The Singapore Family Physician

The College of General Practitioners Singapore
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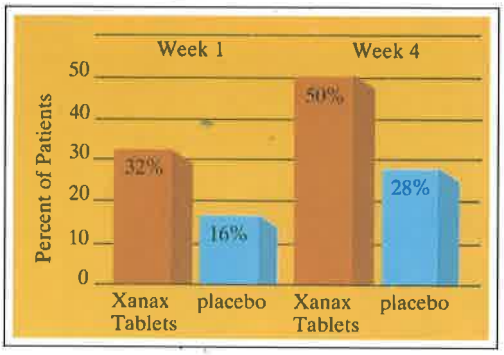

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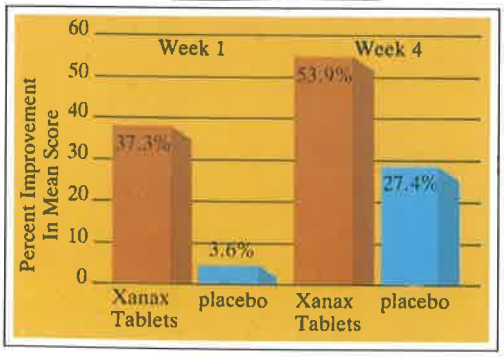

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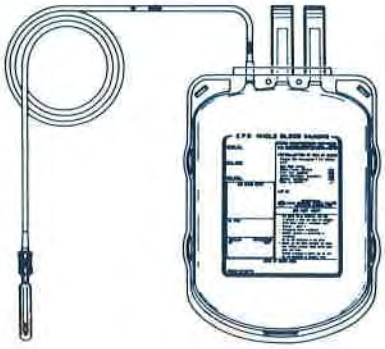

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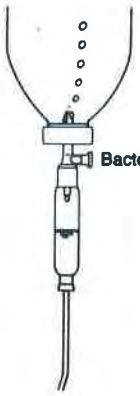

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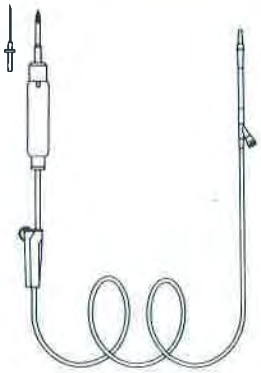

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FAMILY MEDICINE AS A SPECIALTY

The question of whether Family Medicine should be developed into a Specialty has been discussed worldwide. There is a consensus that it should be, if it is to achieve medical excellence and be on par with the existing hospital specialties like internal medicine and paediatrics.

Unlike the existing specialties which are specialties in depth, Family Medicine is a specialty in breadth covering not only curative but also promotive, preventive and rehabilitative services at the ambulatory care level. Its breadth covers not only the biomedical framework of disease but the comprehensive biopsycho-social framework of illness. It is now clear that only a fraction of complaints presented to the doctor are biomedical, the rest having an admixture of psychological, social and somatic issues. The breadth of Family Medicine also covers the different stages of the individual as he goes through his life cycle of childhood, adolescence, adulthood and old age. It also covers the various phases in the life cycle of the family. The important role that Family Medicine plays in the health care of the country is beyond doubt. To implement a good family medicine service in the country there is a need to have well trained family physicians.

Family Medicine as a postgraduate degree is well accepted in the U.S.A., Canada, U.K., countries in the European Community, Australia and New Zealand. As a specialty it is beginning to be accepted in our part of the world and corresponding with the acceptance, the institution of a post graduate higher qualification that is recognised for career development will be necessary. In Malaysia, the Government has accepted in principle the institution of the Master of Medicine (Family Medicine). This will be a four year programme and is of the same duration of training with the existing specialties.

In Singapore a proposal has been made to the School of Postgraduate Medical Studies by the Steering Committee for Family Medicine Training (consisting of representatives from the Ministry of Health, the College of General Practitioners Singapore (CPGS), and the Department of Community, Occupational and Family Medicine (COFM), National University of Singapore) for the institution of a degree of Master of Medicine in Family Medicine. It was submitted in February 1991.

Postgraduate training in Family Medicine to varying degrees of vigour has been an activity since the inception of the College of General Practitioners Singapore in 1971. The latter has been running continuing medical education programmes and has also, since November 1972 been conducting a postgraduate family medicine programme leading to the MCGP(S) Diplomate Examination.

In 1988, the Ministry of Health, together with the CGPS and Department of COFM, NUS, jointly initiated a training programme in Family Medicine. The pilot training programme was of a two year duration, with twenty-one months being spent in rotating hospital postings and the remaining three months in the outpatient setting covering the Government polyclinic, school health and general practice. The first batch of trainees have since completed the two year programme and four out of the six were successful in the MCGP(S) Diplomate Examination.

A review of the programme was conducted in 1990 during the visit of Professor J H Barber from the Department of General Practice, University of Glasgow as HMDP of the Ministry of Health. The programme has since been extended to include a year in the outpatient setting making it a three year programme. This has been proposed as the definitive programme for the Master of Medicine in Family Medicine for Singapore.

In the endeavour to upgrade Family Medicine, the existing general practitioners should not be overlooked and thought should be given to develop a broader system that will enable all general practitioners to upgrade themselves to a higher level of expertise to provide uniformly good quality of ambulatory care. The following is one option for the profession to debate upon.

To be in line with the existing Master of Medicine programmes in the School of Postgraduate Studies, the M. Med. (FM) should be an entry examination. This means that upon acquiring the qualification, the recipient has to spend another three years in the specialty and then submit himself for review to be recognised as a specialist. This recognition is the A.M. for the existing specialties in Singapore. The corresponding recognition for Family Medicine could be the A.M. or the FCGP(S) or both.

Using this as a framework, it would be good if existing holders of the MCGP(S) can also register to be on the accreditation programme and after the requisite number of years submit themselves for review to be given the A.M. or FCGP(S). The content of the review programme can be identical to M. Med. (FM) holders to include postgraduate teaching, research, evidence of acceptable standards of practice and medical record keeping.

What about existing general practitioners who have been in practice for a large numbers of years, say 10 years and more? The Australian Royal College of General Practitioners has the track of FRACGP by assessment. This requires presentation of evidence of acceptable practice or postgraduate endeavour like submission of written projects and the like. Perhaps, the College of General Practitioners Singapore and the profession should explore this further as a means of accreditating general practitioners who have been in practice beyond 10 years.

In the setting of Family Medicine as a specialty care the Family Physician's terms of reference should be enunciated. He should be a doctor who is committed to providing continuing and comprehensive care to his patients and their families. This would include health promotion, disease prevention, care of illness, rehabilitation and prevention as well as referring only those who really need specialised care. As such his services will include (1) initial care of indifferentiated medical problems presented to by the community; (2) curative care in relation to accepted norms; (3) rehabilitative care; (4) palliative care and care of the bereaved; (5) follow up and continuing care of chronic problems; (6) preventive care, such as immunisation and screening; health education and health promotion.

