 : Parents alone in middle years (from last child leaving home till retirement of the parents)

8 : Retirement and later years (from retirement to the death of both parents)

Each stage brings with itself new and special developmental tasks for the family and medical tasks for the family doctor. While the newly marrieds learn to communicate effectively with and adjust to each other and help one another meet social, economic and sexual needs, their doctor can advise on work, family planning and child bearing. When the parents deal with the stresses of fatigue, financial strain, decreased leisure activity and new schedules with the birth of their first child, the family doctor is at hand to teach mothercraft and parenting skills, to care for the sick child and to advise on childhood behavioural problems. When the children grow up into teenagers and prepare to leave home, the family tries to encourage development of the children's independence while simultaneously maintaining closeness and family cohesiveness; the family doctor advises the teenagers on developmental changes, intersibling relationships and possible conflicts with social restrictions, and advises the family on coping strategies during the disengagement process. When the parents are left alone and finally retire, they have to re-align their priorities, adjust to retirement and new roles, and cope with the aging process and with decreasing social circle and loneliness — their doctor advises on keeping fit, helps with degenerative medical problems and provides psychological support to overcome loneliness and deal with bereavement.

The structure and functions of the family alter with the passage of time; failure of the members to adapt to the changes can be a source of ill-health. In each family unit, the family doctor must understand not only the stresses inherent to the structure, but also the stresses from the processes which have led to that structure.

Family Dynamics and Illness

One of the primary concerns of Family Medicine is to understand the individual patient in the context of his family, the dynamics within the family, and the relationship of the family to the community. Human relationships, especially those within a family, are important determinants of health and disease, and family dynamics are connected vitally to the basic issues of physical or mental health. Medical historian Michel Foucault states: "The natural locus of disease is the natural locus of life — the family: gentle, spontaneous care, expressive of love and a common desire for a cure, assists nature in its struggle against the illness ....."[3] The doctor must fully understand these relationships and recognise that such relationships are an important "forth dimension" to the physical, mental and spiritual aspects of all people to be able to fulfill his role of family physician. Illness is a process shaped by biological, psychological and interpersonal processes, each of which must be addressed if the intervention is to be successful[4].

The family plays a big part in defining and producing illness in its members. Every family has its own belief about what constitutes illness i.e. a situation in which the sufferer may legitimately relinquish his usual roles and responsibilities and be allowed certain priveleges; family traditions and medical history may affect the way in which the illness is suffered and sometimes the site and nature of the symptoms; families may "use" one of their members e.g. by obtaining assistance in a crisis through him or by expressing guilt through him.

While the sick family member deals with his physical illness and with his new sick role, the other family members have to deal with any financial difficulty or extra work that it puts on them, besides sharing the mental anguish of sickness within the family. The new needs, roles and responsibilities in the ill one and in the other family members can, especially with serious or chronic illness, lead to conflicts and produce resentment or depression. The family with strong interpersonal bonds will accept illness as a challenge to adapt, to grow up and to make relationships stronger.

The attending family doctor will have to carefully work out who in the family is (or are) really the patient (not necessarily the one whose behaviour or health is complained about), whether the complaint can be explained in terms of conflict between family roles or conflict between family rules and the patient's emotional need for independence from the rules, and how he can

really help the patients and the family maintain and strengthen interpersonal relationships. He will find that there is a difference between being a family doctor and being a personal doctor to all the members of a family. He may view the treatment of the entire family as one unit in itself and he may try to help them to be and behave like a family unit, but when the interests of individual members are in real conflict, his relationships with each one of them are individual, and he will find that methods of help or treatment that worked in times of family harmony or solidarity can fail miserably in times of internal disruption. Being a holistic family physician to even a simple family can be a very complex job.

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THEME EDITORIAL

SCREENING FOR CORONARY ARTERY DISEASE: WHAT IS THE GP'S STAND?

Ischaemic and other heart diseases have ranked second in the list of leading cause of death in Singapore in recent years. Classical risk factors for coronary artery disease (CAD) are well known, and there have been extensive public education campaigns to increase awareness, as well as to urge people to change their lifestyles, even to the extent of incorporation of this knowledge in the health education textbooks of primary school children.

Increasingly, therefore, GPs get requests from adult patients of all ages to test for their cholesterol levels, do their resting electrocardiograms, and so forth. Should we, then, accede to every request, or should we be more selective in selecting who should be screened?

Let us first examine several issues pertaining to screening for coronary artery disease in general practice. Firstly, it is now well established that the progression of preclinical coronary artery disease can be altered by risk factor modification or drugs. This makes the identification of patients with high risk, or those in the early stages of disease, important.

Secondly, once the presence of coronary artery disease, has been diagnosed effective treatment is available for such patient. Prognosis can be improved by earlier institution of treatment or through appropriate referral.

Thirdly, in the process of screening, the doctor is given the chance to advise and educate the examinee about his lifestyle and other relevant matters, which would not otherwise have taken place with patients coming in only for curative care.

Having said that, one must then look at the other side of the coin. Isolated screening of cholesterol levels or electrocardiograph abnormalities are by themselves inadequate. It is necessary to identify the presence of other cardiovascular risk factors such as positive family history of ischaemic heart disease, co-existence of hypertension or diabetes, obesity and smoking. This should be offered as a 'package' to the patients who request for isolated screening tests. Shaper et al reported that their opportunistic identification of high risk groups based on a 'risk score' identified 53% of men who subsequently developed ischaemic heart disease over the next five years. The addition of serum total cholesterol concentration and electrocardiographic evidence only slightly improved the prediction, but would considerably increase the cost and effort of screening.

In doing screening tests for patients, we must also ask the question: 'In whose interest is it to test?'. Commercial interest is widespread in this area, whether for laboratory investigations or for drug treatment of hyperlipidaemia. Does the patient benefit from these costly laboratory charges and costlier drugs? Is it necessary to screen those not at high risk, or to treat those with isolated, borderline raised lipid levels? Is it necessary to screen those who do not require treatment at all? Doctors must guard against the thought of seeming to be preventive-minded, by providing an extra service.

Then there is this question of what to do with borderline results and unexpected findings. It is true that 'the more you look, the more you find', though it is not entirely true that, to save the trouble, you should not seek to know at all. It is, however, important that the patient is not needlessly

disadvantaged as a consequence of the examination, and that doctors will need to defuse the panic caused by simplistic, arithmetical interpretation of isolated or borderline test abnormalities.

Finally, is it not adequate to just screen and convey the results of screening? If any abnormality is found, it is the responsibility of the GP concerned to counsel, manage and follow-up the patient, with referral where appropriate. There must be intervention for those identified. This takes time and effort, especially in ensuring change of lifestyles and compliance. The long-term effects are usually not immediately obvious, and one must guard against discouragement and complacency.

With all that has been said above, there remains one factor which may discount all the logical arguments put forward. And that is 'patient autonomy'. All who have experience in general practice know that patient autonomy ranks very high in outpatient patient care. Patients' fears, views and concerns may be very different from accepted standard practice, statistics and figures from population studies or conclusions from well known research. To the patient, he (or those close to him) is the centre of his universe, and he needs to have his anxieties addressed. The GP has an easier task if his patient is receptive to his advice. To those who insist on getting what they want, it remains for the individual GP to decide how far to accommodate such requests, bearing in mind what is known in the current state of medical knowledge, and other considerations.

Hence, whilst epidemiological and clinical research set the trend and direction for risk factor identification and patient management, at the individual level, it is the patient and his doctor, and their relationship, that determine how these will be carried out.

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Hong Ching Ye

ASSESSING THE PATIENT WITH CORONARY ARTERY DISEASE

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The public in general, and even doctors and medical personnel, are confused by the vast amount of literature that quote various results from trials with varying medications on lipids lowering in the prevention or reduction of the incidence of cardiovascular disease (CVD). The main issue in the analysis of these trials and the discussion that arises therefrom is the relation between cholesterol and coronary heart disease (CHD). The questions that we need to ask ourselves, when reading or listening to the presentation of these trials are:

1. Is serum cholesterol truly important in the genesis of CHD?
2. Is the treatment of hypercholesterolaemia useful in the prevention of CHD?
3. Has such cholesterol-lowering treatment untoward effects?
4. How do other lipid fractions (HDL-C, LDL-C, TG) contribute to atherosclerosis and CHD?
5. What is the contribution to atherosclerosis of other non-lipid risk factors (cigarette smoking, hypertension, hyperinsulinaemia, diabetes mellitus and lack of exercise)?
6. Has treatment for non-lipid risk factors (e.g. the use of diuretics and ß-blocks for hypertension) adversely contributed to rises in lipid and non-lipid risks factors (e.g. hyperinsulinaemia)?

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The answers to all these questions are not necessarily answered in any one of the research papers that we have read. It is not convenient, for example in a paper that analyses the cholesterol lowering effect of a particular agent, to discuss problems of hypertension, or the contributions of glucose and diabetes mellitus to the development of CHD. Basically, trial papers show us what favourable features we may expect when we follow the protocol used to treat our patients with hyperlipidaemia e.g. hypercholesterolaemia. However, when we are confronted with a patient with hypercholesterolaemia, or ischaemic heart disease (IHD) or CHD, our assessment of our patient, and the direction through which we will proceed with his treatment, must necessarily include all his risk factors – in particular with relevance to the questions we have asked ourselves as listed above.

Most importantly, it is to establish the presence in the family of risks for cardiovascular disease (CVD). Does our patient have a family history of myocardial infraction, cerebral strokes or peripheral vascular ischaemic disease? Does our patient have a family history of diabetes mellitus? Does our patient have a family history of hypertension? Diabetes mellitus, hypertension and hyperinsulinaemia are related and are often found in one and the same person. Although the pattern of inheritance of these polygenic diseases is at present uncertain, it is nevertheless true that clusters of these diseases are common in one and the same family. Diabetics and hypertensives are often also hyperinsulinaemic. Insulin promotes growth of smooth muscle cells of the vascular wall, dyslipidaemia (high serum triglyceride), hypertension, and as a consequence, early or premature atherosclerosis (Reaven's Syndrome)

and early cardiovascular and coronary heart disease. We have found in our study of young patients (<45 years), with normal body mass-index and non-smokers, that their risk to their having suffered myocardial infarction was hyperinsulinaemia (insulin area under the curve of an IVGTT : MU/L : AMI: 3648 ± 302, control 2530 ± 184 : p = 0.002) when compared with age and weight matched non-coronary-infarct individuals. Most of these young patients who had suffered a myocardial infarction, had serum cholesterol level within the normal range (cf fasting serum cholesterol : mmol/L : AMI 4.8 ± 0.1, control 4.8 ± 0.1).

Our study interest of late has been in hyperinsulinaemic states and the coronary risk factors associated with its presence -- hypertension, dyslipidaemia, impaired glucose tolerance / diabetes mellitus. Together with hyperinsulinaemia, these three risk factors have been called by Kaplan the deadly quartet. The presence of these risk factors in any one individual (Reaven's Syndrome) was first described by Professor G Reaven from Paolo Atto in 1988 (Banting lecture 1988).

Reaven's Syndrome compounds the problems of atherosclerosis. It no way negates the importance and care which one must give to the study and understanding serum cholesterol levels in our patient. In our daily practice of measuring serum cholesterol, we will have noticed that some of the patients who have normal serum cholesterol level (<5.2 mmol/L) have had a myocardial infarction. Others, with very high serum cholesterol levels (>6.0 mmol/L) do not have ischaemic problems in their cardiovascular system. In the former group, we have explained that they have Reaven's Syndrome, a syndrome that predisposes them to early atherosclerosis and myocardial infarction. The latter group, with high serum cholesterol, may yet succumb to cardiovascular disease at a later date. No better study expresses the relations between serum cholesterol and frequency of coronary heart disease than the MRFIT study and the recently concluded Shanghai study. Both these studies show that even at low levels (Shanghai study) of serum cholesterol and progressing to higher levels of serum cholesterol (MRFIT study), there is a "linear" relation between serum cholesterol and the frequency of coronary heart disease. It is always important to lower serum cholesterol, via hygienic means e.g. life style change or, if necessary with medications.

How should we assess our patient who presents with chest pain of (coronary) ischaemia origin? Table 1 summarises the components of global risk that our patient with coronary heart disease could be confronted with. This assessment of the global risks of CHD is essential to all decisions concerning the management of our patient — the advice on life-style change, weight loss, what drug therapy to be used and goals we wish to achieve not only in lipid lowering, but also in reversal of atherosclerosis. Indeed, today, in our management of our patient with coronary heart disease, it is not just the lowering of cholesterol "numbers" but more importantly, the reversal of atherosclerosis. The reversal of atherosclerosis plaques is now made possible with newer and more potent cholesterol lowering agents (e.g. HMGCo-A reductase) or with a combination of two or more lipid lowering agents.

Table 1

Coronary heart disease: the components of global risks (modified from B Lewis 1992).

Clinical findings:

Age
Male sex; post menopausal state in women.
Personal history of coronary or peripheral vascular disease.
Family history of coronary disease presenting before age 60.

Treatable risk factors:

Cigarette smoking
Hypertension
Hyperinsulinaemia
Elevated plasma cholesterol as a result of raised levels of low-density or intermediate density lipoprotein.
Low levels of high density lipoprotein cholesterol (<0.9 mmol/L in men, <1.1 mmol/L in women)
Elevated plasma triglyceride when accompanied by low level of HDL-Cholesterol.
Diabetes mellitus
Obesity

Today, we routinely asses all patients presenting with coronary heart disease as follows:

Physical	:	Height / Weight / Body Mass Index
	:	Waist / Hip ratio
	:	Blood pressure
Biochemistry	:	Lipid profile (T. Cholesterol, TG, HDL-C, LDL-C)
	:	Lp (a)
	:	Fasting Blood Glucose
	:	Fasting Serum Insulin
	:	HbA1C

Together with the family history of myocardial infarction, these indices give us a comprehensive profile of our patient's risk for cardiovascular heart disease. Measurement of Lp (a) although important, especially amongst our local Indians and diabetics, is as yet not routinely available, except at some special laboratories. Fasting blood glucose (Normal: <90 mg/dL) and corresponding Fasting Serum Insulin (<10 mU/L) gives us a general index to the patient's "pre diabetic - hyperinsulinaemia" state. As described, there is a strong relation between hypertension and hyperinsulinaemia. HbA1C tells us not only the ambient glucose levels over the past three months but gives a crude assessment of the degree of glycoxylation of tissue protein. This should give us a "mental" picture that glycoxylation of LDL-C also does occur to some degree. This glycoxylation of LDL-C enhances its incorporation within the atherosclerotic process.

