The Singapore Family Physician

The College of General Practitioners Singapore
Vol. XII No. 3
July/Sept 1986

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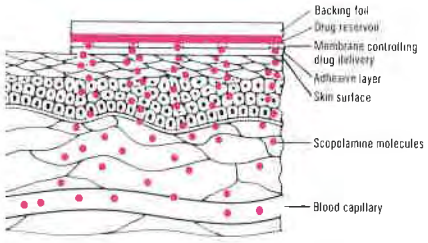

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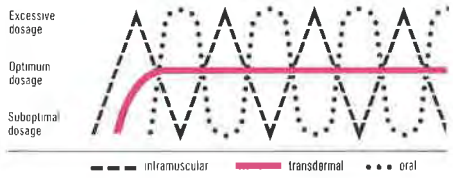

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CONTENTS

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- Simple dosage, easy for the patient
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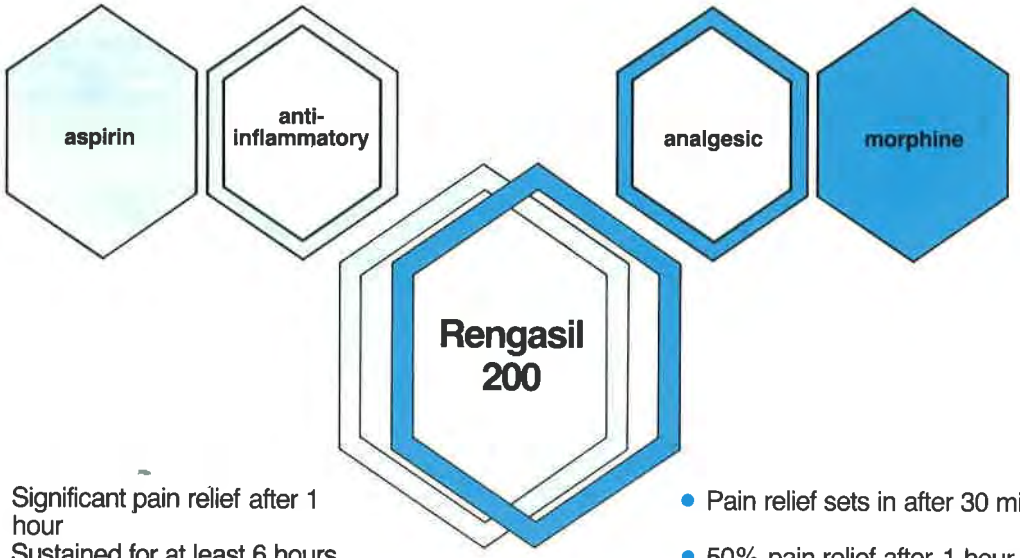
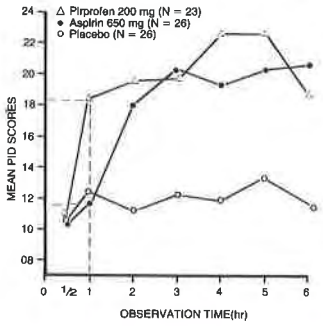


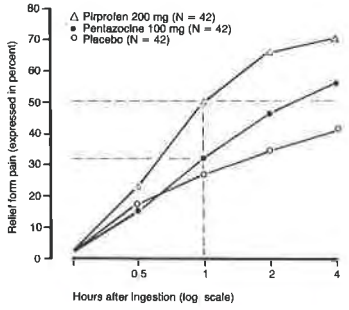
- Significant pain relief after 1 hour
- Sustained for at least 6 hours

Single-dose trial in postpartum uterine cramp pain[1]



- Pain relief sets in after 30 min
- 50% pain relief after 1 hour

Single-dose trial in oral surgery[2]



Outstanding pain relief right from the very first dose

1 Adapted from Bloomfield SS, et al In Current Therapeutic Research. Vol 30. No IS. July, 1981

2 Adapted from Sperr W.: A new analgesic-antirheumatic agent: pirprofen Int Symp , IXth Europ Congr. Rheumatol, 1979.

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EDITORIAL

ACADEMIC GENERAL PRACTICE — FACULTY DEVELOPMENT

The College of General Practitioners Singapore has since its inception played an active role in the National University of Singapore's programme of medical undergraduate teaching in General Practice/Family Medicine. Its Undergraduate Teaching Committee has organised didactic lectures, tutorial sessions, and the attachment of the students to Clinical Teachers — members of the College who volunteer to take the students into their practices to give them an insight into the realities of "front-line" medical care delivery.

The appointment of these volunteer teachers has hitherto been done under difficult conditions in that the "selection process" was hampered by the demand for teachers being far greater than the supply. In spite of this the College has over the years built up a core of dedicated and committed teachers who take in students year after year, attend meetings of the group and participate in teacher-training courses specially organised by the College. The number of such teachers is however still small and needs to be increased.

When the University sets up a Department (or Division) of General Practice/ Family Medicine, as it surely must if it is receptive of societal needs for cost-effective doctors of first contact, the importance of a procedure of selection of such teachers will be even more acutely felt. The Undergraduate Teaching Committee has reviewed the processes employed in other countries, and has proposed essential criteria and guidelines to be used locally.

Personal qualities required of the would-be teacher include a desire to teach, time to teach (or readiness to make time), membership of the College, clinical competence, additional academic qualifications, and a positive attitude to medical students and to Family Medicine itself. These teachers must participate regularly in the teaching of undergraduates, attend courses for teachers, and be willing to submit themselves to academic and operational assessment. Well-staffed and adequate practice premises, with suitable equipment and sound organisation, and an organised efficient record system are the other proposed requisites.

What are the "rewards" for these teachers? The University lists these Clinical Teachers in the University Calendar, makes University Library facilities available to them, and pays each an annual honorarium. The College gives recognition to their valued contribution by special mention in its annual dinner programme, and since last year has awarded Certificates of Appreciation to those with ten or more years of such service.

While the more altruistic of the teachers have continued in the programme for the simple reason of wanting to "help the College", surely more attractive perks are required if more teachers are to be enlisted, especially if the stringent eligibility criteria are brought into play. One that comes easily to mind is direct access for these teachers to the Clinical Departments of University and Government hospitals for admitting patients, instead of having to go through the Government OPD Clinics and/or the Accident & Emergency Departments as at present. Another would be the replacement of the honorarium by a stipend appropriate for the time and effort spent.

It will be an even more difficult job to recruit full-time members of the faculty. Convincing individuals to forego established and successful private practice for an academic career is a complex task. Some of their virtues which had helped assure success in private practice, especially rugged individualism, might hamper their effective functioning in the academic setting. Some practitioners may want to enter the halls of academia simply

because they want to teach, and be ill-prepared for the additional faculty tasks of research, committee work, administration, and curriculum development, not to mention medical writing, that would be put on them; this might lead to disillusionment and their eventual departure from academia.

Because the department, indeed the discipline, will be only as strong as its individual faculty, it will be critical to assure the continued productivity and vitality of Family Medicine/General Practice faculty. Development programmes must be set up to improve the skills of the faculty, both part-time and full-time, in teaching, research, clinical work and administration, so that the productivity of the department is increased. Such faculty development activities and the consequent development of skills by the teachers might even make some part-time faculty become more comfortable and committed in their academic roles and thus more attracted towards full-time teaching. Only with these faculty development programmes can it be ensured that the viability of the department, and the discipline, of General Practice/Family Medicine is not threatened.

MV

MANAGEMENT OF ANGINA PECTORIS

Dr Ding Zee Ping*
MBBS, M Med

Assoc Professor Arthur Tan**
M Med, FRACP (Cardiology), FACC

I. INTRODUCTION

Angina pectoris is a symptom of myocardial ischaemia. It is, however, a late manifestation of myocardial ischaemia. When coronary occlusion occurs, the first evidence of myocardial ischaemia is regional myocardial contraction abnormalities, followed by elevation of left ventricular end diastolic pressure, electrocardiographic changes and then angina pectoris. As a result, patients can have episodes of myocardial ischaemia without symptoms. These episodes of silent myocardial ischaemia can be as dangerous as symptomatic episodes.

II. Diagnosis of angina pectoris

The diagnosis of angina pectoris depends on the **history**. There are few clinical signs during angina. Patients are usually symptom free when seeing a doctor and there may be no clinical signs. A carefully taken history is the basis for diagnosis of angina pectoris.

The relevant features of the history are:

(1) Site and radiation of pain
(2) Precipitating cause
(3) Duration of the pain
(4) Character of the pain
(5) Associated features of the pain
(6) Any objective evidence of myocardial ischaemia.

*Cardiology Fellow, Department of Cardiology
Singapore General Hospital

**Head, Department of Cardiology
Singapore General Hospital

(1) Site of chest pain

As the heart is a visceral structure, pain arising from the myocardium is frequently referred to the corresponding dermatomes. The chest pain in angina is usually located retrosternally or over the praecordium. Other sites of angina include the neck, the left shoulder, medial aspect of the left arm, back of neck, right shoulder and epigastrium. These may occur in isolation, with or without any retrosternal pain. However, though different in site, the pain has other features suggestive of angina pectoris: a typical character, duration and effort relation. Hence, a different site of pain does not exclude the diagnosis of angina pectoris. Radiation of pain occurs up both sides of the neck or medial aspect of the left arm, though not commonly noted.

(2) Precipitating cause

An important feature of the history of angina pectoris is the precipitating cause of these episodes. In typical effort angina, the attack is induced by exertion, usually more strenous than normal effort, such as running upstairs or going uphill. These episodes of angina are more easily induced after meals (post-prandial angina) or during cold weather. The distance precipitating the angina can be fairly constant though in some instances variable. This is related to variable myocardial oxygen consumption with different aerobic activities and different degrees of coronary vasospasm.

(3) Duration of pain

The duration of angina pain is usually 3 to 10 minutes. Pain that occurs fleetingly in seconds is almost invariably not angina. Prolonged episodes of pain imply greater severity of myocardial ischaemia.

(4) Character of pain

An accurate description of the character of pain can be difficult to obtain from our patients. Classically, angina pectoris is described as a "crushing", "constrictive" or "tight" feeling in the chest. Some patients describe it as a "burning" or "chilly hot" sensation in the chest. Sharp or pin-pricking chest pain is not suggestive of angina pectoris. It is difficult to be conclusive about the diagnosis purely from description of the character of chest pain. Consideration must be given to the other features of angina.

(5) Associated symptoms

Associated symptoms of angina pectoris are an important part of the history. Patients usually describe a fear or apprehension with the chest pain, which they are unable to explain. This could be due to breathlessness which occurs frequently in patients having angina.

(6) Objective Evidence

It is always wise to obtain objective evidence of myocardial ischaemia in the patient with angina pectoris. Angina pectoris is however, a transient phenomenon and a normal electrocardiogram when the patient is pain-free does not exclude the diagnosis. In a patient who is having an attack of angina a 12-lead electrocardiogram should show electrocardiographic evidence of myocardial ischaemia, such as ST segment depression or T wave inversion. In cases when the history is doubtful, a stress test should be done for diagnostic and prognostic reasons before life-long therapy is started. If electrocardiographic changes of myocardial ischaemia are present during spontaneous angina a stress test is then not necessary for diagnosis.

The diagnosis of angina pectoris is incomplete without a clinical classification and establishment of **aetiology.** Angina pectoris can be divided into 1) Stable effort angina, 2) Unstable angina and 3) Prinzmetal angina.

(1) Stable Effort Angina

Stable effort angina is diagnosed when angina is precipitated by similar levels of physical activities. Such patients can be managed as outpatients with assessment of the risk factors, prognostic stratification and drug therapy.

(2) Unstable Angina Pectoris

Unstable angina pectoris has different definitions. As a clinical subset it implies a worse prognosis than stable effort angina.

In unstable angina there is a change in the symptoms of the patient with previously stable effort angina. The angina is more easily induced, the episodes are longer, more severe or occur at rest. Unstable angina implies a change in the dynamic equilibrium between myocardial oxygen supply and demand. As many patients with unstable angina develop myocardial infarction, they should be admitted and stablised.

(3) "Printzmetal" Angina

"Printzmetal" angina is a subset of angina due to coronary vasospasm. It was initially defined as episodes of rest angina associated with ST segment elevation. It has subsequently been shown that ST segment depression, T wave inversion can occur during episodes of coronary vasospasm, although at times it may be silent. In the classical vasospastic angina, the patient does not have significant coronary artery disease. He is able to exercise maximally in the day without effort angina and yet at rest, especially in the early hours of the morning during sleep, he is awakened with typical severe angina pectoris. It is important to identify patients with coronary vasospasm as it may be aggravated by beta-blockers. Calcium channel blockers are the therapy of choice in vasospastic angina.

Coronary artery spasm may also occur in patients with atherosclerotic disease, which may explain why patients with fixed atherosclerotic disease may have variable level of exertion before angina pectoris occurs.

Pathogenesis of Angina

A knowledge of the pathogenesis of angina is important as it forms the rationale for therapy.

Angina pectoris is due to an imbalance between coronary blood flow and myocardial oxygen demand. The imbalance results from a reduction in coronary flow, as in vasospasm, or an increase in myocardial oxygen demand as during exertion. This leads to myocardial ischaemia giving the subjective feeling of angina pectoris.

In patients with fixed atherosclerotic coronary artery disease, angina occurs when increased myocardial oxygen demand exceeds that which can be supplied by the narrowed, atherosclerotic coronary artery. It has been shown that due to large coronary reserve, there can be 75% diameter or 90% cross sectional luminal reduction before increased myocardial oxygen demands during exercise cannot be met. Therefore, when exertional angina pectoris occurs there is already fairly severe coronary atherosclerosis. In patients with coronary artery spasm, the coronary arteries are usually normal and angina occurs, not from increased myocardial oxygen demand but from a dramatic reduction in myocardial blood flow due to vasoconstriction of major epicardial arteries.

Coronary atherosclerosis is the most common cause of angina pectoris. However, it must not be forgotten that patients with severe left ventricular hypertrophy from hypertension or idiopathic hypertrophic cardiomyopathy, may have angina despite normal coronary arteries especially from subendocardial ischaemia. Similarly, in patients with aortic stenosis, the combination of left ventricular hypertrophy and reduced perfusion pressure from the stenosis causes angina pectoris even with normal coronary arteries.

III. Prognosis in Angina Pectoris

"How severe is my angina?"

The prognosis of angina pectoris is dependent on the severity of underlying coronary artery disease. Prognosis can be assessed from the 1) history, 2) resting electrocardiogram, 3) stress electrocardiogram 4) ambulatory electrocardiographic monitoring, 5) stress electrocardiogram in combination with nuclear cardiology techniques and 6) coronary angiography.

(1) History

The classification of angina pectoris into different subsets gives an idea of prognosis. Patients having unstable angina have an adverse prognosis and will require further evaluation.

Patients with previous myocardial infarction or a family history of premature death should also undergo further evaluation.

(2) Resting Electrocardiogram

The resting electrocardiogram can contribute to the prognostic evaluation of the patient. The presence of resting ischaemic ST segment changes or widespread ST segment changes during chest pain suggests the presence of severe coronary artery disease.

(3) Stress test with Electrocardiographic Monitoring

The stress test is one of the most useful methods for diagnostic and prognostic evaluation of patients with coronary artery disease. The stress test can be safely done with a risk estimated at 1:10,000 cases. It is mandatory that the test be done by trained medical personnel with adequate cardiopulmonary resuscitative equipment and correct case selection. The accuracy of the stress test depends on the clinical subset of the patient and must never be interpreted independently of the clinical picture. The sensivity and specificity of the stress test are about 60% and 70% respectively, depending on diagnostic criteria. Patients unable to do moderate exercise, who develop angina and electrocardiographic changes of myocardial ischaemia at a low workload or who do not have a systolic blood pressure rise with exercise have adverse prognosis.

