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1. Skoutakas V.A. et al. Review of diclofenac and evaluation of its place in therapy as a nonsteroidal antiinflammatory agent. Drug Intell Clin Pharm. 22, 850 (1988).
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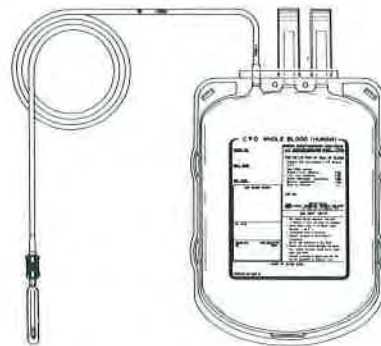
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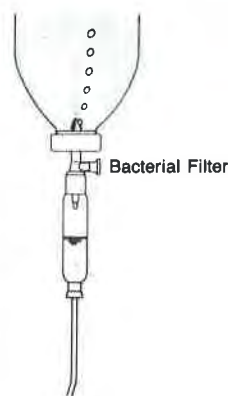
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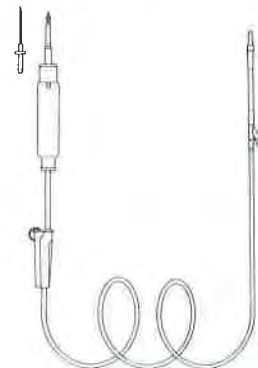
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PRIVATE MEDICINE AND GATEKEEPING

With the rise of private medicine, it has become harder and harder to hold down the surging tides of escalating costs. Patients' expectations become heightened by the numerous newspaper reports of new hearts, new fertility, and the many wonderful things that hi-tech medicine can bring. They too, want to be part of the number in being beneficiaries of such medical advances, not realising that not all can benefit, will benefit and worse of all can afford the experience. The concept of the need for allocation of scarce resources may not be clear to them or to those in the midst of hi-tech medicine. Company managers are increasingly vociferous of the question "Is this really necessary?"

ALTERNATIVE HEALTH DELIVERY SYSTEMS

To deal with the scenario of escalating costs, alternative health delivery systems are being pressed into service: health insurance schemes, health maintenance organisations (HMOs), preferred provider organisations (PPOS) and other innovative schemes where some health care provider becomes the contracted gatekeeper. America leads the way in these innovative schemes.

AN EXERCISE IN FUTILITY?

The question is whether these will solve the problem. Probably not until and unless there is a rethinking and reordering of priorities by doctors, politicians and health care users on the very philosophy of health care. Questions like the following should be addressed: Is it a necessity or luxury? A private or public good? Should it be made as affordable as possible? Should the accounting be based on outcome or based on dollars

generated? Meanwhile, somebody has to keep the gate against the surging tides of rising costs. The family physician is often wooed to be the contracted gatekeeper.

PERILS OF THE CONTRACTED GATEKEEPER

Two papers on gatekeeping in the *Journal of Family Practice* make illuminating reading. The affirmative view that the doctor can be the gatekeeper is that the same professionalism that has fended off change in the control of medical care over the years may minimize some the dilemmas and potential conflicts of interest inherent in the gatekeeper-based system.¹

The opposing view is provided by Gayle Stephens.² His answer to the idea of a contracted gatekeeper system is unequivocally "No!". It is based on more than two years' experience as a gatekeeper in two health maintenance organisations (HMOs) for about 900 subscribers. His belief is that the gatekeeper role, in present day and age of America, is hopelessly conflicted, ethically unmanageable, clinically naive, professionally ungratifying, and historically unnatural for a family physician. He said that on balance, gatekeeping has turned him and his patients "into a gang of wheelers and sharpies, each trying to outfox the other for petty privileges and paltry savings, both manipulating the 'Plan' for bigger ticket items like year-end kickbacks, expensive elective medical procedures, and a better contract at the next renewal date".

Several encounters occurred each week, mostly around requests for referrals. "Patients wanted to see dermatologists, ortho-

paedists, allergists, otolaryngologists, psychologists, even chiropractors for minor, chronic problems that I felt competent to treat. It was difficult to say no to a referral, then shift into the role of a therapist."

"Many requests for referral came by telephone, which was clearly against the rules, and were handled by the nurse, who incurred resentment by insisting that the patient had to see the physician before a referral could be given. Some of the requests were for retroactive referrals, also against the rules... The nurse spent in a good part of each day on the telephone about administrative issues arising from the gatekeeper role".

"The collective impact of these negative encounters, though each in itself might have been minor, created a climate of suspicion, cynicism, readiness to fight, and a sense of being used that permeated my office in a way that I had not known before".

WHO SHOULD KEEP THE GATE?

In the atmosphere of private medicine, Stephens has this to say: "If watch-dogging is a necessary job in current and future systems of medical care, let it be done by technocrats who have no stake in intimacy".

GLG

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EDITORIAL

TO CURE OR TO CARE

"To cure sometimes, to relieve often, to comfort always"

— Ambrose Pare

With the advances in medical treatment, doctors are taught the science of curing but have lost the art of caring. Preston pointed out the irony that the ability to cure which is a historically recent achievement has made the modern physician less inclined to healing. He found that medical students are taught to react to medical problems with curative measures — how to correct diabetic acidosis, how to reconstruct a deformed limb, or how to treat hypertension — but not how to recognise nonphysical sources of distress and the need for healing.¹

Consequently, doctors tend to suffer from a tunnel vision with regard to the psychological and emotional distress of their patients. The Future General Practitioner drew attention to this deficiency of hospital training:

"The doctor may insist on focussing on certain aspects of the patient's problem because they are the easiest for him to handle. He will then refuse to allow the patient to tell him anything else, or refuse to hear.

To obtain his greatest satisfaction, the doctor usually wants to find a patient with a serious, acute illness that has interesting features - elicited by him with great acumen - and one that responds rapidly, completely and gratefully to proper therapy.^{2"}

CURING AND HEALING

We need to make a distinction between curing and healing. Curing has been defined by Preston as the elimination or alleviation of disease through physical processes.

Healing, on the other hand, is defined as the emotional state in the patient that may or may not coexist with a cure and is effected through emotional and psychological mechanisms.

Curing a patient from his disease does not necessarily mean that he has been healed from his illness. Neither does healing requires that the patient be cured of his disease.

For example, a worker may be cured of his fractured tibia but may continue to experience pain from a compensation neurosis. On the other hand, there is the patient who is healed of her migraine through prayer.

Preston noted that curing depends on biophysical remedies while healing is often the result of caring. Benoliel used the terms cure and care to differentiate between the two distinct goals in medical care. She used the term "cure" to refer to the diagnosis and treatment of disease and the term "care" to the assessments and interventions made with regard to the welfare and well-being of the patient. In practical terms, cure is "doing things to" the patient while care is "doing things with" the patient.

THE DISEASE AND THE ILLNESS

Cure is orientated towards the biophysical processes while care is directed towards the effects of the disease on the patient. The failure to recognise the difference between disease and illness is often the cause of a dysfunctional medical consultation. The patient may feel ill but the doctor may be unable to find the pre-

sence of any disease process. For example, a backache arising from muscle spasm caused by emotional stress is just as real to the patient as the backache caused by a slipped disc. On the other hand, the doctor may find a disease process such as hypertension without the patient feeling ill at all.

While it is important to treat the underlying physical pathological processes, it is just as important to recognise and manage the emotional distress experienced by the patient. Preston noted that the emphasis on biophysical abnormalities directs attention to disease and not health. Consequently, the patient with psychosomatic symptoms but who has no disease is of no interest to the physician because there is no abnormality to treat. Such a patient is also in danger of being treated unnecessarily with drugs or surgery when the need for caring is not recognised by doctors who are preoccupied with a cure.

Nixon observed that the doctor who is preoccupied with diseases in organs and parts is unable to understand what is going on in the mind of the patient nor teach him how to cope with the demands of his environment in a healthier way. He also faces difficulty in dealing with specific ill-health which are caused by failure to cope, inability to adapt, loss, bereavement or anger.

TO CURE AND TO CARE

Virshup observed that a definition of success in medicine that is restricted to curing patients, restoring health and preventing health builds in a high probability of failure. He drew attention to the two parallel goals in medicine. On one hand we have to treat the disease process which we as doctors have been trained to do. On the other hand, we have to help the patient to understand, adjust to and cope with his illness as well as to facilitate the healing process of the body. Both these processes are critically, inextricably intertwined and interdependent. However, when the second goal is neglected through a focus of attention on the first, there will be a lack of success in the treatment of the patient. Virshup made a distinction between our goals and our responsibility in treatment.

Our responsibility lies not in the product but in the process. Our goal may be health, or cure, or staving off death, but our responsibility is for competence and for doing our best, for demonstrating care, and for helping the patient cope with his illness.

Preston attributed most of the problems of health care to a mismatch between the expectations of the patient and the doctor. Most patients want or need healing (in the form of simple caring) but the doctors of today want to supply curing. Furthermore, the patient misinterprets the doctor's attempts to cure as an expression of caring while the doctor misinterprets the patient's plea for caring as a need for curing. In his view, it is the lack of caring in physicians that drives people to quacks and healers who cannot cure but supply healing and it is unfortunate that the patient has to choose between the two.

The failure to care is also a result of the myth of omnipotence in the doctor perpetuated by the act of curing which leads a doctor to believe in his own personal powers of healing and to take a god-like stance. Preston noted that physicians tend to be arrogant in order to sustain belief in their own omnipotence and omniscience.

However, Meares recommended the adoption of an attitude of sincere humility instead of a veneer of omnipotence in order to achieve the necessary prestige in the eyes of the patient which will facilitate the process of healing.

In order to provide both cure and care, Preston recommended the development of primary care physicians who will serve as patient-advocate physicians. His vision of primary health care should serve as a vision for all of us in general practice:

"Society should urge the medical profession to develop patient-advocate physicians, primary-care physicians whose economic and professional incentives would be patient oriented. Each patient should have such a physician who would be responsible for all medical treatment, except for emergencies when he is not

available or not expert enough to counsel the patient. This new breed of physicians would have the authority within the profession to make, with the patient, all medical decisions, including those requiring the use of advanced technology and specialised surgical procedures. He would use the expert advice of specialists, who would appropriately be seen as technicians able to diagnose disease in their special fields and perform necessary procedures. The primary physician would be expert at acquiring and interpreting data so as to be able to make well-informed decisions in all areas of medicine. With the resources of modern medicine to draw on but free from the myth of the healer, he could perform the three most humanitarian functions of a doctor: to comfort, to teach, and to provide curative therapy when necessary."¹

PK

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TOTAL CARE IN STROKES

Tjia H MBBS (S'pore), MMed (Int Med) (S'pore)

Epidemiologic evidence indicates both the frequency and severity of cerebrovascular disease has diminished over the past 20 years. Such progress has probably been due to the control of blood pressure and other stroke risk factors some of which have been more recently suggested e.g., smoking, alcohol, diet and coffee consumption.

RISK FACTORS

Some risk factors appear to be definite and others possible.