His tasks, apart from the provision of the services listed, will be to co-ordinate the activities of the local health team; to mobilise community resources for patient care; to train and supervise other members of the primary care facility and to lead in continuing medical education activities; to be responsible for the management of the services provided at the primary care facility; to make appropriate referrals to specialists and to follow-up on patients discharged from specialist care; to participate where appropriate in shared care schemes with specialist colleagues; to keep adequate records of patients seen and managed; to manage patients in a holistic fashion taking into account the social and cultural characteristics.

Family medicine as a specialty is a challenge which is not beyond the reach of each of us. We may not reach it today — we may reach it tomorrow, or even the day after, or on some distant date but we will reach it, if we have the professional will to do so.

GLG

ORIGINAL

A GENERAL PRACTITIONER'S UPDATE ON CATARACTS

T H TAY, MBBS, FRCS(G), FCOphth (UK)

SUMMARY

With the increase in life expectancy, the prevalence of cataracts in Singapore is also on the rise. Cataracts are the major cause of 'treatable' blindness in the Asian region. It is imperative that primary health care providers remain updated on this condition. This article reviews the latest management of cataracts and answers some often posed questions.

Key words: Cataract, red reflex, intracapsular extraction, extracapsular extraction, intraocular lens implant.

INTRODUCTION

Cataracts are a potentially curable cause of poor vision in the elderly. With the increasing size of this cohort the number of patients presenting to General Practitioners for complaints of poor vision due to cataracts is also correspondingly on the rise.

As primary health care providers it is wise for this reason to remain updated on cataracts and their present treatment modalities. This will aid not only in proper diagnosis but also in providing adequate information to the patient to allay their fears and doubts before a referral to an opthalmologist occurs. One can only ponder on the number of misinformed and frightened elderly patients who shy away from seeing an opthalmologist because of their ignorance and fear of the consequence of eye surgery.

SAF Ophthalmologist
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CAUSES

The world's major preventable blinding conditions are cataracts, trachoma, keratomalacia and onchocerciasis.[1,2] Of these cataracts account for more than 50% or the 'curable' blindness in developing countries.[2]

Cataracts are essentially opacities in the usually clear crystal-like human lens. This impedes the path of light into the eye and diminishes the stimulation of the retina leading to a decreased visual acuity.

The causes of cataracts are numerous (TABLE 1). However, the most common and on the whole the most amenable to successful surgical results are the age related cataracts. Other causes on a case to case basis may have excellent visual acuity post-operatively so it is worth while being familiar with the commonest few conditions and their post operative prognostic factors as a rough basis for doctor-patient discussion.

DIAGNOSIS

In mature cataracts, the diagnosis is very obvious when the normal dark pupil has turned white because of the underlying cataract. This can be easily and dramatically shown and explained by a simple mirror and comparison with the pupil of the other eye. Providing of course that the other eye is not similarly affected to the

same degree or to a degree to which visual acuity is so poor!

In immature cataracts the diagnosis becomes slightly more difficult but this is easily overcome by examining for the red reflex using a direct ophthalmoscope. Firstly, rack up the O Dioptre lens on the ophthalmoscope. Next, stand approximately 3 to 4 ft away from the patient and direct the light beam into the eye under examination. If the patient is instructed to look into the beam, the observer looking through the ophthalmoscope will be able to see a clear red reflex in the pupillary area (Fig 1). This is actually light being reflected off the retina and choroid having passed into and out of the eye unhindered by the clear lens. Any opacity (cataract) in the lens will cause the red reflex to be obscured either partially or totally (Fig 2).

The cause for poor vision of course may not solely be due to cataracts as on occasion multiple pathologies can occur in the same eye. These of course have to be looked for in the history and examination eg. trauma, diabetes.

A useful method is to compare the visual acuity of the eye concerned with the amount of red reflex loss found on ophthalmoscopy. This is far from accurate in all cases and requires a fair amount of experience on the part of the practitioner but having said this, obvious discrepancies is a clue of additional pathology. A diabetic patient for example, with 20% loss of red reflex would not be expected to have a visual acuity of counting fingers closely and the decrease in vision may well be mainly due to diabetic retinopathy (Beware of refractive error when considering all factors).

TREATMENT

The treatment for cataracts remains surgical.[3] Although eye drops are available which supposedly reduce the rate of cataract formation these are not proven and once the cataract is formed have little if any effect in improving vision.

Cataract surgery in its modern history first began with the itrascapsular extraction method (Fig 3). Here the cataractous lens is removed in toto rendering the eye aphakic. This method was however subsequently found to have a 1% post-operative retinal detachment rate and further required the patient to wear thick aphakic 'fish bowl' glasses of 10 Dioptres or more. These glasses besides being heavy have their own optical problems because of the high power.

With the introduction of intraocular lenses combined with extracapsular cataract extraction both these problems were overcome (Fig 4). The incidence of retinal detachment was decreased by leaving the thin posterior capsule of the lens intact while removing the anterior capsule and cataractous cortical lens material. This intact posterior capsule acts as a tamponade preventing undue forward movement of the vitreous which would otherwise cause traction on the retina. This traction is considered one of the main causes of aphakic retinal detachments.

The intraocular lens (Fig 5) provides the main bulk of refractive correction lost when the human lens is removed hence thick aphakic glasses can be done away with.

TABLE 2 shows the number of intraocular lens implants done in the Singapore General Hospital for the year 1990. These included implants performed as secondary procedures on already aphakic eyes and those combined with trabeculectomies. These figures show the overwhelming "popularity" of intraocular lens implants. This is compared with those who were rendered aphakic without intraocular lens implants.

QUESTIONS

The patient in discussing cataract surgery may bring up the following questions:

1) WHEN WILL MY CATARACT BE MATURE ENOUGH FOR OPERATION?

Many patients were told in the earlier stages that their cataracts were immature and therefore not ready for operation. Although mature cataracts are marginally easier to extract and the immediate improvement in visual acuity is much more dramatic, there is actually no contraindication to performing early cataract operations. The main indicator should always be the patient's visual

requirement. An elderly lady who only stays at home and does minimal housework could wait longer till her cataract matures while a still active accountant for example may need an operation when his visual acuity has dropped to 6/18 or worse.

2) HOW LONG WILL THE SURGERY TAKE?

Most uncomplicated cataract operations with intraocular implants can be done within 20 to 45 minutes. Most patients will require a 2 - 3 day's hospital stay. However there is a move to make cataract surgery an outpatient day procedure but this requires careful patient selection by the opthalmologist and is only suitable for the co-operative patient who is otherwise fit enough to look after himself and apply the adequate post-operative medication or one who has supportive and knowledgeable family members to provide this simple care.[4] This decision is best left to the ophthalmologist.