The predictive accuracy of the stress test can be enhanced by use of radionuclides such as Thallium myocardial perfusion scans and Technetium radionuclide ventriculography.

(4) Ambulatory electrocardigraphy monitoring

Ambulatory electrocardiographic monitoring can be used for the diagnosis and assessment of prognosis in patients with angina pectoris. It is particularly useful in patients with frequent episodes of angina. It can also be used for assessing antianginal drug efficacy. Its disadvantage is that the results are not immediate. The tape has to be analysed and is dependent on the patient's physical activities. It is difficult to quantitate the severity of myocardial ischaemia as this is dependent on the degree of exertion by the patient. Ambulatory monitoring has enabled us to recognise that many episodes of angina are "silent" but manifested by electrocardiographic abnormalities.

(5) Coronary Angiography

Coronary angiography is the gold standard for diagnosis of coronary atherosclerosis. However, the presence of coronary atherosclerosis does not equate to angina pectoris and, many patients with significant coronary artery disease, may have chest pain not due to myocardial ischaemia. There is no replacement for a carefully taken history in the assessment of patients with coronary artery disease.

Coronary angiography provides vital prognostic information about the patient. It has been shown that the prognosis of the patient is influenced by the presence of left main coronary artery disease, the number of diseased vessels and the degree of left ventricular dysfunction. Coronary angiography can now be safely done with minimal morbidity or mortality and is a pre-requisite before coronary artery bypass surgery.

IV. Management of Angina Pectoris

The aims of management of angina pectoris are two-fold:

(1) To relieve symptoms

(2) To improve prognosis

(1) To relieve symptoms

(1) Acute symptomatic relief

The drug of choice for rapid relief of angina pectoris is sublingual nitrate. This takes effect in between 2 to 3 minutes and lasts for 5 to 10 minutes. Newer preparations such as buccal nitroglycerine and nitroglycerine spray have a quicker onset of action. If the angina is not relieved by 2-3 tablets of sublingual nitroglycerine the patient should seek medical attention immediately. In patients with unstable angina or recurrent rest angina despite maximum oral medical therapy, a continuous infusion of nitroglycerin is particularly effective.

(2) Prophylaxis of angina

The drug of choice for the prevention of angina are the **beta-blockers.** Beta-blockers work by reducing heart rate and blood pressure response to exercise as well as myocardial contractility. As a result, myocardial oxygen demand does not increase as rapidly during exercise enabling increased exercise. There are several classes of beta-blockers, giving frequent confusion over selection. Cardio-selectivity is a useful property but dose-related: cardio selective drugs in high doses become non-selective. In patients who have asthma or brittle diabetes it is safer to use drugs other than beta-blocker even if the beta-blocker is cardio-selective. Close monitoring of lipids must be done as beta-blockers increase triglycerides levels with chronic usage. It has been suggested that beta-blockers with intrinsic sympathomimetic activities do not upset lipid metabolism in patients. Contraindications to beta-blockers include bronchial asthma, chronic obstructive airway disease, severe diabetes, A-V conduction abnormalities and peripheral vascular disease. In such patients, calcium channel blockers will be preferable. Another common problem in patients undergoing treatment with beta-blockers is fatigue and malaise while on therapy. In a patient who is actively working such impairment of physical capacity may be unacceptable and non-beta-blocker drugs may have to be used.

Nitrates

Oral nitrates can be effectively combined with beta-blockers in patients with angina. Due to extensive hepatic metabolism larger doses are required compared to sublingual preparations. Nitrates can also be applied cutaneously; this is especially useful for nocturnal angina.

Nitrates are less effective than beta-blockers or calcium channel blockers when used as monotherapy. Initial nitrate therapy is frequently associated with headache and giddiness. A useful clinical tip will be to use lower doses of nitrates initially with gradual titration for maximum efficacy. The patient can be assured that side effects will lessen with continued usage.

Calcium Channel Blockers

Calcium channel blockers have been a recent exciting development in drugs for angina. At present, three groups of calcium blockers are available: verapamil, nifedipine and diltiazem. Though classified together, these drugs have different pharmacological and electrophysiological properties. Knowledge of their properties enables one calcium channel blocker to be used in preference to the other. Both verapamil and diltiazem act not only on the coronary vasculature but also on the sinoatrial and atrioventricular node. They can easily be used as monotherapy for angina, in contrast to nifedipine, when reflex tachycardia can occur if the patient is not on beta-blockers. The reflex tachycardia may be deleterious as myocardial oxygen demand is increased. However, the absence of any electrophysiological effects on the sinoatrial or atrioventricular node makes nifedipine an ideal drug to combine with a beta-blocker.

Calcium channel blockers will be the drug of choice in patients with coronary artery spasms. A carefully taken history may identify those with more frequent episodes of coronary vasospasm than effort angina. A second group of patients in whom calcium channel blockers are preferable, will be those having contra-indications or intolerance to beta-blockade.

A favourable "cocktail" used by cardiologists in patients of angina pectoris will be a combination of a beta-blocker, calcium channel blocker and nitrates. In such patients having triple therapy, close monitoring of the haemodynamic and electrophysiological effects of these drugs must be done.

IV. Prognosis

The prognosis of a patient with angina can be improved by:

(1) Modification of risk factors

The risk factors for coronary artery disease can be effectively altered and this has been shown to improve prognosis. One of the most important risk factors is cigarette smoking. It has been shown that even in those who have sustained a myocardial infarction the risk of subsequent coronary event will be reduced by cessation of smoking; after three years the risk approximates that of a non-smoker. The Lipid Research Clinic trials have demonstrated that patients with elevated cholesterol levels of more than 265 mg/dl have a reduction in subsequent coronary events in association with a reduction in blood cholesterol levels.

(2) Effective symptomatic treatment and prevention of myocardial ischaemia

With current medical therapy the majority of patients will have satisfactory control of their symptoms. Those symptomatic despite optimal therapy require further investigations for coronary angioplasty and bypass surgery.

(3) Assessment for coronary artery bypass surgery

This can be done by stratification of the patient into different risk categories, as described above. Patients with moderate or high risk should undergo invasive investigations.

Conclusion

Coronary heart disease is a leading cause of mortality and mobidity in our country. Knowledge of the clinical presentation, pathogenesis and management will enable better recognition of the unique problems of this condition.

OSTEOPOROSIS: CURRENT PREVENTIVE AND THERAPEUTIC OPTIONS

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INTRODUCTION

Osteoporosis is becoming a major problem in our elderly population. Unfortunately the condition often presents at a far-advanced stage with multiple fractures. The current emphasis should be on the prevention and management of osteoporosis at the early postmenopausal stage and the primary care physician has an important role to play in this respect.

Definition

Osteoporosis is characterised histologically by decreased bone mass per unit volume leading to multiple fractures. Unlike osteomalacia, the ratio of mineral to matrix is normal. Osteopenia refers to the radiological evidence of decreased bone mass from osteoporosis or osteomalacia. When there is Xray evidence of osteoporosis, bone loss of 30-50% is already present.[1] The condition can be diagnosed at the early stage by dual photon beam absorptiometry.

Pathophysiology

In about 5% of cases, osteoporosis is secondary to medical causes such as steroid therapy, renal failure or hypogonadism.[2] The remaining large majority are classified under the category of senile or postmenopausal osteoporosis. Although bone mass decreases with age, many other risk factors may contribute to the pathogenesis. (Table I)

Women with small stature at maximal skeletal maturity at 15-30 years will have less bone mass throughout life. Moreover lean women will produce less oestrogen than obese ones because of the decrease in the production of oestrogen from the lipocytes. Cigarette smoking, excessive alcohol, and caffeine stimulate osteoclastic action and inhibit osteoblastic activity.[3]

TABLE I
RISK FACTORS[3]

Female sex	Premature menopause (natural or acquired)
Small stature/leanness	Inadequate exercise
Family history of osteoporosis	Inadequate calcium intake
Cigarette smoking	Decreased calcium absorption
Excessive alcohol or caffeine	Impaired adaptive mechanism to a low calcium diet
Subclinical lactase deficiency	

The three major factors in the pathogenesis of osteoporosis are:

(1) Inadequate calcium intake, impaired absorption and adaptive mechanism in the elderly,

(2) Low oestrogen status following menopause,

(3) Inadequate physical activity.

Role of Calcium in osteoporosis

In the normal elderly there is decreased calcium intake, impaired gastrointestinal absorption and adaptive mechanism to a low calcium diet[4]. The recommended RDA for calcium is 800mg/day. The average elderly consumes a low intake of 450-500mg of calcium per day leading to a negative deficit which can contribute to a bone loss of 1.5% per year.[1] Heaney et al showed that 0.987 g of elemental calcium per day is required to maintain zero balance in premenopausal women but this requirement in postmenopausal women increases to 1.45 g/day.[5] This higher requirement for zero balance is due to the decreased efficiency of calcium absorption in the elderly. Finally the adaptive response to the low calcium intake and absorption is impaired because of the low level of 1.25 $(OH)_2$ Vit D_3[4].

The three processes of calcium intake, absorption and adaptation are more impaired in the elderly with osteoporosis. The impaired absorption is unlikely to be secondary to a primary intestinal transport problem because it can be correctable by 1.25 $(OH)_2$ Vit D_3.

Following the fall in oestrogen after menopause, bone resorbing cells have an increased sensitivity to endogenous PTH (parathyroid hormone) while the kidney retains its normal sensitivity. Thus the bone becomes a dominant and easily accessible source of calcium to meet the needs of calcium haemostasis. The small transient rise in serum calcium produces a mild suppression of PTH. The subsequent increase in serum phoshate suppresses the production of 1.25 $(OH)_2D_3$ from 25 (OH) D_3. The decrease in oestrogen also has a direct effect of decreasing the plasma Vit D binding protein leading to the low 1.25 $(OH)_2$ D_3 level. (Fig. I)

1.25 $(OH)_2D_3$ activity in osteoporosis[4]

The main defect in osteoporosis lies in the decreased level of this activated Vit D. This can be due to:

(1) effect of aging on 1α Hydroxylase which converts 25 (OH) D_3 to the activated form in the kidney,

(2) decreased 1α hydroxylase secondary to renal impairment in the elderly,

(3) low PTH level in osteoporosis,

(4) combination of the above 3 factors.

Based on the activity of PTH there are two distinct subsets in osteoporosis.[4]

Subset I

The majority of osteoporotics have a decreased PTH level leading finally to decreased Vit D_3 level.

Subset II

In 10-30% of cases, the PTH level is normal or even raised from secondary hyperparathyroidism. The decrease in the activated vit D_3 is due to an intrinsic defect of 1αhydroxylase in the kidney associated with normal aging or underlying renal impairment.[4] This particular group of osteoporotics are not responsive to oestrogen and the supply of calcium and Vit D_3 are more important than oestrogen therapy.[6]

Management of osteoporosis

The management of osteoporosis can be planned with regards to

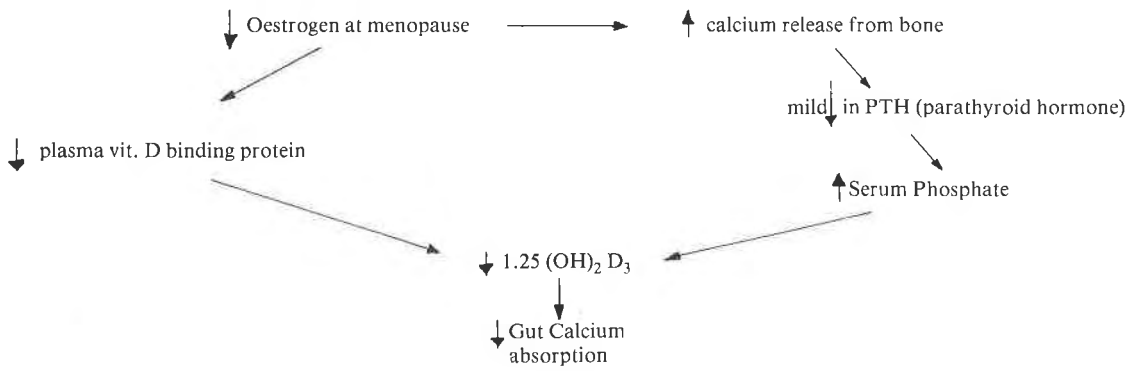
(1) the fracture threshold phenomenon and

(2) the bone remodelling stages.

Fracture threshold phenomenon[3,7]

Osteporotic females begin with a low bone mass which decreases slowly over the years to the critical fracture threshold level that predisposes them to fractures. There is a subgroup of females who are prone to develop osteoporosis at a younger age; they can be identified by presence of multiple risk factors. In the near future we can routinely identify this subgroup with confidence by measuring their bone mass objectively using dual beam photon absorptiometry.[8]

FIGURE I
RELATION OF CALCIUM/OESTROGEN/ AND VITAMIN D IN OSTEOPOROSIS[4]



Bone loss can be prevented by physical exercise and the use of *anti-resorbing agents* such as calcium, vitamin D, oestrogen and calcitonin. Calcium supplements and adequate exercise can be recommended for all post-menopausal females especially in the first three years after menopause when bone loss occurs most rapidly. In view of the costs and potential risks, oestrogens and calcitonin should only be administered to the high risk groups or established osteoporotics in the early stages.

Once the low bone mass has progressed to a significant stage which predisposes to fractures, *positive bone-formers* such as fluoride should be added to the therapeutic program.

Therapy in relation to bone remodelling stages[9]

Apart from the concept of fracture threshold phenomenon, therapy can be guided by recognising the two different phases of osteoporosis. There is an initial high turnover stage immediately following menopause characterised by elevated bone resorption and formation. Though both processes are active, resorption exceeds formation leading to negative calcium balance. The condition later progresses to a low turnover state with decreased bone formation and resorption with both processes being in equilibrium. The nett effect is a decrease in bone mass leading to fractures. Once these occur, there is a flare up of rapid bone loss associated with a high turnover state. This may revert back to the original low turnover state when bone remodelling is restabilised.

In the initial stage of osteoporosis with high turnover bone remodelling, bone resorption can be retarded by antiresorbers such as calcium, oestrogens and calcitonin. But once a low turnover phase is established, fluoride may be added to stimulate bone remodelling.

Calcium supplements

Due to the poor absorption in postmenopausal females, the large dose of 1 gram of elemental calcium is recommended in addition to the normal dietary intake of 500 mg per day.[1]

Calcium carbonate is preferred as it contains 40% of elemental calcium while calcium gluconate and calcium lactate produce 9% and 13% respectively.[1] If the usual calcium carbonate is given as the 500 mg tablet, at least 5 tablets will be needed daily to supply the required elemental calcium per day. Calcium carbonate is often well tolerated though it may produce constipation and flatulence at times.[1]

The usual Calcium et Vit D tablet contains 400 IU of Vit D, 450 mg of calcium lactate and 150 mg of calcium phosphate. This produces 80 mg of elemental calcium per tablet and on this basis about 12 tablets of Cat et Vit D need to be consumed per day. The cumulative dose of vitamin D would exceed the recommended daily requirements.

Calcium Sandoz (forte) provides a combination of calcium lactate, gluconate and carbonate which produces 500 mg of elemental calcium per tablet and this can be given twice per day for maintenance therapy in osteoporosis.

Calcium supplements should be avoided in those with urinary calcium of more than 300 mg per day, chronic renal failure and past history of urinary stones. Calcium may be combined with vitamin D but the dose of latter needs to be well controlled.

Vitamin D

The main defect in osteoporosis lies in the impaired conversion of 25 OH Vit D_3 to the activated form 1.25 $(OH)_2$ D_3. About 10-30% of osteoporotics have an intrinsic defect of the 1α hydroxylase system and they are thus nonresponsive to either oestrogen or the usual vitamin D_2 supplements. As the use of oestrogen remains controversial because of its safety, it appears rational to treat the defect directly by using 1.25 $(OH)_2$ D_3 supplements[6].