- 1) Hypertension has long been recognised as the most important risk factor for stroke, both for ischaemic and primary intracerebral haemorrhage. The stroke risk rises with both systolic and diastolic blood pressure. This is true for both sexes and at all ages. Even though systolic and diastolic pressures are closely related, the risk of stroke is just as high in systolic hypertension per se.¹ So logically, all hypertensive patients need to be treated to reduce their stroke risk.
- 2) Smoking increases the stroke risk to a much lesser extent than increasing the risk of peripheral vascular and coronary heart disease. The effect of stopping smoking is not known at present.
- 3) Diabetes mellitus is a risk factor for ischaemic stroke in large vessel disease but is questionable in small vessel disease. The risk of diabetes mellitus in haemorrhagic stroke is yet to be clarified. There is evidence that the control of hyperglycemia can reduce the severity of cerebral damage during the acute stroke period.
- 4) Transient ischaemic attack (TIA) increases the individual's stroke risk con-

siderably. The most widely accepted estimate of the incidence of stroke after TIA's is that of the Mayo Clinic experience which is 7% per year, whereas the normal stroke rate in an asymptomatic population of >60 yrs is about 1% per year.³

- 5) There is evidence that acute alcoholic episodes and chronic alcoholism are important risk factors for both ischaemic and haemorrhagic strokes. However occasional or modest use of alcohol was not shown to increase a person's stroke risk.⁶
- 6) Obesity is a risk factor for heart disease and may be a secondary risk factor for stroke.
- 7) Heart disease is an important risk factor for ischaemic stroke.

Pathogenesis

The pathogenesis of cerebral ischaemic has been postulated to be the result of the following mechanisms.

A Carotid artery disease:

- 1) Hemodynamic compromise occurs when a tightly stenotic or occluded carotid artery results in poor distal, perfusion especially when there is inadequate collateral circulation.
- 2) Artery to artery embolisation occurs especially when there is an ulcerated plaque at the carotid bifurcation.

B Cardiac emboli resulting from heart disease or arrhythmias account for about 10% of all cerebral infarction.

C Lacunar infarction which is a special group of small deep cerebral infarction most often encountered in the setting of hypertension. They occur as a result of occlusion of the penetrating branches of the lenticulo-striate arteries

*Consultant Neurologist and Head
Department of Neurologist
Tan Tock Seng Hospital
Moulmein Road Singapore 1130*

**Symposium paper presented at the
2nd Annual Scientific Conference**

and branches of the posterior cerebral artery and involves mainly the basal ganglia, internal capsule, thalamus and brainstem.

D Intracerebral haemorrhages including subarachnoid haemorrhage.

Strokes have traditionally been classified as thrombotic, embolic or haemorrhagic. However in spite of increasing sophistication in diagnostic technology there appears to be an increasing number of patients especially young patients who have infarcts of unknown cause (IUC).

In a retrospective study of 713 stroke patients from the Stroke Data Bank in the New York Neurological Institute 24% of patients were in the category of IUC.⁴ The most important characteristics of this group of patients were their young age, presence of a superficial infarct, prior transient ischaemic attack, low weakness score and presentation with a non-lacunar syndrome. Continued use of this category of IUC may help further clarify other yet unknown risk factors and stroke subtypes and may allow newer mechanisms of ischaemic strokes to be uncovered.

LABORATORY INVESTIGATIONS

1) *Ultrasound*

The B-mode ultrasound is particularly useful for screening patients with suspect extracranial carotid artery disease. The transcranial doppler will complement this technique as it can evaluate blood flow in the circle of Willis and posterior circulation.

2) *Echocardiography and Holter monitoring*

These techniques are used in the evaluation of patients with sufficient indication of a cardiac cause of embolism.

3) *Neurovascular Imaging Techniques*

i) CT scans show intracerebral haemorrhage and areas of focal infarction. In the case of infarction, the CT scan may not show up an area of hypodensity in the first 24-48 hours of infarction.

- ii) Magnetic resonance imaging (MRI) is superior to the CT scan in cases of early localisation of lacunar and brainstem infarction.
- iii) Digital subtraction angiography and conventional angiography. The indications for angiography are cases of suspected aneurysms, arterio-venous malformations, and arterial dissection.

The diagnosis of a stroke is often straight forward. Patients may fall into the following categories.

- 1) Transient Ischaemic Attack.
- 2) Progressive Strokes.
- 3) Completed stroke — mild/severe.

The progressive stroke is said to occur in a patient when his deficits become more severe or when his deficits spread to involve contiguous areas over a period of 24-36 hours. The pathophysiologic event in progressive strokes has been attributed to extension of a thrombus.

The management of a stroke patient would depend on complicating neurological and medical conditions.

Coma in strokes is usually the result of massive hemispheric infarcts with edema, brainstem infarctions and intracerebral and subarachnoid haemorrhage. Seizures or even status epilepticus can occur in a fresh stroke.

STRATEGIES TO PREVENT AND TREAT A STROKE

The first line in stroke prevention is detection and adequate treatment of manageable risk factors.

Over the past 10 years, numerous clinical trials have advocated aspirin in the primary and secondary preventions of strokes.

The largest study on the role of aspirin in the primary prevention of stroke is the US Physicians Health Study. Alternate day therapy with 325mg aspirin given to 22,000 healthy US Physicians reduced the occurrence of myocardial infarction, but

with no detectable reduction in the occurrence of strokes.⁵

In the secondary prevention of stroke, aspirin after transient ischaemic attacks has been shown to reduce the incidence of stroke and death especially in males.⁶ The optimum dose of aspirin is still not known and conflicting opinion persists.

Benefit has been reported with 1200 mg daily aspirin. No trial has yet been completed on whether low dose aspirin (300 mg) is beneficial. It is not clear at present whether aspirin can prevent stroke recurrence after cerebral infarction.

Ticlopidine has recently been available as a new antiplatelet agent. In the Canadian American Ticlopidine study, ticlopidine reduced the stroke recurrence by 30% in those with mild strokes. It appeared to be beneficial in both men and women.⁷

To date there are about 10 major studies on the use of anticoagulants as medical treatment for transient ischaemic attacks conducted over the past 25 years. Survival rates appear to be unaffected by therapy but all TIAs ceased. Stroke incidence ranged from 0-7% in the treated group vs 15-53% in the control groups. These patients are usually treated for 3 months after which the slope of events is essentially parallel over the next 5 years.

The Extracranial-Intracranial Bypass (EC-IC Bypass) introduced more than 10 years ago, aimed at improving cerebral blood flow in symptomatic patients with carotid occlusion or surgically inaccessible stenosis was found to be not beneficial in a multi-centre study published two years ago.¹¹ The most pressing question now is the role of Carotid Endarterectomy (CEA) in stroke prevention. No clinical trial has adequately assessed the benefits or demerits of CEA. Most would agree today that anticoagulation with heparin and warfarin is reserved for an acute stroke occurring as a result of cardiac embolisation.^{2,11} Others may extend its use in carotid dissection and the so-called progressive strokes.¹

REHABILITATION

Rehabilitation of stroke victims is an important medical and social need. The sequelae of strokes may take the form of neurological cognitive or behavioural deficits. A large proportion of stroke patients experience to a certain extent improvement in neurological deficits within a few months of the stroke. Rehabilitation is to assist and accelerate recovery of impaired functions.

Hence the aims of rehabilitation are:

- 1) Improvement of motor, speech, cognitive and other impaired functions.
- 2) Mental and social readaptation of patients.
- 3) Where possible a return to the activities of daily living.

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THE ROLE OF THE FAMILY PHYSICIAN IN PRE-PREGNANCY COUNSELLING

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SUMMARY

A gigantic step has been taken in recent years with the improvement in the science of maternal and child care. This has resulted in marked reduction in maternal and infant morbidity and mortality. Further scientific advances in this area is unlikely to result in further spectacular improvement in the near future. Preventive medicine should take its place in medical care and pre-pregnancy counselling should not be neglected.

Pre-pregnancy counselling, defined as the physical and mental preparation for childbearing of both parents, helps the couple enter pregnancy at the optimal time and offers alternatives to couples who should not procreate naturally.

Family doctors are of paramount importance as they are most likely to be the first doctors to encounter couples who seek medical advice.

INTRODUCTION

In recent years we have witnessed marked improvement in maternal and child care. This is shown by a striking reduction in maternal and perinatal mortality and morbidity. Our achievements have been made possible by rapid advances in the field of medical science. We have identified risk factors in pregnancy, developed new methods of fetal and maternal monitoring during pregnancy and labour as well as introduced active and intensive management of neonates. All these have resulted in improvement in the survival of the very sick or very small babies.

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Further advances will continue. The question we have to ask ourselves today as health providers is whether we should continue to screen women only after they have become pregnant, after the added risk of conception has been imposed on them or should counselling and risk identification begin before conception?

The reproduction of our species should not be haphazard, and preparing for childbearing is a large component of health education. Preventive medicine should be applied whenever possible and pre-pregnancy counselling is one of the methods. The concept of pre-pregnancy counselling is not a new one. It is defined as 'the physical and mental preparations for childbearing of both parents'. Pregnancy entered into by couples who are prepared will have a better chance of good outcome than one that comes at a time when the woman's body is ill prepared for carrying the fetus. A decision must be made as to the ideal timing for conception, the added risk that pregnancy may imposed on some couples and indeed the question of whether pregnancy should be undertaken at all when the risk is considered unwarranted should be tackled prior to pregnancy. An option should be offered to couples who should not embark on pregnancy at all.

In spite of the stress on the need for early antenatal visits, antenatal care still starts too late for some. Embryogenesis occurs from three to seven weeks after the last menstrual period and is usually completed by the tenth week of gestation. Few women seek medical attention before six or eight weeks of gestation and by that time, teratogenesis would have occurred. Furthermore, for genetically high-risk women pregnancy is not the optimal time for referral to the geneticist for they have already lost the chance of benefiting from other available options. Counselling at this stage will only increase anxiety for the couple.

AIMS OF PRE-PREGNANCY COUNSELLING

The main aims of pre-pregnancy counselling are:-

1. To screen for hitherto undiagnosed medical conditions which when untreated may make pregnancy hazardous.
2. To control prior to pregnancy medical problems which may affect pregnancy outcome.
3. To assess and advise couples whose previous pregnancies were complicated by obstetric problems.
4. To investigate couples with recurrent pregnancy failure.
5. To provide genetic counselling to couples at risk of producing children with inheritable disorders.
6. To reassure couples who for whatever reason are fearful of pregnancy.

SCREENING FOR PRIOR UNDIAGNOSED MEDICAL PROBLEMS

Certain medical disorders may be asymptomatic. A common condition is hypertension. A woman is likely to embark on pregnancy without being aware that she suffers from hypertension. She will not realise the problems that she may encounter during the pregnancy and that hypertension may be aggravated by pregnancy and conversely, that it may complicate the pregnancy.

ASSESSMENT AND CONTROL OF EXISTING MEDICAL PROBLEMS

Other conditions like diabetes mellitus and cardiac diseases are usually previously diagnosed and control of these illnesses will improve perinatal outcome.