3) WILL I NEED TO WEAR GLASSES AFTER OPERATION?

In most cases post operative patients will need to wear glasses to obtain 6/6 to 6/9 vision (assuming of course that no other ocular pathology exists).

There are several reasons for glasses:

(1) The intraocular lens is unable to mould like the normal lens. Therefore accommodation is not possible. Usually an IOL power is chosen that will render the patient slightly myopic ie. he would be able to read without glasses. However, distant vision will then be slightly blurred without using glasses. Thus the patient may require glasses when he goes out to catch a bus for instance.

(2) Suturing of the cornela wound induces post-operative astigmatism. Although this settles with time (usually 6 - 8 weeks, occasionally longer) there usually remains a residual impairment which requires correction with glasses.

The most important thing to stress however is that these glasses are not the 'fish bowl' aphakic gasses and hence do not suffer from their associated problems. In fact the glasses will not differ from those worn by the majority of the ammetropic population.

It must once again be emphasised that the need for glasses is tied to the patient's visual requirements. An elderly lady whose main task is looking after her grand-children at home may be extremely happy after a cataract operation which improves her vision from counting fingers to 6/18 and may not bothered with 'troublesome' glasses even though with them she may obtain 6/9 vision.

4) DO INTRAOCULAR LENS HAVE TO BE CLEANED?

This question is often asked because patients mistake IOLs as being similar to contact lenses. As the name implies intraocular lens are implanted within the eye and will therefore remain in situ without any need to be removed.

5) HOW LONG WILL MY IOL LAST?

This is a question that has not been resolved. Nevertheless with improved IOL design complications are being seen less frequently and in the elderly patient it is safe to say (barring complications) that the implant will last for the remainder of the patient's life. Indeed with the increased biocompatibility of the IOLs, younger and younger patients (eg. cases of trauma) are having these inserted with the expectation of life-long durability.

6) HOW SOON AFTER SURGERY WILL I BE ABLE TO SEE?

As soon as the post-operative eye dressing is removed the patient should experience an improvement over his pre-operative vision. This is especially so when pre-operatively the vision was counting fingers or worse.

However due to the post-operative oedema and wound suturing induced astigmatism, some blurring may still commonly be expected. This may take several weeks to

finally settle before fine tuning is done with the prescription of glasses.

7) I HAVE HAD A CATARACT OPERATION DONE SEVERAL YEARS AGO WITHOUT AN INTRAOCULAR LENS IMPLANT, NOW THAT MY OTHER EYE REQUIRES A CATARACT OPERATION SHOULD I HAVE AN IOL IMPLANTED?

This question commonly arises in patients who have had 1 eye operated on 8 or more years ago (before the IOL became common procedure). It is a question that requires careful discussion with the patient.

If the patient has been wearing aphakic glasses since the operation and has been coping well with them then obviously the problems inherent with these glasses have been overcome and the patient has been using the operated eye as his master eye, as the vision (if satisfactory) in the non operated eye would be blurred by the aphakic lens in front of that eye (NB. Remember that aphakic glasses are usually prescribed bilaterally even if the need is unilateral because of problems in balance and image size disparity if they were not). In such cases the second eye can be operated on without an intraocular lens implant and the patient would be happy with 'balanced' vision using aphakic glasses.

If however the patient says that he gave up using aphakic glasses after a while and has been depending on the non-operated eye to see (before that eye's cataract has matured) then this patient will probably be better off with an Intraocular lens implant. If the patient is happy with the IOL a secondary operation may be considered to implant an IOL in the initially operated aphakic eye.

8) WHAT ABOUT BIFOCAL AND MULTIFOCAL LENSES?

These are relatively new developments in the local scene. Bifocal IOLs are a combination of 2 lens powers arranged such that the patient should have clear near vision as well as clear distant vision. Images in-betweeen would be somewhat blurred. Multifocal lens work on principles of diffraction as opposed to the usual refractive type of IOLs.[5] As the name implies they theoretically allow for multiple foci of clear vision. However as these lenses are still relatively new their use should be discussed on a case to case basis with the ophthalmologist.

CONCLUSION

With the number of the elderly increasing in Singapore, the number of patients requiring cataract extraction will increase. Being well versed with the answers that patients often ask will be important in allaying unnecessary fear and anxiety.

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TABLE 1 CAUSES OF CATARACT

1. Senile/Age related
2. Traumatic
3. Metabolic –
   eg. i) Diabetes
   ii) Galactosaemia
   iii) Wilson's disease
   iv) Hypocalcaemia
4. Toxic – eg. Corticosteriods
5. Secondary –
   i) Anterior uveitis
   ii) Hereditary retinal and vitreoretinal disorders
6. Associated Syndromes –
   eg. i) Down's
   ii) Alport's
   iii) Lowe's
7. Hereditary

Table 2 NUMBER OF OPERATIONS PERFORMED SINGAPORE GENERAL HOSPITAL – 1990

Extracapsular cataract extraction with IOL implant	IOL implant as a secondary procedure	IOL implant/ cataract extraction and trabeculectomy	No IOL (Aphakic)	Total
3951	70	40	211	4271
4061			211	4271