This active metabolite of vitamin D has a tendency to produce hypercalcemia in view of its potency and lack of physiologic feedback mechanism.[10] Thus the dose should be kept as low as 0.5 ug per day and regular serum and urinary calcium monitoring must be performed.

Recent studies have shown the usefulness of activated D_3 in increasing the bone mass in

conjunction with calcium supplements.[11] However, there is no large scale trial presently to confirm the safety of routine use of this vitamin together with calcium. The Hospital for Special Surgery in New York currently recommends the usual conservative vitamin D_2 (400 IU/day) in addition to calcium carbonate (3.9 g/day) for routine prophylaxis for all postmenopausal women.[11]

Calcitonin

Salmon calcitonin is a recent addition in the treatment of osteoporosis. This 32 amino-acid polypeptide acts by inhibiting osteoclastic activity and stimulating new bone formation leading to a significant increase of total body calcium.[12] Apart from the antiosteolytic action, calcitonin also possesses analgesic property via calcitonin receptors in the human brain.[8] Salmon calcitonin thus serves as a useful therapeutic agent for osteoporosis with high turnover state especially in those with bone pain associated with vertebral fractures. When given at an intramuscular dose of 100 IU daily in conjunction with calcium and vitamin D, the hormone exerts its full effect in about five to seven days and pain abates after one week of therapy. For osteoporotics with acute fractures, calcitonin may be continued for a brief period of 3-5 weeks.[8]

The side-effects are usually mild and subside spontaneously. 10% of patients develop nausea and vomiting which can be overcome by reducing the dose.[12] Another 5-10% may develop slight facial flushing and heat sensation. Rare cases of hypersensitivity may result but skin tests can be done routinely to exclude the hypersensitive ones. Prolonged treatment of bedridden patients must be accompanied by frequent urinalysis as coarse granular casts may occur.

Salmon calcitronin is a good therapeutic agent for high turnover osteoporosis but presently its use is limited by the expense and injection route. These disadvantages will soon be overcome by the introduction of the intranasal form of salmon calcitonin.[8]

OESTROGEN

Mechanism of action

As there are no oestrogen receptors in the bone, there is no direct action of the hormone on bone remodelling.[3] Oestrogen inhibits bone resorption indirectly by stimulating the release of calcitonin which acts on specific calcitonin receptors in the bone.[1] The absence of oestrogen may also render the bone more sensitive to the decalcifying action of parathyroid hormone.[3]

Efficacy

- Oestrogen has the proven efficacy of slowing bone loss in postmenopausal women.[13]
- Oestrogen also reduces the risk of hip or forearm fractures by 50-60% in those who use it for 6 years or more.[14] But no studies have conclusively shown the reduction of vertebral fractures after oestrogen therapy.[3]
- There is controversy in the relation of oestrogen to increased bone mass. Some studies have indicated that oestrogen therapy within 3-6 years after menopause will increase the bone mass initially but once symptomatic osteoporosis occurs, oestrogen can only help to halt further bone loss.[13] Other studies have shown that long term inhibition of bone resorption by oestrogen may finally lead to a decrease in bone formation.[1]
- There is no doubt that oestrogen slows down bone loss leading to decreased incidence of hip and forearm fractures but the longterm effect on bone mass is questionable.

Indications for oestrogen therapy

In view of the potential risks involved, oestrogen is limited to the following patients:-

(a) Postmenopausal women with high risk factors for developing osteoporosis especially those with premature menopause. (table I)

(b) Females with low bone mass at menopause.

(c) Osteoporotics with high turnover state.

(d) Patients must be compliant and willing to undergo frequent gynaecological check-ups.

(e) At-risk groups are excluded:- those with impaired liver function tests, past history of breast carcinoma, endometrial carcinoma and deep vein thrombosis or pulmonary embolism.

Form and dosage

Conjugated oestrogen (Premarin) is given at a low dose of 0.625 mg per day for a 21 day period followed by 10 mg of progesterone (Provera) per day for the last 7 days of the month. the addition of the progestational agent decreases the risk of endometrial carcinoma.[3]

Timing and duration of therapy

- Oestrogen should be started within the first 3 years of menopause when bone loss is most rapid.[3]
- The duration of therapy remains uncertain. In one study, loss of bone mass was prevented up to 8 years while on continuous treatment but the loss of bone mass resumed after 8 years despite continued therapy.[15]
- At present oestrogen should be continued for an indefinite period till results of longterm studies are publicised.

Side-effects

- Endometrial and breast carcinomas are established risks of longterm oestrogen therapy. The incidence of endometrial carcinoma increases by eight times after ten years of therapy.[1]
- Gallbladder disease occurs 2-3 times more commonly in those treated with oestrogen.[1]
- Patients with past history of myocardial infarct should not be denied oestrogen as there is no increased risk of infarcts in females while on long term hormonal therapy.[3]
- The risks of developing hypertension, pulmonary embolism and deep vein thrombosis remain controversial.[3]
- oestrogen has a positive effect on all-cause mortality related to its effect of raising HDL cholesterol.[16]

Is oestrogen therapy in postmenopausal females justifiable?

The beneficial effects must be weighed against the potential risks:-

Positive points

Slowing down of bone loss especially in the first 3 years

Reduction of hip and forearm fractures

Negative aspects

Definite risks of endometrial and breast cancer

No reduction of vertebral fractures

No actual increase in bone mass

Not so effective if started 6 or more years after menopause

10-30% of osteoporotics may be non-responsive to oestrogen

Based on current clinical evidence, the routine prophylactic use in all postmenopausal women is not recommended.[7] Those with high risk factors for developing osteoporosis early should be started on oestrogen treatment within the first 3 years after menopause. Photon absorptiometry may soon be established as a routine test to quantitate the bone mass of all postmenopausal women and definite recommendation for oestrogen therapy can be based on this objective test.[11]

Sodium fluoride

Sodium fluoride stimulates osteoblastic bone formation and is useful especially for elderly females with low bone turnover states.[8] As fluoride bone has increased crystallinity and decreased elasticity, the increase in the bone mass does not imply an increase in bone strength.[17] Because of this mineralisation defect, calcium in adequate dose of 1 gram per day must be given together with fluoride. Sodium fluoride is usually started at small doses and built up gradually to 50 mg per day. The main side effects are gastrointestinal upset (21%) and joint pain or periarticular fasciitis of the knees and ankles (34%).[18] side effects are usually minor and may be alleviated by enteric coated tablets.

The current unresolved problem is the length of therapy needed. It takes about 2 years at a dose of 40 mg per day before new fractures cease and treatment may be continued for a period of 3-4 years.[8] The study by Riggs et al showed individual variation in responsiveness to fluoride.[17] In this study 60% of fluoride-treated patients had radiographic evidence of increased vertebral mass associated with reduced fracture rates. These patients took at least one year to reach the critical threshold level of vertebral bone mass to prevent further fractures. In the remaining 40%, the level was not reached till 4-6 years of treatment. This individual difference in responsiveness was postulated to be due to an intrin-

sic abnormality of osteoblastic function present in some patients, which prevented the stimulation of bone mass to the critical level. Thus sodium fluoride therapy may also serve as a diagnostic means to identify the subset of osteoporotics with impaired osteoblastic activity.[17]

EXERCISE

Physical exercise is a good and safe prophylaxis against involutional bone loss as activity is an important determinant of bone remodelling. One prospective study of 31 postmenopausal women showed that moderate physical exercise increased the lumbar spine bone mineral content by 3.5%.[18] Nursing home residents who participated in another exercise problem had a 4.2% increase in the bone mineral content.[19]

Though exercises and proper posture of the axial loaded skeleton may be useful for the prevention of osteoporosis, postmenopausal women with established osteoporosis may run the risk of developing further fractures. Therefore mild and practical exercises should be recommended and motivation is needed for the successful longterm prophylaxis of spinal osteoporosis.

CONCLUSION

The ideal aim in the overall management of osteoporosis is the maintenance of a positive bone balance at all times to prevent fractures. This is best achieved by the prevention of bone loss in the early postmenopausal years by good nutrition, adequate calcium and vitamin D supplements and an effective exercise program. The use of oestrogen is at present limited to the susceptible groups with low bone mass and multiple risk factors. Calcitonin may soon be available as a convenient intranasal form for the treatment of high turnover osteoporotic states. Sodium fluoride, a positive bone-former, may be usefully combined with anti-bone resorbers in the later stages of osteoporosis.

Despite better understanding of its pathophysiology, treatment of osteoporosis remains unsatisfactory. Thus the current emphasis should be on prevention. Effective prophylactic measures started in the early postmenopausal years may reduce the morbidity of fractures in our elderly population.

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NORPLANT: A SUBDERMAL CONTRACEPTIVE IMPLANT SYSTEM

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INTRODUCTION

Fertility regulation has been a high priority of the Government of Singapore since 1965. Major efforts have been made to increase the number of users of modern methods of contraception and in spreading the reach of the National Family Planning programme. The primary objective of this programme is to provide a wide variety of contraceptives from which potential acceptors can choose.[1, 2]

The present array of modern contraceptive methods is less than perfect. The most effective reversible methods have troublesome side effects and for some women important health risks. Methods with the least side effects on the other hand tend to be less reliable in preventing pregnancy. Moreover, the perfect contraceptive is not currently available and may never be developed. However, considerable progress has been made through evolutionary changes based on existing methods. Today's spermicides, condoms, oral contraceptive pills, IUCDs and surgical procedures have major advantages in terms of safety or acceptability over their early prototypes[3, 4]. *Current oral contraceptives, for example, contain lower doses of oestrogens, a change which reduces side effects and health risks. Advances in female sterilization technique have made tubal occlusion an outpatient surgical procedure. At the same time, through long term post-marketing epidemiological research and surveillance, the potential risks of various methods such as oral contraceptives or IUCD's for specific kinds of users are more precisely understood and simple standardized screening procedures have been developed to detect women at risk. Consequently methods which continue to carry risks for a small number of women can be used with relative confidence by the greater majority of women*[3,4].

Nevertheless, each year somewhere between 40-45 million women undergo induced abortions — almost half of them illegal procedures and one estimate is that 20 million of these procedures follow contraceptive failure — a strong indication of the inadequacy of current contraceptive choices and the need for development of a wider choice of safe, effective, acceptable and affordable contraceptive[5-8].

Development of Norplant Implants

In 1967, Segal and Croxatto proposed that subdermal capsules of silastic could serve as the basis for long term reversible steroidal contraception[9]. Since then, laboratory research and clinical trials have resulted in the development of subdermal Norplant implants as a novel contraceptive method. The only drug used in the implants is long acting low dose progesterone, levonorgestrel, which is widely used in oral contraceptives and has regulatory approval for that purpose in several countries including the USA[10, 11].

The Norplant system was developed by the International Committee for Contraception Research (ICCR) of the Population Council, New York, USA. It is commercially

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manufactured at present by Huhtamaki Oy/Leiras Pharmaceutical of Finland under licence from the Council. Clinical studies began in 1975 and to date more than 200,000 women months of experience have been collected and analysed. In October 1984, the World Health Organisation (WHO) concluded after a specially convened technical review that "the norplant system is an effective and reversible long term method of fertility regulation. It is considered suitable for use in family planning programmes along with other currently available contraceptive preparations and devices, since it provides an important option for women desiring long term contraception"[12]. Similarly in 1985, the Department of Obstetrics and Gynaecology with the approval of the Ministry of Health undertook a study to evaluate the efficacy, acceptability and safety of this method of contraception in our local population and the preliminary results to date are encouraging[13].

Mode of Action

The Norplant system consists of six capsules each containing 36 mg of Levonorgestrel and having a diameter of 2.4 mm and a length of 3.4 cm. The six capsules appear to release levonorgestrel at a rate of approximately 80 ug per 24 hours during the first few weeks of use, declining over the next 18 months to a constant rate of approximately 30 ug of levonorgestrel per 24 hours. This latter rate of release is maintained for at least 5 years. The blood level is approximately equivalent to that attained daily by the use of progestogen-only mini pills but in the latter case of blood levels fluctuate with daily spikes. The steady release rate of Norplant, with a constant low level in the blood stream, probably accounts for the much lower pregnancy failure rates than with the mini pill. As with other progestogen only contraceptives, Norplant provides contraception by the following mechanisms:

(i) ovulation is inhibited in 50% of menstrual cycles. Levonorgestrel acts on the hypothalamus and pituitary and suppresses the LH surge responsible for ovulation.

(ii) the cervical mucus becomes thickened and impedes sperm penetration.

(iii) it has a suppressive effect on the endometrium. It limits the ability of the implantation of any eggs that do get fertilized by virtually halting the production of progesterone by the corpus luteum.

Selective Criteria and Contraindications

Since Norplant is a new method of contraception, there has been insufficient time for large scale, long term studies of rare adverse effects. Thus contraindications and possible warning regarding Norplant use must be based on extropolation from information in other hormonal methods. Until further information is available, the principle criteria for patient selection are: they have to be 18-40 years old, of demonstrated fertility (at least one birth), be neither pregnant nor breastfeeding at the time of insertion and have none of the contraindications to the use of steriods like undergoing anti-coagulant therapy, having undiagnosed abnormal uterine bleeding, haemorrhagic diathesis or active hepato-cellular disease.

Placement and Removal of Implants

The capsules are inserted subdermally usually in the inner aspect of the upper arm or in the palmar aspect of the forearm. The skin is first painted with an antiseptic and a local anaesthetic is applied. A single 2 mm incision is made and the capsules are introduced through the incision by a specially designed 10-gauge trochar. Capsules are fed through the trochar and placed just beneath the skin in a fan shaped pattern. Placement of the capsules requires about 5-7 minutes. Sutures are not required and a single butterfly bandage suffices.

Insertions should ideally be carried out during the first seven days after the onset of menstruation in order to minimise the risk of inserting Norplant in the presence of an undiagnosed pregnancy. Insertion can also be carried out immediately post-abortion and immediately post-partum in non-breastfeeding women. At this stage of knowledge, it is not possible to make a recommendation on the use of Norplant in lactating women. Local complications following insertion are uncommon and the cumulative local infection rate is 0.3%. Expulsion of the capsules is rare and has occured only in the presence of infection[14, 15].

The capsules should be removed after 5 years of use as the effectiveness of the method declines after this time. No harmful effects are caused if the capsules are not removed at this time other than the risk of unexplained pregnancy. Removal of the capsules is accom-

plished by a procedure similar to placement and again requires antiseptic measures, local anaesthesia, an incision and forceps. Removal of the capsules is more time consuming than the insertion and the mean time for removal is about 21 minutes.

Effects on Reproductive System

Besides changes in the cervical mucus, there have been no significant effects on the cervix, vagina or vulva. There were no significant pathological changes in the endometrial biopsies examined during the nine years of Norplant use. Transient ovarian enlargement was occasionally noted[16, 17].

Endocrine Effects

Some studies claimed decrease in testosterone and androstenedione levels,[18] while other studies demonstrated contrary results[19, 20]. With regards to effects on adrenal function, a decrease in peripheral levels of cortisol of approximately 30% over 12 months has been observed although the values are within the normal range. Norplant does not appear to disturb thyroid function significantly[20, 21].

Metabolic Effects

Liver function has been evaluated and besides slight non-significant increases in total serum protein and albumin, no other changes in liver function were seen[22].

Measurements of urea, nitrogen, uric acid, potassium, sodium and calcium show no changes[23].

Carbohydrate metabolism appears not to be effected by Norplant although mean blood glucose levels have been shown to be elevated but within the normal range after 12-51 months of Norplant use[21].