Progression to treadmill and other form of exercise studies may be necessary to complete the assessment of our patient. Our aim is always to minimize the cardiovascular risk and to identify the high-risk patient for necessary counselling. The type of therapy follows this assessment. For the patient who has already had a myocardial infarction, the same approach to assessment should follow. In him, treatment aims are not only to reduce cardiovascular risks but also to reverse atherosclerosis that has set in. Unfortunately, not always are we successful in reduction of cardiovascular risks, reversal of atherosclerosis and pevention of a fatal myocardial infarction. The risks factors are many, varied in type and severity. We should persist with public education and the creation of an awareness to the dangers of fatal coronary heart disease. Deaths from coronary heat disease in Singapore have levelled off in the past three years, and in 1992, have begun to show a fall. This is heartening indeed. Our message of life-style change for our citizens is effective and successful, and with so much in the literature as our guide and so many facilities at hand to assess our patient, we should seriously consider the next myocardial infarction in our patient as a failure in our practice of medicine!

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HOW TO MANAGE PATIENTS AFTER CORONARY ARTERY BYPASS SURGERY?: AN ABC GUIDE

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SUMMARY

Successful management of patients after CABG requires the total commitment of the patient and the family, the cardiologist, cardiac surgeon and the family physician. The patient must be reminded that **there is no cure for coronary arterial disease** and it is futile to depend on a single wonder drug from hearsay or faith! However, regular interactions of the patient with the team will minimise the failure of the grafted vessels and the progression of atherosclerosis of the native vessels.

The **early phase** of management is to look out for surgical associated complications, prevent graft thrombosis and to rehabilitate the patient back to "normal" life as soon as possible. The **longterm** management is to minimise the risk of occlusion of the grafted and native vessels. Remember, the pathophysiology of atherosclerosis of native and grafted is complex. This will require careful and regular monitoring of the known coronary risk factors and to impressing on the patient the importance of drug compliance. The longterm goal of management is to prevent future cardiac events and to ensure that the patient has a reasonable quality of life.

INTRODUCTION

Coronary arterial disease is the primary cause of morbidity and mortality in developed societies. The mainstay of treatment is drug therapy and maintenance of a healthy lifestyle. However, select patients will require either balloon angioplasty or coronary arterial bypass grafts (CABG). As this is such a common problem in the community, it is very likely that the family physicians will come across such patients and have to manage them. The management of such patient is the same as for patients with established coronary arterial disease except for the additional pathology of the venous or arterial conduits used for CABG. The aim of this article to rationalise the role of family physicians in the management of patients after CABG.

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ABC OF CORONARY ARTERIAL BYPASS GRAFTS

* The primary role of CABG is to **redirect** blood flow distal to the stenoses of the affected coronary arteries. Hence, this is essentially a **palliative** and not a curative procedure.

* The **saphenous veins** and the **internal mammary arteries** are the most commonly used conduits. The venous grafts are usually less durable and about 50% will have significant disease after ten years as compared to the less than 10% of arterial grafts.

* **Early** graft occlusion is **thrombotic** in origin whereas **late** occlusion is an **accelerated form of atherosclerosis.**

WHAT DO I DO WITH PATIENTS AFTER CABG?

Management of these patients can be broadly divided into three stages.
(A) The first three months
(B) The first year
(C) Longterm management

(A) The First Three Months Post-CABG

After the patient is discharged from hospital, he will require a lot of encouragement and close supervision to monitor the complications associated with CABG (Table 1). **Wound infections** of the sternum and legs are uncommon but do take special care in diabetic patients. Elderly and diabetic patients with bilateral internal mammary grafts are more prone to sternal wound breakdown. Occasionally, the patients complain of **swollen legs**. This is related to the removal of the saphenous veins used for CABG and hence it will take time for the deep venous system to compensate and accommodate the venous return from the lower limbs. Simple measures including elevating the affected legs when not ambulant and wearing the special stockings for the first three months will relieve the oedema. In the occasional patient with pronounced oedema, diuretics can be used sparingly. Remember to exclude other causes of oedema including renal impairment, hypoproteinaemia and cardiac failure.

Table 1 : Early Complications after CABG

Cardiac
arrhythmias especially atrial fibrillation
pericardial effusion
Thoracic
Pleural effusion
pneumonia
pneumothorax
Wound infections
sternotomy
legs
Leg oedema
Haematological
anaemia
Neurological
sleep disturbances
anxiety neurosis

If the patient complains of **difficulties in breathing or cough**, consider pleural effusion, chest infection or pericardial effusion. The pleural effusion usually settles spontaneously but occasionally it has to be drained. A clinically large pericardial effusion is potentially serious and is readily confirmed with an echocardiogram. Remember, the angiotensin converting enzyme inhibitors can also cause troublesome cough!

Cardiac arrhythmia after CABG is uncommon. The most common dysrhythmia is **atrial fibrillation**. This is usually a self-limiting arrhythmia and can be readily treated with either digoxin, beta blockers or calcium antagonists. **Ventricular arrhythmias** are potentially more serous especially in patients with impaired left ventricular function. It is important to check the serum potassium as this can be readily corrected. If this simple measure fails, do refer the patient to the cardiologist for further management.

The first three months after CABG can be quite "stressful" for the patient as he is on a lot of **medications** (Table II). The single most important drug that the patient must take is the antiplatelet agent i.e. salicylate. This is to prevent the early thrombotic occlusion of the grafted vessels. There are many types of salicylates in the market but be familiar with the one that you use. There is a general trend to use **low dose** salicylate as it is just as effective in preventing arterial thrombosis but with less risk of gastritis. The current trend is to use 100 mg of salicylate per day but there is interesting data to use as low as 50 mg per day! To date, there is no proof that the addition of dipyridamole to salicylate will lower the risk of graft closure. However, for patients who cannot tolerate salicylate, one can use either ticlopidine or dipyridamole. Very occasionally, the patient is on anticoagulants i.e. warfarin. This is usually reserved for patients who have had endarterectomy of the native vessels in addition to the grafts. Remind the patient to look our for the early sings of bleeding from the stomach i.e. black stools, as this can happen anytime during the course of treatment.

Table II : Medications after CABG

Antiplatelet agents
salicylates
dypridamole
ticlopidine
Anticoagulants
Blood pressure lowering agents
Glucose lowering agents
Lipid lowering agents
Gastric medications
Tagamet, Zantac, Losec
Others
Vitamin A & E
fish oil
lecithin
garlic pills

Medications are commonly taken for the associated medical pathologies i.e. hypertension, diabetes mellitus and hyperlipidaemia. Lastly, the patient may take fish oil, vitamin E, lecithin and garlic. However, there is still no solid data to ascertain their beneficial effects in this context. Furthermore, the optimal dose of Vit E is still undefined. The general rule is for the patient to take the essential medications as they are of proven value and to continue on the healthy lifestyle.

The patient and relatives want to know **"how much exercise he can perform"** after open heart surgery. There are no hard and fast rules but general principles apply. The patient should gradually increase his effort tolerance until he is limited by fatigue and breathlessness. The patient is discouraged from driving for the first month but can usually resume full activities by the third month. The same principle applies for resumption of sexual activities after CABG.

Another common question asked is **"what food can he take?"** This is the best opportunity to interact with the family and the patient as the patient is most sensitive to recommendations. Stress on healthy diet which is low on animal and saturated fat but more on fish, fibre and vegetable. Moderate alcohol consumption is not contraindicated but cigarette smoking is taboo!

Important check lists include the weight, blood pressure, glucose and lipids. If the patient is already on hypolipidaemic drugs, the lipids should be checked at least three monthly. The current recommendation is to **keep the LDL cholesterol to less than 100 mg/dl.** There is ample data to show that lowering of cholesterol can prevent the progression and promote the regression of atherosclerosis in grafted vessels. If the patient is on hypolipidaemic drugs, it is important to check the liver function tests periodically as the dose may need to be adjusted or even stopped.

(B) The first year after CABG

After the third month, the patient is usually back to normal activities. The major thrust of management is to prevent thrombotic occlusion of the grafted vessels. Hence, the patient must be reminded to take the antiplatelet agents regularly. In addition, strict control of the coronary risk factors and maintaining a healthy lifestyle is essential to the longterm potency of the grafts and native vessels.

(C) Longterm management of patients with successful CABG.

After the first year of surgery, the doctor must be on guard as the patient may feel so well that he may go back to his bad old ways! **The main thrust of management is to prevent late occlusion of the native and grafted vessels.** The pathophysiology of late graft occlusion is different. Thrombosis of the grafted vessel is rare but there is now an accelerated form of atherosclerosis especially of the venous conduits as they adapt to the new role. However, salicylate must still be taken regularly until a better alternative is found.

The patient's blood pressure, glucose, lipids and weight must be monitored regularly as these risk factors may manifest themselves in the patients' lifetime. In addition, the patient is constantly reminded of the value of healthy lifestyle i.e.

healthy diet, regular exercise, not to smoke and learning how to cope with stress.

Lifelong and compliant drug therapy is essential to the longterm prognosis of these patients. Hence, it is important to monitor side effects from such drugs i.e. GIT bleeding from antiplatelet agents especially the salicylate preparations. As these patients are also on medications for associated medical pathologies i.e. hypertension and diabetes mellitus, they must be motivated to take the mouthful of tablets. To minimise the cost of longterm management and compliance with medications, it is essential to review the medications regularly and to use the essential ones only.

Furthermore, the patient must be **closely monitored for early signs of occlusion of the native and grafted vessels**. If the patient has recurrence of angina, he should be referred to the cardiologist for further evaluation as he may require an exercise stress test or coronary angiogram. Remember, if the patient has recurrence of angina, it does not always mean that he will require repeat CABG as balloon angioplasty can complement the management of these patients.

One of the major challenges is to co-ordinate the management of these patients as not uncommonly, they are also attended to by other doctors who may adjust their medications.

HYPERTENSION IN THE ELDERLY

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SUMMARY

Only lately has the issue of managing the elderly hypertensive become clearer. All the recent major trials demonstrate the benefit of treating both systolic-diastolic hypertension and isolated systolic hypertension in the elderly. Nevertheless, in this age group the doctor should be mindful of the patients' increased susceptibility to iatrogenic side-effects and consider the treatment's risk-benefit ratio carefully. Non-pharmacological therapy can still have validity and thiazide diuretics have consistently shown efficacy in the elderly. Calcium-antagonists, ACE-inhibitors and beta-blockers provide other alternatives, and the final selection is often influenced by the patients' other concomitant diseases..

Key words:
Drug-selection, elderly, evaluation, hypertension, non- pharmacological, treatment-benefits, treatment-risks.

INTRODUCTION

Though extremely common, it has only been of late that clarity has emerged about how hypertension can be best managed in the elderly. Even in the early 1980s, it was felt that there were no significant benefits in prescribing antihypertensives to the older patient, whatever the blood pressure. Very few clinical trials of antihypertensive therapy included the elderly cohort. However, in the latter half of the last decade as well as more recently, several studies have been published which have specifically looked at this problem[1-6].

In this brief review, the epidemiology and pathophysiology of hypertension in the elderly are first dealt with, followed by its clinical evaluation. The management section will focus primarily upon the risks and benefits of therapy, the different modalities of treatment and the principles of choosing the appropriate drugs.

DEFINITIONS

Two hypertensive entities require definition in the elderly group of patients. In essence these definitions are empirical and the following are derived from the recommendations of the National Heart, Lung and Blood Institute in the United States:

- **Systolic-Diastolic Hypertension (SDH):**
  Systolic blood pressure (SBP) > 140 -160 mm Hg and
  Diastolic blood pressure (DBP) ≥ 90 mm Hg

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- **Isolated Systolic Hypertension (ISH):** SBP ≥ 160 mm Hg and DBP < 90

EPIDEMIOLOGY

The Influence of Age

The average SBP increases throughout life in most developed countries while the average DBP rises till the age of 55 to 60 years. However, this rise in the average blood pressure has not been observed in primitive societies indicating that hypertension is not an inevitable consequence of ageing.

Prevalence and Incidence

Prevalence estimates of hypertension are influenced not only by the defined values but also by the number of times the blood pressure is checked before the diagnosis is made. Based on studies which utilize measurements on more than one occasion, it appears that in the west, the prevalence of SDH is about 20%, while that of ISH ranges from 10 to 20%[7]. The annual incidence of hypertension in the elderly has been estimated to be 6 to 8% with no sex differences being noted[8].

Risks

In the Framingham study[9], the hypertensive elderly, aged 65 to 74, had a three times greater risk of cardiovascular disease, a twice greater cardiovascular mortality and twice the risk of cerebrovascular accidents than comparable normotensive persons. This risk persists even in older individuals and in the SHEP and STOP-Hypertension Studies, the benefits of antihypertensive therapy were visible till the age of 80 years.

Although the management perspective of hypertension has classically focused upon DBP levels, epidemiological data[10] indicate that for middle-aged and older adults, SBP is more predictive of future cardiovascular morbidity and mortality than DBP. With regard to cerebrovascular accidents, both the SBP and DBP are highly predictive.

PATHOPHYSIOLOGY

It is thought that losses of vascular distensibility and elastic recoil contribute significantly to prominent rise of the systolic blood pressure in the elderly.