ILLUSTRATIONS

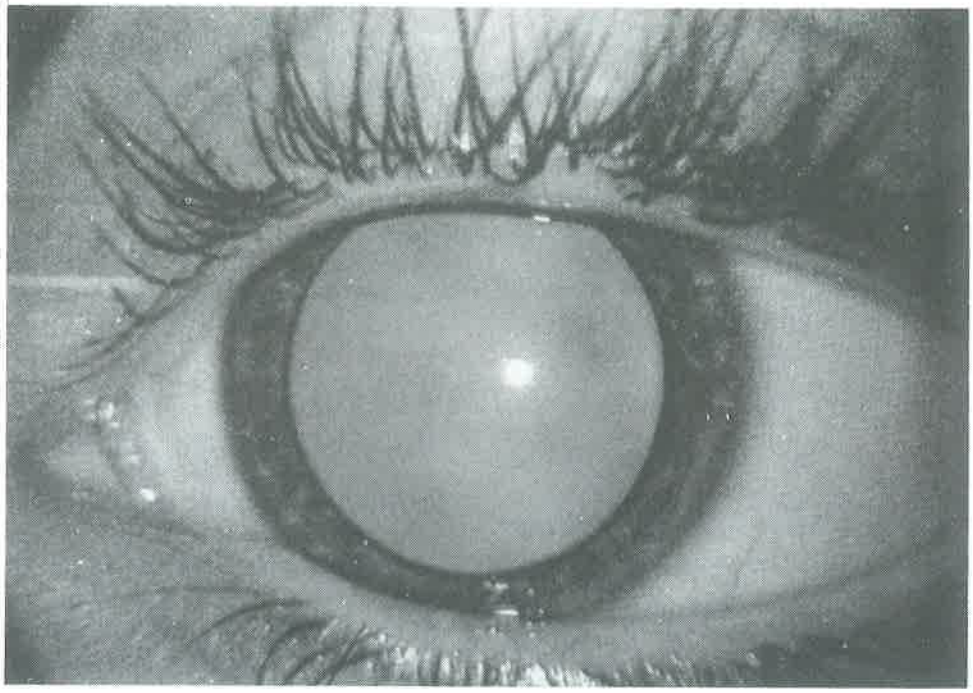

Figure 1. Normal Red Reflex

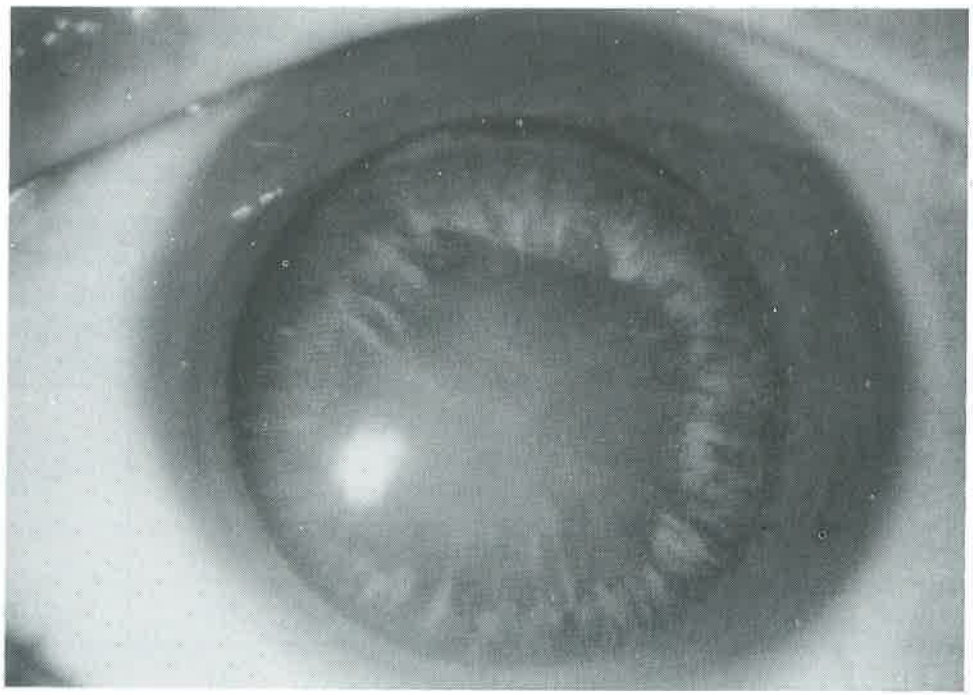

Figure 2. Cataract Obscuring Red Reflex

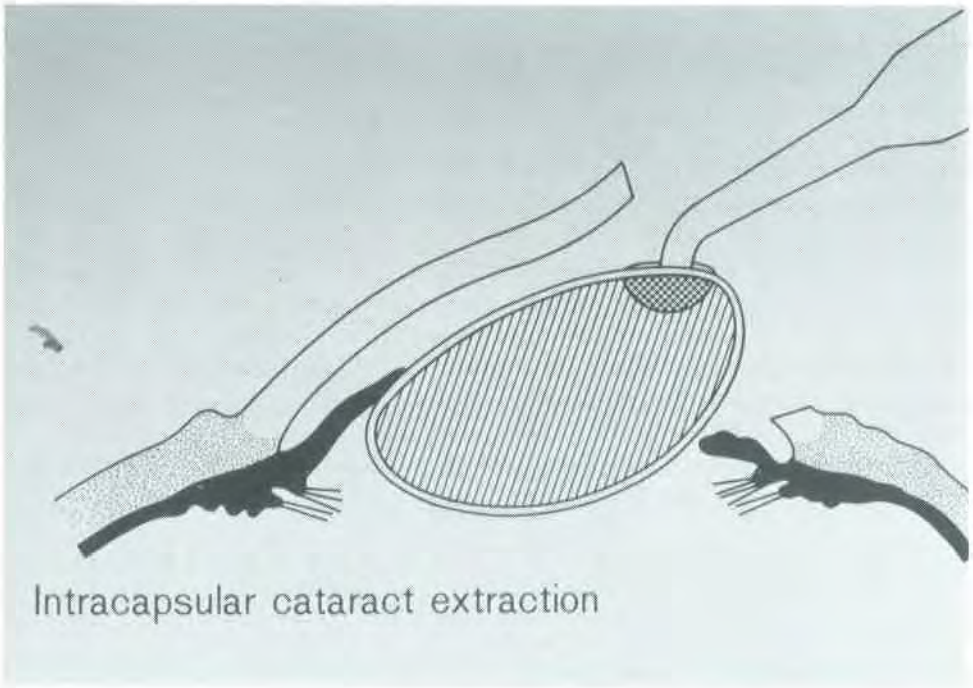

Figure 3. Intracapsular Cataract Extraction

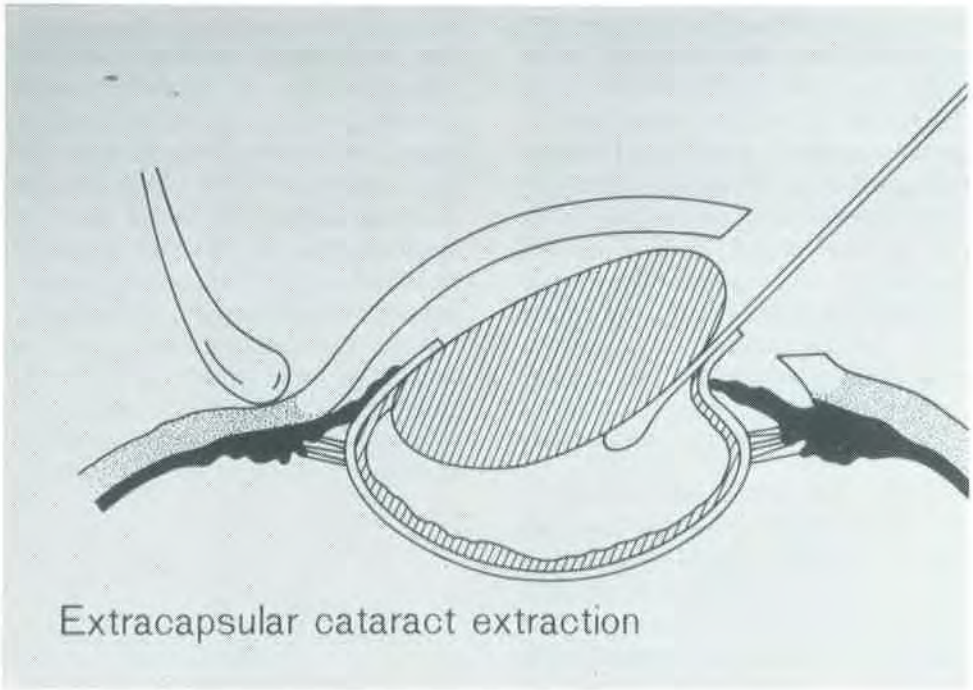

Figure 4. Extracapsular Cataract Extraction

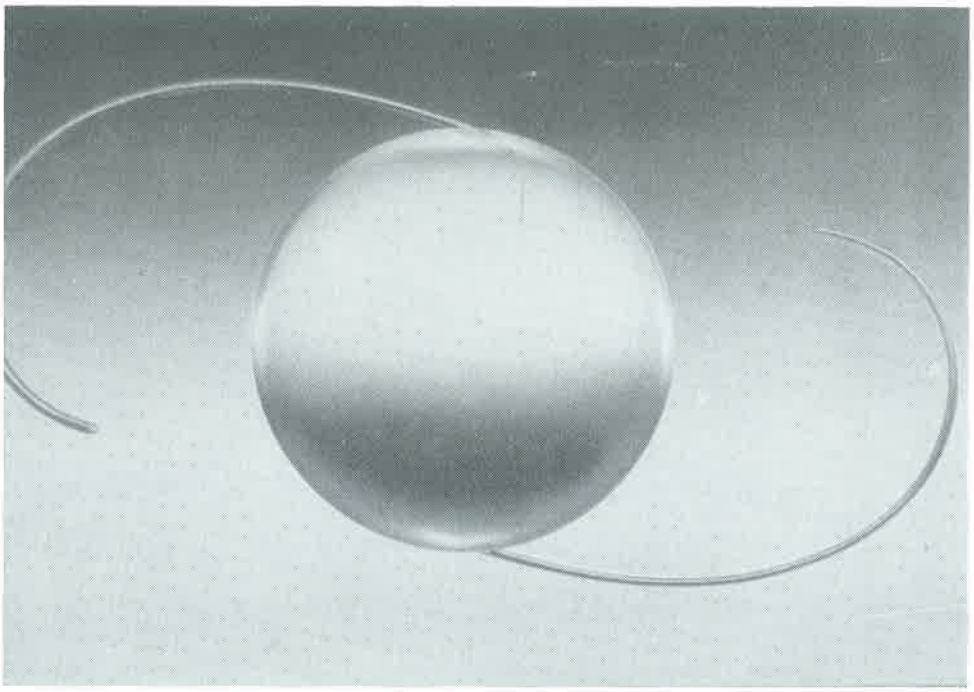

Figure 5. Intraocular Lens Implant

ORIGINAL

GALACTORRHOEA – A CLINICAL APPROACH

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INTRODUCTION

Galactorrhoea is not a particularly well-known clinical problem. Its exact frequency is difficult to estimate. It may be more common than reported as many patients do not seek treatment for this problem. Nevertheless it is not entirely uncommon to encounter a patient who complains of galactorrhoea. The cause of galactorrhoea are many, ranging from pituitary tumours to no known cause. It can result in a lot of anxiety in the person affected. A rational clinical approach is necessary to avoid missing important pathologies while at the same time avoiding unnecessary investigation.

DEFINITION

There is no universally accepted definition for galactorrhoea. Galactorrhoea may be defined as 'a persistent discharge of milky or milklike secretion from the breast in the absence of parturition or beyond six months postpartum in a non-nursing mother'.[1] Other forms of non-milky discharge from the breast (serous, purulent, sanguineous or bloody) do not constitute galactorrhoea. Galactorrhoea is not associated with any increase risk of malignancy of the breast although this may be a common fear amongst women suffering from this symptom. Galactorrhoea may occurring one or both breast and varies in severity from occasional discharge only on expressing to copious spontaneous discharge.

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CAUSES OF GALACTORRHOEA

The list of possible causes of galactorrhoea is long. Figure 1 lists some of the common causes. It should be noted that a large number have no identifiable cause (Idiophathic). The major groups of causes are those related to abnormal prolactin levels, other endocrine causes, drug-related causes and others.

Galactorrhoea with pituitary tumour

This constitutes about 20% of cases of galactorrhoea.[2] In the vast majority of cases, it is a prolactin-secreting tumour. Occasionally, galactorrhoea is seen in acromegaly or even more rarely, in Cushing's disease.