Lipid metabolism studies have given inconsistent results. Triglycerides, cholesterol and low density lipoproteins were reported to be significantly decreased with up to 37 months of use[20, 24, 25]. Wide discrepant changes in high-density lipoprotein (HDL) — cholesterol have been reported: no changes[20], no change initially but a significant increase at 12 months[24], and decrease[25]. In the centre reporting increasing HDL-cholesterol levels, the cholesterol/HDL cholesterol ratio was reduced at 6 months and further at 12 months. This would appear beneficial to the patients. However, further studies are still needed to clarify the effects of levonorgestrel released from Norplant on lipids and lipoproteins.

Coagulation studies showed significant increase in factor VII and a decrease in antithrombin III in Norplant users after 6 months[26]. These changes were significantly less than those observed in the groups taking oral contraceptives. Moreover, the oral contraceptive users showed significant changes in many of the other factors measured whereas the Norplant users did not.

Menstrual Pattern Changes

The most frequently reported side effects is disruption of menstrual rhythm in the early months of Norplant use. Most abnormal bleeding episodes are characterised by an increased frequency, an increased number of bleeding days or irregular bleeding and spotting. Heavy bleeding episodes are uncommon. Amenorrhoea may also occur. The incidence of these menstrual irregularities seems to diminish with time[27-29].

Since Norplant capsules release a synthetic progesterone, levonorgestrel, without the balance of an exogenous oestrogen or oestrogen stimulant, the disruption of menstrual function is expected to be pervasive[30]. In consequence, if women are told before the insertion of implants that they should expect altered menstrual patterns, then the tend to tolerate these menstrual irregularities relatively well and do not classify them as symptoms of ill health.

Non Menstrual Side Effects

These are usually few and insignificant. In a cohort of 992 women, a mean increase in weight of 1.4 kg occurred during the first year of use of Norplant. However although weight was increased in 52% of women, 35% of the women showed a decrease[31]. No significant changes in systolic or diastolic blood pressure were found. In the studies reported by the ICCR, headache was the second predominant spontaneously reported complaint after menstrual disturbances[31]. However, it has not appeared to be a problem in most clinical trials of Norplant in other countries[33, 34].

Use Effectiveness and Continuation Rates

Norplant has shown a very low pregnancy rate and a high continuation rate[27, 32-34]. The annual pregnancy rates range from 0.2 to 1.3 per 100 women per year during the five year clinical trials conducted by ICCR. The gross cumulative pregnancy rate at 5 years is 2.6 per 100 women years. This is indeed a very low use effectiveness rate for a reversible method. The method does not appear to increase the incidence of ectopic pregnancy. However, attention must be paid to the possibility of an ectopic pregnancy if clinical symptoms are suggestive. The Norplant system is considerably less effective after the five year period. Effectiveness of the method compares favourably with oral contraceptives and IUDs.

Evidence shows a high initial acceptability. The continuation rates at the end of the first year range from 60% to over 90% and by the fifth year around 50%[13, 32]. These continuation rates are comparable to those commonly quoted for the IUDs. Menstrual irregularities are the main cause of discontinuation and the aggregate cumulative discontinuation rate has been quoted as being 1.4 per 100 women at years.[32]. However it should be noted that these data are from clinical trials and therefore may not be representative of discontinuation rates that might be seen in programme use. It is difficult to compare the results of these studies with those on other contraceptive methods, since the contraceptive in question has a known duration of action of 5 years and the acceptors are thus likely to be highly motivated to continue contraceptive use for such a period.

Return of Fertility

The return of fertility is not delayed following the removal of Norplant. When the capsules are removed because of complications related to the method or if the woman wants to conceive, blood levels of levonorgestrel decline rapidly, becoming undetectable within 2 days and ovulation returns promptly. Of patients having Norplant removed in order to conceive, 40% had become pregnant by 3 months, 76% by 1 year and 90% by 24 months[31]. The rates are similar to normal rates of fecundity.

Service Delivery Issues

Service delivery issues arising from the introduction of Norplant must be addressed if utilization of this method in family planning programmes is to be successful. The cost effectiveness of the method relative to other methods of contraception remains to be determined.

Since Norplant utilizes as new mode of drug delivery requiring insertion and later removal, attention must be given to training of both physicians and health personnel for these procedures. In addition since Norplant is a progestogen only method associated with alteration of the menstrual cycle, specific and appropriate counselling is required for potential acceptors. Accordingly, the training of medical personnel in the use of the method and of staff responsible for counselling potential acceptors is the most important factor in the sucessful introduction of the Norplant method. It is important to identify specific centres that can serve as national or regional training centres. Similarly, back-up and referral facilities need to be identified.

Thus from the outset, it is essential that government health agencies be involved in the introduction of the method. Experience with the introduction of other methods of contraception has shown the essential role that 'pre-introductory trials' play in this process. These trials can be of use in identifying training requirements and other method related service delivery issues to ensure wide scale Norplant acceptance.

Conclusion

In summary, the findings on Norplant both in Singapore and in other countries are encouraging and indicate that this delivery system has the potential for a wider use in the future. It would appear that Norplant approaches the ideal contraceptive method for women who have had all the children they desire but do not want any irreversible contraception such as tubal sterilization. It further allows a woman to defer tubal sterilization until neurodevelopmental status of her children can be confidentially assessed; this is an important issue in a situation where two-children families are encouraged. Thirdly it provides a convenient method of contraception with low patient compliance and motivation in fertile women who wish long term pro-

tection but yet desire children in the future. Again it provides women with a non-coitus-related method of birth control.

The oestrogen free composition of the implant may have a special appeal for women seeking contraception during lactation, for older women for whom pills containing oestrogen may be contraindicated and for women seeking hormonal contraception who for logistical or motivation reasons are unsuccessful in using oral or injectable contraceptives over an extended period of time.

Future Developments

A second generation system is also being developed by the Population Council. This utilizes only two implants and is known as Norplant 2. This system consists of two rods only and the rods are 2.4 mm in diameter, 4.4 cm long and contain 70 mg of levonorgestrel. They have been shown to deliver amounts of levonorgestrel equivalent to those released by the regular six capsule Norplant. Norplant 2 is currently being tested in phase III clinical trials.

The only other implantable system for contraception that could be available to family planning programmes this decade is the Capronor device. Capronor consists of a single bio-degradable capsule that delivers Levonorgestrel for a period of approximately 18 months. This system is at an early stage of development. Preliminary clinical trials have been completed and studies will shortly be expanded to cover safety and efficacy[35, 36].

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ASSESSMENT OF FITNESS FOR ANAESTHESIA

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The aim of this presentation is to outline briefly the salient factors in the operative assessment of patients and clear some of the doubts regarding fitness for anaesthesia. This discussion is tailored toward the needs of the General Practitioner and the Community Health Service doctors.

As a preamble to this, perhaps a brief look at anaesthesia and the anaesthetist may not be out of place. The General Practitioner may meet the subject matter in a number of situations.

1. Knowledge of the subject matter (Anaesthesia)
   a) Patient may want to know.
   b) The GP may need to choose an anaesthetist.
2. Liaison with anaesthetist directly or indirectly
   a) To inform e.g. the drugs the patient is on.
   b) To follow up patient, especially in ICU.
3. Simple Anaesthetic procedure in office practice e.g. digital block.
4. Pain clinic, where the doctor may need to refer patients for chronic pain treatment.

Now, the subject Anaesthesia is very broad but suffice to say in essential two methods are presently available, viz.:

1. Local anaesthesia, including spinal, epidural etc.
2. General anaesthesia, which includes the triad of hypnosis (sleep), relaxation and analgesia (pain relief).

Nowadays, apart from administration of anaesthesia, the anaesthetists also become more and more involved in:-

a) Resuscitation
b) Intensive Care
c) Function as Respiratory Physiologists
d) Pain Clinic

It is understandable therefore that patients have to conform to certain requisites prior to anaesthesia. This could be called "tests". In terms of investigation per se, there are three areas to be thought of

a) simple bedside tests e.g breath holding tests which can be done by all the General Practitioners;
b) Clinical Laboratory tests — some General Practitioners may have facilities for testing e.g. Hb, Blood Sugar, etc.;
c) Research Lab: e.g. Lung Function Tests usually done only at hospital level.

In this hospital for instance, we have laid down certain tests to be followed routinely:-

I Patients over 40 years for any form of surgery should have the following tests done:
   a) Hb, TW and Platelet Counts;
   b) Urea/Electrolytes;
   c) Chest X-ray;
   d) ECG;
   e) Prothrombin Time/Partial Thromboplastin Time for major surgery;
   f) Blood Gases for very obese patients.

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II Patients below 40 for major surgery:-

a) Hb;
b) Urea/Electroyltes;
c) CXR

III Any other investigations that have relevance (e.g. blood sugar for diabetics)

The rationale for these is elucidated in the following discussion.

In the meantime, it has to be emphasized here that we are referring only to elective cases. Further, fitness for anaesthesia must imply fitness for general anaesthesia, as often times one may have to "convert" to general anaesthesia the local procedures.

The discussion here will give the doctor a background knowledge which will enable him:-

a) To render advice when asked.
b) To help the hospital and patient avoid unnecessary bed occupancy in the event the patient is not fit.
c) To extend his ability to provide total care to the patient.

It would not be a good reflection of a doctor if his main aim was to write a scratchy letter and advise the patient that the rest would be taken care of when he reached the hospital. Psychological support, advice on the probable course of events and adequate communication with the surgeon will prevent anxiety and unnecessary delay in treatment. The family physician can hence contribute much to ensure that the patient is in the best of health or as fit as possible. In order to achieve this, a few guidelines as follow may be of value to General Practitioners.

I History

An adequate history is of paramount importance in all medical examinations. In particular any relevant past medical illnesses and anaesthetic experiences could be noted. Effort tolerance is a simple qualitative gauge of fitness. The patient's ability to climb few flights of stairs easily contrasts with one who is dyspnoeic at rest.

II Physical Examination

Obvious defects like poor dental condition alerts the doctor. A loose carious tooth could be an unforeseen danger. Dental clearance may be needed prior to surgery. Examination of the heart and lungs is mandatory. The ability to diagnose the underlying pathology for a heart murmur or arrhythmia may not be absolutely necessary. A record of these defects would suffice to increase the awareness of the abnormality and the appropriate medical specialist would be consulted in the hospital for further management.

II Simple Laboratory Tests

Some clinics may have facilities for simple tests which become eventually very helpful. e.g. haemoglobin level, chest x-ray, electrocardiogram (ECG) and blood sugar.

The above investigations, together with urea, electrolytes and blood sugar, are routine for all patients for elective operations who are more than 40 years of age. For a number of patients below the age of 40 years, these parameters may have to be measured if the surgery is extensive.

The investigations done at the clinic level may save much time and trouble. Here is an example: A middle aged patient was recently admitted for elective surgery. The family physician wrote that the haemoglobin was 14 mg %, the electrocardiogram, chest x-ray, urea, sugar and electrolytes tested were normal one week prior to the date of admission. We accepted the patient as fit for anaesthesia as far as investigations were concerned, and surgery was carried out fairly expeditiously.

The American Society of Anaesthesiologists classifies patients into a number of grades according to their general condition. The modified classification is given here.

Class

1. Normal healthy patient.
2. A patient with mild systemic disease.
3. A patient with a severe systemic disease that limits activity but is not incapacitating.
4. A patient with an incapacitating systemic disease that is a constant threat to life.
5. A moribund patient.

A rough classified communication like this would assist the anaesthetist on the problem he is about to face.

Some common diseases increase the morbidity or mortality of patients undergoing elective operations. Risks and benefits must be weighed. No amount of risk should be taken for elective, non-urgent surgery. Some common conditions are discussed below.

1 Infections

Common bacterial infections include acute bronchitis and urinary tract infections. The most common viral infection is upper respiratory tract infection. All suspected bacterial infections should be treated first. All fevers should be diagnosed and treated. A patient with upper respiratory tract infection should be advised not to go for surgery until he is well. Exceptions include pus with fever, or a patient with mild upper respiratory tract infection going for superficial surgery where postponement is inconvenient. This has to be discussed with the anaesthetist. Perhaps it is important to explain this to the patient. General anaesthesia reduces the body's defence mechanism, the immune response. The combination of viral and anaesthetic suppression of one's immune response is most undesirable and could be detrimental. Cellular functions of white blood cells are also adversely affected. Prolonged general anaesthesia may cause bone marrow depression and leukopenia, with their attendant dangers.

Instrumentation of the airway (intubation) aggravates a viral sorethroat. A blocked nose prevents a patient from breathing adequately after general anaesthesia, and recovery may be stormy.

2 Respiratory Diseases

One of our chief concerns is the asthmatic patient, who is not uncommon. Bronchospasm creates much problem during anaesthesia. All asthmatics should be treated and stablised before operation. The medications should be continued to the day of the operation. It is a good practice to advise patients to bring along their medications including the inhalers. Asthmatics on steroid therapy, could suffer from adrenal suppression and Addisonian crises if the drug is suddenly withdrawn.

It is our practice here to advise patients to stop smoking 2-3 weeks before operation. Patients are told that:

a) Smoking increases irritability of the airways. This induces coughing when anaesthetic gases are introduced. It may affect the smooth induction of anaesthesia.

b) Smoking affects ciliary function. This may lead to mucus retention, postoperative pulmonary infection or collapse.

c) Smoking increases carboxyhaemoglobin levels and contributes to poor oxygen transport.

3 Cardiovascular System

Most patients with well-compensated heart disease undergo non-cardiac elective surgery safely in the hands of good anaesthetists. The family physician should, however, alert the surgeon or anaesthetist on the cardiac conditions of his patient, so that further consultations and necessary management could be instituted on time.

Hypertension is a common condition. The World Health Organisation adopted the figure of above 160/95 as significant. Whatever criteria are adopted, all patients should be advised to continue treatment until the day of the surgery. In this hospital, we adopt a value of diastolic 100 mg Hg as the cut off point above which operations are postponed and treatment instituted. Hypertension ($\geq$100 mm Hg diastolic) is associated with higher complication rates during and after anaesthesia.

Patients who have had recent acute myocardial infarction should not go for elective operations. A delay of 6 months to 1 year is advisable as reinfarction rates are higher in the early months following a fresh infarction.

4. Anaemia

What is the acceptable level of haemoglobin in a patient going for an elective operation? Most doctors will still accept the magic figure of 10 mg % as a cut off level of the haemoglobin. Many anaesthetists however believe that low haemoglobin is not an absolute contraindication for elective surgery,

the most obvious being the patient with chronic renal failure. They are often refractory to oral therapy and blood transfusion predisposes to myocardial failure. In addition transfusion causes suppression of erythropoeisis.

Anaemia can be due to failure of production of red blood cells or blood loss. Most important here is for the General Practitioner to recognise treatable anaemia. Here, we refer to the nutritional anaemia. Simple dietary advice and vitamin/iron therapy may correct the haemoglobin to the normal level.

If the General Practitioner has failed to treat the anaemia or if he has reasons to believe that the condition is due to a chronic loss of red blood cells, then he can still refer the patient to the surgeon. In the hospital the Medical Officer or Surgeon has to decide on the merits of blood transfusion bearing in mind that transfusion itself carries its own risk.

If the decision is to transfuse, than it is best done 2-3 days in advance. Chronic anaemia involves a compensated physiological state. There is usually an increase in the total blood volume. This is brought about by hypoxic vasodilation of the small vessels, hypotension and renal compensation by fluid conservation. There is thus a further haemodilution. Further, there is an increase in cardiac output due to decreased peripheral resistance consequent upon vasodilation. The viscosity of the blood is reduced. The increase in cardiac output increases tissue perfusion and compensates for the decreased oxygen carriage in the blood. There may be an increase in the level of 2-3 DPG. This substance is found in the RBC and decreases the affinity of the Hb for oxygen, which helps the unloading of oxygen at tissue level. Hence in the presence of increased blood volume and cardiac output, it is not physiological to give more blood especially in large quantities e.g 2-3 litres over a short period. This may tip the balance in those who are on the verge of cardiac failure. Therefore, we must transfuse slowly and with diuretics if necessary.