Diabetes mellitus is associated with an increased perinatal mortality and morbidity. This is because of an increased incidence of congenital anomalies, the possibility of unexplained stillbirths, birth trauma associated with macrosomia and many neonatal problems. Not only is the fetus at an increased risk, a poorly controlled pregnant diabetic is more likely to develop ketoacidosis which when not recognised and treated promptly, results

in the death of the mother.

Management of cardiac disorders has changed in recent decades and as cardiac surgery becomes more accepted, correction of any surgically correctable cardiac disorder prior to pregnancy should be the aim. On the other hand, there is a group of patients in whom pregnancy is associated with an unacceptably high maternal mortality, for example patients suffering from Eisenmenger's syndrome, who should be advised frankly not to conceive at all.

Another good example of a condition that should be controlled prior to pregnancy is epilepsy. Most anti-convulsant drugs are known teratogens and it would certainly be beneficial for the patient to be converted and stabilised with the lowest dosage of the least teratogenic drug prior to conception. Once pregnancy has occurred, any change in the drug used will be useless as the teratogenic effects would have occurred by the time the pregnancy is diagnosed. It will in fact be dangerous to change the drug during pregnancy as it may precipitate a seizure.

The number of similar conditions is big and it is impossible to make an exhaustive list.

ASSESSING PATIENTS WITH PRIOR PREGNANCY COMPLICATIONS

A number of people who for a variety of reasons have unhappy memories of their past obstetrical experience require counselling. These couples may have suffered physical and emotional trauma during previous pregnancy and labour and wish to know whether such experiences could be repeated. A mother who delivered by Caesarean section after prolonged labour may wish to know about her future reproductive capacity or when she can plan for future pregnancies. True, nobody can predict the future with absolute confidence but considered counselling will add confidence to the couples.

Women with the problem of previous premature labour are more likely to have another premature labour and they should be informed of the possibility and advised to take steps to minimise the risk factors in

subsequent pregnancies. A number of patients with gestational diabetes are actually established diabetics, whilst pregnancy induced hypertension may actually be essential hypertension. Let's not wait for the next pregnancy to find out or worse still, to be just as undecided as in the previous pregnancy.

INVESTIGATING PATIENTS WITH PREVIOUS PREGNANCY FAILURE

Recurrent abortion is a common source of anxiety amongst couples. Patients with recurrent abortions or previous stillbirth have many questions. They include the whys, hows and what next. A thorough workup is sometimes required before their questions can be tackled. They need investigations prior to their next pregnancy and not at the time when they are stressed with another threatened abortion or when another stillbirth has occurred. Investigations may find the cause of the repeated failure. If a cause is found, it may be amenable to treatment, for example, in cases with genital tract infection. Unfortunately, fifty to sixty percent of spontaneous abortion during the first trimester are due to a chromosomal abnormality where no therapy can be offered to reduce the chance of the abnormality. In such cases prenatal diagnosis can be offered to detect the abnormality before fetal viability.

GENETIC COUNSELLING

The value of genetic counselling is well recognised. It is becoming even more significant because genetic disorders are now an important cause of infant morbidity and mortality. The Medical Research Council in a review of clinical genetics emphasized that 'Handicaps due to a genetic disorder or congenital malformation are the major child health problem today.'

From the individual couple's point of view, birth defects have an even more important implication as they affect their very lives. Some couples have refrained from having children because they have had a child suffering from crippling inheritable congenital abnormality or because some relatives suffer from genetic diseases.

With increasing understanding of medical genetics, development in biochemistry, cytogenetics and tissue culture and advances in recombinant DNA technology, we are now armed with new approaches in recognition, management and prevention of birth defects.

We must advise our patients on the risk of producing an abnormal child and the ideal time for genetic investigation and counselling is prior to pregnancy. The option becomes limited once pregnancy has occurred. Termination is an option but in couples in whom abortion is unacceptable, the pregnancy will have to continue in spite of the abnormality. On the other hand, if the information is transmitted prior to pregnancy, the couple may decide that the problem is so insurmountable that they prefer not to attempt natural parenthood and elect the use of effective contraception and adoption as the alternative. Methods in prenatal diagnosis needs to be discussed and the option of elective termination of an abnormal foetus and the possibility of intrauterine surgery may be discussed. When appropriate, artificial insemination of donor's sperms or in-vitro fertilisation with donor's ovum may be viable alternatives.

Prenatal diagnostic methods do have their false negatives and in cases where prenatal diagnosis fails to identify an abnormal foetus and an abnormal child is born, neonatal alternatives like immediate surgery and social support for the handicapped child should be discussed.

Never has the demand for genetic counselling been so urgent as now in modern day Singapore, where there is an ageing obstetric population aggravating the probability of chromosomal anomalies and an extremely competitive society with very limited resources for the handicapped.

REASSURING THOSE WHO FEAR PREGNANCY FOR WHATEVER REASON

Besides all the above where pre-pregnancy diagnosis and counselling is important, there is no doubt that all couples planning to start a family will benefit from

pre-pregnancy counselling. They should be advised on the subject of diet, smoking, alcohol consumption or drug addiction and immunisation against infection. Planned parenthood vastly improve pregnancy satisfaction.

THE ORGANISATION OF THE PRE-PREGNANCY COUNSELLING SERVICES

The family health care providers are in the ideal position to initiate pre-pregnancy counselling programme. Understanding the principle of family care as well as being the most likely doctors that the general public will approach, they can efficiently coordinate the process of counselling. They cannot however be expected to run the whole process by themselves and will from time to time require other expert opinion, for example, that of the geneticist, the physician, and obstetrician, to name just a few.

In general, the counselling clinic should be in two sessions. The couple should be seen together to prevent any misunderstanding and misinterpretation of data when information is passed from one to the other at home. This will also permit the couple, who are likely to be worried about different aspects of the same problem to ask their own questions and hear the answer themselves. This prevents unnecessary guilt feeling and blame-placing on any one party. The couple should be given sufficient time to discuss their problems and to clarify their doubts during both sessions.

The first session involves taking a medical history with particular concentra-

tion on the specific problem for which counselling is sought and a general physical examination. Blood investigations can be done when necessary. Between the two sessions, the practitioner can research the case at hand in order to provide up-to-date information as well as to invite participation from other specialists. During the second session, these specialists may be invited to answer further questions. The investigation results of the couple should be reviewed and discussed. Further investigations and referral to other specialists may be recommended when indicated.

THE FUTURE

Pre-pregnancy counselling should eventually become part of patient care as vaccination, routine check-ups and antenatal care are.

Maybe one day we can organise a self-help group not unlike that of Alcoholic Anonymous where couples can be enlightened and can share their experiences with others having the same problems as well as help each other when faced with any unpleasant encounter in pregnancy.

I visualise that with determination and co-operation between the different specialities involved in maternal and child care we will meet in the year two thousand in the 'World Congress of Pre-pregnancy Counselling' and one of us will be presenting a paper of the thousands of Mr and Mrs ABC who are happy parents or grandparents because pre-pregnancy counselling has given them the courage and the hope to have healthy children.

OFFICE EVALUATION OF THE DIZZY PATIENT

P N Chong MBBS, MMed (Int Med) MRCP (Ireland)

Dizziness is one of the most common symptoms presenting to the general practitioner. It is a difficult problem, made worse by its very subjective nature and the many disorders that can cause it. Few doctors will not feel a sense of despair when confronted with a patient whose main complaint is that of dizziness. This communication hopes to bring some order to the evaluation of the dizzy patient in the context of a busy office practice. It will not deal with specific diseases. Instead, it aims to provide a framework whereby the doctors can make some sense of the patient's complaints, and manage his patient in a more systematic way.

NORMAL AND ABNORMAL BALANCE

An understanding of the normal balance mechanisms is essential to the evaluation of the dizzy patient. The organs maintaining normal balance involve sensory input from three sources, viz., the vestibular labyrinth, the eyes, and the somatosensors from muscles and joints. These inputs about our environment are collated in the brainstem, acted upon by the motor system, modified by the cerebellum and the basal ganglia, and sent to the thalamus and cerebrum for conscious appreciation (Fig. 1).

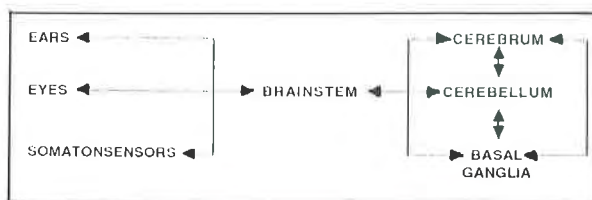


FIG. 1. SCHEMATIC REPRESENTATION OF THE VESTIBULAR SYSTEM AND ITS CONNECTIONS

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Dizziness is a sense of abnormal balance, and results from disturbance of one or more of the organs maintaining balance. However they do not all result in the same kind of symptoms. The first and most important step in evaluation is to determine exactly what the symptoms are. This is achieved by careful history taking.

THE HISTORY

The main objective of history taking is to categorise the patient's symptoms into one of four groups. These groups are viz.:-

1. **Vertigo** — a sense of unreal movement often associated with nausea and vomiting.
2. **Unsteadiness** — characterised by a tendency to fall.
3. **Lightheadedness** — presyncopal feeling. May be relieved by assuming a supine position.
4. **Giddiness (hing-hing)** — nonspecific. Cannot be easily put into any recognisable pattern. In the elderly, consider a problem of multisensory deficits. These sufferers may have cataracts, neuropathy, limited neck movements, and aging of the vestibular system.

Differentiation into these categories must be attempted despite the obvious difficulty in doing so, because identification into a group have implications regarding the organ/s at fault (Fig. 2), and therefore, the appropriate examination and investigations.

The first symptom to confirm or discard is **VERTIGO**, because it is characteristic and limits the lesion to

TABLE I: SYMPTOMS AND THEIR ANATOMIC CORRELATION

| Primary Symptom | Other Symptoms | Target Organ/s |
|-----------------|--|---|
| Vertigo | Deafness, tinnitus Diplopia, dysarthria | Labyrinth Brainstem Facial numbness Hemiparesis Hemiparaesthesia Unconsciousness |
| Unsteadiness | Falling (at night) Wobbliness Weakness | Somatosensors Eyes Cerebellum Basal Ganglia |
| Lightheadedness | Dim vision Position-induced Hyperventilation Pallor "Distant sounds" | Cerebrum Heart |
| Giddiness | Hyperventilation Headaches | Anxiety Multisensory deficits ? Cerebrovascular insufficiency |

the vestibular system. Unsteadiness, while sharing a tendency to fall as vertigo, will not have the sensation of unreal movement. Lightheadedness is less well defined but may be experienced at some point of one's "normal" life as in getting up from the toilet bowl suddenly or while standing in a parade under the hot sun. Giddiness is almost nondefinable, and the Hokkien description of "hing-hing" cannot be more appropriate.

Having made the first step to determine the presence or absence of vertigo, evaluation of the rest of the history follows as in the following algorithm (Fig. 2).

By the end of the history you would have decided on a particular group that your patient might fit in. A focussed examination on suspicious areas is then in order.

THE NEUROLOGICAL EXAMINATION

A "complete" neurological examination is not realistic nor desirable in the assessment of the dizzy patient or any other neurological problem. The history should point to the organs most likely to be involved and where attention should be focussed. Nonetheless the following examination scheme should be adopted in all patients complaining of dizziness. With practice, it should take about 5 minutes to complete.