If there is a prolactinoma, prolactin levels will be grossly elevated. Levels above 5000 mU/I are almost always associated with prolactinoma whilst levels below 1500 mU/I rarely are.[3] Mildly elevated prolactin levels should not be loosely attributed to prolactin secreting adenoma. More often these pituitary tumours are non-functioning and the mild elevation of prolactin level is due to disruption of the hypothalamic-pituitary axis. Suprasellar tumours (eg. craniopharyngioma, meningioma, etc) as well as infiltrative lesions in this area (eg. craniopharyngioma, meningioma, etc) as well as infiltrative lesions in this area (eg. sarcoidosis, histiocytosis X, etc) may also cause mild elevation of prolactin levels.

The mainstay of treatment of prolactinoma is the ergot derivative, bromocriptine. Introduced in the early 1970s, this drug has been very effective in lowering the prolactin levels and restoring menstrual cycles as well as fertility in affected women.[4] It has also been shown to reduce the size of pituitary tumours.[5,6] Sometimes this effect may even be rapid (in a matter of days)

and dramatic. Surgical removal or radiotherapy may be reserved for cases that are not responsive to bromocriptine or patients who are intolerant of this drug. Surgery may also be indicated in the cases where the vision is severely threatened. There are also a number of newer agents that have been shown to be effective in treating prolactinoma and these may become more widely used in the future.[7,8,9]

Galactorrhoea and drugs

This is not an uncommon causes of galactorrhoea. The number of drugs implicated are many but most commonly they are the phenothiazine group of drugs used for treating major psychiatric illnesses. There is often an accompanying mild elevation of prolactin level. This resolves usually on stopping these drugs. However, the wisdom for stopping these drugs with a possible relapse in symptoms must be weighed against the need to treat galactorrhoea which may not be distressing to the patient. Often patients can be reassured that galactorrhoea is benign and no specific treatment is needed.

Of interest is the implication of benzodiazepines as a cause of galactorrhoea. This is occasionally encountered but the mechanism is unknown. Other drug include tricyclic antidepressants, metoclopramide, methyldopa, reserpine, isoniazid, cimetidine and others. A thorough drug history is therefore necessary in approaching a problem of galactorrhaea.