Secondary causes of hypertension such as Conn's Syndrome or phaeochromocytoma are far less common in this age group. Reno-vascular disease secondary to atherosclerosis can be encountered in 5 to 10% of the cases. Generally such patients have a relatively sudden onset of moderate to severe hypertension that may be refractory to therapy.

CLINICAL EVALUATION

The basic aims of the clinical evaluation will be to accurately determine the presence of hypertension, to look for end-organ damage and, in certain cases, to rule out secondary causes of the elevated blood pressure.

Determining Hypertension

The most important principle here is that of measuring the blood pressure more than once and preferably on two to three occasions. This is because of the high variability of blood pressure. Also relevant is to note that blood pressure in the elderly should be assessed in both the supine and erect postures; orthostatic hypotension increases with age and can have important implications for therapy.

Recently the entity of pseudo-hypertension in the elderly has been mentioned. This occurs when there are thickened or calcified arteries which are not easily compressed by the blood pressure cuff. Hence the estimates of such indirect measurements are higher than those obtained via direct intra-arterial assessment. This however is not considered to be highly prevalent.

The Osler Manoeuvre can be used to pick up pseudo-hypertension: when the cuff is elevated to above systolic pressures, one attempts to see whether the radial or brachial arteries are still

palpable. If so, the patient has rigid arteries and can have pseudo-hypertension. This needs only to be done whenever the condition is suspected, e.g. when there are significant elevations of blood pressure without evidence of end-organ damage and in those treated hypertensives who have features of low blood pressure (e.g. fatigue or giddiness) and yet whose cuff pressures are normal.

End-organ Damage

This can bee assessed by looking at the retina for hypertensive changes, evaluating the renal function and doing a chest x-ray and ECG to see if there are features of left ventricular hypertrophy. The presence of end-organ damage when the blood pressure is still borderline behoves the doctor to initiate therapy.

Secondary Causes

As mentioned above the most common secondary cause is reno-vascular stenosis; other conditions are much rarer. Secondary hypertension can be suspected for example, when

- there is refractory hypertension (DBP still above 100 mm Hg despite use of triple antihypertensives)
- there is sudden onset of significant hypertension (DBP > 105 mm Hg)
- there is spontaneous (and not drug induced) hypokalemia or clinical features very suggestive of phaeochromocytoma.

MANAGEMENT

Benefits of Therapy

At this point in time, all the major trials indicate that it is beneficial to treat both SDH and ISH in the elderly. Evidence is also fairly clear that these benefits extend right up to the age of 80 years; thereafter the evidence becomes less impressive.

Total cardiovascular events (coronary heart disease, heart failure and stoke) were reduced significantly in five trials[2-6] with reductions ranging from 17 to 40%. This can be predominantly attributed to significant falls in both fatal and non-fatal strokes; though a reduction was demonstrated for fatal and non-fatal cardiac events in all trials, this decline achieved significance only in the STOP-Hypertension trial.

Risk of Therapy

At least theoretically, there are reasons why the risk-benefit-ratio for the treatment of hypertension can increase in the elderly. It is known that the elderly are especially susceptible to the adverse effects of antihypertensives. Thus they are more likely to develop hyponatremia and hypokalemia with diuretics, experience depression or confusion with antihypertensives that affect the central nervous system and develop postural hypotension because of impaired baroreceptor reflexes.

Also there have been recent reports of a J-shaped relationship between treated DBP and mortality from myocardial infarction[11,12]. What these descriptive studies have shown is that while the reduction of DBP can be associated with decreasing rates of myocardial infarctions, there is a limit to this relationship and beyond a certain level, further reductions in DBP begin to be associated with an increasing frequency of fatal myocardial events. This issue however is controversial and there is a possibility that in these studies, the subjects with low DBP had more serious cardiovascular disease to begin with. Nevertheless it seems prudent not to treat SDH in the elderly in an aggressive manner. It is recommended that the treatment goal be to levels just below the defining threshold of 140-160/90 mm Hg.

In sum, the evidence todate indicates that the benefits of therapy definitely outweigh the risks, at least up to the eighth decade of life.

Non-pharmacological Therapy

This includes weight reduction, sodium intake reduction, moderate consistent aerobic exercise and relaxation therapy. They can be of relevance even in the elderly; however more research is needed to determine the ability of elderly people to change lifelong habits.

Of all the modalities, weigh reduction (if a patient is overweight) is the most effective non-pharmacological treatment of hypertension. One third of hypertensive patients will also respond to sodium restriction if the sodium intake can be decreased to below 80 mEq/day. Especially if the DBP is only mildly elevated (between 90 to 100 mm Hg), one may perhaps be more inclined to give these modalities a chance for a few months before resorting to antihypertensive medications.

Drug Therapy

The antihypertensive agents whose efficacy in the elderly has been proven consistently in trials are the thiazide diuretics. In some instances, potassium supplementation is necessary with diuretics. Beta blockers have a more doubtful value in the elderly. In fact, in a recently concluded Medical Research Council trial, thiazide diuretic was found to be better than atenolol[6]. The newer classes of agents such as calcium channel blockers and angiotensin converting enzyme (ACE) inhibitors, while providing more alternatives for therapy, have yet to be studied specifically in this context.

Generally speaking when antihypertensive drugs are prescribed, the starting dosages should be about half the standard dose used for younger patients. One should also await a longer period of time before prescribing dose increases. The adage is, "Start low, and go slow."

Concomitant diseases can play a significant part in the selection of the specific antihypertensive drug. Thus if heart failure is present, a diuretic or ACE inhibitor can be used as first line drugs. With angina, a calcium antagonist or beta blocker are appropriate first choices. If the patient has asthma or chronic obstructive airway disease, beta blockers are contraindicated and with significant peripheral vascular disease, calcium antagonists are suitable as starters. Diuretics should be avoided in the setting of gout and when there is diabetes mellitus, ACE inhibitors are a rational first line choice. If the patient has no other co-existing disease, diuretics can be prescribed because of their proven efficacy.

Other Coronary Risk factors

Since hypertension is very firmly associated with coronary artery disease, one is also obliged, when lowering the elevated blood pressure, to look actively for other co-existing and modifiable risk factors for ischaemic heart disease. In particular, any hypercholesterolaemia should be treated with dietary restrictions and, if necessary, with medication. The smoking habit, if present, should be strongly discouraged.

Indications for Referral

These can be essentially summarized as follows:

- when the hypertension is refractory to therapy
- when a secondary cause for hypertension is suspected
- when a hypertensive emergency or urgency is encountered.

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MALIGNANT HYPERTENSION — AN ABSENT DISEASE?

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Of the many chronic medical problems that bring patients to their physicians, perhaps, none is more common than hypertension. A large data-base developed in the United States to provide post-marketing survey for drug reactions, showed that the common chronic diseases that led to an office visit included hypertension, followed in order by diabetes mellitus, coronary artery disease, chronic obstructive pulmonary disease (COPD), cardiac arrhythmia and congestive heart failure. All these common chronic diseases are frequently and concomitantly associated with hypertension. In fact, this same market analysis has shown these common disease problems were seen in 47% of those patients attending for treatment of hypertension.

In 1974, Dr LP Low and the late Prof CS Seah found that 14% of the people (18 years and above in age) whom they surveyed were hypertensive (BP > 150/90). No recent study has been conducted to show if there is an increase in the incidence or prevalence of hypertension in Singapore. Like any chronic disease that is related to life-style changes and obesity, it is most likely that the prevalence of hypertension in Singapore could be higher than 14% today. Hence, blood pressure measurements of all patients attending our clinic today must be routinely done. This is a very simple procedure, not necessarily very time consuming, and does not inconvenience the patient. In fact most patients would welcome their blood pressure check.

Today, with better understanding of the pathophysiology of hypertenison and a whole host of very potent agents for its treatment, no patient with hypertension should go without proper management. For not to detect hypertension through a very simple measurement of the blood pressure, and to give inadequate treatment is to subject our patient to severe consequences of end-organ damage. We describe a case study of a patient presenting with malignant hypertension.

S, a 28 year old Chinese man was admitted on 29.12.92. He complained of headaches for about 4 months with increasing shortness of breath both on exertion and on lying down. He had vomited intermittently. His vision was progressively blurred and he had begun to experience right leg weakness just prior to admission. He had sought treatment in Indonesia for his complaints but experienced no improvement with medication. There was no previous history of note.

On examination, his blood pressure was measured at 184/124 mmHg. Both eyes showed papilloedema. His heart was enlarged and he was in cardiac failure. There was a triple rhythm. The chest x-ray showed cardiomegaly and pulmonary oedema. Because of his raised blood pressure, cardiac failure and bilateral papilloedema, he was diagnosed to have malignant hypertension. The ECG showed left ventricular hypertrophy with strain.

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Fortunately, today, malignant hypertension is a very rare disease. Our patient is unfortunately, a young Indonesian sailor with chronic glomerulonephritis. His hypertension is subsequent to his chronic glomerulonephritis and a lack of its treatment, even though today there is a whole host of medications that would bring down the raised blood pressure and thus avoid end-organ damage. In the early 1960s, malignant hypertension was often seen. Then the medication for lowering blood pressure was Reserpine (Rauwolfia). This drug was not sufficiently effective. Our prognosis for our patients then was a survival period of 6-12 months only. Today, with newer agents of hypertension therapy, dialysis and renal transplantation, our patient may be restored to better health again.

Hypertension is not a benign disease. Depending on the height of the blood pressure, it eventually leads to end organ—heart (LVH, CHD) blood vessel (fibrinoid necrosis), kidney (glomerulosclerosis) — damage. If the pressure is very high and the rise has been quite abrupt, it often leads to deterioration of health and damage of the resistance blood vessels— small arteries and arterioles. This rise in damaging blood pressure is called malignant hypertension. The pathophysiological lesion is fibrinoid necrosis in these vessels. Fibrinoid necrosis of the vessel wall leads to severe reduction in the lumen of the vessel. Besides an increase of blood pressure, these area of fibrinoid necrosis lead to weakness of the vessel wall (cerebral haemorrhage) and the rapid development of microinfarcts, haemorrhages and papilloedema of the retina — the classical changes in the fundi seen in malignant hypertension. Fibrinoid necrosis, unfortunately, occurs in any form of hypertension, from whatever cause, provided that the pressure is high!

The treatment of hypertension does not only include the lowering of the blood pressure, although this may be easily achieved with all blood pressure lowering medications from diuretics to ACE-inhibitors. The more important aspect of treatment is the prevention of end organ damage from persistent raised blood pressure e.g. fibrinoid necrosis of arterioles and small arteries, left ventricular hypertrophy and renal sclerosis and failure. All these pathophysiological changes portend to life-threatening consequences. Fibrinoid necrosis of the cerebral vessels is the common cause of cerebral strokes and haemorrhage. Left ventricular enlargement has been shown to be a greater predictor of mortality and morbidity than the level of raised blood pressure. It predisposes to ventricular arrhythmia and sudden cardiac death.

The kidneys occupy a special role in hypertension. They may be the cause of the hypertension — as in the patient described above who has chronic glomerulonephritis. They may in turn be damaged by hypertension and then contribute to the cause of the hypertension. Hence, the kidney has a dual role in the perpetration of raised blood pressure. This is because the kidney has an intrinsic hormonal system — the renin - angiotensin - aldosterone system — that contributes to the persistence and aggravation of the blood pressure once it is raised. Angiotensin II is a potent vasoconstrictor and growth promoter. It promoters vascular smooth muscle cell growth and sclerosis of the glomerulus.

Our choice of medication for our patient with hypertension must include drugs that do not increase risk factors e.g. raise levels of lipids and insulin through promotion of insulin resistance (diuretics and ß-blockers). ACE-inhibitors and calcium blockers are today's best choices for the treatment of hypertension. ACE-inhibitors directly block the production of angiotensin II and have been shown to be "renal-protective". They reverse protein loss (microalbuminuria) and prevent further proteinuria after a one year trial in those patients with gross protein loss through the kidneys. Calcium inhibitors are just as useful as ACE-inhibitors. They reduce intracellular calcium and also the response to angiotensin II.

Hypertension, we realise, in a condition, initially, with no symptoms. Despite the popular opinion that headaches are a common symptom of thigh arterial pressure, properly controlled studies of an unselected population have shown this not to be so. Hence, there is no better method of detecting hypertension than measuring the blood pressure. The treatment of blood pressure, today, is tailored to the individual with the disease. It is very amenable to treatment and management. Patient education and understanding of this chronic disease is most essential. But the first step is to measure the patient's blood pressure!

A PRIMARY HEALTH CARE CORONARY RISK SCREENING PROGRAMME: Correlation Of Electrocardiographic Abnormalities With Risk Factors For Coronary Artery Disease

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SUMMARY

The Coronary Risk Screening Programme is a Community Service conducted to identify individuals at high risk of coronary artery disease in Singapore. Data of 2125 persons who underwent screening from 1.1.1990 to 31.6.91 inclusive were analysed and their electrocardiograms coded using the Minnesota code. Of those screened, 18% were below 40 years of age. The majority were non-smokers (85%) and Chinese (92%). Seven out of ten subjects had a positive family history of hypertension and / or diabetes. About half that number had a past history of the same. Six per cent of subjects without past history of hypertension were noted to have elevated blood pressure. Of those without past history of diabetes, 26% had elevated random sugar levels requiring further evaluation. As for the presence of other coronary heart disease risk factors, about half the subjects had mildly raised serum cholesterol levels, and one-third of the subjects were overweight at the time of screening. Eighteen per cent of subjects had major or minor electrocardiographic abnormalities indicative of possible coronary artery disease. Important risk factors associated with electrocardiographic abnormalities include older age, male sex, raised blood pressure, positive past history of hypertension and diabetes, increased body mass index, raised serum cholesterol and history of smoking. Of these, older age, male gender and hypertension were the most significant. An increasing proportion of subjects with electrocardiographic abnormalities were associated with increasing number of cardiovascular risk factors.