5. Other Diseases

Diabetes mellitus, thyrotoxicosis, epilepsy and other medical conditions should be stablised in the preparation for anaesthesia.

Muscular diseases are associated with malignant hyperthermia and adversely react with muscle relaxants. The history should be clearly written.

6. Electrolytes

Most important of the electrolytes is potassium. Potassium levels may be low due to chronic diuretic therapy without potassium supplements. A potassium level below 3 mmol/L is significant and replacement should be instituted. Hyperkalemia above the normal range of 3.5-5.5 mmol/L should be treated before operation. During anaesthesia, especially during intubation with succinylcholine , the potassium level may rise acutely. The toxic level is 7 mmol/L. Hypokalemia carries the risks of cardiac dysrhythmias, potentiation of digitalis, muscle weakness, inability to reverse relaxants and renal failure. Hyperkalemia leads to ventricular fibrillation and cardiac standstill.

Drug History

On many occasions during the pre-anaesthetic visits, we encounter patients who are unable to give the names of medications that they have been taking, sometimes even for many years. Here we are concerned in two areas:

1. Drug interactions i.e. between patient's drugs and anaesthetic agents.

a) Propranolol and calcium antagonists (e.g. Nefidepine, Verapamil, Diltiazem) affect myocardial contractility and conductivity which will be exhanced by anaesthetic agents like halothene. Heart block and cardiac failure may ensue.

b) Antibiotics, especially of the aminoglycoside class (e.g. Kanamycin, Neomycin, Streptomycin), potentiate neuromuscular blocking agents.

c) Psychotropic Drugs

Tricyclic antidepressants block reuptake of monoamines into adrenergic nerve terminals so that circulating cathecholomines are increased. Sinus tachycardia and ventricular dysrhythmias can occur if adrenaline is used during surgery.

Monoamine oxidases may interact with pethidine and morphine and potentially fatal reactions have been reported.

2. Adverse Drug Reactions —

Adverse drug reactions can be subdivided into three types:

a) Extreme pharmacological action of a drug. Methyldopa may precipitate postural hypotension and cause giddiness in some patients. Side effects of many drugs fall under this class.

b) Genetic problem, e.g. G6PD deficiency.

c) Drug allergy. This topic is very much in the limelight and therefore needs no further elucidation here.

In conclusion, the above presentation on pre-operative assessment of patients before surgery, often referred to as "fitness for surgery" is briefly outlined for the edification of the General Practitioners. Hopefully this would help in "arranging" the patient for elective surgery. While many of the clinical investigations are done to give a base-line reading and provide confidence to the anaesthetist, very often they are done not necessarily to confirm what we already know, but to know what we do not (e.g. chest x-ray in lung cyst).

GASTROINTESTINAL TUBERCULOSIS — THE GREAT MIMIC

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There are no classical clinical features in abdominal tuberculosis and manifestations are protean. The gastrointestinal tract, lymphatic system, peritoneum and solid viscera may be subject to varying degrees of involvement, alone or in combination with extra-abdominal disease.

The commonest symptom in GIT TB is abdominal pain, this being reported in 50-60% of cases in various series. Pain is most commonly due to acute or chronic obstruction and is thus associated with vomiting. Thus may it mimic GIT malignancy. Pain can also be due to tuberculous peritonitis presenting as an acute abdomen. This occurs mainly in young children and adolescents and may mimic acute appendicitis. Pain may also be due to abdominal distension caused by tuberculous ascites. Pain can also be caused by perforation. This is relatively uncommon occurring in 0-10.5% of various series. This is because of fibrous adhesions and induration at the base of tuberculous ulcers.

The next most common group of symptomatology is pyrexia, night sweats, asthenia and abdominal distension. These are non-acuate cases and the accuracy of diagnosis is only 50%, even in India where TB is endemic.

Another very common presentation is that of a right iliac fossa mass. Ileo-caecal tuberculosis is the most common site for TB, and usually presents as a right iliac fossa mass. It may then mimic Crohn's disease, appendiceal mass, colonic/caecal malignancy, actinomycosis, amoeboma or diverticulosis. It has been noted to be present in as high as 85% of some series of GIT TB.

Diarrhoea as a presenting symptom occurs only in less than 10% of patients with GIT TB. It is due to small bowel ulcerations or to TB colitis. There may occasionally be blood and mucus in the stools although it is rare when it will mimic other inflammatory bowel diseases.

Occult blood loss is a common sign with an incidence of about 30%. Massive haemorrhage is however very rare. In most large series from India, this problem was not encountered. Tuberculous gastrointestinal lesions do not bleed massively because of associated endarteritis. Vimla et al (1982) reported only two cases of ileo-caecal TB presenting with massive malaena, in 16 years of working in an endemic region. The two cases occurred in young persons who presented with recurrent episodes of malaena. There were no constitutional symptoms and no demonstrable pulmonary lesions. In one of their patients, superior mesenteric angiogram was normal. In both cases, the diagnosis was not clear before surgery and all had a right hemicoletomy or limited right hemicolectomy done at laparotomy for ileo-caecal tuberculosis.

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Pathological features of Abdominal TB

GIT TB can be either primary or secondary, primary TB being due to ingestion of Tuberculosis bovis. Secondary TB is of the hominis type and is secondary to tuberculosis elsewhere, usually PTB, either via the blood stream or through ingestion of sputum.

From a clinico-pathological view, there are 3 main groups. Firstly, tubeculous peritonitis and secondly gastrointestinal tuberculosis and of course a combination of the two.

In tuberculous peritonitis, there is usually a straw-coloured fluid ascites. The peritoneum and greater omentum is studded with fine white tubercles and there is thickening of the walls of the intestine and other solid viscera.

In the gastrointestinal group, 3 types of lesion are seen:

1) The ulceration type
2) The fibrous type
3) The hypertrophic type

Tuberculous enteritis is commonest in the ileo-caecal region (21.2 to 87% in various series) because of relative stagnation of contents and abundance of lymphoid tissue but TB may involve any part of the small or large intestine. The wall of the intestine is thickened with granulomatous infiltration and fibrosis and may form a mass. There are enlarged lymph nodes and tubercles may be found over the serosa and mesentery. There may be skip lesions in the mucosa. There are always superficial ulcerations of the mucosa with irregular and undermined edges, associated with pseudopolyps leading to narrowing of the lumen. These ulcers do not usually penetrate the muscularis mucosa but deep to this are masses of granulomas which may coalesce and often show central caseative necrosis.

Histological examination shows little difference between ulcerative lesions in the small intestine and the hypertrophic mass in the ileo-caecal region. Histology may reveal classical granulomas with epitheloid cells, Langhan's giant cells, central caseation and an outer rim of lymphocytes. In hypertrophic lesions, there is an excessive amount of fibrosis in the thickened wall and the hard white tissue may then mimic malignancy. It has been suggested that secondary infection of TB ulcers with pyogens may contribute to cause hypertrophic fibrosis. Granulomas are scattered in all layers of the intestine and may involve the serosa. Caseation is not often present. It was present in only 32.7% of the Mayo Clinic series (1950). When caseation is not present, the histological differentiation between TB and Crohn's may be difficult. The only certain way of diagnosis is the demonstration of the tubercle bacillus in the section. Positive identification was achieved in 4 to 75% of cases in various series from 1964 to 1983.

The final stage on the TB inflammatory process in the small intestine is fibrosis which leads to multiple strictures and eventually intestinal obstruction.

Investigations in gastrointestinal tuberculosis

1) The results of haematological and biochemical investigations will indicate a chronic inflammatory process.
   - — normocytic, normochromic anaemia
   - — low serum iron with a normal iron binding capacity
   - — low serum albumin
   - — disturbed alpha 2 — globulin fraction
   - — increased ESR, although often it is normal

2) Heaf and Mantoux are positive in 30 to 100% in various series.

3) Identification of the tubercle bacillus is extremely important. It can be cultured or guinea pig innoculated from the stool, ascitic fluid, peritoneal biopsy, liver biopsy, gastic aspirate. The bacilli can also be stained in tissue preparation. Culture is often negative and takes 6 weeks. In the presence of extensive pulmonary infection, the tubercle bacilli may be cultured from the stools without any demonstrable evidence of gastrointestinal infection. The converse is also true, i.e. a negative stool culture in an isolated gut disease does not exclude active tuberculosis. Das and Shukla found peritoneal biopsy to be a useful diagnostic procedure with positive results in 88 percent of patients with ascites and 42.1 percent of those without ascites.

4) **Xrays:** A plain Xray of the abdomen may show small bowel fluid levels with or without ascites in cases with intestinal obstruction. There may be diffuse calcification or evidence of localized abscess. In ileo-caecal tuberculosis there are characteristic radiological appearances on Barium enema. The caecum disappears and the ascending colon shortens. The ileum may retain its normal calibre but passes vertically upwards into the colon. A typical colonic tuberculosis may stimulate colonic malignancy with characteristic shouldering. Rarely disease of the oesophagus, stomach and duodenum may be demonstrated on barium meal examination.

5) **Ultrasonic Characteristics**

Bluth et al reported a case of ileal tubercu-

losis identified by the pseudokidney sign. This sign was first described in 1979 as an ultrasound pattern characteristic of a gastrointestinal lesion. The pattern consists of a strong echogenic centre surrounded by a sonolucent rim. The common factor present in all the abnormalities causing the characteristic pattern is bowel wall thickening. It is said that tuberculosis of the small bowel must be considered as a differential diagnosis of a pseudokidney sign in the right lower quadrant of the abdomen.

6) **CAT SCAN Evaluation**

Abdominal lymphadenopathy is the most common manifestation of tuberculosis on CT Scans. Adenopathy of course is found in a multitude of conditions but tuberculous adenopathy exhibits several characteristic but not pathognomic features:

a) There is a striking tendency for involvement of the mesenteric and peripancreatic nodes overshadowing the extent of retroperitoneal involvement.

b) Abdominal TB shows a gradient of retroperitoneal involvement, most severe in the upper abdomen; the lower nodes are less commonly involved because they drain the lower extremities which are less likely to be affected by haematogenous TB except when the infection is overwhelming.

c) Lymph node masses even when large do not usually cause obstruction of the biliary ducts, ureters or bowel. Obstructive jaundice due to adenopathy in the porta hepatis has been reported as a rare complication of TB in North America.

d) Low density centres within TB abdominal lymph nodes are suggestive but not diagnostic. They may be found in secondary malignancy, lymphoma (especially when treated), pyogenic infection and Whipple's disease. TB shows this presumably due to central caseative necrosis.

e) It had also been suggested that high density ascites may be characteristic of TB. Fluid density measurements ranged from 20 to 45 Hounsfield units (HU) (average 30 HU). This can be explained by the high protein and cellular contents of a TB effusion. In the earlier transudative stage, the density may be near water (0-10 HU).

f) In all patients with ascites, there was abnormal thickening of soft tissue and nodularity associated with the peritoneal surfaces, mesentery and omentum. Findings of tubo-ovarian abscesses, adenopathy, peritoneal exhancement or a dirty-looking mesentery suggest a complex nature to the ascites and TB as a diagnosis.

7) **Laparoscopy**

Laparoscopy is a safe and effective method of obtaining an early diagnosis in patients with suspected tuberculous peritonitis. Diagnosis by culture of ascitic fluid is unreliable and often gives false negative results. Blind percutaneous needle biopsy of the peritoneum was advocated but it has a variable success rate. Open peritoneal biopsy was advocated then as being safer and more reliable. Laparoscopy however avoids laparotomy and is reliable and safe. Laparoscopy should be used with caution in a patient with an obviously "doughy" abdomen, caused by matted tuberculous adhesions, lest bowel should be adherent to anterior abdominal wall.

8) **Colonoscopy**

Numerous reports have stressed the need for surgical intervention to establish the diagnosis but recenly it has been shown that GIT TB is adequately treated by anti-TB drugs alone. Colonoscopy thus provides histologic and bacteriologic material for definitive diagnosis. It has been shown by Brieter et al that multiple target biopsies from the deeper portions of the ulcer bed and **margins** very often reveals granulomas or stains acid fast bacilli in spite of claims that TB is largely a disease of the submucoasa.

9) **Exploratory Laparotomy**
If all esle fails, exploratory laparotomy may have to be resorted to for a definitive diagnosis.

Surgical Management

The surgical management of abdominal tuberculosis depends on whether the condition is acute or non-acute. For non-acute situations, surgery is mainly employed for diagnosis.

Laparotomy should however be avoided if possible and even patients requiring surgery benefit from the preoperative anti-tuberculous chemotherapy.

There are several occasions when surgery is indicated.

1) Perforation of a tuberculous ulcer either into the free peritoneal cavity or with localized abscess formation.
2) Acute or subacute intestional obstruction by fibrotic or hypertrophic TB gut or due to mesentery shortening causing kinking.
3) Massive gastrointestinal haemorrhage.

Perforation may be managed by double layer closure with resection of the perforated segment. Simple closure may be done with or without omental reinforcements. It has been suggested that simple closure is contraindicated when perforation has occurred in a segment of bowel where there is much granulation and caseation. It is also contraindicated when there is a stenosing stricture or obstruction distally.

In obstructed cases, if the disease is limited to a short segment, segmental resection is the treatment of choice:

1) in view of complications — cicatrization due to healing fibrosis
b) decreases duration of medical treatment

Obstruction may also be treated by a bypass procedure, e.g. gastrojejunostomy for duodenal tuberculosis, whenever resection is not possible because of widespread peritoneal tubercles or massive lymphadenitis. Plastic corrective ileo-caecoplasty has been used for ileo-caecal obstruction.

The treatment of choice for gastrointestinal tuberculosis presenting with massive haemorrhage is segmental resection.

Surgical management should always be followed by anti-TB drugs and even preceded by them if diagnosis is already certain.

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HOME STUDY SECTION

COMMON SKIN INFECTION: VIRAL INFECTIONS OF THE SKIN

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INTRODUCTION

Not long after the discovery of bacteria it became apparent that there was a subdivision of micro-organisms viz. the viruses, very different from the bacterial pathogens, that cause common infections as measles and herpes simplex.

Viruses are living organisms since they contain nucleic acids which apart from their small size are identical to those found in all other living organisms. The intact skin provides a powerful defence against direct external viral infection but the skin is a common target organ in systemic viral infections and the manifestation of infection in this organ is frequently the most characteristic manifestation of the disease. Cutaneous manifestation of systemic viral infections e.g. measles and chickenpox will not be discussed here. This paper discusses the common localized cutaneous viral infections.

HERPES SIMPLEX VIRUS INFECTION

Herpes simplex virus (HSV) is divided in subgroups A and B on the basis of their behaviour in cell cultures. The viruses in group A are readily released from infected cells and include HSV type 1 and 2 of man. The group B viruses are released from cells with difficulty, and include varicella-zoster virus, cytomegalovirus and Epstein-Barr virus, all of which infect man. HSV enters the host through the mucous membranes of the nasopharynx, conjunctivae and genitalia, or through traumatized skin. The virus multiplies at the point of entry; and is spread via the blood or peripheral nerves. After resolution of the primary infection, the virus becomes dormant and persists intracellularly, usually in the nerve cells in the ganglia. There is usually sufficient circulating antibody after a primary infection to inactivate extra-cellular virus for some time.

Man is the only known natural reservoir of HSV and the virus is ubiquitous in mankind; 70-90% of adults possess neutralizing antibodies to HSV Type 1 (as evidence of past HSV infection).

Primary infection with HSV Type 1 usually occurs before 5 years of age but is rare in infants under 6 months old as infants are protected by passively transferred maternal antibody. The virus spreads from person to person by direct contact and probably indirectly from eating or drinking utensils contaminated with infected saliva. HSV Type 2 is sexually transmitted except in neonatal infections where infection with the virus occurs during parturition.