Galactorrhoea and oral contraceptives

This is another major category amongst the causes of galactorrhoea. The problem often appears after stopping the oral contraceptive pill. It may or may not be associated with post-pill amenorrhoea. The mechanism may be due to a change in the hormonal balance due to withdrawal of estrogens. The prolactin level is often normal. The problem usually resolves spontaneously.

Galactorrhoea and hypothyrodism

Although this only occasionally encountered, it is worth highlighting as the diagnosis of hypothyroidism may be missed.[10] The patients are often euthyroid clinically and hypothyroidism is detected only on blood tests. Prolactin levels are normal in half of the cases.[2] Treatment with thyroxine resolves the problem. Galactorrhoea may even be seen in subclinical hypothyroidism.[11] It is therefore worthwhile to screen patients with galactorrhoea for hypothyroidism.

Miscellaneous causes of galactorrhoea

There is a number of rare causes of galactorrhoea and these include major surgery, eg cholecystectomy, oopherectomy, etc as well as surgery of the chest wall (thoracotomy). Lesions on the chest wall like herpes zoster have also been associated with galactorrhoea.

Idiopathic galactorrhoea

This is the largest single category of causes of galactorrhoea. More than a third of cases of galactorrhoea would have no identifiable cause. Most of these patients have normal prolactin levels. Some may have amenorrhoea or irregular menstrual cycles but most have normal periods. It has been found that some with normal menses may have unexplained subfertility.

It is believed that galactorrhoea in these patients are due to minor abnormalities in the prolactin levels that are not obvious on normal testing but yet can cause galactorrhoea. Most of these cases occur in women who have previously delivered although a third of cases may be in women who never had children. Attempts to perform 24 hour sampling of prolactin levels have shown variable results with some authors discovering that mean prolactin levels to be higher than normal.[2,12,13]

Treatment in this capacity is usually dependent on whether fertility is desired and whether the patient is distressed by the problem. In many cases, the patients need only reassurance. When treatment is needed, bromocriptine is again the drug of choice. Here, a small dose (starting at 1.25mg or 2.5mg) given every night is usually effective. Although it has been found that some patients with unexplained subfertility and galactorrhoea become pregnant when treated with bromocriptine,[14] other patients with galactorrhoea may also become pregnant without any treatment.

Galactorrhoea in male patients

This is not commonly encountered. It accounts for about 5% of cases of galactorrhoea. Often

there is some form of hormonal disorder or imbalance. It may be associated with acromegaly, gynaecomastia, Klinefelter's syndrome, sex hormone therapy or androgen deficiency.

APPROACH TO GALACTORRHOEA

The approach to a patient with galactorrhoea is firstly to determine clinically if there is an identifiable cause. In particular a detailed drug history is necessary. The next important step is to determine the prolactin level. If prolactin level is elevated, a repeat is recommended as 'stress' may cause mild elevation of this hormone. (see Figure 2)

An elevated prolactin level would usually require further investigation to rule out the existence of a pituitary tumour or a tumour affecting the hypothalamic-pituitary axis. This is usually done by a CT Scan of the head and pituitary fossa.

Thereafter, depending on the cause that has been discovered, the patient is treated accordingly. It is worth reiterating that most patients only need reassurance and galactorrhoea may need no treatment unless it is severe. Often there is a need to allay the fear of an underlying breast malignancy. If any breast lumps are felt, a mammography to exclude malignancy may be ordered.

Bromociptine remains the mainstay in drug therapy. Small doses are sufficient in most cases. In idiopathic cases, a trail of short course (few months) usually suffices. It should be mentioned, however, that a proportion of patients may have relapse of their symptoms on withdrawal of bromocriptine. Some authors advocate long term follow-up of patients who have galactorrhoea with yearly estimation of prolactin levels to detect any emergence of a prolactin secreting microadenoma.[2]

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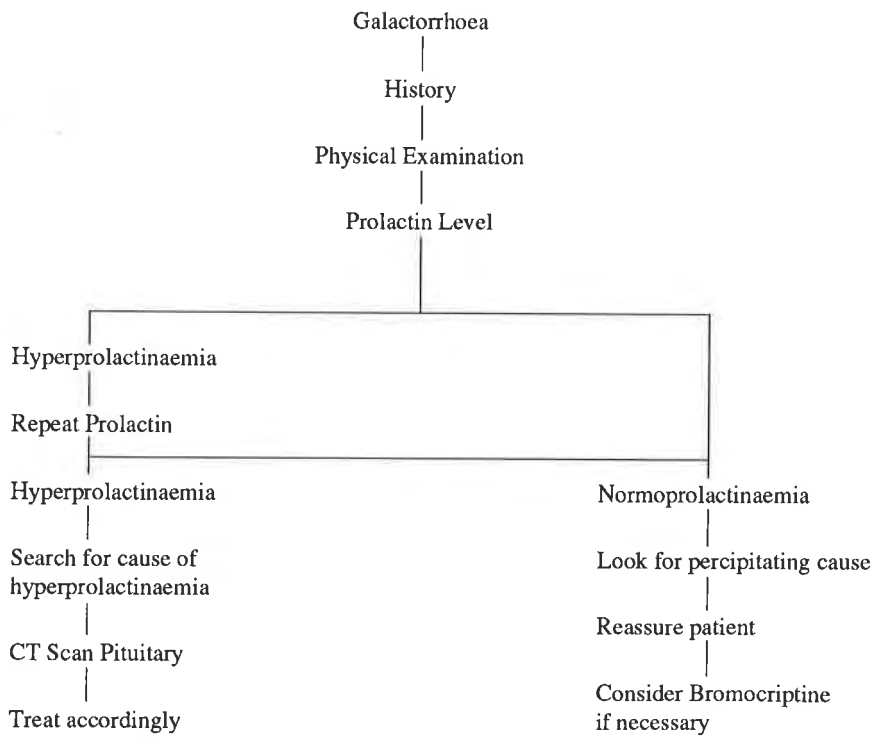
Figure 1: Causes of galactorrhoea

Pituitary Tumor	Prolactinoma
	Acromegaly
	Others
Drugs	Phenothiazines
	Tricyclic Antidepressants
	Metoclopramide
	Benzodiazepines
	Others
Oral Contraceptives	
Hypothyroidism	
Miscellaneous	Major Surgery
	Chest wall conditions
Idiopathic	With Amenorrhoea
	With Menses

Figure 2: Approach to a patient with galactorrhoea



3RD ANNUAL SCIENTIFIC CONFERENCE:
College of General Practitioners Singapore, 20 October 1990
Seminar on Recent Advances in Diagnostic Techniques

MRI AND OTHER IMAGING TECHNIQUES

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INTRODUCTION

What is MRI? MRI stands for Magnetic Resonance Imaging. MRI is a totally different imaging modality which does not involve X-rays in any way.