Key words:
Coronary risk screening, risk factors, electrocardiographic abnormalities

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INTRODUCTION

In recent years in Singapore, coronary heart disease ranks second to cancer as a principal cause of death[1]. Whilst death due to coronary heart disease is reported to be on the decline in most industrialised countries except eastern Europe[2], the trend in Singapore is still on the increase, and will continue for some years till a decline is seen in the younger birth cohorts[3]. In spite of the great advancements in the field of cardiology with sophisticated treatment methods for coronary artery disease, these are costly and benefit only a small proportion

of patients who are in an advanced stage of the disease. The appropriate focus on management should, therefore, be on prevention.

Primary care physicians, by virtue of being at the "grassroot" level, are well placed to promote primary and secondary prevention. They have an important role in advising individual patients on healthy lifestyle, as well as in the early detection and treatment of coronary artery disease. The tools available to the primary care physician are simple, and consist of a vigilant outlook, clinical methods and basic laboratory investigations.

The Coronary Risk Screening Programme conducted by the Primary Health Division of the Ministry of Health uses just such methods to detect individuals with high risk of developing coronary artery disease. It identifies those with known risk factors for coronary heart disease and institutes preventive measures by advice on dietary modification and lifestyle changes. Those judged to benefit from intervention strategies are referred to secondary care physicians for further evaluation and management.

Risk factors associated with coronary artery disease are well known, and include advancing age, male sex, hypertension, cigarette smoking, elevated serum cholesterol levels, glucose intolerance and obesity[4]. Certain electrocardiographic abnormalities are also known to be associated with an increased risk of coronary artery disease and its complications[5-12].

This study looked at the risk profile of individuals who presented for screening at the Programme, the correlation between risk factors for coronary artery disease and electrocardiographic abnormalities, and discusses the implications for the practising primary care physician.

MATERIAL AND METHODS

Study Population

The study population consisted of a total of 2125 subjects who attended the Coronary Risk Screening Programme at the Toa Payoh Polyclinic between 1.1.1990 and 30.6.1991 inclusive. The Toa Payoh Polyclinic is the only government polyclinic offering this programme for the whole of Singapore. The subjects were from all over Singapore, and were elf-selected in that they made appointments for screening on their initiative.

Study Protocol

The Coronary Risk Screening Programme was started in June 1988 drawing on the experiences of the Framingham Coronary Risk Appraisal Programme[13]. It was focused primarily on individuals aged 40-65 years with no previous history of coronary heart disease. The objectives of the programme were to identify and to reduce risk factors in those screened and to institute intervention measures. The screening procedure was conducted by a trained staff nurse, who took a detailed history with a structured questionnaire, which included information about smoking, past and family history of heart disease, diabetes mellitus, high blood lipids and stroke, among others. The subject was then examined by the nurse, who took the height measured in metres (taken to the nearest 0.01m), weight in kilograms (to the nearest 0.1kg), and blood pressure (after 20 minutes' rest) using the standard mercury sphygmomanometer with the subject seated and the cuff applied to the right arm. Systolic blood pressure was read at the appearance of the first Korotkoff sound and the diastolic pressure at the final disappearance of the Korotkoff sound. A random blood sample was taken and sent to the private laboratory located within the Polyclinic. Blood glucose estimation was done using the Beckman Glucose Analyser, total blood cholesterol by an enzymatic method using Technicon RA 1000 Reagent, high density lipoprotein (HDL) cholesterol was estimated after precipitation of chylomicrons and very low density lipoprotreins (VLDL). A twelve-lead electrocardiogram was taken with the subject lying supine, using a Hewlett Packard Pagewriter II Self Interpretative Electrocardiogram machine.

Electrocardiographic Coding

The electrocardiograms were coded using the Minnesota Code[14,15] by one physician coder. For the purposes of analysis, the ECG abnormalities

Table I: Prevalence of Coronary Risk factors and Electrocardiographis Abnormalities by Gender

RISK FACTORS / ECG ABNORMALITIES	SUBJECTS MALES N= 892 Number (%)		FEMALES N = 1150 Number (%)	
FAMILY HISTORY				
Coronary disease	102	(11.4)	145	912.6)
Stroke	149	(16.7)	143	(12.4)*
Hypertension	386	(43.3)	490	(42.6)
Diabetes	287	(32.2)	319	(27.7)*
Hyperlipidaemia	25	(2.8)	44	(3.9)
PAST HISTORY				
Stroke	4	(0.5)	6	(0.5)
Hypertension	188	(21.1)	179	(8.8)*
Diabetes	51	(5.7)	54	(4.7)
Hyperlipidaemia	64	(7.2)	72	(6.3)
SMOKING HISTORY				
Never smoked	613	(68.7)	1121	(97.5)*
Stopped smoking	139	(15.6)	6	(0.5)*
Current smokers*				
1 - 10 / day	61	(6.8)	12	(1.0)
11 - 20 / day	53	(5.9)	9	(0.8)
>20 / day	25	(2.8)	1	(1.0)
BODY MASS INDEX				
> 25 - 30 (overweight)	295	(33.1)	298	(25.9)*
>30 (obese)	40	(4.5)	59	(5.1)
BLOOD PRESSURE				
Systolic ≥ 160 mmHg	49	(5.5)	86	(7.5)
Diastolic ≥ 95 mmHg	93	(10.4)	61	(5.3)*
TOTAL CHOLESTEROL				
> 5.2 - 6.5 mmol/L	137	(40.4)	500	(43.5)
> 6.5 mmol/L	179	(20.1)	259	(22.5)
HDL CHOLESTEROL				
≥ 1 mmol/L	529	(59.3)	891	(58.3)
RANDOM BLOOD GLUCOSE				
> 5.5 - 11.1 mmol/L	249	(27.9)	284	(24.7)*
>11.1 mmol/L	25	(2.8)	19	(1.7)*
ECG ABNORMALITIES				
Major	76	(8.5)	74	(6.4)
Minor	123	(13.8)	89	(7.7)
Other	94	(10.5)	90	(7.8)

** refers to significant difference in proportion between males and females at = 0.05*

were grouped into major and minor abnormalities according to the criteria set out by the Pooling Project[16] and the Three Chicago Epidemiologic Studies[17]. Codes 1.1 and 1.2 (Q waves indicative of myocardial infarction) were included as major abnormalities. The major, minor and other abnormalities were defined as in Table II.

Statistical Analysis

Statistical analysis was done using the SAS System for Personal Computers Release 6.04, and the Epistat statistical programme, comparing sub-sets of subjects by gender, and the existence of electrocardiographic abnormalities against coronary risk factors.

RESULTS

A total of 2125 subjects attended the Coronary Risk Screening Programme in the study period. Of these the electrocardiograms of 77 subjects (3.6%) were missing. The demographic profiles of those with and without electrocardiograms were similar. Of those with electrocardiograms, five subjects had a history of heart attack or angina. One ECG was coded as 9.8.1, which was 'technical problems which interfere with coding'. These subjects were excluded from further analysis.

Sociodemographic Profile

Of the 2042 subjects included in the analysis, 43.7% (892) were males and 56.3% (1150) were females. A great majority of those screened were ethnic Chinese comprising 91.5% (1875); whilst only 2.4% (48) were Malays, 4.7% (95) Indians, and 1.5% (30) belonged to other races.

Less than four fifths of those screened, 78.0%, (1593) were within the targeted 40-65 age group, with 17.6% (360) less than 40 years of age and 4.4% (89) above 65 years.

About two-thirds, 68.2%, (1391) of those who came for screening had secondary education and above. 35% (127) of those less than 40 years old and 25.5% (429) of those 40 years and above were professionals or held executive posts.

Risk Factor Profile

Table I showed the prevalence of coronary risk factors among the screened population. Seven out of ten of those who came for screening had someone in the family with hypertension and / or diabetes. One in ten had a positive family history of coronary artery disease or stroke. A past history of hypertension, diabetes or hyperlipidaemia was found in one-third of patients.

The majority, 85% (1734) of those screened were non-smokers. Only about 8% (161) were current smokers. About one third of subjects screened were overweight or obese.

Around 14% (289) of all subjects had raised systolic (>160 mmHg) and / or diastolic (>95 mmHg) blood pressure when measured at the time of screening. In the 367 patients with a past history of hypertension, 33% (122) were noted to have raised systolic and / or diastolic blood pressure at that time. Of the remaining subjects without past history, 6% (106 / 1675) were noted to have elevated blood pressure at the time of screening.

As for estimation of random blood sugar levels, it was found to be elevated in 28% (577) of subjects. Of those with a past history of diabetes mellitus, 28% (29 / 104) had a random blood sugar level of >11.1 mmol/L, and could be considered to be unsatisfactorily controlled at the time of screening. Of those without any past history of diabetes, a full quarter (480 / 1919) were noted to have borderline raised blood sugar level of >5.5 - 11.1% requiring further investigations such as the glucose tolerance test and follow-up, and 0.8% (15) were discovered to be frank diabetics (random blood sugar >11.1% mmol/L).

A large proportion of subjects with mildly raised values of serum cholesterol were noted on screening. Forty six percent (936) of the subjects screened had borderline raised cholesterol levels of >5.2 - 6.5 mmol/L, and 21.4% (438) had levels of >6.5 mmol/L.

Table II: Prevalence of Electrocardiographic Abnormalities by Gender

Abnormality Minnesota codes		Male N	(%)	Female N	(%)
MAJOR ABNORMALITY					
1.1	Major Q waves	1	(0.05)	2	(0.10)
1.2	Major Q waves	14	(0.69)	8	(0.39)
4.1 & 4.2	ST depression	1	(0.05)	3	(0.15)
5.1	T wave inversion	2	(0.10)	1	(0.05)
5.2	T wave inversion	21	(1.03)	26	(1.27)
6.1	Complete AV block	0	(0.00)	0	(0.00)
6.2	2° AV block	0	(0.00)	0	(0.00)
7.1	L bundle branch block	1	(0.05)	1	(0.05)
7.2	R bundle branch block	14	(0.69)	9	(0.44)
7.4	Intra-ventricular block	17	(0.83)	4	(0.20)
8.1	Ventricular ectopics	9	(0.44)	24	(1.18)
8.3	Atrial flutter / fibrillation	0	(0.00)	0	(0.00)
	TOTAL	80	(3.93)	78	(3.83)
MINOR ABNORMALITY					
1.3	Borderline Q wave	50	(2.45)	28	(1.37)
2.1	Laxis deviation	28	(1.37)	24	(1.18)
2.2	R zxis deviation >= 120	0	(0.00)	0	(0.00)
3.1	QRS high voltage, L	50	(2.45)	10	(0.49)
3.2	QRS high voltage, R	0	(0.00)	0	(0.00)
4.3	ST depression, bordeline	0	(0.00)	0	(0.00)
5.3	Twave inversion	16	(0.78)	37	(1.81)
6.3	1 AV block	8	(0.39)	6	(0.29)
9.1	QRS low voltage	0	(0.00)	0	(0.00)
	TOTAL	152	(7.44)	105	(5.14)
OTHER ABNORMALITY					
2.3	R axis deviation <120°	14	(0.69)	16	(0.78)
3.3	high R, <3.1	23	(1.13)	6	(0.29)
6.4	WPW pattern	1	(0.05)	1	(0.05)
6.5	Short PR interval	0	(0.00)	6	(0.29)
6.6	Aberrant AV conduction	0	(0.00)	4	(0.20)
7.3	Incomplete RBBB	22	(1.08)	17	(0.83)
7.5	R-R' pattern	39	(1.91)	34	(1.67)
8.2	Ventricular rhythm abnormality	0	(0.00)	1	(0.05)
8.7	Sinus tachycardia	3	(0.15)	11	(0.54)
8.8	Sinus bradycardia	10	(0.49)	6	(0.29)
8.9	Other arrhythmias	0	(0.00)	1	(0.05)
9.2	ST elevation ≥ 1mm	13	(0.64)	4	(0.20)
9.3	P wave ≥ 2.5mm amplitude	1	(0.05)	3	(0.15)
9.4.1	Early transition	592	(28.99)	834	(40.84)
9.4.2	Late trasition	15	(0.73)	17	(0.83)
9.5	T wave >12mm amplitude	4	(0.20)	0	(0.00)
	TOTAL	737	(36.11)	961	(47.06)

Note: Each ECG may have more than one abnormality.

Electrocardiographic Abnormalities

As for electrocardiographic abnormalities, 18% (362 / 2042) of those screened had a major or minor abnormality indicating myocardial ischaemia (Table II), and these were in people with no previous history of coronary artery disease. Of note in particular are the small number (15 males, and 10 females) with major Q waves (codes 1.1 & 1.2) indicating past silent through-and-through myocardial infarction. Other abnormalities not indicative of increased risk of coronary artery disease, but quite commonly found include right axis deviation <120° (1.5%), high R less than Code 3.1 especially in males (males 1.1%, females 0.3%), incomplete right bundle branch block (2.0%), and R-R' pattern (3.6%). Early transition was particularly common (males 29.0%, females 40.8%).

As codes 9.4.1 for early transition and 9.4.2 for late transition were very common occurrences, and, as these do not carry any prognostic significance for coronary artery disease, they were included in the 'normal' group for further analysis.