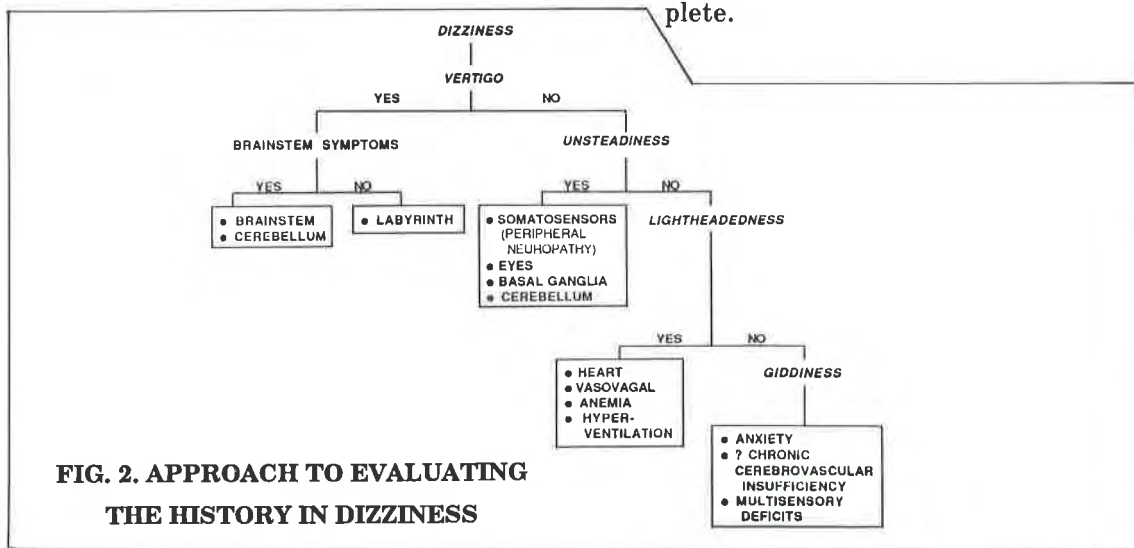


FIG. 2. APPROACH TO EVALUATING THE HISTORY IN DIZZINESS

**EXAMINATION SCHEME:
ESSENTIALS**

Patient Sitting:

Cranial nerves (diplopia, corneal reflex, hearing, facial muscles)
Nystagmus (see below)
Power (wrist extension)
Coordination (finger-nose)
BP and pulse

Patient Standing:

Rhomberg's
Gait (walk to couch)

Patient on Couch:

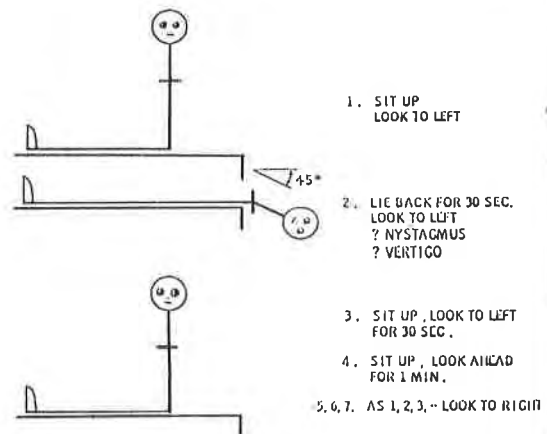
Reflexes
Position sense
BP and pulse
Heart murmurs and carotid bruits

In patients who complained of vertigo, a more detailed examination of the vestibular system is warranted. This involved the evaluation of nystagmus and the execution of positional tests (Dix-Hallpike or Nylen-Barany maneuver). Table II lists the more useful characteristics to differentiate peripheral (labyrinthine) from central (brainstem) nystagmus.

**THE DIX-HALLPIKE
(NYLEN-BARANY) MANOEUVER**

This is done with the patient sitting on the couch and suddenly lowering the patient to a position below horizontal and with the head turned 45° to the side. The patient is left in this position for about 30 seconds before returning to the sitting position with the head looking at the same direction for another 30 seconds. The test is then repeated with the head turned to the other side (Fig. 3). Severe vertigo and nystagmus occurring some seconds (i.e. with latency) after lowering the patient indicates a vertigo of peripheral origin. If fatigable (disappears after repeated testing) it is virtually diagnostic of BENIGN POSITIONAL VERTIGO.

If there is no latency and there is no fatigability, a posterior fossa tumour has to be excluded.



**FIG. 3 DIX-HALLPIKE MANOEUVER
LABORATORY INVESTIGATIONS**

The choice of investigations depends on the clinical evaluation, and there is no standard protocol. However the following are probably to be considered in the evaluation of the dizzy patient:-

**TABLE II: DIFFERENTIATING BETWEEN PERIPHERAL (LABYRINTHINE)
AND CENTRAL NYSTAGMUS**

| | |
|--|---|
| Horizontal ± / - Rotatory | Vertical Horizontal + / - rotatory |
| Fatigues (disappears on repeated testing) | Persistent |
| Diminished by visual fixation | Unchanged |
| Associated severe vertigo | Usually not true spinning |
| Always conjugate | May be dysconjugate |
| Direction of nystagmus always in one direction irrespective of direction of gaze | Direction of nystagmus changes with direction of gaze |

1. Blood count
2. Chemistry
3. ECG
4. EEG
5. Brainstem evoked potentials for brainstem abnormalities.
6. Audiometry
7. Caloric tests
8. CT scan or MRI

SOME PRACTICAL CONSIDERATIONS

- Exclude vertigo first
- Vertigo is usually caused by peripheral lesions (labyrinthine). Other supporting symptoms are severe movement hallucination, influenced by head position, intense nausea and vomiting, relieved partly by visual fixation. Absence of CNS signs. Associated tinnitus and deafness.
- Repeated vertigo in the absence of other brainstem symptoms/signs is usually not vertebrobasilar insufficiency. This is an over diagnosed entity. Brainstem "vertigo" usually do not produce true spinning. Nystagmus is usually very prominent. To diagnose vertebrobasilar insufficiency, it is necessary to elicit symptoms like diplopia, speech disturbance,

hemiplegia, hemianaesthesia, ataxia, etc. Loss of consciousness in the absence of brainstem symptoms or signs is not a common presentation of vertebrobasilar insufficiency.

- If anxiety is suspected, hyperventilation may reproduce the symptoms.
- In some elderly patients where there is no specific/significant physical abnormality, consider the diagnosis of multisensory deficit.
- Indefinable dizziness usually have no significance pathology.

SYMPTOMATIC TREATMENT OF THE DIZZY PATIENT

Specific treatment of the underlying condition should be given when possible.

However symptomatic therapy is often necessary. The following flow chart may be useful (Fig. 4).

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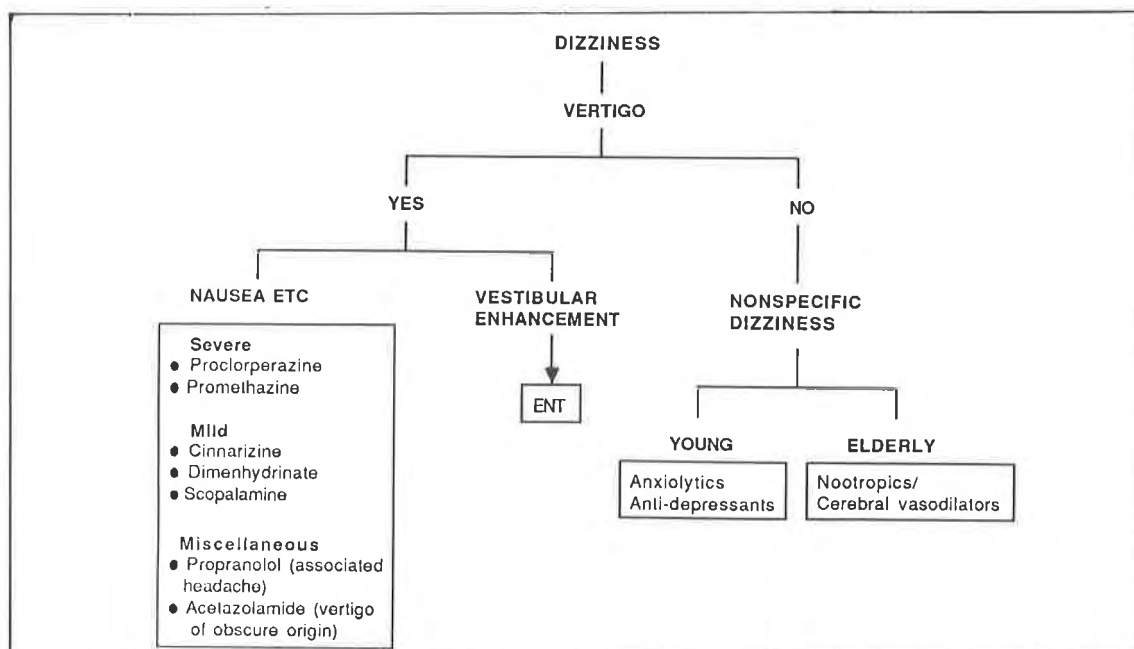


FIG. 4 SYMPTOMATIC TREATMENT OF THE DIZZY PATIENT

ANTITHYROID DRUG THERAPY FOR GRAVES' HYPERTHYROIDISM — ANY NEW ANSWERS TO OLD QUESTIONS?

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INTRODUCTION

Antithyroid drugs have been the mainstay of the treatment of Graves' hyperthyroidism for more than 40 years.^{1,2} The most commonly used drugs in Singapore are carbimazole and to a lesser extent propylthiouracil. Both are almost equally effective and most patients can be rendered euthyroid within 3 to 6 weeks of antithyroid drug therapy.

Hyperthyroidism due to Graves' disease is a common clinical problem in Singapore. In a population-based study in 1988, the prevalence of thyrotoxicosis was found to be 1.28%.³ Although the efficacy of antithyroid drugs in normalising thyroid function in the short term is unquestioned, its ability to achieve long term remission after drug therapy is stopped remains far short of that desired. Various studies reported rates of relapse varying between 40% to 70%. This remains the biggest draw-back of antithyroid drug therapy. This gives rise to the question of whether relapse of hyperthyroidism after drug therapy can be predicted and whether higher rates of remission can be achieved with drug therapy. In this article I shall discuss what is presently known about answers to these two questions.

CAN RELAPSE OR REMISSION BE ACCURATELY PREDICTED?

A large number of factors, both clinical and laboratory, have been proposed as predictors of relapse (Table 1). Clinical signs

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that may suggest a higher risk of relapse include large goitres, exophthalmos, thyrotoxic periodic paralysis and a history of previous relapse(s). Laboratory factors that may be predictive of relapse include the presence of HLA DR3, persistence of thyrotrophin receptor antibodies, abnormal T3 suppression test and abnormal TRH test at the end of drug therapy.