Magnetic Resonance Imaging involves placing a patient in a magnetic field so as to align the patient's protons in a particular direction. Subsequently, the protons are manipulated by applying electromagnetic pulses in the radiofrequency range and by collecting the signals that return, we are able to obtain an image. The characteristics of the tissue that is being studied will depend on the intrinsic properties of the tissue, i.e. its T_1 and T_2 relaxation times as well as the density of the protons in the tissue.

CONTRAINDICATIONS TO MRI

There are certain constraints that preclude an MR study in view of the fact that the patient is in the presence of a magnetic field.

Some contraindications to placing the patients in such a magnetic field are:

(1) Patients with cardiac pacemakers and neurostimulators. These instruments will be deprogrammed in the room and will not function normally.

(2) Patients with the history of metallic fragments in the eyes should not be scanned as the metal in the eyes may move and cause catastrophic effects.

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(3) Patients with implants, eg cochlear or stapes implants may not be scanned.

(4) Specific aneurysm clips in the groin are also ferromagnetic and may move, causing haemorrhage. Unfortunately, patients are not going to be able to tell us what type of aneurysm clip he has in his head so generally all patients with aneurysm clips should not be in the same vicinity as the magnet.

SPECIAL CIRCUMSTANCES

(1) If your patient has any type of metal in his body, please check with the radiologist if the examination can proceed. Small pieces of shrapnel for example in non-sensitive areas like finger-tip can be reinforced with tape and the examination can still proceed.

(2) **Pregnancy** is not a true contraindication. There are no known deleterious effects but MR should not be ordered too flippantly. Each patient should be screened carefully. Preferably, the examination, if indicated, should be postponed beyond the first trimester.

(3) Patients too ill or dyspneic to lie still may be difficult to image as we require the patient to lie flat on his back for at least half an hour. MR therefore should not be done for ill or unstable patients unless the patient is able to breathe on his own and is able to co-operate.

(4) We also have a problem with claustrophobia. If your patient is claustrophobic, please consider another modality or sedation if MR is necessary.

MAIN INDICATIONS FOR MRI

What are the indications for an MR examination at this present time?

(1) MRI has proven to be extremely useful and sometimes diagnostic for examining the central nervous system.

(2) The other region for which MRI is often used is the musculoskeletal system specifically for the spine, the major joints and for staging of musculoskeletal tumours.

CENTRAL NERVOUS SYSTEM

For the study of the central nervous system, MRI has a great advantage compared to Computed Tomography (CT).

(1) In the posterior fossa, the CT image is often degraded by streak artifacts from the adjacent petrous temporal bone. With MR , we have good visualisation of the brain stem and surrounding structures.

(2) We can also obtain images in multiple planes, coronal, sagittal, axial planes without moving the patient around. This is especially useful for assessment of tumours and their extent.

(3) There is a high level of contrast seen between gray and white matter so that problems of demyelination are easily diagnosed.

(4) The craniovertebral junction is also easily evaluated while previously, delineation of this region required complex motion tomography or CT myelography. With MR, the brain stem, spinal cord and ventricular system are seen in continuity in a single sagittal plane.

MUSCULOSKELETAL SYSTEM

Spine

(1) MR is used extensively for studying disc herniations especially in the lumbosacral spine and cervical spine. It has almost replaced myelography as the examination of choice for patients with suspected lumbar disc disease. Myelography is now usually reserved for cases of doubt or controversy.

(2) In patients with suspected spinal cord compression, MR gives us the ability to identify the site, extent and cause of obstruction without any invasive procedure.

(3) There is great potential for studying the spinal cord in cases of trauma but again, the necessity for patient selection limits the availability of the examination. MRI may show haematomas or contusions of the spinal cord without having to move the patient too much.

The Major Joints

(1) Shoulder Joint

Previously, patients with suspected rotator cuff tendonitis or tears had to undergo arthrography which required us to inject contrast into the shoulder joint, a procedure that is not too pleasant. Now with MR, we have direct visualisation of the rotator cuff tendons.

Similarly for assessment of tears of the glenoid labrum, arthrography is now a thing of the past. With MR, this can be achieved without causing the patient discomfort.

One disadvantage is in assessing tears of the anterior capsule of the shoulder. Unfortunately, MR cannot visualise this and CT arthrography is still the modality of choice.

(2) The Knee Joint

This is a major area for MR imaging. Tears in the menisci and cruciate ligaments may be easily visualised without any interventional procedure. Comparison of MR with arthroscopy shows a negative predictive value of 94%. An additional advantage of MR compared to arthroscopy is that para-articular masses may be examined as well for example, cysts and ganglia.

(3) Temporo-mandibular Joint

The TMJ may also be elegantly visualised with MR. Its motion can be evaluated and the disc may be visualised directly.

However, the resolution of MR is still not good enough to show tears or perforations of the disc and this can only be properly assessed via arthrography.

BONE AND SOFT TISSUE TUMOURS

Bone and soft tissue tumours may be properly staged with MR. Especially useful is the multiplanar capabilities which can define the true longitudinal extent of the tumour.

Note, however, that MR may miss calcification. Calcification cannot be consistently seen on MR and even when there is a large amount of calcification see on plain radiographs, on retrospective evaluation of the MR image, the calcification cannot be detected.

OTHER APPLICATIONS OF MRI

Other systems that are examined but less frequently by MR are in the:

(1) Evaluation for liver metastases. When metastases are suspected or when suspicious of additional liver metastases, a properly conducted MR study can be as sensitive as a good dynamic CT examination. However, many factors may influence the quality of the study such as patient motion, respiratory motion and peristalsis.

MR can also be used to study possible haemangiomas of the liver. MR is supposed to be able to differentiate haemangiomas from liver metastases in the majority of the cases.

(2) Staging of pelvic tumours, especially for endometrial cancers and cervical cancers in females and prostatic tumours in males. MR is also proving to be good for staging bladder tumours.

(3) In the thorax, MR has the advantage of not requiring any intravenous contrast to differentiate blood vessels from enlarged lymph nodes. It is therefore excellent for evaluating hilar and mediastinal lesions.

However, the acquisition times are still too long for evaluating lung parenchymal lesions. CT is still superior in this respect.

CONCLUSION

The main indications of MR at this present time have been mentioned. There is continuing work in progress and with faster scan times and newer techniques, MR is showing great potential for studying the heart and vessels.

In the very near future, MR angiography and MR fluoroscopy will be available. Further refinements in techniques will also increase the applicability and probably the sensitivity and specificity of MRI.