Univariate analyses of major and minor electrocardiographic abnormalities with coronary risk factors are shown in Table III. Apart from the non-modifiable risk factors of age and sex, other factors such as family history of hypertension and ischaemic heart disease were not found to be

Table III: Association of Coronary Risk Factors with Major and Minor ECG Abnormalities

Risk Factor Screened	Number	ECG Abnormilties major (%)	minor (%)	P-value (Chi-sq)
Sex				
male	892	8.5	13.8	
female	1150	6.4	7.7	0.000
Age				
< 40 yr	360	2.8	7.8	
≥ 40 yr	1682	8.3	10.9	0.000
Body Mass Index				
< 25	1346	7.1	8.8	
≥ 25	696	7.8	13.5	0.007
Systolic BP				
< 160	1907	6.8	10.3	
≥ 160	135	15.6	11.1	0.001
Diastolic BP				
< 90	1888	6.7	9.9	
≥ 90	154	15.6	16.2	0.000
Total Cholesterol				
< 5.2	590	6.8	8.8	
≥ 5.2	1452	7.6	11.0	0.029
HDL Cholesterol				
< 1.00	532	9.8	12.4	
≥ 1.00	1510	6.5	9.7	0.015
History of hypertension				
Yes	367	14.7	14.4	
No	1675	5.7	9.5	0.000
History of diabetes				
Yes	105	13.3	16.2	
No	1937	7.0	10.7	0.005
Smoking history				
Never	1734	6.9	10.2	
Past	145	12.4	11.7	
Current	161	7.4	11.2	0.021

significantly associated. Of the modifiable risk factors, blood pressure, obesity and diabetes were more closely associated with electrocardiographic abnormalities than serum cholesterol levels or smoking. When all the factors were entered in the full model for stepwise multiple logistic regressions, only older age, male gender and hypertension emerged as significant predictors of both major and minor electrocardiographic abnormalities. The adjusted odds ratios are indicated in Table IV.

About 70% of subjects screened had at least one risk factor, regardless of whether they had in addition electrocardiographic abnormalities as well. The likelihood of ECG abnormalities increased with increasing numbers of risk factors (both modifiable and non-modifiable) present in the individual, as shown in table V.

Table IV: Significant Risk Factors of ECG Abnormalities (Major and Minor) from Step-Wise Multiple Logistic Regression

Variable	Odds Ratio 95% Confidence	Interval
MAJOR ABNORMALITY		
Sex	1.54	1.08 - 2.20
Age	1.54	1.28 - 1.81
History of hypertension	2.39	1.62 - 3.52
MINOR ABNORMALITY		
Sex	2.01	1.48 - 2.73
Age	1.31	1.13 - 1.52
Diastolic blood pressure	1.75	1.07 - 2.86

Odds ratios for each risk factor adjusted for all other risk factors.

Table V: Prevalence of ECG Abnormalities by Number of Modifiable and Non-Modifiable Risk Factors

ECG abnormalities	Number of risk factors#			Number of modifiable risk factors		
	None	1-3	4-9	None	1-2	3-5
Number of subjects	124	1206	712	311	1303	428
Presence of major or minor abnormalities	7 5.6%	179 14.8%	176 24.7%*	39 12.5%	215 16.5%	108 25.2%*

\# *Include modifiable and non-modifiable risk factors*
* *p value (Chi square for trend) = 0.000*

Modifiable risk factors:
1. body mass index >25 kg/m^2
2. raised blood pressure (systolic >160 mmHg and diastolic >95 mmHg)
3. raised serum total cholesterol (>5.2 mmol/L)
4. raised random blood sugar level (>5.5 mmol/L)
5. positive smoking history

Non-modifiable risk factors:
1. Age ≥ 45 years
2. Male sex
3. Race (non-Chinese)
4. Family history of coronary heart disease

DISCUSSION

The results of this study raise several interesting points for the practising clinician. Firstly, although most of the subjects were the right people screened, there was a minority who were outside the intended population of the screening programme. About one-fifth of those screened were not in the target age group of 40 - 65; more females than males came forward to be screened; and there was a lower representation by Malays and Indians when compared with the racial composition of Singapore (1990) of 77.7% Chinese, 14.1% Malays, 7.1% Indians and 1.1% other ethnic groups[18]. Mortality data on coronary heart disease suggested deaths to be higher in the older age group, in males and among Malays and Indians compared to the Chinese[19]. Some of the possible explanations for the presence of this situation include a larger participation form professionals and executives, usually less than 40 years old, better educated, more knowledgeable and hence more interested in their health. The cost and necessity to pay a fee of S$50.00 is a deterrent. The operational time of the Programme during office hours may be inconvenient for those who are working. The screening of people with low risk makes poor epidemiological and economise sense, but is a reality in primary care, where patient autonomy and right rank very high. These important task of the primary care physician in such an instance is to heighten the awareness of those at high risk while at the same time discourage those at low risk from coming for screening.

The second point of note is that a large proportion of subjects found to have borderline abnormal results and were asymptomatic. Half (50%) had mildly raised serum cholesterol levels, one-third were overweight or obese, and one quater had elevated random blood glucose levels not amounting to frank diabetes. These subjects stand to gain with lifestyle and behaviour modification, such as a change in diet to one of lower intake of simple sugars, saturated fats and cholesterol, and an increase in frequency of exercise. Those with elevated blood glucose levels should be further investigated to determine if they are diabetic or have impaired glucose tolerance. Follow-up and proper management of these subjects will result in fewer developing frank diabetes and complications, and also coronary artery disease.

A third finding was that blood pressure emerged as the single strongest factor associated with ischaemic electrocardiographic abnormalities, which in themselves are powerful predictors of subsequent major coronary events[5, 7-12, 20]. Hypertension is a major risk factor for the development of cardiovascular disease and the prime risk factor for coronary artery disease[21]. In coronary artery disease, taken together with the fact that the population attributable risk for hypertension (51%) is far above that for cholesterol (18%)[22], the necessity to achieve and maintain good blood pressure control in hypertensives cannot be overemphasised. In this study is was found that control of hypertension was not satisfactory in 33% of those with history of hypertension. The primary care physicians play an important role in the continuing care of chronic disease such as hypertension in ensuring a stringent control of blood pressure by patient education, motivation, compliance and regular follow-up. The same can be said for the follow-up management of diabetes.

Lastly, electrocardiographic abnormalities are commonly encountered in any screening electrocardiogram. About 27% of electrocardiograms in this study were found to have some form of abnormality. Some of the abnormalities which carry no prognostic significance for coronary artery disease, such as mild right axis deviation or minor intraventricular conduction defects (e.g. R-R' pattern) can be noted as a matter of interest and dismissed. As early transition was especially commonly

encountered, it should always be looked out for before interpreting the significance of any tall R in the left chest leads. As for abnormalities which were included in the major and minor categories (18%), the problem faced by the practising primary care physician is always whether or not to refer the subject for further evaluation by the cardiologist. This is especially difficult with abnormalities such as ST-T wave changes which have been attributed to a variety of causes including ethnic groups[23] and emotion[24], and at the same time have been showed to be indicators of increased risk in coronary artery disease[25-26] and especially in males[27]. The presence of additional coronary risk factors in the same person warrants further referral. The more risk factors present, the severer the grade (e.g. higher blood pressure, diabetic instead of impaired glucose tolerance), the greater the indication for further cardiac evaluation. Ultimately the decision will be on a case to case basis, tailored to the needs of each person,. taking into consideration his freedom of choice.

CONCLUSION

In the preventive strategies directed at the development of coronary artery disease, the reduction of risk factors is the key to a decrease in morbidity and mortality in the community. This effort indirectly leads to a reduction in health care costs. Two complementary strategies are required for effective prevention[28], namely the public health education of the population, directed at those with low and moderate risk, and the identification of individuals at high risk, for intensive evaluation, counselling and treatment.

Primary care doctors are in unique position to carry out preventive activities, as in the screening and early detection of risk factors and the advice on life-style changes. Regular follow-up, supervision and monitoring are important, and this is possible at a relatively low cost at the primary health level.

Coronary Risk Screening Programme aims to introduce intervention and risk factor modification early in aborting coronary events. In the words of those who estimate the impact of such a programme, 'population-wide gains in life expectancy form single risk-factor modifications are modest, but gains to individuals at risk can be more substantial'[29], at the same time minimising cost and wastage. 'Even though the population effect would be minimal... a focus on certain subgroups, such as those aged 40-59 years, can achieve substantial results within those groups,'[30]. The concern of primary care doctors in the coronary risk screening programme is thus to identify potential persons who would benefit most from the programme, to detect potential risk factors in high risk groups and to intervene by modifications and treatment of potential patients of coronary artery disease.

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UNEXPECTED RECOVERY FROM SERIOUS ILLNESS: HOW THEY DID IT

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INTRODUCTION

The causes for cancer, arthritis, diabetes, multiple sclerosis — all the incurable — have eluded medical science.

The mysteries of the crippler and killer diseases will be solved only by careful study of the very unusual people who defy the odds by recovering from serious illnesses.

Claude Bernard and Pasteur carried on a controversy that spanned decades, with Bernard maintaining that illness hovered about continuously, and could not take root unless the terrain, i.e. the body, was receptive. He claimed the appropriate focus of study must therefore be the terrain itself. Pasteur, of course, along with Koch and others of his generation, was involved in the valiant attempt to identify and conquer the germ, and thereby rid humankind of disease. The story goes that on his death bed, having succumbed to one of the germs he had tried in vain to eradicate, Pasteur conceded that Bernard, alas, was right. the microbe is nothing, the terrain everything.

CASE REPORTS OF UNEXPECTED RECOVERY FROM SERIOUS ILLNESS

William Boyd, Emeritus Professor of Pathology, University of Toronto in his Monograph: 'The Spontaneous Regression of Cancer' (1966) noted: "The all-important biological fact is that something has happened, as a result of which the cancer has been destroyed in whole or only in part. In both cases there is evidence of some defensive force, healing process, immunological reaction, call it what you will. Some day we must discover the secret of this mysterious process and apply it with success to the treatment and the cure of cancer. In the meantime it might be well to follow Brunschwig's suggestion of establishing a registry for living patients whose histories are acceptable as instances of spontaneous regression. It is pleasant to think that we have a patron saint for spontaneous regression, namely St. Peregrine, the cancer saint. As a young priest, he suffered a cancer of the leg and was scheduled for operation. The night before, he prayed fervently to be saved from amputation and he dreamed that he was cured. On awakening, he discovered that it was more than a mere dream — he was completely cured. He lived to his eightieth year, dying in 1345 without further evidence of cancer. During his life, he dedicated himself to the relief of such suffering, and he was canonized St. Peregrine in 1726. Perhaps we could term tumours that disappear spontaneously 'St. Peregrine Tumours".

Tilden Everson, Clinical Professor of Surgery, and Warren Cole, Professor of Surgery and Head, University of Illinois, in a study and abstract of reports in the world medical literature and of personal communications concerning spontaneous regression of malignant disease titled "Spontaneous regression of cancer" (1966), a total of 176 cases of cancer considered to have adequate documentation (including histologic confirmation of malignancy), pointed out in their conclusion: "The existence of spontaneous regression of cancer, in at least some cases, supports the concept of biologic control of cancer and reinforces the hope that a more satisfactory method of treating cancer than surgery or irradiation may be found in future years."

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Klopfer (1957) reported: "Mr. Wright had a generalised far advanced malignancy involving the lymph nodes, lymphosarcoma. Eventually the day came when he developed resistance to all known palliative treatments. Also, his increasing anaemia precluded any intensive efforts of X-ray or nitrogen mustard, which might otherwise have been attempted. Huge tumour masses the size of oranges were in the neck, axillae, groin, chest and abdomen. The spleen and liver were enormous. The thoracic duct was obstructed, and between one and two litres of milky fluid had to be drawn from his chest every other day. He was taking oxygen by mask frequently, and our impression was that he was in a terminal state, untreatable, other than to give sedatives to ease him on his way. In spite of all this, Mr. Wright was not without hope, even though his doctors most certainly were. The reason for this was that the new drug that he had expected to come along and save the day had already been reported in the newspapers! Its name was "Krebiozen" (subsequently shown to be a useless, inert preparation). Then he heard in some way that our clinic was to be one of a hundred places chosen by the Medical Association for evaluation of this treatment. We were allotted supplies of the drug sufficient for treatment of 12 selected cases. Mr. Wright was not considered eligible, since one stipulation was that the patient must not only be beyond the point where standard therapies could benefit, but also must have a life expectancy of at least three, and preferably six months. He certainly did not qualify on the latter point, and to give him a prognosis of more than two weeks seemed to be stretching things. However, a few days later, the drug arrived, and we began setting up our testing program which, of course, did not include Mr. Wright. When he heard we were going to begin treatment with Krebiozen, his enthusiasm knew no bounds, and as much as I tried to dissuade him, he begged so hard for this "golden opportunity", that against my better judgement, and against the rules of the Krebiozen committee, I decided I would have to include him. Injections were to be given three times weekly, and I remember he received his first one on a Friday. I did not see him again until Monday and thought as I came to hospital he might be moribund or dead by that time, and his supply of the drug could then be transferred to another case. What a surprise was in store for me! I had left him febrile, gasping for air, completely bedridden. Now, here he was, walking around the ward, chatting happily with the nurses, and spreading his message of good cheer to any who would listen. Immediately I hastened to see the others who had received their first dose at the same time. No change, or change for the worse, was noted.