Clinical Features

The skin, oral cavity, vagina, conjunctiva and nervous system are the most frequent sites of HSV infection. This usually begins with the formation of a painful clear flaccid vesicle which ruptures easily producing a shallow ulcer. Either Type 1 or Type 2 HSV can be associated with any of the clinical sites of infection. Type 1 virus is usually isolated from oral lesions but occasionally Type 2 viruses can be isolated from this site. Herpes genitalis is normally caused by Type 2 virus, but isolates from up to 5% of cases have been found to be Type 1 virus. In general however, lesions above the waist are associated with Type 1 infection, and those below with Type 2.

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Primary infection with HSV Type 1

Mucous membranes:

Gingivostomatitis. The mouth and lips are the most frequent sites of primary infection in children. Many children may develop quite trivial or unrecognized lesions. Lesions are initially vesicular but rapidly evolve into greyish-yellow plaques and ulcers (fig. 1). Affected mucous membranes are red and swollen and there is an oral fetor, drooling, pain and regional lymphadenopathy. Systemic symptoms of fever, malaise, and headache may occur. Healing of ulcers takes 10 to 14 days.

Skin:

Primary herpetic dermatitis: The disease is usually seen in young children and is characterized by a localised eruption and systemic symptoms of malaise and fever. The rash appear in crops and resemble the rash of varicella but the vesicles are grouped (fig. 1). Occasionally the lesions appear to occur along the distribution of a sensory nerve and have a zosteriform appearance (fig. 2).

Traumatic herpes: This results from infection with HSV which enters through a break in the skin and these infections may be caused by either Type 1 or 2 virus. The condition usually occurs on the limbs where minor abrasions and cuts commonly occur.

Herpetic whitlow: This is recurrent herpes infection on the finger. In all cases there was a history of contact with patients with active herpetic lesions. It is not uncommon. The distal segments of the fingers, usually the thumb and index finger, are the usual sites. At first there is erythema and tenderness at the site of trauma and shortly afterwards vesicles appear. The vesicles then enlarge and coalesce to form a blister with a surrounding bright border of erythema. An abscess may develop. In the primary infections there may be systemic symptoms of fever and malaise, but this is rare in recurrent eruptions which usually occur at the site of the initial lesion. The disease is self-limiting and lesions take two or three weeks to resolve if left untreated. A prodrome prior to vesicular eruptions in recurrent lesions is common.

Eczema herpeticum (Kaposi's vericelliform eruption): This is a severe infection by HSV and is most commonly seen in patients with atopic eczema. It can also occurs in patients with Darier's disease (an autosomal dominant skin disorder). The HSV appears to enter the diseased skin producing an acute onset of vesicular lesions over eczematous areas of the skin (fig. 3). In patients with severe atopic dermatitis, generalised vesicular lesion may be seen. The vesicles appear in crops over 3-4 days and may remain for as long as nine days. The patients are usually febrile and the illness may at times be severe. Large areas of eptithelium may be denuded causing loss of body fluids and dehydration. Secondary bacterial infection is not uncommon. Severe HSV infection may present as multiple superficial erosions on background eczematous skin.

Primary Infection with HSV Type 2

Herpes genitalis

Genital herpes was recognized by the 19th Century physicians although they were unaware of its viral aetiology. It is usually caused by HSV type 2.

Type 2 infection may produce illnesses of varying severity and in the majority of infections in women no significant symptoms develop except in the initial (primary) infections. Lesions are most commonly found on the vulva, vagina, cervix and the perineum, and occasionally on the mons pubis, perianal region, thighs and buttock. The symptoms associated with herpetic vulvovaginitis are burning sensation, itch, and hyperaesthesia on the vulva and in the vagina. Dysuria, dyspareunia and vaginal discharge are common. Severe initial (primary) HSV infection is often associated with dysuria, urinary retention, inguinal lymphadenopathy and constitutional symptoms. The herpetic lesions are morphologically similar to herpetic lesions on other parts of the body, consisting of erythematous papules and vesicles that rapidly rupture leaving shallow painful ulcers (fig. 4). On the moist membranes of the vagina and cervix, vesicular lesions are uncommon and diffuse erythema with superimposed mucoid patches and ulcers covered with yellowish-grey pseudomembrane may be seen on speculum examination.

Necrotic cervical ulcers may develop. The initial (primary) HSV infection in females is often associated with severe erosions (fig. 4),

secondary infection, regional lymphadenitis and marked prostration that requires hospitalization.

Infection in the male normally produces vesicular eruption on the glans, prepuce or shaft of the penis (fig. 5). The lesions may be itchy and painful and a watery urethral discharge and dysuria may occur. Symptoms of urethritis and prostatitis can occur in the absence of overt lesions on the penis in some patients. As in females the initial or primary herpetic infection tends to be more severe lasting 10 to 14 days and recurrent infection milder and clears spontaneously after about 5 to 7 days if no secondary infection occurs.

Recurrent HSV Infection

Even though antibodies to HSV develop within a few weeks of the primary HSV lesion and persist throughout life, the infection characteristically recurs, and often at the site of the original lesions.

Recurrent herpetic infection tends to be less sever than initial (primary) infection and such patients often experience prodrome of itch, tingling or burning sensation before recurrences. This then is followed by papular, vesicular and then superficial ulcers occasionally with crusting, the lesions evolving over 5 to 7 days. Classicially the recurrent lesion recurs at or near the site of primary eruption.

The intervals between recurrences are unpredictable and varies from a few days to years. A variety of stimuli may induce recurrences. Local trauma including sexual intercourse, ultraviolet light are common provoking factors: many patients get attacks after sunbathing. Fever is perhaps the most common stimulus, and cold sores frequently follow respiratory tract infections such as the common cold. Emotional stress and upsets can produce outbreaks in some people.

Neonatal herpes infection

This HSV infection occurs in infants within the first four weeks of age. Infection occurs during parturition in the infected mother. About half of neonates with HSV infection show evidence of central nervous system involvement, almost all cases with hepatic involvement, and about one third develop vesicular skin rash with mucous membrane (mouth and eyes) involvement. On the whole however, relatively few infants born to mothers with genital herpes develop neonatal herpes. In one study it was found that although 50% of infants born to mothers with primary disease became infected, only 4% of offspring born to mothers with recurrent disease were affected. There is an increased abortion rate among women with active herpes genitalis infection during the first twenty weeks of pregnancy, and also an increased prematurity rate in women with genital herpes after thirty weeks of gestation.

Diagnosis of HSV infection

Direct demonstration of the virus or viral antigens in lesions can be rapidly obtained from vesicle fluid, or scraping from the base of lesions. Examination for Tzanck's cell (giant cells from the base of the ulcers) is also diagnostic. However, culture facility is usually not available in private practice.

Antibodies (serological test) against HSV is useful only in initial (primary) HSV infection. A four fold rise in antibodies titre against HSV over a 2 to 3 weeks period is diagnostic of recent HSV infection. Single positive HSV antibody is not indicative of recent infection. A high IgM antibody may be indicative of recent HSV infection but does not differentiate between a primary and recurrent infection. In addition the presence of HSV Type 2 IgM antibody does not necessary indicate veneral HSV infection since Type 2 virus can be non-venereal infection. Serology findings alone are thus academic and useless in deciding whether the HSV infection is venereal in origin or not. The result has to be assessed with the clinical history and findings. Serology tests are generally useless for recurrent HSV infection as the rising HSV antibody titre during recurrent HSV infection is usually minimal. The diagnosis of HSV infection can be made clinically usually. However, it may be important to confirm genital herpes by viral culture where medicolegal litigation is expected. Pregnant females with suspected genital HSV infection should have the infection confirmed by cultures and if found positive should undergo weekly HSV cultures after 35 weeks gestation; a Ceasarian section is recommended if any HSV culture was positive.

Treatment of HSV infection

Most patients with HSV infection of the skin and mucosa require symptomatic treatment only and antibiotics are indicated only with secondary bacterial infections. Systemic antiviral agents are required only in special cases and it is generally unnecessary to treat common HSV infection of the skin and mucosa with systemic antiviral agents. Personal hygiene is all that is required in most cases. Normal saline wash to the lesions with analgesics are adequate in most cases. Systemic or topical antibiotics should be given where secondary infection is present. Most available topical antiviral agents are not effective in alleviating the symptoms and duration of HSV infection.

Clinical studies indicated that acyclovir (acycloguanosine) is effective in shortening the duration of HSV infection if administered early in the course of the disease. Acyclovir is available as oral tablets and as topical cream and acts by inhibiting DNA synthesis in the presence of HSV thymidine kinase and therefore is specific against HSV only. Intravenous acyclovir is indicated only in severe life-threatening HSV infection. Acyclovir is expensive and the oral tablet is generally indicated in severe initial (primary) HSV infection where painful and severe erosive lesions are often present. Acyclovir has to be given within 4 days of symptoms and signs of HSV infection to be effective. It is particularly useful in females with initial HSV infection. It must be remembered that acyclovir merely hastens the recovery of herpetic lesions and often does not eradicate HSV infection and does not prevent recurrent HSV infections. Patients with frequent recurrent HSV infection may have their recurrences aborted if acyclovir is taken early, preferably during the prodrome phase but this is an expensive way to prevent recurrences. Topical acyclovir has also been reported to be effective in shortening the course of HSV lesions if applied early but again does not prevent recurrences.

There are several other topical and oral antiviral agents, including a HSV vaccine in the market which are promoted to be effective in the treatment HSV infection, but there is no convincing scientific evidence to support their efficacy.

HERPES ZOSTER

Varicella and zoster are caused by the same agent, varicella zoster virus (VZV). Like HSV, it belongs to the member of the herpes virus family. Varicella (chickenpox) is caused by primary infection with this virus and zoster is the localised segmental recurrent lesion.

Herpes zoster usually occurs in a patient with past history of varicella and the condition is due to the reactivation of latent VZV. The triggering factors for reactivation of VZV include trauma, sunburn, immunosuppressive therapy or during altered immune status as seen in patient with malignancy and severe infections, although in many cases no triggering factor can be identified. Immunity to VZV infection is long lasting, but the neutralizing antibody produced does not protect from reactivation of the latent virus.

Clinical Features

Eruption of herpes zoster is usually preceded by prodromal sensory symptoms over one or two dermatomes. This is characterised by paraesthesia and burning sensation or shooting pain in the affected dermatomes. The overlying skin may be tender to touch. This prodrome usually usually lasts 2 to 4 days. Initially the skin is oedematous and red. Then grouped vesicles containing clear fluid develop at intervals over the affected dermatomes (fig. 6). The severity of the lesion varies. Secondary bacterial infection may occur. The vesicles dry up with crusting after 7 days and the scabs peels off after about 10 to 14 days. Patients should be advised not to pick on the scabs as this may lead to unsightly scarring.

The ophthalmic division and maxillary divison (fig. 8) of the trigeminal nerve, C3, T5 and L2 dermatomes for some unexplained reasons are more commonly affected with herpes zoster. Occasionally more than one segment are involved. Involvement of the VII cranial nerve (facial nerve) produces Ramsay Hunt syndrome (fig. 7), a condition which presents with vesicular zoster lesions on the affected ipsilateral ear, cheek, buccal mucosa and palate and associated with lower neurone ipsilateral facial palsy (Bell's palsy). Occasionally abberrent vesicular lesions may appear on areas of skin distant from the dermatomes of the herpes zoster lesions. In immunocompromised patients disseminate

herpes zoster may occur.

Complications of herpes zoster

Pain during the prodrome and following zoster eruption and post herpetic neuralgia are not uncommon in older patients. Chronic post-herpetic neuralgia can be sometime be so severe in the elderly as to cause suicidal tendency.

During acute zoster eruption, stubborn constipation and cystitis can occur if the lower thoracic segment and sacral segment are involved respectively.

Cutaneous involvement of the nasociliary nerve is a good pointer of possible occular complication. Vesicles appearing on the medial eyelid margins are invariably associated with some ocular involvement. These patients require ophthalmologic treatment early to prevent scarring.

Diagnosis of herpes zoster

The diagnosis of herpes zoster can be made clinically on most occasions. Smear and culture of VZV is unnecessary for the confirmation of the diagnosis. However during the prodrome period herpes zoster may be confused with several acute medical conditions such as pleurisy, prolapse intervertebral disc. A full examination, and rectal examination should be carried out. For normal screening, a full blood count, ESR, liver function test, urine microscopy and chest X'ray required.

Treatment of herpes zoster

In mild attacks, rest, analgesia and prevention and control of secondary infection are all that is required. Paracetamol or aspirin is adequate to relieve mild neuralgia. Mefanamic acid or codeine compound may be indicated in severe neuralgia. A mild sedative may be useful.

There is no effective topical or oral antiviral agent in the treatment and control of VZV infection. Systemic antiviral agents are seldom indicated except in severe disseminated herpes zoster and even then the efficacy of these antiviral agents remains doubtful.

Acyclovir is much less active agains VZV than it is against HSV. Large doses of acyclovir (up to 3 to 4 times normal doses for HSV infection) may be required to effectively shorten the duration of zoster lesions.

Oral prednisolone has been reported to reduce the risk of post-herpetic neuralgia in the elderly. Systemic steroid has the theoretical risk of disseminating the viral infection but such complication has not been recorded. It may be worthwhile treating older patients (arbitrarily those more than 55 years old), after exclusion of underlying systemic disease, with a short course of oral prednisolone in doses of 30 mg daily during the initial phase of herpes zoster and quickly tail down the dose over 7 to 10 days.

Post-herpetic neuralgia is treated with analgesics and mild tranquilizers. In recalcitrant neuralgia, oral pimozide has been reported to be effective in controlling the pain.

MOLLUSCUM CONTAGIOSUM

This is caused by a pox virus. Man is the only known natural host for molluscum contagiosum virus. The mode of transmission is uncertain, but appears to be by direct contact from an infected person. It can be also be sexually transmitted.

Clinical features

The incubation period is estimated to be from 14 to 50 days. The lesion is characteristic; presenting as pearly-white to skin-coloured umbilicated dome-shaped papules varying from 1 to 10 mm in diameter (fig. 9). Occasionally giant nodular lesions may be seen. Spontaneous involution of the lesions can occur after several months. Extensive lesions may occur in immune-suppressed individuals. They are common in children. Commonly affected sites include the anogenital region, flexures and trunk. It occasionally occurs on the face and scalp. They are often multiple and Koebner phenomenon may be evident. Lesions over the genital lesions are usually sexual transmitted (fig. 19).

Diagnosis

The presence of molluscum body on direct microscopical examination of curetted lesions crushed on a slide rapidly confirms the diagnosis. The histology of the lesion is also characteristic. However the diagnosis of molluscum contagiosum can be made clinically without these investigative procedures.

Treatment

The duration of the individual molluscum lesion is variable. The molluscum lesions in most patients clear within 6 to 9 months, but some lesions can persist up to five years. The treatment of choice depends on the age of the patient and the distribution and number of the molluscum lesions. For small lesions, currettage without anaesthesia is preferred. Application of trichoroacetic acid or 20% podophyllin in 95% alcohol into the punctum of the lesion may be effective but care must be taken to avoid contact with the surrounding skin as these preparations are highly irritant to normal skin. Large lesions can be curetted under local anaesthesia. The patient should then be followed up over the next 3 months to ensure that no untreated lesion remains.

VIRAL WART

Infectious wart is caused by a human papillomavirus (HPV). They characteristically produce benign skin tumours. Some genus of the wart viruses have oncogenic potential. There are numerous different strains of papillomaviruses and some strains appears to be more frequently associated with certain type of lesions, eg. HPV-1 is often associated with plantar warts and HPV-2 and HPV-3 with multiple common warts.