Of the clinical factors identified, a past history of relapse and thyrotoxic periodic paralysis are probably the most useful. Although the risk of relapse may not be much higher after the first relapse,⁵ the rate thereafter becomes much higher. Many doctors use this as an indication that ablative therapy should be offered. Thyrotoxic periodic paralysis is uncommon in the Caucasian population and is not mentioned as a predictor for relapse in Western literature. However, Yeo et al reported a relapse rate of 75% within 4 years in his group of patients.⁶ The presence of a large goitre is a well known risk factor for relapse. However the estimation of size of goitre is a subjective one and the value of this parameter in predicting relapse for each patient is probably limited. Similarly exophthalmos although mentioned as a predictor of relapse also has limited value in this respect.

Amongst the laboratory factors, the value of HLA has been debated with some studies suggesting that it is useful and some refuting it. Overall, HLA is not useful in the local context as it is probably only predictive in the Caucasian population if at all. The other 3, TSH receptor antibody, T3 suppression test and TRH test, are approximately equal in predictive ability, quoted in various studies to be of the order 70 to 85% of case relapsing if these factors are

present. These 3 are likely to be useful indicators of when drug therapy can be terminated without a high risk of relapse.

The large number of factors identified as possible predictors of relapse is an indication that none of these factors are sufficiently accurate in predicting relapse or remission. Each factor on its own can predict about 70 to 80% chance of relapse if that factor is present as compared to about 30 to 40% if it is absent. This degree of accuracy is not sufficient when applied to a single patient - e.g., it does not seem satisfactory to subject a patient to ablative treatment with surgery or radioiodine on the strength of a 70 to 80% chance of a relapse or to take a patient off therapy knowing that there is still a 30% chance of relapse.

A number of studies, notably the prospective multicentre trial in Europe,⁵ has looked into the use of combinations of more than one factor. Surprisingly, this does not give rise to a higher level of sensitivity nor specificity in its predictive value. The risk of relapse or remission is not better predicted by using more than one factor unlike the additive effects that have been used for coronary risk factors. This makes it difficult to solve the question of predicting relapses. More studies are therefore necessary to define how these predictive factors especially laboratory ones can be used in the context of decision making in antithyroid drug therapy.

CAN A HIGHER RATE OF REMISSION BE ACHIEVED WITH ANTITHYROID DRUG THERAPY?

Since there is still no simple answer to the question of prediction of relapse, we should look next into whether a higher rate of remission can be achieved. The usual regime of treatment with decremental doses of antithyroid drug for a duration of 12 to 18 months is associated with a relapse rate of about 50 to 70%.

The issue that is central here is whether antithyroid drug therapy alters the natural history of the disease or merely induces euthyroidism whilst awaiting a natural remission. If antithyroid drugs have no influence on the natural history of the disease, relapse rate should be similar

for treated and untreated cases of Graves' hyperthyroidism.

It is obviously not possible to perform any prospective trial to observe the relapse rate of treated and untreated hyperthyroidism. There are however a number of studies that have compared the relapse rates of cases treated with propranolol or antithyroid drugs. These appear to show a higher rate of remission for patients treated with antithyroid drugs suggesting that these drugs do alter the natural remission rate of the disease.¹² There is evidence particularly in-vitro evidence to suggest that anti-thyroid drugs exert an immunosuppressive effect.¹³ This may explain the higher remission rate in cases treated with antithyroid drugs.

It is therefore tempting to consider next whether higher doses of antithyroid drugs or longer duration of therapy can result in higher remission rates. The most convincing evidence comes from the study by Romaldini et al who showed that treatment with a high dose (mean dose of carbimazole of 60mg per day) resulted in a higher rate of remission.¹⁴ A remission rate of 75.4% for high dose regimen as compared to 41.6% in the low dose regimen was reported. The obvious concern with high dose therapy is the incidence of side effects. The most feared side effect from antithyroid drugs, agranulocytosis, was not more common in the high dose group although milder side effects were more frequent - eg arthralgia, pruritus, gastritis, headache, etc.¹⁴ Cooper, in his review on antithyroid drugs,¹⁵ quotes the risk of agranulocytosis as 0.1 to 1% with higher risk for doses of carbimazole exceeding 40mg per day. The occurrence of agranulocytosis is almost always in the first three months of treatment, although it has been reported to occur on reintroduction of the drug after completing a previous course.

The other way of achieving a higher rate of remission may be a longer duration of antithyroid drug therapy. A number of studies have suggested that a longer duration of therapy achieves higher remission rates without undue risk of side effects.^{10,16} This is again in keeping with the concept of antithyroid drugs having an immunosup-

pressive effect.¹³ Of great interest is the study by Yamamoto et al¹⁰ which reported that 126 out of 193 patients had normal T3 suppression after long term treatment of up to 8 years and of these 96% remained in remission after a mean follow-up of 4 years. A significant number (67 out of 193) however were not benefited by long duration of therapy and there is no clear answer on how this group can be identified (vide supra). Nevertheless data such as these argue in favour of long term antithyroid drug therapy as a means towards higher remission rate. Although the exact duration that is optimal is not clear, the use of one or more tests that can predict relapse eg T3 suppression test used by Yamamoto et al,¹⁰ together with a long duration of treatment may be the answer.

CONCLUSION

There is at present no completely satisfactory answer to the problem of antithyroid drug therapy. High dose (with thyroxine supplement) and long duration of therapy appear to induce higher rates of remission but these approaches have certain drawbacks. However, ablative therapy, surgery or radioiodine, is also not without disadvantages. Surgery is associated with a small but finite surgical risk as well as risks of relapse or hypothyroidism, approximately 10-20% each¹² though the rate varies from study to study. Radioiodine is associated with 10-20% chance of relapse and 30-50% risk of ultimate hypothyroidism¹² the rates depending on the dose of radioiodine given. The practice of giving a large dose of radioiodine to render the patient unequivocally hypothyroid and starting replacement therapy immediately does not seem satisfactory either, curing one illness but conferring a second.

Medical treatment of Graves' hyperthyroidism therefore remains an important mode of therapy for this condition. Based on our present knowledge, I feel the following approach is worth considering:

1. The use of high dose carbimazole (40mg per day) with thyroxine supplementation for 12 to 18 months to induce a remission, especially in new cases, may be considered.
2. For patients who have relapsed, the

use of TRH test (or T3 suppression test) together with TSH receptor antibody to identify patients at high risk of relapse may be used and if these tests predict a relapse, long term antithyroid therapy until these tests are normal may be considered.

3. Ablative treatment may be considered for patients who are not compliant with drug therapy, who have adverse drug reaction, who have 2 or more relapses or a relapse after one form of ablative therapy.

TABLE 1 FACTORS THAT PREDICT A HIGHER RELAPSE RATE FOR GRAVES' HYPERTHYROIDISM

Clinical:

- Large goitre (Ref 4, 5)
- Exophthalmos (Ref 4)
- Thyrotoxic Periodic Paralysis (Ref 6)
- Past history of relapse (Ref 4, 5)

Laboratory:

- HLA DR3, HLA B8 (Ref 5)
- TSH receptor antibodies (Ref 4, 7, 8, 9)
- T3 suppression test (Ref 5, 10)
- TRH test (Ref 5, 11)

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THE OSTOMY PATIENT & THE FAMILY PHYSICIAN

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ABSTRACT

This paper, based on a retrospective study on 117 colostomies and 32 ileal conduits performed at the Department of Surgery, Toa Payoh Hospital from 1972 through 1987 is intended to outline the role the General Practitioner/Family Physician, can play in the aftercare of the ostomy patient after discharge from hospital.

In order to be able to give basic enterostomal care, the GP must be familiar with the various ostomy appliances and skin care methods/products and know the source of locally available expert care i.e., the GP must be able to treat the common dietary, psychosexual and skin problems likely to affect his patients. In addition, he must be able to recognise the major complications (the majority being surgical) which mandates referral to a skilled enterostomal therapist or surgeon.

INTRODUCTION

Stomal care is an area where most doctors including the surgeons who created stomas in the first place, find themselves ill-equipped to handle because of inadequate knowledge of the basic elements of stoma care. General practitioners in particular should be able to treat the majority of stomal problems and refer only the serious complications to the expert.

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To leave the care of the ostomate to our health institutions and voluntary organisations is simply not enough as this care is a dynamic and continuing process between therapist and patient — it requires the personal touch of a professional who is both knowledgeable and understanding. There is no other person better qualified to undertake this role than the modern Family Physician who by dint of his training can counsel and rehabilitate the ostomate in relation to his family and society as well as handle problems pertaining to the ostomy per se.

It is hoped that this communication will be of value to the Family Physician who is prepared to take on this important task.

CARE OF THE STOMA

(1) Choice of Appliances

The simplest type is a one piece "stick-on" disposal plastic bag. In older patients, those with watery stools not easily correctable by diet manipulation and in those with urostomies (e.g. ileal conduits), a drainable bag may be appropriate. The latter is also useful for those with irritable skin intolerant to everyday changing of the appliance. In this respect the patient has the alternative of using special bags provided with a stomahesive platform or karaya gum seal especially when dermatitis has supervened. If contour or shape changes occur too often, then recourse may be made to the modern Sur-fit appliance that conforms to the surface on which it is applied. A 2 piece bag consisting of a belt and plate/flange with a bag are to be worn when there is a sticking problem.

(2) Daily Bag Changes

Apparatus required consists of: a bowl of lukewarm tap water, mild toilet soap, some tissue paper, the required bag and

skin protective aids if needed. A paper bag for disposal of used material is also required.

The Procedure:

- a) Remove the used bag from top downwards,
- b) Clean peristomal area with wet tissue paper, using soap if desired,
- c) Rinse peristomal skin thoroughly,
- d) Dab dry and apply the new bag bottom upwards, making sure the flange is free from creases to avoid leakage,
- e) Tincture Benzoin Co can be used on the peristomal skin to increase adhesiveness for the bag. Do not apply direct to sore skin.

(3) Irrigation Method for Bowel Regulation

While the above method of bag changes can allow for several bowel actions a day, regulation of colonic evacuation by irrigation each morning can do away with the wearing of a bag and is particularly suitable for the young and intelligent patient, motivated sufficiently to be taught this method.

CARE OF SORE SKIN

Sore skin should not occur if proper skin care is given and fitting appliances utilised. However, if sore skin develops, then the following aids can be used:

- a) *Skin Aids:* Stomahesive, Karaya Gum Seal or Ostomy Adhesive Disc.
- b) *Skin Lotions:* Tinc Benzoin Co on top of Gentian Violet 1%. Lotion F (if available), Flavine Lotion, or Calamine Lotion.
- 3) *Skin Creams:* Orabase, Chiron Barrier Cream, Karaya Paste, Hollister Gel or Tulle Gras with Karaya Gum on top.

If calamine lotion is used, then the powder should be removed when the lotion is dry before applying the bag. For slight soreness, any lotion can be used before sticking the bag on.

Where there is excoriation, the skin aids such as stomahesive should be applied on top of the skin after the lotion is used. The stomahesive should be left for 3-5 days or even longer if there is no leakage. In this

way the skin is allowed to heal. A drainable bag should be used during such treatment.

Tulle gras dressings can be used in the absence of other aids. The tulle gras dressing should be evenly spread over the sore area, then a layer of karaya gum added on to hold the sticking surface onto the bag.