Only in Mr. Wright was there brilliant improvement. The tumor masses had melted like snowballs on a hot stove, and in only those few days, they were half their original size! This is, of course, far more rapid regression than the most radio-sensitive tumor could display under heavy X-ray given every day. And we already knew his tumor was no longer sensitive to irradiation. Also, he had no other treatment outside of the single useless "shot". This phenomenon demanded an explanation, but not only that, it almost insisted that we open our minds to learn, rather than try to explain. So, the injections were given three times weekly as planned, much to the joy of the patient, but much to our bewilderment. Within 10 days (Mr. Wright) was able to be discharged from his death-bed, practically all signs of his disease having vanished in this short time. Incredible as it sounds, this terminal patient, gasping his last breath through an oxygen mask, was now not only breathing normally, and fully active, he took off in his plane and flew at 12,000 feet with no discomfort! This unbelievable situation occurred at the beginning of the Krebiozen evaluation, but within two months, conflicting reports began to appear in the news, all of the testing clinics reporting no results. At the same time, the originators of the treatment were still blindly contradicting the discouraging facts that were beginning to emerge. This disturbed our Mr. Wright considerably as the weeks wore on. Although he had no special training, he was, at times reasonably logical and scientific in his thinking. He began to lose faith in his last hope which so far had been life-saving and left nothing to be desired. As the reported results became increasingly dismal, his faith waned, and after two months of practically perfect health, he relapsed to his original state, and became very gloomy and miserable. But here I saw the opportunity to double-check the drug and maybe, too, find out how the

quacks can accomplish the results that they claim (and many of their claims are well substantiated). Knowing something of my patient's innate optimism by this time, I deliberately took advantage of him. This was for purely scientific reasons, on order to perform perplexing questions he had brought up. Furthermore, this scheme could not harm him in any way, I felt sure, and there was nothing I knew anyway that could help him. When Mr. Wright had all but given up in despair with the recrudescence of his disease, in spite of the wonder-drug which had worked so well at first, I decided to take the chance and play the quack. So deliberately lying, I told him not to believe what he read in the papers, the drug was really most promising after all. What then, he asked was the reason for his relapse? "Just because the substance deteriorated on standing", I replied, "a new super-refined, double-strength product is due to arrive tomorrow which can more than reproduce the great benefits derived from the original injections." This news came as a great revelation to him, and Mr. Wright, as ill as he was, became his optimistic self again, eager to start over. By delaying a couple of days before the shipment arrived, his anticipation of salvation had reached a tremendous pitch. When I announced that the new series of injections was about to begin, he was almost ecstatic and his faith was very strong. With much fanfare, and putting on quite an act (which I deemed permissible under the circumstances), I administered the first injection of the doubly potent, fresh preparation — consisting of fresh water and nothing more. The results of this experiment were quite unbelievable to us at the time, although we must have had some suspicion of the remotely possible outcome to have even attempted it at all. Recovery from his second near-terminal state was even more dramatic than the first. Tumor masses melted, chest fluid vanished, he became ambulatory, and even went back to flying again. At this time he was certainly the picture of health. The water injections were continued, since they worked such wonders. He then remained symptom-free for over two months. At this time the final AMA announcement appeared in the press — "nationwide tests show Krebiozen to be a worthless drug in the treatment of cancer." Within a few days of this report, Mr. Wright was readmitted to the hospital in extremis. His faith was now gone, his last hope vanished, and he succumbed in less than two days.

Mr. Wright's experience tells us that it was his total belief in the efficacy of the worthless drug, Krebiozen, that mobilized a healing placebo response by activating all the major systems in healing, especially the immune system."

In 1976 Ainslie Meares wrote to the Medical Journal of Australia seeking referral of suitable cancer patients to conduct a small private experiment to see if the progress of their condition could be influenced by intensive meditation. Only 3 patients made themselves available and two of them soon dropped out of the experiment. The third patient, a single woman, age 49, had continued steadfastly in the experiment for six months. She had pathologically proven carcinomas of both breasts. She had been given radiotherapy to both breasts with initial regression of the tumours. However, they soon recurred and she developed radiologically proven metastases in the spine She underwent oophorectomy. Her condition rapidly deteriorated and she developed ascites. In the six months the patient had attended more than 100 sessions of intensive meditation. She also practised by herself many hours of meditation at home. There was a dramatic change of her condition after intensive meditation. At follow-up 18 months later she was found to be well, strong, free of pain and had gained 25 lbs in weight and led a very active life.

In 1978 Meares reported in the Medical Journal of Australia a case of 'Regression of Osteogenic Sarcoma Metastases associated with Intensive Meditation'. The patient, aged 24 underwent a mid-thigh amputation for osteogenic sarcoma 11 months before he first saw Meares. He had visible bony lumps of about 2 cm in diameter growing from the ribs, sternum and the crest of the ileum, and was coughing up small quantities of blood. There were gross opacities in the X-ray of the lungs. The patient had been told by a specialist that he had only two or three weeks to live. Now after two and a half years he had moved to another state to resume his former occupation.

Norman Cousins was suffering form a serious arthritic and rheumatoid-like collagen disease diagnosed as ankylosing spondylitis with high fever and pain, and taking 40 tablets of asprin a day with no relief. He documented his recovery in

his book "The Anatomy of an Illness as Perceived by the Patient" (1979), in which he treated himself with generous, positive doses of good humour (primarily in the form of old Marx Brothers movies and reruns of Allen Funt's Candid Camera). Then again, years later, he suffered from a massive heart attack, from which he totally recovered. Cousins summarized his experiences of personal healing by emphasizing that positive attitudes and emotions can affect the biochemistry of the body to facilitate rejuvenation and health. His book "The Healing Heart" was published in 1983. He subsequently became adjunct Professor at UCLA's School of Medicine, studying the biochemistry of emotions.

Martin Rossman reported in his book Healing Yourself (1987): "When I was in my second year of practice, working in the country medical clinic, a middle-aged woman named Edna came in for a checkup. She was a likable, talkative person who said she was there because "the doctors worry me so and tell me I better keep an eye on my blood pressure". Her chart revealed that she had been diagnosed with a precancerous condition of the uterine cervix over two years previously, and the gynecologists she had seen wanted to take biopsies and remove the affected areas. Edna had turned this recommendation down four times, and each successive note by her gynecological consultants was more frustrated and concerned in tone. There was mention of possible psychopathology and irrational beliefs about healing. When I asked Edna why she was unnecessarily risking her life, she smiled broadly and told me that "Jesus will heal me, and I don't need surgery". She said she prayed and talked to Jesus every day, and he promised he would heal her if she put her trust in him. I asked her how she communicated with Jesus, and she told me, "I see him when I pray, and he talks to me just like we're talking now." I again explained the medical concerns I and the other doctors had about her. Then I told her I had no doubt that Jesus could heal her if he wanted to, but wondered how long it would take. She was a bit surprised when I asked her if she would be willing to get in touch with him and ask him if he'd agree to heal her in the next six weeks. She closed her eyes, and after a few minutes smiled and nodded her head. "Yes, he says he can heal me in six weeks."She agreed to have another pelvic examination and Pap Smear at that time and also agreed to have a cone biopsy if the Pap Smear was still abnormal. "But it won't be," she said, "I know that now." And she left, smiling more widely than ever. I felt good to have been able to get a commitment out of her to have a biopsy if her prayer proved ineffective. Six weeks later she returned. Her cervix looked normal on examination. Three days later her Pap smear report came back, perfectly normal. Edna's story points to the potent healing effects of faith and belief."

Why do some patients get well and others die when the prognosis is the same for both?

From experience with hundreds of patients at their Cancer Counselling and Research Centre in Texas, the Simontons have found a scientific basis for the will to live. The psychological techniques that the Simontons developed do not replace standard medical procedures, but are used in conjunction with them. The object is to help patients help themselves by creating the best environment — internal and external — for their own recovery. Specifically they describe how an individual's reaction to stress and other emotional factors may have contributed to the onset and progress (or recurrence) of the disease and give detailed instructions to help patients recognise and deal with these elements in their lives., Their treatment program — including techniques for learning a positive attitude, relaxation, visualisation, goal-setting, managing pain, exercising, and building an emotional support system — will not only enhance patients' chances for recovery but will substantially improve the quality of life. Positive expectancies and a habit of psychological self-awareness and self-care can play a significant role for anyone in maintaining health, and for the cancer patient in getting well again. Their results were amazing.

Dr Bernie Siegel, an American surgeon, has showed how 'terminal' patients in his care have taken control of their illness — how, through the healing power of love, they have changed, enriched and prolonged their lives far beyond medical expectation through techniques such as meditation, visualisation and relaxation.

In the 1970s, Spiegel and his colleagues at Stanford University combined group therapy with hypnosis and relaxation, in weekly sessions over a year, for

a group of women with advanced breast cancer. An equal number, who were randomly assigned to a control group received the same medical care but did not participate in group therapy. The goal of the group was simply to help the women cope with the stress of a painful, terminal illness, and it succeeded in that. But what surprised Spiegel and his colleagues was that the women in the support groups lived an average of 18 months longer. Spiegel published his results in the lancet in 1989.

Esther Goshen-Gottstein (1988) documented her husband, an eminent linguist in his 60s, who suffered severe brain damage as a result of complications during a coronary bypass operation. Against all the statistics, against all reasonable expectations, he recovered . He emerged form a coma of many weeks' duration. He finally achieved almost complete neurological and intellectual recovery. Within 18 months of his terminal brain damage, he had written two books and countless scholarly articles, attended conferences, lectured widely, and been appointed to visiting professorships at Harvard and Brandeis Universities. His dramatic recovery was made possible by the intensive rehabilitative efforts, the support and resourcefulness of his family, who never lost faith in the possibility of a complete recovery and, not least, by his own indomitable will.

PERSONAL CASES

A 37-year old man was very much distressed when he was diagnosed having cancer of his stomach and requiring surgery. He was very depressed, fearful and unable to sleep. Two hypnotic sessions turned him from a state of complete despair to one of confident hope. He required no night's sedation before surgery, the amount of thiopentone used was much reduced, he needed only one single injection of omnopon after the operation, and his post-operative convalescence was remarkable. The operation was done in 1964. The pathology report showed poorly differentiated adeno-carcinoma of stomach, and there was right regional node involvement. After the operation the relatives asked the surgeons about the prognosis and they were told that the patient might not live for more than four months. Now after nearly 30 years he is still alive, fit and well, and recently has been checked by the specialist with no evidence of cancer.

A 44-year old married woman underwent radical mastectomy for cancer of the breast. After the operation she was very miserable, complaining of insomnia, gastric pain, irritating cough and was very depressed. Hypnotherapy relieved her of all these symptoms. She was then conditioned for the deep X-ray therapy which she was going to have the following week and of which she was doubtful, fearful and reluctant to undergo. Hypnotherapy removed all her doubts and fear, and suggestions were given that she would be able to take the deep X-ray well and would have no discomfort or burns. Relaxation and visualisation were used. She went through deep X-ray therapy 20 times with no discomfort or burns. During the conditioning sessions the will to live was strongly instilled into her. The operation was done in 1967 and she is now still alive and well.

A 55-year old seaman had painless haematuria. IVP showed a big hypernephroma of the left kidney. He was persuaded to go for an operation to cure him. He accepted the advice and was soon operated on. Immediately after the operation while he was recovering in the ward, he was told that he had been successfully operated. He had an uneventful post-operative convalescence and underwent a course of deep X-ray therapy. The pathology report showed clear cell renal carcinoma with chronic pyelonephritis in the adjacent renal tissue. There was an embolus in the renal vein. The operations was done in 1971. He lived well for 11 years and died of secondaries in the lungs in 1982.

Perhaps, a Registry of Unexpected Recovery from Serious Ilness should be developed, which may give further insight into hitherto poorly recognised psychological, pathological, psychological, and other factors responsible in healing.

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HOW TO MANAGE CARDIAC ARRHYTHMIAS? A PRACTICAL GUIDE

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SUMMARY

Cardiac arrhythmias are not uncommonly seen in our daily practice. Management of arrhythmias is both challenging and interesting. Some of these arrhythmias do not require further investigations nor treatment. It is very important not to scare the patient. The role of a family practitioner is to identify patients who will require further investigations and treatment. The goals of treatment must be clearly explained to the patients to prevent disappointment. It is extremely important to identify the high risk group for sudden arrhythmic deaths. In general, patients with large hearts and poor left ventricular function have poor prognosis even if treated aggressively. Furthermore, they are at high risk for proarrhythmic effects. Remember, if you have to use anti-arrhythmic drugs (i) use drugs that you are familiar with and (ii) use the minimal number of drugs to improve compliance. Lastly, identify and inform patients who may benefit from curative therapy (radiofrequency catheter ablation) instead of longterm anti-arrhythmic drugs.

INTRODUCTION

It is very likely that you will encounter patients with cardiac arrhythmias in your daily practice. Thus, it is important to be to *recognise* the different types of arrhythmias and then *manage* them appropriately. This article will focus on the fundamentals of arrhythmia management especially on **ventricular ectopics, atrial fibrillation and supraventricular tachycardias**. There will be just a brief mention of the less common cardiac arrhythmias.

TYPES OF CARDIAC ARRHYTHMIAS

As indicated in Table 1, there are many types of cardiac arrhythmias. However, not all are dangerous nor require treatment. Some of them are no more than just a nuisance. Therefore, it is important to recognise potentially dangerous arrhythmias, their modulating factors and substrate. It is very important to define the arrhythmogenic substrate and modulating factors as they determine the potential outcome of the underlying arrhythmias. For example, ventricular ectopics in the acute phase of myocardial infarction is potentially dangerous. Whereas, it is usually benign in otherwise healthy adults. In general, **tachyarrhythmias are** always of concern and present early to the doctors. **Remember, ventricular fibrillation and ventricular tachycardia can kill a patient quickly!**

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Table 1. Types of Cardiac Arrhythmias

Atrial
- ectopics
- flutter
- tachycardia
- fibrillation

Atrioventricular
- atrioventricular junctional/nodal re-entrant tachycardia (AVJRT / AVNRT)
- atrioventricular re-entrant tachycardia (AVRT)

Ventricular
- ectopics
- tachycardia
- fibrillation

When to Start Patients on Anti-arrhythmic Drugs?

Not all patients with cardiac arrhythmias require treatment. Table 2 outlines the principle of anti-arrhythmic therapy. It is very important to define the endpoints of therapy as this will decide the choice of drugs. Remember, it is not always possible to achieve the same end points for patients with the same cardiac arrhythmia.

Table 2. Uses of Anti-arrhythmic Drugs

To treat symptomatic arrhythmias
- interfering with day to day activities
- **"malignant"** symptoms i.e. near-syncope and syncope

To reduce the morbidity of
- thromboembolism associated with atrial fibrillation
- syncope associated with tachyarrhythmias

To reduce the mortality of
- sudden deaths from VT or VF
- sudden deaths from atrial fibrillation with antegrade conduction via a fast conducting accessory pathway

Why Not Use Anti-arrhythmic Drugs for All Types of Cardiac Arrhythmias?