The role of immune response to infection by HPV is poorly understood. The cell mediated mechanism may be important in controlling HPV infection. This is evidenced by the raised incidence of viral warts in patients suffering from conditions associated with compromised immune status e.g. Hodgkins disease, renal transplant patients on immunosuppressives, and chronic lymphatic leukemia when compared with a control group.

Wart virus can be autoinoculated from hand to foot or vice versa. Acuminate warts are usually transmitted by sexual contact and epidemiological surveys provide no evidence of an association between common warts and genital warts. Trauma is an important factor favouring successful inoculation with the virus.

Clinical features

The HPV infects epidermal cells by direct inoculation with infected material. The incidence of warts increases during school entry years to reach a peak between 12 and 16 years, declines sharply to the age of 20 and more gradually thereafter. The incubation period varies between 1 to 20 months. The lesions are classified according to morphology and location of the warts.

Common warts

These are firm verrucous or filiform papules that range from 1 mm to 2 cm in diameter (fig. 11). Some lesions can coalesce to form large irregular warty, horny plaques. They are most commonly found on the back of the hands and fingers, knees and other bony prominences but it can occur on any skin surface. Koebner phenomenon may occur with viral warts. Subungual and periungual warts are characteristic and may disturb the growth of nail and cause marked nail plate deformities.

Plantar warts

These warts are seldom elevated above the surrounding skin. They are often surrounded by a thick dense callus but on close inspection the sharp edge of the wart is usually evident in the centre of the callus (fig. 12). These warts are often located on the pressure areas such as the heel, head of the metatarsals and ball of the toes. It may be single or multiple. They can cause severe pain and affect walking at times.

Plane warts

These are small (1 to 5 mm), usually skin coloured or brownish, flat papules occurring on the face and back of the hands. The lesions may coalesce forming small plaques. The surface of the lesions has a characteristic stippling which is best seen with a hand lens. The lesion tends to be multiple especially those occurring on the face and not infrequently several different parts of the skin are affected simultaneously. Koebnerization is commonly seen (fig. 13). Plane warts may disappear suddenly after a few weeks or months.

Acuminate warts (veneral warts, genital warts)

These warts occur around the mucocutaneous junctions (fig. 14) and the intertrigenous areas and are sexually transmitted. The lesions are often exuberant, non-horny but often filiform and soft and are red to pink in colour (fig. 14). Rarely giant

cauliflower-like lesion may develop, especially during pregnancy. Malignant change may occur from acuminate wart infection. There are reports to suggest that genital warts of the cervix in females are associated with a higher incidence of carcinoma of the cervix. Other sexually transmitted diseases should be excluded in patients with genital warts.

Treatment of viral warts

At least 65% of warts disappear spontaneously within 2 years but it is not possible to prognosticate warts in the individual patient. There are many methods used in the treatment of warts, many are relatively ineffective and some are painful and some involve the risk of scarring.

Topical application:

Podophyllin. This is a resinous extract of a plant with cytotoxic property. Podophyllin inhibits mitosis and causes swelling and necrosis of cells. Preparations of 25% or 50% in tincture Benzoin or paraffin and ointments are available. These preparations are effective against most genital warts but generally ineffective against common warts elsewhere. The normal skin surrounding the warts should be covered with some vaseline and podophyllin applied onto the warts and covered with talc or bismuth powder to prevent the irritant effect of podophyllin on surrounding normal skin. The patient should be instructed to wash away the podophyllin paint after 6 hours to prevent irritation. This treatment should not be repeated more than 2 times a week. Occasionally recalcitrant plantar warts may be treated with occlusive podophyllin application.

Other topical agents. e.g. formalin, salicylic acid, lactic acid and glutaraldehyde are generally ineffective against common warts. Caustic agents should be avoided to prevent scarring.

Cryosurgery or liquid nitrogen. This is probably the best available treatment for common warts but may cause pain if used for genital warts. If freezing has been adequate a blister develops in 12 to 48 hours and part or all of the wart is contained in the roof of the blister without scarring. Repeat applications at weekly intervals may be required before the warts can be cleared. For plantar warts the callus must be removed prior to liquid nitrogen application. Liquid nitrogen treatment may be extremely painful for plantar warts.

Electrocautery/Surgery. This method tends to cause scarring. Curettage and excision may be indicated for larger digitate or filiform lesions and in plantar warts but this should be followed by cautery of the base. This is probably the most suitable method for the treatment of common viral wart in general practice where it is not cost-effective to use liquid nitrogen.

HAND, FOOT AND MOUTH DISEASE

This condition is caused by the Coxsackie viruses, usually with Coxsackie A16, of the enteroviruses family. The disease usually appears in epidemic form and often several members of a family are affected simultaneously.

Clinical features

The condition begins with mild fever lasting 4 to 7 days followed by painful stomatitis and malaise. Oral vesicles which rapdily ulcerate are irregularly distributed over the palate, buccal mucosa, gums and tongue; early lesion may present as haemorrhagic spots. Skin lesions are characteristic. Small oval vesicles ranging from 2 mm to 5 mm are filled with purulent or pearly-grey fluid surrounded by a narrow red areola are seen. These skin lesions are usually found on the sides and back of fingers, toes, and margins of palms and soles (fig. 15).

Treatment

The condition is self-limiting and requires only symptomatic treatment. Skin cleanliness and oral hygiene together with antipyretic/analgesics are all that are required. The condition is infectious and infected children should be isolated and kept away from school or playing with other children. Adults can also be infected from infected children.

SUGGESTED READING

Robinson TWE & Heath R B. Virus Diseases and the Skin. Churchill Livingstone. Edinburgh, London 1983.

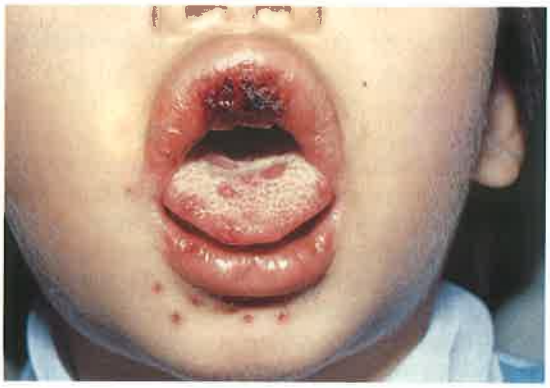


Fig. 1. Herpes simplex infection in a child. Note superficial ulcerations and crusted lesion on upper lip with oedema and aberrant herpetic lesions on tongue and chin.

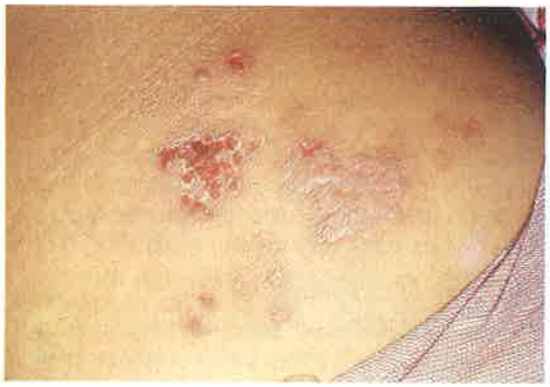


Fig. 2 Herpes simplex on thigh. Note grouped vesicular lesions on erythematous base. Zosteriform distribution may mimic Herpes zoster.

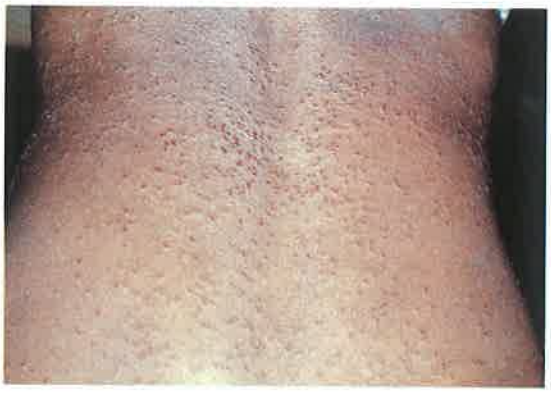


Fig. 3. Eczema herpticum. Severe herpes simplex infection in a patient with atopic eczema. Note widespread superficial erosive lesions on eczematous skin. Lesion started as vesicles which rapidly rupture to form erosions.

Fig. 4. Primary herpes simplex infection on the vulva. Note infected ulcerations and satellite lesion. Primary infection is often severe and painful. A sexually transmitted disease.

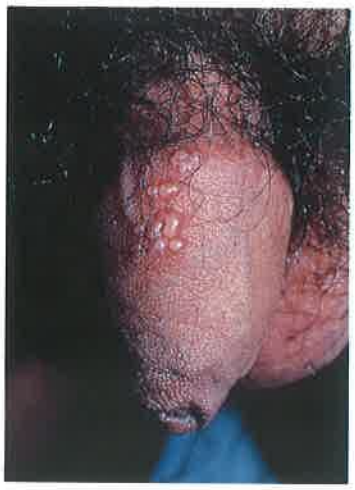


Fig. 5. Classical herpes simplex lesions on the prepuce. Note intact vesicles and erythematous base. Vesicles breaks easily to form superficial erosions which can become infected and chancroidal.

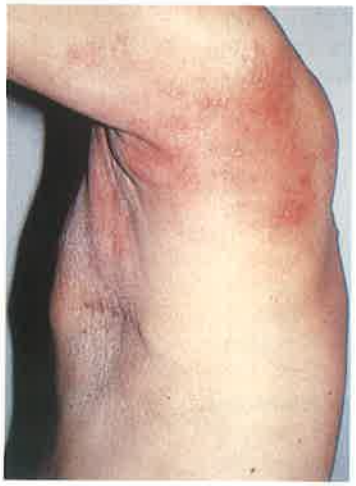


Fig. 6. Early herpes zoster of T1 dermatome. Note erythema which may be associated with pain, and the linear distribution of the vesicular lesions. A search for underlying malignancy is necessary.

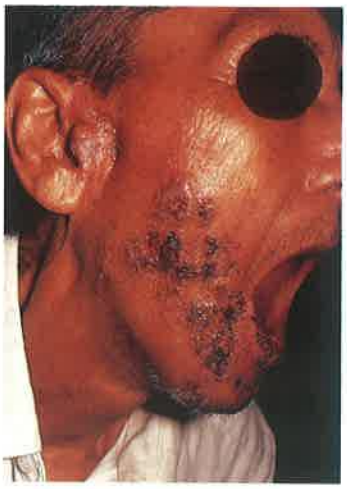


Fig. 7. Ramsay Hunt Syndrome. Herpes zoster of the VII cranial nerve. Note linear vesicular and crusted lesions on distribution of facial nerve. Involvement of ear and buccal mucosa associated with facial nerve palsy is common.

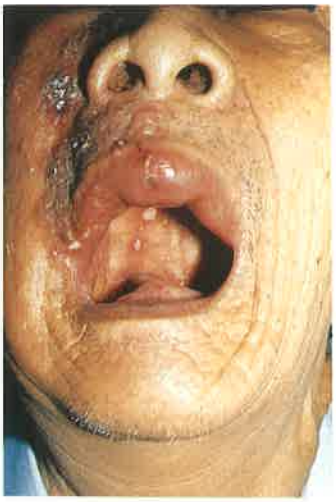


Fig. 8. Herpes zoster of the Maxillary branch of the V cranial nerve. Note involvement of buccal mucosa.

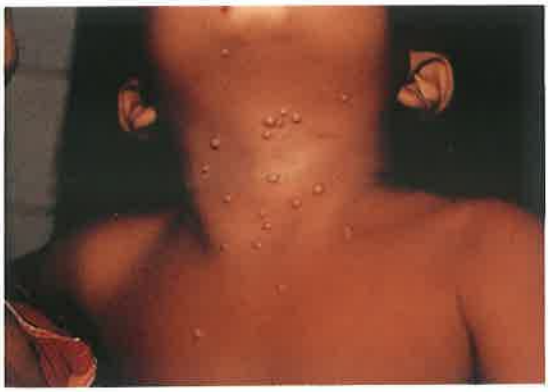


Fig. 9. Molluscum contagiosum in a child. Note characteristic umbilicated skin coloured papulo-nodular lesions. Lesions often appear on flexures in children.

Fig. 10. Molluscum contagiosum on the shaft of penis. Note characteristic umbilicated pearly papules. Lesions may simulate herpes simplex infection but these lesions are not vesicular and do not ulcerate. A sexually transmitted disease.

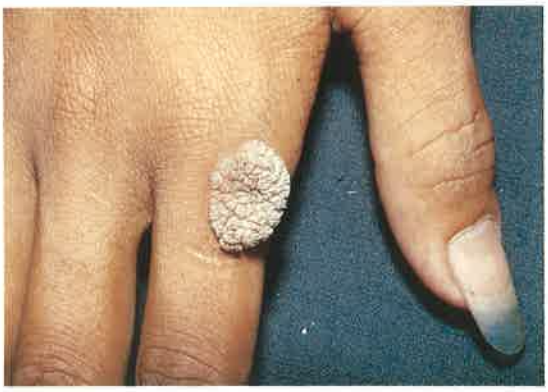


Fig. 11. Common viral warts. Note characteristic verrucous lesion.

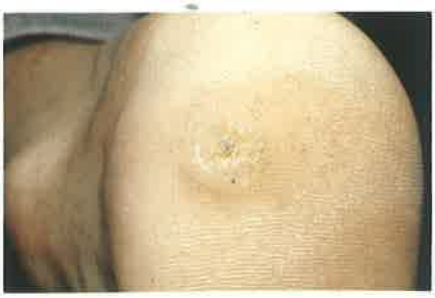


Fig. 12. Plantar wart. Note deep seated warty lesion with surrounding callosity. The wart in the center may not be evident until the overlying callosity is removed.

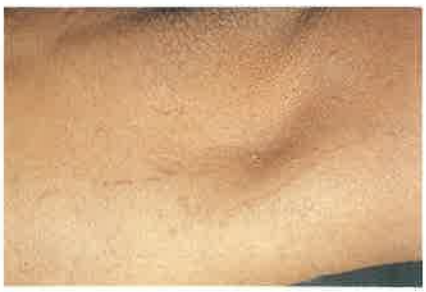


Fig. 13. Plane warts with Koebner phenomenon. Note linear lesions following skin scratches. Surface of individual lesion appears verrucous under magnification. Plane warts often affect the face.

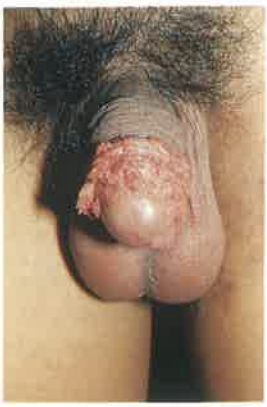


Fig. 14. Genital viral warts. Note filiform, warty appearance. A sexually transmitted disease. Filiform warts usually respond to podophyllin paint. Other sexually transmitted diseases should be excluded.

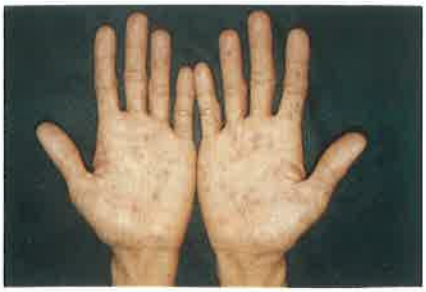
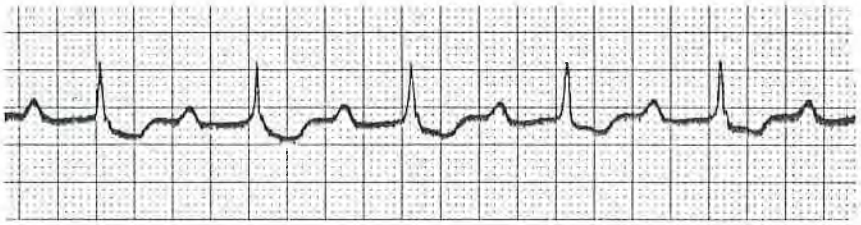


Fig. 15. Hand foot and mouth disease. Note oval haemorrhagic spots with surrounding dusky red areola on palms and finger pulps. Similar lesions are often seen on the soles and buccal mucosa. An infectious condition. The condition is caused by Coxsackie virus and occurs in epidemics.