However, patients' skins vary and not one single method is suitable for all patients. Hence referral to a Stomatherapist may be necessary should any difficulty be encountered.

DIETARY CARE

An ostomate should be advised to take a normal balanced diet in the absence of any medical problem. However mild adjustment is occasionally needed to regulate bowel action. Through the experience of several ostomates, we have noted that 2 glasses of water taken early in the morning helps to induce bowel action and thus the ostomy patient can be free for the rest of the day.

Certain everyday complaints can be eliminated by dietary manipulation:

- a) *Excessive odour:* avoid offending foods eg. onions, garlic, fish, cabbage, asparagus.
- b) *Excess flatus:* avoid troublesome vegetables, carbonated beverages, raw milk, acid fruit drinks. Restrict consumption of nonabsorbable carbohydrates. Cut meals to small portions; several meals per day on a regular basis.
- c) *Watery effluent:* avoid raw milk, fatty foods, fibrous foods and simple carbohydrates.
- d) *Dry, hard stools:* check medications especially antacids. Increase intake of fibrous foods, fruit juices and total fluids.
- e) *Excess undigested matter:* eliminate membranous meats, nuts and corn.

Thus by trial and error, the diet can be selected to avoid foods which cause difficulty. Constipation alone is not a bad fault, but loose stools are hard to control — hence the colostomy patient should keep some anti-diarrhoeal mixture or drug e.g.,

Lomotil tablets at hand for use as the occasion arises.

CARE OF THE PSYCHE

Psychological problems can present in many forms:

- a) *Fear*: this can be fear of touching the stoma, or simply fear of odour, of leakage of pouch, of poor acceptance of partner, of future problems with the wound, of loss of erection in the male or just noise from the stoma.
- b) *Depression*: the nature of the underlying disease, its unpleasant symptoms and treatment, all give ample reason for depression.
- c) *Rejection*: relationships within the family can deteriorate and alcoholic abuse by the spouse and rejection even to the point of desertion can follow.
- d) *Social isolation*: embarrassment and feelings of social inadequacy may lead to isolation.

Careful empathic counselling and regular support is required for both the patient and his entire family. This is where membership of an "ostomy" club can help patients resolve their own problems. Where the problems have become deep seated, recourse to a psychologist or psychiatrist may be necessary. Fears can be very real and in the young the change of body image can present difficulties. Also there is always the fear of losing his job for the breadwinner. Indeed, most ostomates have an inner "fear of the unknown". The problem of noise due to passage of gas from the stoma can be prevented by the oral intake of generous amounts of activated charcoal and the use of "odour-free" bags containing carbon discs or chlorophyll tablets in addition to methods involving dietary manipulation.

CARE OF SEXUAL/ADAPTATION PROBLEMS

Sexual/adaptation problems include:

- a) Poor acceptance of partner
- b) Loss of ejaculation and complete or partial impotence in the male
- c) Dyspareunia in the female
- d) Alcoholic abuse by partner/rejection

and desertion

- e) Maltreatment by errant husbands
- f) Partner unfaithfulness etc.

Counselling directed towards sexual adaptation of the ostomate and his/her spouse should begin as soon as the patient is fit enough to listen and must be followed up by the family doctor.

Sexual information should be given by one who is comfortable with the subject and does not bring his/her values to the bedside. Males will be particularly concerned with impotency and females need reassurance that the vaginal canal has not been impaired. Generally, all ostomates and their spouses need a resource person to resolve their fears and concerns relevant to sexual function. In the male with established impotence, referral to a Urologist for implantation of a penile prosthesis may be appropriate.

CARE OF SURGICAL PROBLEMS

These constitute the major problems which must be recognised and referred for early treatment, viz:

- a) Prolapse
- b) Para-stomal hernia
- c) Stenosis
- d) Peristomal abscess and fistulation
- e) Local recurrence of disease at stoma
- f) Faulty siting
- g) Stomal ulceration
- h) Recession, etc

These physical problems generally require correction by the surgeon.

SUMMARY AND COMMENTS

The number of ostomates is steadily increasing. All Family Physicians must therefore become cognizant of the everyday problems confronting these individuals and be prepared to counsel them towards their total rehabilitation in society.

The number of trained enterostomal therapists is simply just not enough to take on this responsibility single-handed. In this paper, we have outlined the common problems likely to be presented to the Family

Physician. The day to day care of the stoma and the basis for the use of the various types of appliances has been presented together with instructions on how the bag should be changed daily. With this information, we have proceeded on to the management of the commonest dermatological problem of ostomate, namely — the "sore" or dermatitis skin. In treating the latter, the use of lotions should take priority; then in progressive order, creams and finally skin aids can be used in the more difficult cases. The use of any one medication does not preclude simultaneous use of one of the others in a different group. The diet of the patient with an ostomy can be the cause of several complaints that can interfere with the very joy of living. Common dietary offenders have been pointed out and the solutions suggested. Psychological problems commonly occur and the family physician must be able to recognise the emotional hurdles of ostomates and help them and those around them resolve their fears and feelings of inadequacy. If this is not done, the outcome will be depression, rejection and ultimately social isolation. The sexual adaptation of the ostomate and his/her spouse is another important aspect of rehabilitation which the family physician can help as he is usually also the family confidante. The family doctor can counsel and indicate to the patient that there are many options of sexual expression, including physical methods. And last but not least, the Family Doctor must know

when and how to call on the expert for help. In particular, he must not hesitate to refer his patients once one of the major complications (usually surgical) is diagnosed.

With the above knowledge, there should be no need for hasty referral of the ostomate to the surgeon for each and every problem. The Family Physician should give some of his time elucidating the problems of the ostomate and take remedial action himself for the minor complaints, which as can be seen, are all within his purview to treat. If difficulty should arise, there is always the Stomatherapist and Stoma Club available at the Singapore Cancer Society.

CONCLUSION

Family Physicians today cannot reject their responsibility to be part of the stoma rehabilitation team. They should be able to provide general advice, treat the simpler stoma problems and give continuing support to the increasing number of ostomates in the community.

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ORIGINAL ARTICLES

THE MANAGEMENT OF ASYMPTOMATIC GALLSTONES: DO THEY REQUIRE TREATMENT?

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SUMMARY

Many patients with minimal or no symptoms will have gallstones diagnosed on screening ultrasound examination. In the majority of patients with asymptomatic gallstones the lack of evidence supporting prophylactic cholecystectomy suggests that an expectant policy should be pursued and the primary physician should follow up these patients closely to detect symptoms or complications early. However, available evidence seems to support prophylactic surgery for patients with big stones, non-functioning gallbladders or with a porcelain gallbladder.

INTRODUCTION

There has been a trend in recent years for an increasing proportion of the Singapore population to undergo health examination and various screening programmes conducted by physicians working in private clinics and public institutions. This trend is likely to continue. As a result, many individuals with minimal or no symptoms will have gallstones diagnosed on screening ultrasound examination. The primary health care physician will hence

be faced with the problem of whether to refer this patient with asymptomatic gallstones for treatment or to observe them until symptoms develop.

THE CASE FOR PROPHYLACTIC SURGERY

Historically, there has been much confusion regarding the correct course of management. In 1822, Naunyn noted that calculi may remain asymptomatic but do not remain harmless in the long run.¹ Moynihan stated that all gallstones produced symptoms and invariably lead to complications of a severe nature if the patients lived long enough.² Mayo stated that innocent gallstones are a myth.³ On the other hand, other physicians such as Sir William Osler felt that surgery should only be recommended for those with complications and an expectant policy should be pursued for those with asymptomatic gallstones.⁴ The consensus was for operation, and this was supported in a study in 1948 by Comfort who followed-up a group of 112 patients with silent gallstones and reported that 46% of them developed symptoms.⁵ A further study in 1960 by Lund showed that 18 of 34 patients or 53% with silent gallstones observed over a 5-20 year period developed biliary pain or complication.⁶ Hence early opinion in 60's weighted in favour of prophylactic cholecystectomy for asymptomatic stones especially as the mortality of surgery was decreasing.

THE CASE AGAINST PROPHYLACTIC SURGERY

One of the problems of the early studies was defining what were the symptoms of

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gallbladder disease. Belching, dyspepsia, fat intolerance and non-specific upper abdominal pain were often thought to be caused by gallstones. However more recent studies showed that dyspepsia occurs as frequently in persons without gallstones as in person with gallstones.^{7,8,9,10,11,12,13} Therefore dyspepsia should not be attributed to the presence of gallstones until other possible causes are excluded. Using this criteria a reinterpretation of the results by Comfort for example showed that only 19% of the patients developed biliary symptoms. Other studies by Berk in 1964¹⁴ and Gracie and Ransohoff¹⁵ seem to indicate that less than 15% of the patients developed biliary problems in the follow-up of 10-15 years. The more recent study by Friedman¹⁶ of 467 patients seen by primary health physicians indicated that in those with asymptomatic gallstones biliary colic developed in 13% and in only 6% did complications arise. In those who had mild symptoms, 25% developed biliary colic and the complication rate was 8.7%. However, in those with non-functioning gall bladders, biliary colic developed in 22% and complications in 13%. Overall only 1 to 2% of the patients developed complications per year and only 1.6% required cholecystectomy per year. The maximum complication rate was within the first 5 years. Many other methods have been used to assess the benefits of cholecystectomy such as Ransohoff's study¹⁷ where it was calculated that for those below 30 years of age prophylactic surgery resulted in a net loss of 4 days of life and for those over 50 years there is a net loss of 18 days of life. It was also calculated that the health cost was five times more in those who are subjected to prophylactic surgery. Fitzpatrick assessed that good risk, asymptomatic patients where prophylactic surgery was performed, had a mean net gain of 2 weeks of life. In those at poor risk, surgery resulted in the mean loss of 1 month of life.¹⁸

In summary, since 1980 there has been 40 articles written as to the benefits of prophylactic surgery in silent gallstones. Of these 48% recommend prophylactic surgery, 30% recommend expectant treatment and 22% feel that no conclusive argument can be made for or against prophylactic surgery.

SUB GROUPS THAT MAY BENEFIT FROM PROPHYLACTIC TREATMENT

Patients with large stones of 2cm in diameter have 2 times the risk of developing complications¹⁹ and those with porcelain gallbladder have an increased risk of carcinoma of the gallbladder.

The size or number of stones, age and sex of the patient do not provide discriminating features to sway the decision for or against surgery. In diabetics with asymptomatic stones expectant treatment appears to be superior to prophylaxis. In patients with cirrhosis the mortality of surgery is higher and unacceptable.^{20,21}

Alternative modalities for treating gallstones are in vogue. Among them are dissolution therapy, contact dissolution therapy, extracorporeal shockwave therapy and laser lithotripsy. Initial findings are all in symptomatic cohorts and the place of the modalities in asymptomatic cases have not been thoroughly assessed.

CONCLUSION

In conclusion, available evidence seems to support prophylactic surgery for patients with big stones, non functioning gallbladders or with a porcelain gallbladder. Expectant treatment is preferable in patients with diabetes, cirrhosis or those who suffer from concomitant medical problems which make surgery risky.