Anti-arrhythmic drugs should only be used after careful consideration of the potential risks of the underlying arrhythmia that one intends to treat and the potential serious complications of the drugs (Table 3). Remember, anti-arrhythmic drugs can cause more harm than benefit to the patients! In general, ventricualr tachyarrhythmias nearly always warrant special and early attention because of the high attendant risks. Beware of patients with poor left ventricular function and complex arrhythmias. These patients have significant risks with any anti-arrhythmic drugs that we commonly use.

Table 3. Problems with Anti-arrhythmic Drugs

They are not uniformly effective because of
- lack of patients' compliance
- their limited pharmacological properties

They can cause more harm

(a) by depressing the LV function
- class IA drugs (procainamide)
- class IC drugs (flecainide)
- beta blockers (sotalol)
- calcium antagonists (verapamil)

(b) by their proarrhythmic effects (worsening the cardiac arrhythmias) especially in patients with high grade ventricular arrhythmias and impaired LV function

CHOICE OF ANTI-ARRHYTHMIC DRUGS

The choice of anti-arrhythmic drugs (Table 4) depends on many factors (Table 5). This will minimise the risk of complications from anti-arrhythmic therapy. Some of the anti-arrhythmic drugs have multiple actions e.g. amiodarone and beta blockers. In other words, a single drug can be used to treat different types of cardiac arrhythmias.

Table 4. Types of Anti-arrhythmic Drugs

Class IA	procainamide, quinidine, disopyramide
Class IB	lignocaine, mexiletine, phenytoin
Class IC	flecainide, propafenone
Class II	beta blockers (sotalol)
Class III	amiodarone, bretylium, sotalol
Class I	calcium antagonists (verapamil, diltiazem)

Table 5 Factors influencing the Choice of Anti-arrhythmic Drug

Types of arrhythmias

- atrial
- atrioventricular
- ventricular

End points of therapy

- prevention of arrhythmias
- termination of arrhythmias
- slowing the rate of the tachyarrhythmias

Left ventricular function

- normal
- impaired (mild to severe)

Associated pathologies

- "structurally" normal heart
- dilated heart from any aetiologies
- post myocardial infarction
- hypertension
- metabolic disturbances i.e. potassium, calcium, magnesium
- post open heart surgery

PRATICAL APPROACH TO COMMON CARDIAC ARRYTHMIAS

ATRIAL FIBRILLATION (Figure 1)

Not all patients with atrial fibrillation require treatment. Before you treat this arrhythmia, try and establish the underlying cause, e.g.

- thyrotoxicosis
- chronic obstructive airway disease
- hypertension
- ischaemic heart disease
- "large" heart from any cause
- valvular heart disease

Furthermore, empirical treatment of atrial fibrillation in the elderly patients can unmask sick sinus syndrome with traumatic results!

It is "easy" to initiate treatment for patients with atrial fibrillation. However, you must identify your targets of therapy. The **primary goal** is to convert atrial fibrillation to sinus rhythm. However, if this cannot be achieved, you should then focus your attention to reduce the risk of thromboembolism and cardiac failure associated with the fast heart rate. Remember to use drugs that you are familiar with and drugs with the least side effects (Table 6).

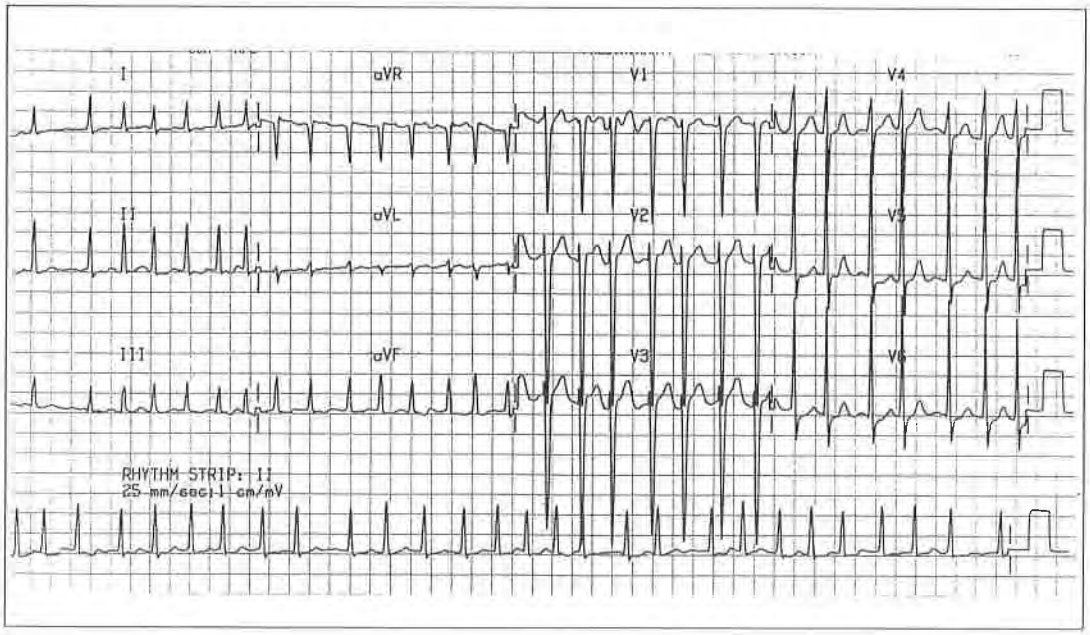


Fig 1 : 80 year old male with palpitations. ECG confirmed atrial fibrillation with rapid ventricular response. In addition, there is left ventricular hypertrophy.

Table 6. Rationale of Treatment of Atrial Fibrillation

1. **Prevent recurrences with atrial related agents**
   - digoxin
   - sotalol
   - class IA (quinidine, procainamide, disopyramide)
   - class IC (flecainide, propafenone)
   - amiodarone

2. **Convert to sinus rhythm**
   - atrial related agents (see above)
   - synchronised DC cardioversion (only selected patients)

3. **Slow down the ventrivular rate during atrial fibrillation**
   - **AV nodal depressant agents**
     digoxin
     beta blockers (propranolol)
     calcium antagonist (verapamil, diltiazem)
     amiodarone
   - **catheter radiofrequency ablation** of the AN node in cases resistant to medical therapy (rarely indicated)

4. **Prevent thromboembolism** (this usually determines the morbidity and mortality of this arrhythmia)
   - **anti-platelet agents**
     salicylates, dipyridamole, ticlopidine
   - **anticoagulations**
     warfarin (low versus therapeutic dose)

Note: It is not necessary to treat patients with chronic atrial fibrillation with anti-arrhythmic drugs if
* the patient is asymptomatic
* the heart rate is within the normal range
* there is no overt heart failure

SUPRAVENTRICULAR TACHYCARDIA (SVT) (Figure 2)

This is not an uncommon arrhythmia. **Not all patients with SVT require investigations and treatment.** Patients should only be referred for further investigations (i.e. electrophysiological study) if they agree to ablation therapy. **Symptomatic** patients should **not** be subjected to lifelong anti-arrhythmic therapy because of its low compliance rate and side effects. Hence, any patient with **regular narrow complex tachycardia** who requires longterm drug therapy is a suitable candidate for ablation therapy.

Rationale of Therapy

* **cure,** especially for patients with WPW syndrome and AVNRTs
  - catheter radiofrequency ablation (treatment of choice)
  - surgical ablation (seldom performed)

* **prevent recurrences with oral medications**
  - generally not effective because of lack of compliance

* **acute termination of SVT**
  - vagal manoeuvres (first choice)
  - drug of choice (i/v verapamil 2.5 to 15 mg)
  - pacing (selected patients)
  - synchronised DC shock (if haemodynamically compromised)

VENTRICULAR ECTOPICS

A majority of patients with ventricular ectopics do not require anti-arrhythmic drugs. Before you commence anti-arrhythmic drug therapy, (i) identify patients who at high risk to develop ventricular tachyarrhythmias e.g. patients with recent myocardial infarction and patients with large hearts and impaired left ventricular function. (ii) define the modulating actors that can lower the threshold for ventricular tachyarrhythmias e.g. hypokalaemia and ischaemia and (iii) in selected patients, define the ventricular profile using ambulatory ECG and exercise stress test.

Treat ventricular ectopics IF

1. patient is symptomatic
   - start with reassurances (++)
   - if this fails, consider an anxiolytic or a beta blocker
   - use anti-arrhythmic drugs until after consulting your cardiologist
   - avoid the use of anti-arrhythmic drugs in pregnant patients

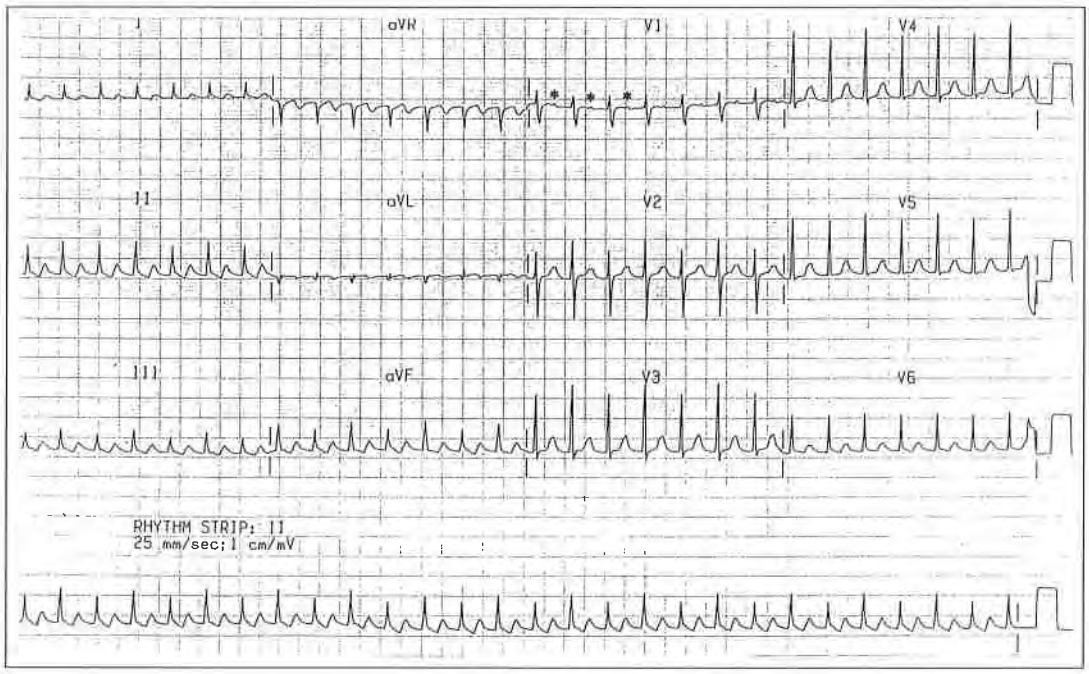


*Fig 2 : 30 year old female with recurrent palpitations. Typical regular narrow complex tachycardia. Note electrical alternans (alternating small and large QRS complexes) and retrograde P waves (marked *). This is compatible with accessory pathway reentrant tachycardia.*

2. there is a high risk of sudden death
   - post myocardial infarction with large anterior myocardial infarction (beta blockers / amiodarone)
   - patients with large heart and impaired LV function (amiodarone)
   - hypertrophic cardiomyopathy (amiodarone)

Remember, antiarrhythmic drugs can kill if used indiscriminately!!

ATRIAL ECTOPICS

These are "irritating" arrhythmias. Occasionally, they can trigger atrial tachyarrhythmias (atrial flutter-fibrillation) in susceptible patients (chronic lung diseases and large hearts). Anti-arrhythmic drug treatment is seldom required.

VENTRICULAR TACHYCARDIA

This is seldom seen in general practice. However, it is very important to recognise this arrhythmia (Figure 3). It is more common in patients with enlarged or "sick" hearts. The prognosis is guarded for patients with enlarged hearts. Treatment is essential but can be difficult. There are significant proarrhythmic effects with anti-arrhythmic drugs. Hence, it is better to let your cardiologist manage them.

However, patients with ventricular tachycardia and structurally normal hearts have good prognosis. These are often mistaken as supraventricular tachycardia with aberration as they are often found in the younger age group and who are clinically well (figure 4). They respond well to sotalol or calcium antagonists (e.g. verapamil). Occasionally, more potent anti-arrhythmic drugs may be required e.g. amiodarone. If this fails, consider catheter radiofrequency ablation.

VENTRICULAR FIBRILLATION (Figure 5)

This is invariably a **lethal** arrhythmia. Thankfully you do not see them unless you happen to own an ECG machine! The only treatment is immediate defibrillation!