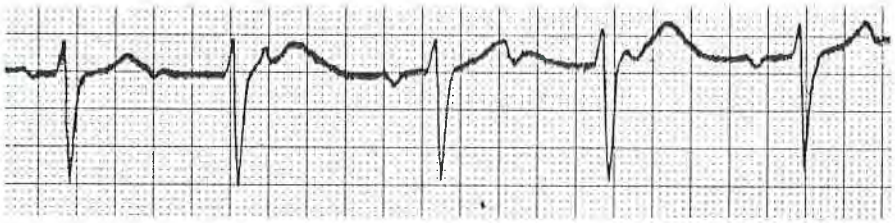
ECG QUIZ

Study the following ECG rhythm strips and make a diagnosis in each of them. Under what circumstances may these arrhythmias be seen and what is the treatment if any?

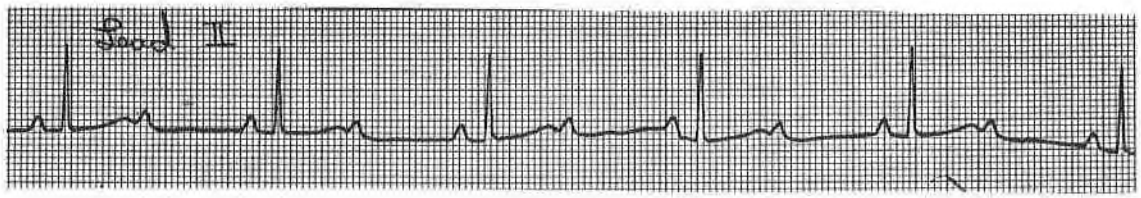
Strip 1



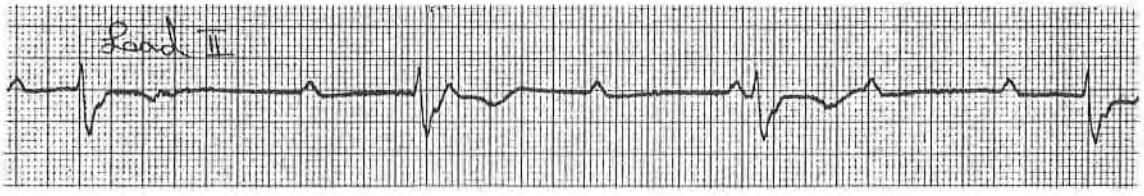
Strip 2



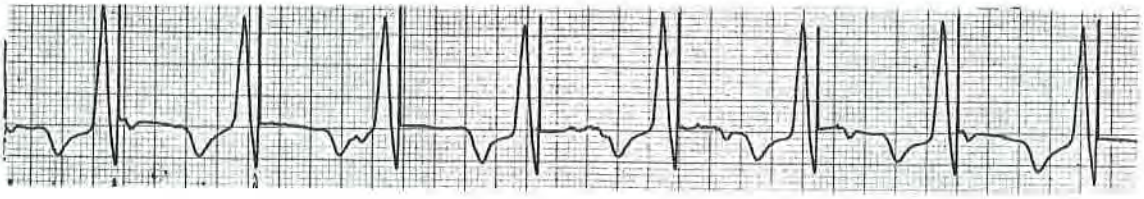
Strip 3



Strip 4(a)



Strip 4(b)



This ECG Quiz is contributed by Dr Baldev Singh MBBS (S), M Med. (Int. Med.) MRCP (UK)

ANSWERS

Strip 1:

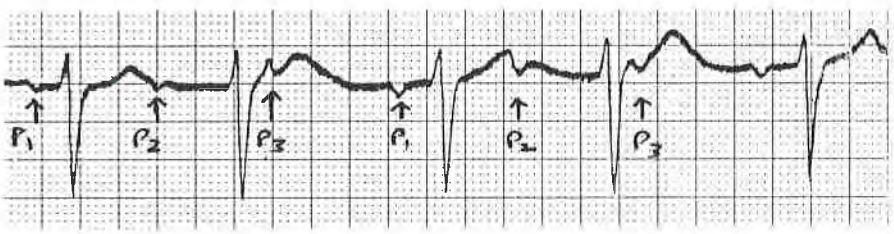
First Degree Heart Block

The PR interval is prolonged to 0.3 sec. and all the P waves are conducted. The normal PR interval should not exceed 0.2 sec. No specific treatment is necessary. It can occur under conditions of increased vagal tone, digoxin administration and rheumatic carditis.

Strip 2:

Second Degree AV Block. Mobitz Type I (Wenckebach block)

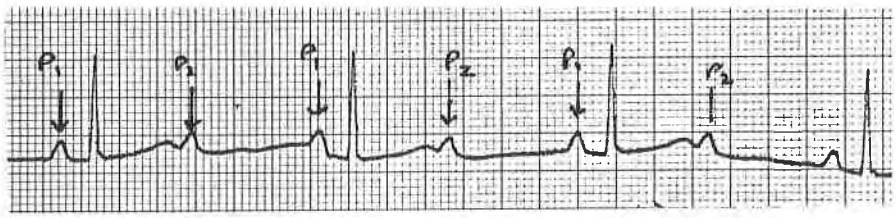
The strip below shows the P waves marked out. The PR interval of the 1st complex is longer than that of the 2nd complex and the 3rd P wave is not conducted. The cycle repeats itself. This is due to progressive decrease in conduction velocity. This conduction disorder is usually transient and may be seen in inferior myocardial infarction, myocarditis, digoxin toxicity and treatment is directed to the underlying cause. It may also be seen following cardiac surgery and even in healthy well-trained athletes due to increased vagal tone.



Strip 3:

Second Degree A-V Block, 2:1, Mobitz Type II

As shown in the strip below P_1 has a fixed relationship with the following QRS complex and P_2 is not conducted.

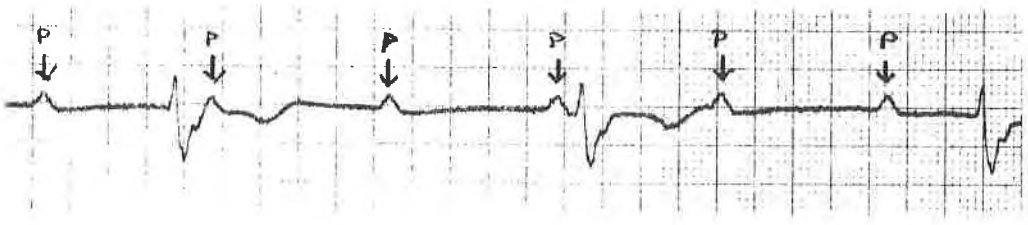


Unlike Mobitz Type I, Type II A-V block tends to be persistent and may progress to Third Degree or complete Heart Block. It usually results in the setting of an acute anterior myocardial infarction. Atropine administration may be tried but is unlikely to succeed and artificial transvenous cardiac pacing is usually necessary.

Strip 4a and 4b:

Third Degree AV Block of Complete Heart Block

The atrial rate is about 65/min and the ventricular rate about 28/min. The P waves bear no relationship to the QRS complexes.



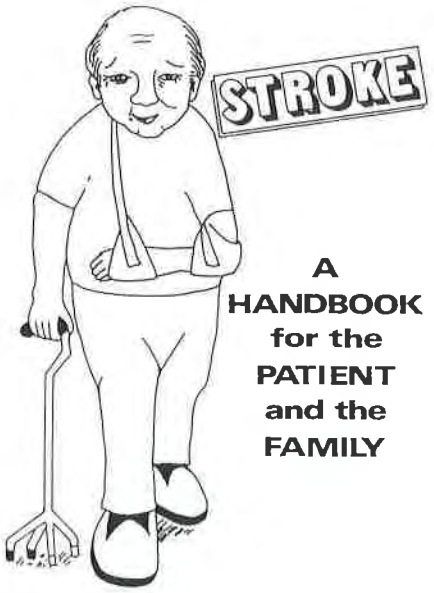
The QRS complexes are wide (greater than 0.1 sec) indicating an infra-nodal block. The pacemaker therefore lies below the bifurcation of the bundle of His. The intrinsic firing rate for such a low block is usually 30-40/min and episodes of ventricular asystole may occur. This usually indicates extensive conduction system disease and may complicate acute anterior infarction. In this situation a temporary transvenous pacemaker will always be required. Intravenous isoprenaline infusion may be of value in maintaining or accelerating ventricular rate while awaiting pacemaker insertion but this must be given cautiously as myocardial ischaemia may be aggravated and ventricular tachycardia or fibrillation may ensue.

When Third Degree AV block occurs at the A-V node level, a junctional or A-V nodal escape pacemaker initiates ventricular depolarisation. This is usually a stable pacemaker and the ventricular rate is between 40-60/min. This type of Third Degree AV Block occurs in the setting of an inferior infarction and results from an increase in vagal tone at the level of the A-V node. It is usually transient, but the patient must be monitored closely.

Strip 4b shows the pattern following ventricular pacing. Every pacing spike is followed by a QRS complex indicating full capture.

BOOK REVIEW

STROKE — A HANDBOOK FOR THE PATIENT AND THE FAMILY



This handbook is a joint project of the Singapore Association of Occupational Therapists and the Home Nursing Foundation. It covers information about common causes, contributing factors and warning signs of a Stroke, results of a Stroke, treatment, rehabilitation, activities of daily living, maintenance exercises, hints, prevention and where to go for help. It is aimed at helping stroke victims and their families better manage the condition and play an active role in the rehabilitation process, in order to help the patient achieve maximum indepedence, within the limits of his disabilities.

The relevant and comprehensive text is versed in simple language which makes for easy reading, and the numerous accompanying illustrations are clear and extremely helpful. The family physician or general practitioner will find this book easily recommendable to stroke patients and their families, and will definitely want his own personal copy.

Both the Singapore Association of Occupational Therapists and the Home Nursing Foundation are to be congratulated for addressing themselves to a common and important problem.

The Handbook is priced at S$5.00 per copy, and is available at:

1) All Government Polyclinics and Outpatient Dispensaries.
2) Home Nursing Foundation Headquarters 26 Dunearn Road, Singapore 1130.
3) HNF Senior Citizens' Health Care centres at:
   (a) Block 151 Lorong 2 Toa Payoh, Singapore 1231
   (b) Block 10 Eunos Crescent, Singapore 1441

MHV

NEWS FROM THE COUNCIL

1. College Convocation and Sreenivasan Oration

The Twelfth College Convocation and Annual Dinner will be held at the Shangri-la Hotel Singapore on Sunday, 9 November 1986. The Ninth Sreenivasan Oration will be delivered the same evening and at the same venue by Mr Gopal Baratham, Neurosurgeon and Head of the Neurosurgery Unit, Tan Tock Seng Hospital. The title of his oration will be "The Practice of Medicine is sustained by doubt".

2. Continuing Medical Education Programme

The Internal Medicine (I) Update Course conducted by the Continuing Medical Education Committee of the College was a tremendous success. Over 170 general practitioners/family physicians registered for the course, and attendances at the sessions averaged about 130 each week. The enthusiasm of the doctors, and the sacrifice made in attending the lectures, is really very encouraging — 123 registrants recorded 75% attendance, 53 of whom attended all the eight sessions.

The next module will be on Internal Medicine (II). It is scheduled to commence on Friday, 24 October 1986 and will cover eight theory sessions.

3. Post-Renovation Opening of the College of Medicine Building (COMB)

The renovation work at COMB is being carried out in full swing and is expected to be completed by March 1987. The official post-renovation opening of COMB is sheduled for 9 May 1987 and in this connection it is proposed to organise an Exhibition, hold Seminars, Conferences and Scientific Meetings. The Exhibition will be held from 9 to 22 May 1987 and will focus on the history of medical services, the medical school and the building itself.

We are looking for information, articles and exhibits related to the three Exhibition themes. Examples of exhibits we are looking for are:

a) graduation scrolls, caps and gowns;
b) medical, surgical, dental and pharmaceutical instruments;
c) medicine, bottles, old medical prescription forms and vaccination certificates;
d) trophies; old photographs of hospitals, medical school or an area of medical activity;
e) medical, dental and pharmaceutical journals; publications e.g. books, newsletters, etc.
f) oil paintings of pioneers of medicine; oral history of selected events in the history of the medical services or medical school, and
g) other artifacts of historical significance.

All contributions for the Exhibition may be forwarded direct to:
The Director, National Archives, Hill Street Building, 140 Hill Street, Singapore 0617

or to the College Secretariat.

4. A Meeting with the Minister of State, Ministry of Health

The President, Vice President, Censor-in-Chief and Honorary Secretary from our College met Mr Yeo Cheow Tong, Minister of State, Health, Dr Kwa Soon Bee — Director of Medical Services, Singapore and other representatives from the Ministry of Health, Singapore, on July 17, 1986, for a formal discussion on the role that the College could play in helping improve the standard of Primary Health Care in Singapore. The discussion included:

Vocational Training, Continuing Medical Education, Undergraduate Training on Family Medicine/General Practice, College Diplomate Membership Examination, Public Health Education, and Health Financing.

5. New Members

The following have been accepted by Council into the membership of the College during the months of July/September 1986:

Dr Liew Yin Choo	— Ordinary Membership
Dr Ong Siong Hoon, Dicky	— Ordinary Membership
Dr Tan Kiat Piah	— Ordinary Membership
Dr Tham Pak Onn	— Ordinary Membership
Dr Lim Hui Moey, Evelyn	— Associate Membership

WONCA Regional Conference
Asia Pacific Region
and
Hong Kong Medical Exposition '87

"Family Medicine — Crossing the Frontiers"

Hosted by: The Hong Kong College of General Practitioners
5th — 9th September 1987
Hong Kong

香港
HONG KONG

Registration enquiries are now being accepted for an **important Conference in General Practice/Family Medicine** — the WONCA Regional Conference — Asia Pacific Region which will be held in Hong Kong. **Hong Kong** has everything to make a visit to the Orient specially memorable. The delegates and accompanying persons can enjoy a combination of excellent tours around Hong Kong and China, spectacular culinary dishes and, of course, the endless delights of shopping.

A stimulating and fulfilling Scientific Programme has been planned and will include: four **Plenary Sessions and Workshops** for which outstanding speakers will be invited to address the delegates on The Stable Core of Family Medicine/General Practice; Family Medicine/General Practice in the Asia Pacific Region; Geographical & Economic Frontiers, Medical Frontiers and Socio-cultural Frontiers.

Update Sessions on various new developments in medicine;

Free Standing Papers on various aspects of general practice;

Special Interest Groups for discussion on such topics as Undergraduate Teaching, Traditional Medicine, Balint Group, Sports Medicine, Computers, Practice Management, Professional Standards, Geriatrics, Vocational Training, Occupational Health, Audit and Adolescent Medicine etc.

Poster Sessions for papers on any area of medicine.

CALL FOR PAPERS

You are invited to submit original papers on various areas of General Practice and Family Medicine, especially those related to the Conference and session themes, for presentation in:

- **UPDATE SESSIONS**
- **FREE STANDING PAPERS**
- **SPECIAL INTEREST GROUPS**
- **POSTER SESSIONS**

Abstracts of your papers should be submitted before 15th January 1987.

For registration details and further information of the Conference, and Guideline for Preparation and Submission of Abstracts, please contact your local College of General Practitioners/Family Medicine or write to:

HKCGP
恩致全豚

Contact: Administrative Secretary,
The Hong Kong College of General Practitioners,
8th Floor, 15 Hennessy Road, Hong Kong.

FAMILY MEDICINE
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