In the majority patients with asymptomatic gallstones the lack of evidence supporting prophylactic cholecystectomy suggests that an expectant policy should be pursued and the primary physician should follow up these patients closely to detect symptoms or complications early. It is necessary also to investigate for conditions such as peptic ulcer disease, pancreatitis and reflux esophagitis in those who are mildly symptomatic before attributing the symptoms to gallstone disease.

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HOME STUDY SECTION

CALCIUM-CHANNEL BLOCKERS

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INTRODUCTION

The introduction of calcium-channel blockers (calcium antagonists) represents an important advance in the treatment of cardiovascular disorders. Amongst the increasing number of calcium antagonists now available, verapamil, diltiazem, nifedipine and nicardipine are the most widely used locally.

MODE OF ACTION

All calcium-channel blockers have the ability to prevent calcium influx into cardiac and vascular smooth muscle cell by blocking the "slow calcium-channel" which is sensitive to changes in cellular transmembrane potential. As calcium entry is essential for smooth muscle contraction, these drugs cause widening of the lumina of coronary and systemic arteries. This direct vasodilator action has been used in the management of angina and hypertension. As pacemaker cells of the atrio-ventricular nodal tissue are also dependent on calcium ingress, a therapeutic effect on fast supraventricular arrhythmias can be anticipated. On the negative side, calcium-channel blockade may reduce myocardial

contractility, a problem largely confined to verapamil. Relaxation of smooth muscle at other, anatomical sites e.g., gut wall, uterus and bronchi, can also be demonstrated.

The available drugs show substantial variation in their chemical structures and appear to bind at different sites relative to the orifice of the slow calcium-channel. It is hardly surprising that they have differing properties. Verapamil (a phenylalkylamine derivative) exerts a powerful effect on cardiac conduction whereas drugs with a dihydropyridine structure, such as nifedipine and nicardipine, are particularly effective peripheral vasodilators. Diltiazem (a benzothiazepine derivative) fits somewhere in between. All will dilate coronary arteries. These disparities in pharmacodynamic action have important relevance to the indications for and side effects of each individual drug. (Table 1)

PHARMACOKINETICS

Calcium antagonists are rapidly and completely absorbed after oral administration

TABLE 1
PHARMACOLOGIC EFFECTS OF CALCIUM-CHANNEL BLOCKERS

| | VERAPAMIL | DILTIAZEM | NIFEDIPINE | NICARDIPINE |
|--------------------------|----------------------|-----------|------------|--------------|
| Heart rate | - | - | + nc | + - |
| Myocardial contractility | - | - | + nc | nc |
| Nodal conduction | - | - | nc | nc |
| Peripheral vasodilation | + | + | + | + |
| Maximum duty dose | 480 mg | 360 mg | 80 mg | 120 mg |
| Key | - indicates decrease | | + increase | nc no change |

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and undergo extensive first-pass hepatic metabolism. Caution is therefore necessary when prescribing in liver disease. Bioavailability ranges from around 20% for verapamil, 35%

for nifedipine to 45-60% for diltiazem and nifedipine. The resulting inactive metabolites are excreted in the urine. All calcium antagonists have a relatively short half-life, necessitating a three times daily dose regimen, though sustained released formulations are now available.

CLINICAL USES

The therapeutic value of calcium-channel blockers is best established in angina. Calcium antagonists reduce myocardial work, and increase myocardial blood flow and oxygen supply. They achieve this by reducing contractility, by reducing arterial tone (afterload), by causing direct coronary arterial vasodilation, and, possibly, by reducing venous tone (preload). The more rate limiting calcium antagonists (i.e. those with the greatest effect on atrioventricular conduction) may also be beneficial by reducing the heart rate during exercise and, often, at rest (Table 2).

Dihydropyridines (nifedipine and nicardipine) are particularly indicated for vasospastic (variant) angina and have a useful synergistic effect with beta-blockers in the treatment of chronic stable angina. This combination (i.e. beta-blocker and calcium antagonist) is not recommended for verapamil. The role of these drugs as monotherapy in chronic stable angina is less clear. There are some reports that agents with arteriolar dilator effect can aggravate angina, by reducing diastolic perfusion pressure, inducing coronary 'steal' and causing reflex tachycardia. In patients with unstable angina and myocardial infarction, these drugs can also aggravate myocardial ischaemia and are best used in conjunction with a beta-blocker. Calcium antagonists should be preferred to beta-blockers in patients with angina and peripheral vascular disease, chronic obstructive airway disease or asthma.

Calcium entry blockers are being increasingly used for the treatment of hypertension. The currently available calcium antagonists are similar in antihypertensive efficacy but differ in their effects on the atrioventricular node and the degree of peripheral vasodilatory action. The second generation of dihydropyridine calcium antagonists exhibit more specific vasodilatory actions with a less negative inotropic effect, which may effect their use in patients with congestive heart failure.

Overall the data suggest that calcium antagonists appear to be just as effective as existing first-step therapy in the treatment of uncomplicated essential hypertension. Long-term tolerance does not appear to be a problem and side effects, if they are going to appear, usually occur early. If the side effects can be tolerated they may become attenuated with time. Calcium antagonists are attractive choices for the treatment of many subsets of patients with hypertension. These include patients with concomitant conditions that preclude or discourage the use of other agents, such as bronchia asthma, peripheral vascular disease, diabetes mellitus and gout. Patients with various other diseases may receive additional benefits from calcium antagonists used to treat their hypertension (Table 2). These include patients with migraine, cerebral vascular disease and oesophageal spasm. In addition, calcium-channel blockers may be particularly useful in hypertensive emergencies for which a prompt but controlled fall in blood pressure is desirable. Sublingual nifedipine can be used to reduce blood pressure acutely and safely in patients presenting with severe refractory hypertension.

**TABLE 2
INDICATIONS FOR CALCIUM-CHANNEL
BLOCKERS**

- | | |
|---|--------------------------|
| * Angina | * Raynaud's phenomenon |
| * Hypertension | * Systemic sclerosis |
| * Hypertrophic cardiomyopathy | * Pulmonary hypertension |
| * Subarachnoid haemorrhage (nimodipine) | * Migraine prophylaxis |

Calcium antagonists that slow cardiac conduction are indicated for the acute and chronic treatment of supraventricular tachycardias (SVT). Intravenous verapamil is the drug of choice for paroxysmal SVT, but not in patients receiving a beta-blocker. If hypotension is a concern pretreatment with intravenous calcium gluconate is advisable. Oral verapamil and diltiazem can be used to

control the ventricular response in atrial fibrillation and can also be used as maintenance/prophylaxis for chronic SVT.

Because of its specific effect in dilating peripheral arteries, nifedipine may be used in the treatment of Raynaud's phenomenon and, possibly, for the prophylaxis of migraine, although this is not at present a licensed indication. Many studies are underway in other areas of therapeutics where benefit from smooth muscle relaxation can be anticipated e.g. cerebral insufficiency, peripheral vascular disease, irritable bowel syndrome, bronchial asthma, premature labour. By reducing myocardial contractility, calcium antagonists may be useful in treating conditions in which diastolic relaxation is impaired e.g. hypertrophic cardiomyopathy. Calcium-channel blockers with no negative inotropic effect may be useful in the treatment of congestive heart failure.

CONTRAINDICATIONS/CAUTIONS

Because of its negative inotropic and chronotropic properties, verapamil should not be used in patients with cardiac failure or with abnormalities of cardiac conduction. Combining verapamil with a beta-blocker undoubtedly increases the risk of cardiac decompensation and heart block. These caveats apply also to diltiazem but to a lesser extent as this drug does not have as powerful an action on cardiac conduction and myocardial function as verapamil. Nifedipine and nicardipine do not alter cardiac conduction and their relaxant effect on cardiac muscle is minimal. No evidence for a teratogenic effect has been reported with calcium antagonists. However, they inhibit uterine activity and may be best avoided towards the end of pregnancy. As calcium-channel blockers are biodegraded in the liver, caution is therefore necessary when prescribing in liver disease. (Table 3)

ADVERSE EFFECTS

Calcium-channel blockers are remarkably safe drugs considering the importance of calcium transmembrane flux for so many biological systems. All may produce nausea, sweating, headache, dizziness, flushing, palpitations and hypotension as a consequence of systemic vasodilation. These effects are most

TABLE 3
CONTRAINDICATIONS/CAUTIONS FOR
CALCIUM CHANNEL BLOCKERS

| | |
|--|---|
| * Cardiac failure | * β -blockade ^{1,2} |
| * Severe bradycardia | * Digitalis toxicity ² |
| * Sino-atrial and atrioventricular nodal disease | * Anti-arrhythmic treatment |
| * Liver disease | * Wolf-Parkinson-White syndrome with antegrade conduction |
| * Pregnancy | * Left ventricular outflow obstruction |

¹ Oral verapamil.

² Absolute contraindication to intravenous verapamil.

pronounced with nifedipine and nicardipine and may occur in more than 15% of patients treated without concomitant beta-blockade. Intense peripheral vasodilation may lead to an initial reflex tachycardia with a paradoxical increase in angina frequency. Nifedipine and nicardipine are best combined with a beta-blocker which abolishes this reflex response.

Verapamil may precipitate cardiac failure in patients with compromised myocardial performance. Gravitational peripheral oedema associated with increased capillary permeability but without other signs of cardiac failure can present with all these drugs and is often resistant to diuretic therapy. This occurs particularly with nifedipine in high dosage. Unwanted conduction disturbance with verapamil may develop if there is pre-existing cardiac disease and abnormal conduction on a pre-treatment ECG will identify those patients at greatest risk. Constipation may be apparent on starting treatment with verapamil as a result of relaxation of gastrointestinal smooth muscle.

Diltiazem appears to produce fewer side effects than the other drugs as it has a lesser effect on cardiac function than verapamil. It has less vasodilation than nifedipine. Nevertheless, many of the above symptoms and signs have also been associated with diltiazem deterioration in symptoms of myocardial ischaemia and, rarely, with the production of a myocardial infarction. (Table 4)

**TABLE 4
ADVERSE EFFECTS OF CALCIUM CHANNEL
BLOCKERS**

| | |
|-----------------------------|-----------------------|
| * Headache | * Sedation |
| * Flushing | (prenylamine) |
| * Dizziness | * Risk of ventricular |
| * Ankle oedema (nifedipine) | arrhythmias |
| * Nausea | (prenylamine, |
| * Palpitations | lidoflazine) |
| * Constipation (verapamil) | * Hepatitis |
| * Skin rashes | (nifedipine) |

DRUG INTERACTIONS

Pharmacodynamic interactions with other cardioactive drugs are predictable. Furthermore, the hepatic bio-transformation of calcium antagonists makes them susceptible to pharmacokinetic interactions with drugs metabolised by the same route. (Table 5)

**TABLE 5
DRUGS INTERACTIONS OF CALCIUM CHANNEL BLOCKERS**

Pharmacodynamic

| | |
|-----------------------------|---|
| * -adrenoceptor antagonists | * Potentiation of sino-atrial and atrioventricular nodal effects of diltiazem and verapamil (verapamilis contraindicated) |
| | * Potentiation of negative inotropic and hypotensive effects of calcium channel blockers |
| * Digoxin | * Potentiation of sino-atrial and atrioventricular nodal effects of diltiazem and verapamil |

Pharmacokinetic

| | |
|--------------|--|
| * Digoxin | * Verapamil and diltiazem may elevate circulating digoxin concentrations |
| * Rifampicin | * Reduced circulating concentrations of verapamil |
| * Cimetidine | * Increased circulating concentrations of nifedipine, nicardipine, verapamil and diltiazem |

CONCLUSION

Calcium-channel blockers are an important group of therapeutic agents with a widening range of clinical indications. Their side effects are predictable from their pharmacological properties and relate particularly to slowing of cardiac conduction, impairment of myocardial contractility and sequelae of peripheral arteriolar dilatation.