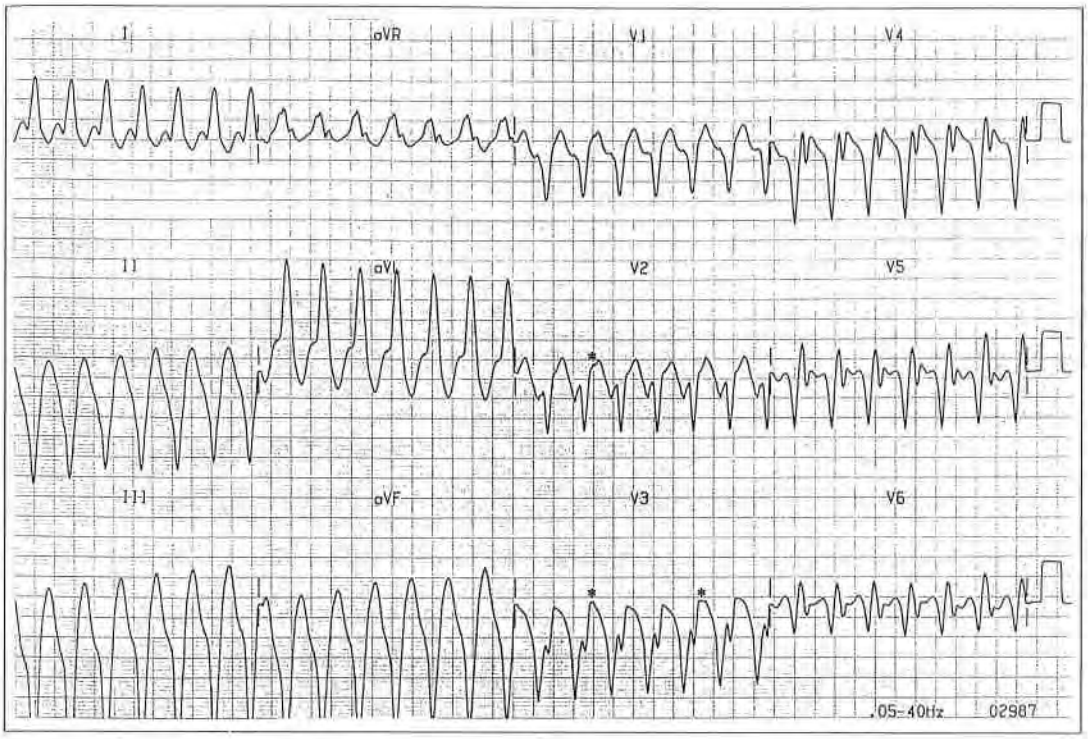


*Fig 3 : 50 year old male with palpitations and dizziness. Past history of myocardial infarction Note the wide complex tachycardia typical of ventricular tachycardia. The QRS complexes are wider than 120 msec and there are AV dissociations (marked *).*

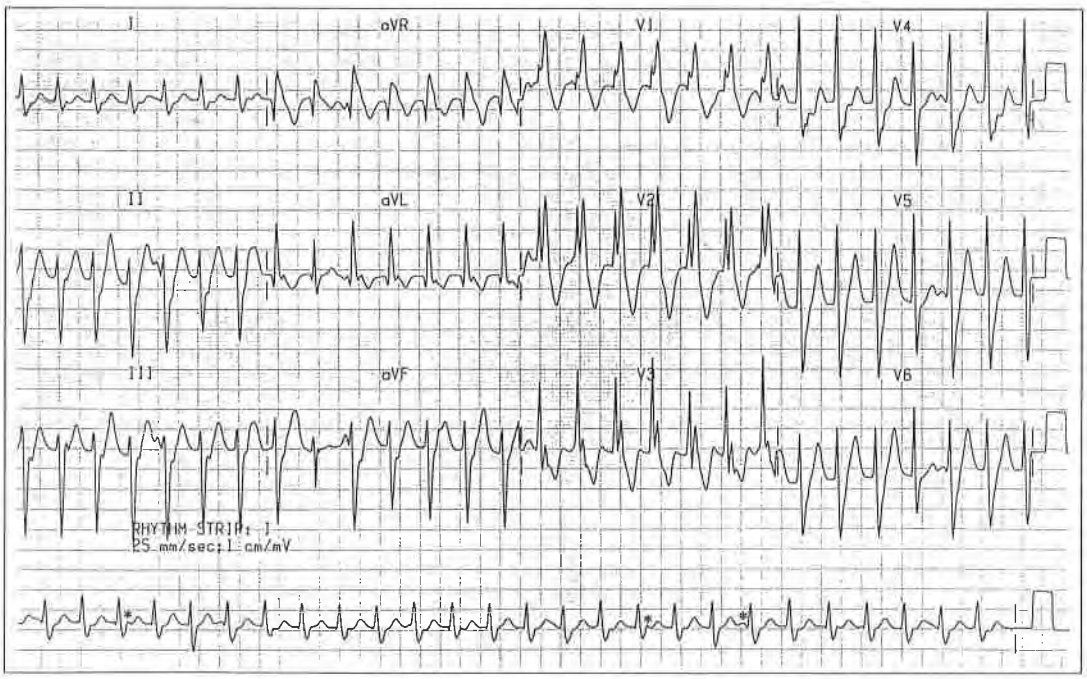


Fig 4 : 25 year old male with recurrent palpitations. Clinically normal. Note the wide complex tachycardia with RBBB pattern. The ECG features of ventricular tachycardia are (i) superior (left) cardiac axis (ii) R wave smaller voltage than S wave in V6 and (iii) AV dissociation.

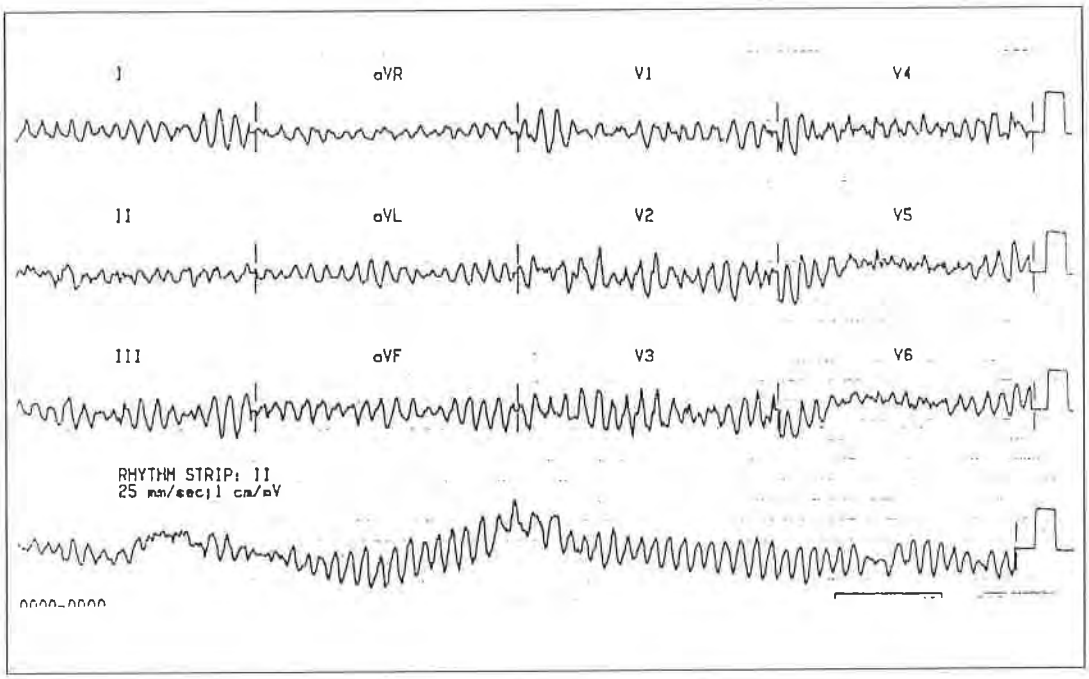
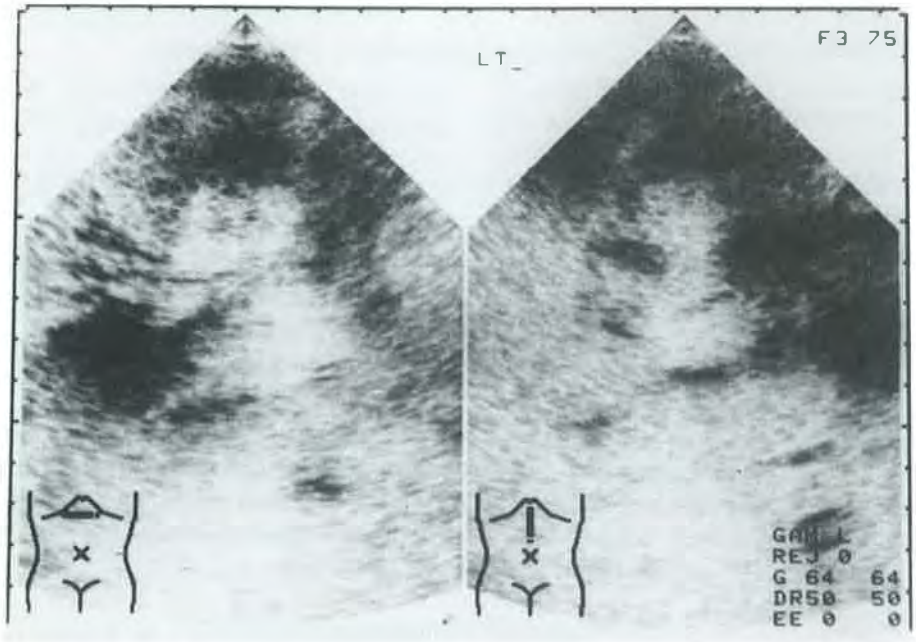


Fig 5 : This 60 year old male presented with severe chest pain. While recording the ECG at the A&E, he fitted! He was succcessfully defibrillated. Note the classical bizarre QRS complexes of ventricular fibrillation.

X-RAY QUIZ

Submitted by Dr Ng Hweena
MBBS (S'pore), FRCR (UK)

History:- Ultrasound images of the liver of a 60 year old man. These sections show liver parenchyma. The gall bladder images show no evidence of calculi.



What is the abnormality?
What are your differentials?

Radiologic Clinic
#05-09 The Promenade
300 Orchard Road
Singapore 0923

Answers on next page

X-RAY QUIZ ANSWERS

These are scans over the midline, probably the left hepatic lobe. The ultrasound shows two areas of increased echogenicity with acoustic shadowing posteriorly.

No areas of cavitation identified.

These suggest calcified lesions.

Causes of Hepatic Calcifications

A. If small and multiple

1. *Healed granulomas* – from previous tuberculosis or histoplasmosis.

B. If curvilinear and patchy

1. *Hydatid cysts* may calcify extensively. Calcification may also be present within daughter cysts.

2. *Abscesses* – usually amoebic and may be along the margins forming rim echogenic layer.

C. Localized in a mass

1. *Metastases* – calcified metastases are uncommon.
But colloid carcinoma of the rectum, colon or stomach calcify most frequently. It may be amorphous, flaky, stippled or granular. These may be solitary or multiple. Calcification may follow radiotherapy or chemotherapy.

Metastases from malignant bony lesion.

2. *Hepatoma* – rare to calcify. Calcifications are also punctate, stippled or granular. It is seen in 10% of cases.

Trauma – Haematomas calcify inhomogeneously.

Vascular – Calcification within thrombus at portal vein or aneurysm of the hepatic artery.

The lesions for this patient turned out to be calcified metastases from carcinoma of the colon.

WORLD HEALTH ORGANIZATION PUBLICATIONS

NEW BOOK ANNOUNCEMENTS

WOMEN AND TOBACCO
C. Chollat - Traquet et al
1992, xi + 128 pages
ISBN 92 4 156147 5

This book explores the many special issues that surround the impact of tobacco use on the health and well-being of women. Noting that most tobacco control programmes fail to address the distinct needs of women, the book concentrates on the identification of gender-specific factors that help explain why girls and women smoke and how tobacco damages their health. The book, which uses data from a wide range of sources, makes a special effort to cover all dimensions of the problem, ranging from conditions in developing countries that deter female smoking to the reasons why women may find it more difficult to quit than men. The impact of the tobacco industry's efforts to recruit female smokers is also considered.

The book's six chapters give detailed country-specific statistics revealing changing trends in female tobacco use and related morbidity and mortality, explain how the constituents of tobacco produce dependence and damage health, linking female smoking to a greatly increased risk of eight forms of cancer and six other major diseases, explore the question of why women start and continue to smoke, revealing the importance of gender difference in the physiology and social psychology of smoking and describe the actions that need to be taken, by government, policy-makers, health professionals, and women's groups, to protect girls and women from starting to smoke and to plan cessation programmes specifically designed to reach and influence women.

GUIDELINES FOR CHOLERA CONTROL
1993, vi + 61 pages

This book sets out the facts and advice needed to guide public health actions in response to an outbreak of cholera.

The opening chapters provide basic information about the disease, common sources of infections, and measures for prevention, with emphasis placed on the paramount need for safe water supplies. Other chapters describe what national programmes should do to be prepared for an outbreak of cholera, outline the actions to take at the earliest stage of an outbreak, and provide guidelines for the management of patients, including advice on the use of oral rehydration therapy and antibiotics. The remaining chapters cover measures for preventing the spread of an outbreak, the epidemiological investigation of an outbreak, the role of the laboratory in diagnosis, and long-term preventive activities.

The second half of the book, which consists of five annexes, provides brief advice on the construction of a ventilated improved pit latrine, followed by a detailed step-by-step guide to the management of cholera patients, a selection of sample health education messages, and nine rules for safe food preparation to prevent cholera. The book concludes with guidelines describing a simple and rapid method for the isolation and identification of *Vibrio cholerae* O1 in diarrhoeal stools.

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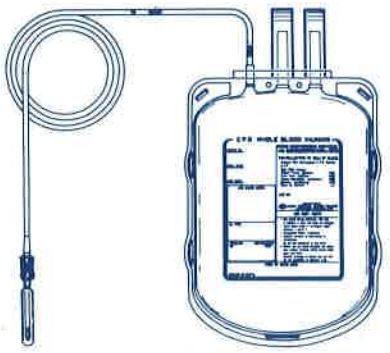
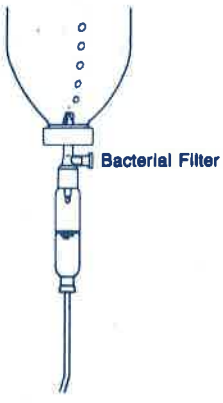
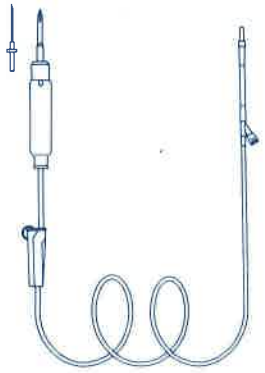


Blood Bag

Transfusion Set



Bacterial Filter



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Further information is available on request.

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