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MUTIPLE CHOICE QUESTIONS

1. In normal therapeutic doses nifedipine:
 - A. may aggravate angina pectoris
 - B. should not be given with beta-blockers
 - C. has pronounced anti arrhythmic activity
 - D. frequently worsens intermittent claudication
 - E. may precipitate a headache.
2. Verapamil:
 - A. may be used to treat atrial arrhythmias
 - B. frequently causes constipation
 - C. should not be given to asthmatic patients
 - D. is often given with beta-blockers in hypertension
 - E. should not be used in patients with cardiac failure.
3. Known side effects of calcium-channel blockers include:
 - A. hyperuricaemia
 - B. hypokalaemia
 - C. ankle oedema
 - D. skin rashes
 - E. dizziness.
4. The following statements are true:
 - A. nifedipine decreases AV node conduction
 - B. diltiazem often increases the heart rate
 - C. cimetidine increases the circulating concentrations of calcium-channel blockers
 - D. digoxin potentiates the SA and AV nodal effects of verapamil
 - E. sublingual nifedipine is ineffective in patients presenting with severe refractory hypertension.

ANSWERS

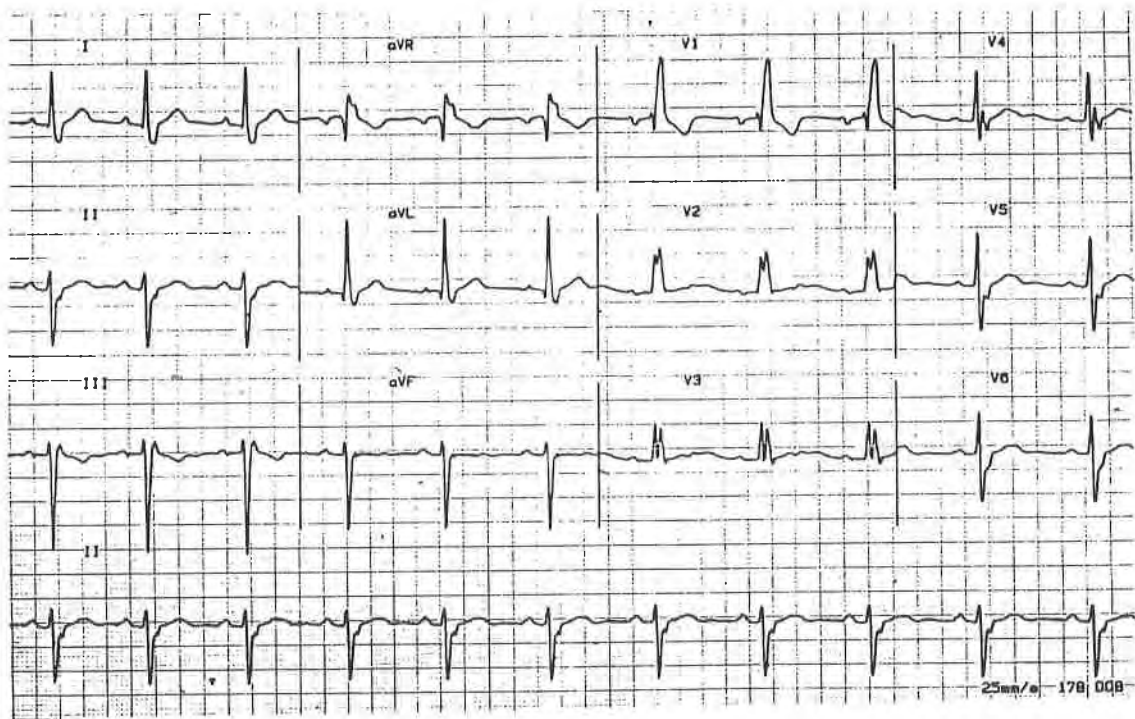
1. A E
2. A B E
3. C D E
4. C D

ECG QUIZ

Contributed by Dr Baldev Singh, MBBS (S'pore), MMed (Int Med), MRCP (UK)

The ECG shown below belongs to a 68-year-old Chinese male who presented with chest pain.

What are the ECG abnormalities present?



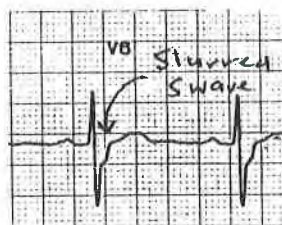
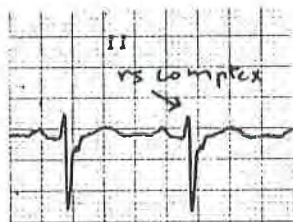
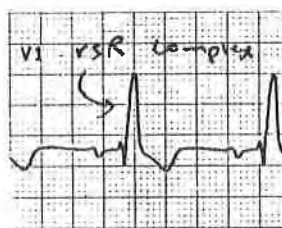
ANSWER TO ECG

1. The ECG shows rsR complexes in V1, V2, and V3. Slurred S waves are seen in I, aVL, V5, V6. The QRS is also widened to 0.12 sec. These features indicate a Right Bundle Branch Block.
2. The ECG also shows a left axis deviation of almost 60°, rS complexes are seen in II, III, aVF and very small q waves are noticeable in I and aVL. These are the features of a left anterior fascicular block.

The patient has a bifascicular block - a combination of Rt BBB and Lt. anterior fascicular block.

The majority of chronic interventricular blocks are caused by degenerative disease of the conduction system or the secondary involvement of the conduction system by calcification or fibrosis. Ischaemic heart disease and cardiomyopathy account for the remainder.

This patient's chest pain was not typical of ischaemia and a thallium exercise stress test also did not show any ischaemia.



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Further reading

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¹ Stevens, C.E., *et al.* J. Am. Med. Assoc. 257: 2612-2616, May 15, 1987.

² Yeoh, E.K., *et al.*: Abstract 282. A comparative study of recombinant versus plasma vaccine in high risk infants. 1987 International Symposium on Viral Hepatitis and Liver Disease, London, The London School of Hygiene and Tropical Medicine, May 25-28, 1987.

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Care required in volume or salt-depleted patients, those previously treated with diuretics and those

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Pregnancy

There are no studies in pregnant women. Should only be used if potential benefit outweighs the risk to the fetus. Caution if given to nursing mother. No paediatric experience.

Diuretics potentiate the antihypertensive effect of 'Zestril'. Symptomatic hypotension can be minimised by discontinuing diuretic prior to 'Zestril'. Avoid use of potassium sparing diuretics and potassium supplements with 'Zestril' especially in patients with renal impairment. If used concurrently, frequent monitoring of serum potassium is required. Indomethacin may diminish the antihypertensive efficacy of concomitantly administered 'Zestril'. 'Zestril' ameliorates diuretic-induced hypokalaemia.

Side effects

Mostly mild and transient: dizziness, headache, diarrhoea, fatigue, cough, nausea, rash, hypotension, orthostatic effects, palpitation, chest pain, asthenia. Angioneurotic oedema has been rarely reported. If it occurs, discontinue 'Zestril' promptly. Treatment with antihistamines or adrenaline may be appropriate. Increases in blood urea and serum creatinine, usually reversible, have been seen. Small decreases in haemoglobin and haematocrit have occurred. Hyperkalaemia may occur.

Overdosage

No data. Correct hypotension with plasma volume expansion. 'Zestril' is dialysable.

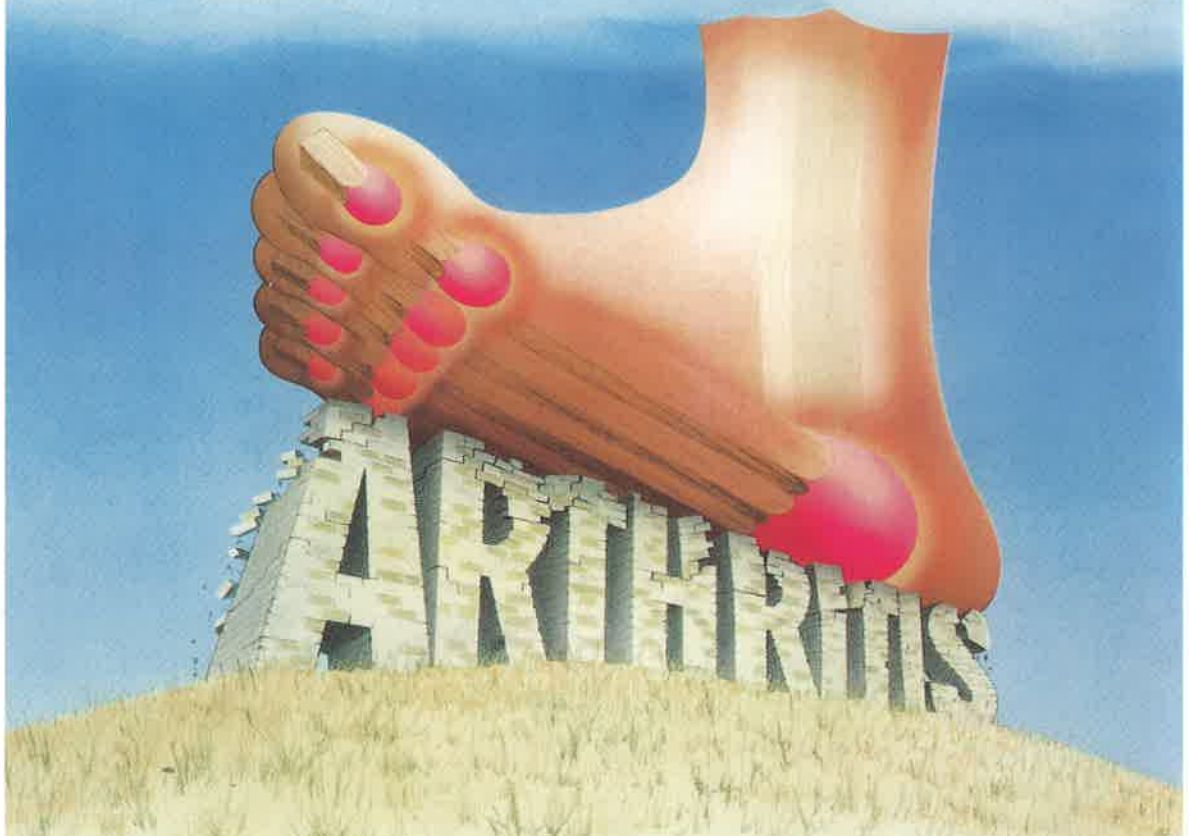
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