3RD ANNUAL SCIENTIFIC CONFERENCE:
College of General Practitioners Singapore, 20 October 1990
Seminar on Recent Advances in Diagnostic Techniques

AIDS MARKERS

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Acquired Immunodeficiency Syndrome was first described in 1981 in United Kingdom as a disease affecting homosexual as well as bisexuals. Thereafter in 1983 the causative agent, a virus, was isolated at the Pasteur Institute in Paris. The virus was first called HTLV 3, later renamed Human Immunodeficiency Virus HIV 1.

A second strain of AIDS virus was identified as HIV 2 in 1986. The virus is prevalent in West Africa and Brazil and thereafter it was detected in USA as well. This virus again can cause AIDS. Soon all blood and organ donors will have to be screened for both HIV 1 and HIV 2.

The virus is spherical, it has a core which has the protein p24. The envelope has on its surface protruding portions which consists of glycoprotein which contains gp 120. The transmembrane portion contains gp41.[1] It is this protruding gp 120 which attaches to the host cells the T4 lymphocytes, when an individual gets infected with the virus. The appearance of antibodies to these 3 proteins are the important markers in diagnosing the disease.

There are 3 main ways by which the infection can be spread[2], and all body fluids and blood are infected with the virus: sexually, parenterally by blood and blood products and vertically from infected mother to child. Sexual transmission is the most important mode of spread.

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All persons at risk for AIDS should be encouraged to seek counselling and advised on testing.

- Homosexuals
- Intravenous drug abusers
- Haemophiliacs who received blood products especially prior to 1985.
- Male and female prostitutes
- Children born of infected mothers and
- Sexual partners of the above

A positive test in a patient can cause serious implication. Therefore pretest counselling and consent should be obtained from the patient. If the doctor is not sure of how to handle the patient he or she should be referred to a specialist clinic.

Of what use is the laboratory test in HIV infection? It confirms the diagnosis of a suspected infection, screens blood for transfusion and detects infection in individuals in high risk groups.

There are laboratory tests for the detection of antibodies to the virus and the tests for the detection of the antigen. Antibody tests can be further subdivided to screening and the confirmatory tests. The ELISA test is a more commonly used test for the detection of antibodies to Human Immunodeficiency Virus. A large number of specimens can be tested using these screening tests while the more laborious but sensitive and specific test such as Western Blot test is used for confirmation.

The principle of the ELISA test is:

Beads or microtitre plates are coated with the HIV antigen. Patient's serum is allowed to react with the antigen for 30 mins. A second antibody anti-igG conjugated to the enzyme is added. To detect whether the reaction has taken place a substrate is added. If the HIV antibody has been

present in the patient's serum a colour change will occur. The darker the colour the greater the amount of antibody in the patient's serum. The machine reads the optical density of the specimens and indicates whether it is positive or negative. Control sera both positive and negative are used in each test run.

The advantages of the test are: it is inexpensive; automation is possible – a large number of tests could be performed at any one time and sensitivity varies from 93% to 99% (with recent kits the sensitivity has approached 99%).

The disadvantages and the limitations of the ELISA method are: The first generation ELISA kits in which the coating antigen consists of lysate of the whole virus associated with a good number of false positives. These false positives are either due to reactions of test sera with cellular components from the cultures used to grow the virus or to cross reactions with other agents infecting the patient. The use of antigen manufactured by recombinanat DNA methods has increased specificity which now approaches 100%. Nevertheless all ELISA positives must be confirmed by a confirmatory test.

Another disadvantage being: ELISA tests for antibody may be negative during the early stage of infection i.e. during the "window" period. During this period antigen for HIV may be positive. This period varies from 3 weeks to 3 months.[4] The test may be negative late in the disease and also in children.

Particle agglutination: The Serodia HIV test kit is particle agglutination assay for screening of antibodies to human immunodeficiency virus. The assay uses gelatin particles as a carrier, coated by antigen prepared by disruption of purified HIV with detergents.[5] The patient's serum is added and if there are antibodies to HIV the gelatin particles are agglutinated. Unsensitised particles are used as negative control. The plate is covered and left at room temperature for 2 hours. It is a one step easy test. The agglutination is read. The particles being coloured the plate could be read by the naked eye.

Particles concentrated in the shape of a compact button with a smooth round outer margin is reported as negative, while particles concentrated in the shape of a compact ring with a smooth round outer margin +/-. When the test is positive the agglutinated particles spread out covering the bottom of the well uniformly.

Western Blot assay is a highly sensitive and specific test for HIV antibody detection. In this method individual proteins of an HIV lysate are separated according to the size of the protein by electrophoresis. The virus protein is transferred on to nitrocellulose paper and reacted with the patient's serum. Any HIV antibody present in the serum is detected by an antihuman immunoglobulin G (IgG) antibody conjugated with an enzyme. In the presence of the substrate coloured bands appear on the strip.[6] In each test run positive and negative control sera are included.

The earliest antibody detected after an infection is p24 and its precursor 55 while antibodies to the envelope protein gp160 and gp120 are detected in all HIV infected persons regardless of the clinical states.

Although the overall sensitivity and specificity of the Western Blot test for the detection of antibodies to the various viral proteins are high, there has been debate regarding the interpretative criteria. Our laboratory follows the Association of State and Territorial Public Health Laboratory Directors (ASTPHLD) CDC's interpretation.

The criteria for a positive is any two of the following bands p24, gp 41 and gp120/160.[6] The criteria for a negative Western Blot interpretation specify no bands. Test results which do not fit into a positive or a negative are reported as indeterminate result.[7]

ASTPHLD definition gives the highest percentage of positive and the lowest percentage of indeterminate results. Most indeterminate patterns involve p15, p24, p55 or any combination of these 3 proteins.[7]

Indeterminate results may be obtained from patients who are sero converting. These patients should be followed up. If the WB results continue to be consistently indeterminate for at least 6 months in the absence of known risk factors and clinical symptoms the patient can be considered negative for antibodies to HIV and these patients

should be reassured that they are not infected with HIV. In contrast if a asymptomatic patient with an indeterminate WB results belongs to a high risk group, the patient requires additional follow-up. These patients with indeterminate results should not donate blood.[7]

Another important marker in HIV infection is antigen detection. The presence of antigen has both diagnostic and prognostic value.[8] Two weeks after the initial infection antigen p24 can be detected in the blood by an enzyme linked immunoabsorbent assay (ELISA). With the appearance of the antibody in the third week the antigen concentrate rapidly decreases to undetectable levels. Thereafter the antigen cannot be detected for several months. Reappearance of p24 antigen occurs with the fall of p24 antibody concentration in the patient's blood. This may be months or more after the onset of the disease. The HIV antiginaemia follows a diphasic course initially appearing during the acute stage and after a varying period of time reappearing in the late stage of infection.[9]

There is a correlation between antiginaemia and the stage of the disease. The presence of antigen in the blood is an unfavourable prognostic sign.

Virus isolation: Isolation of the virus is time consuming and requires special methods. To separate the lymphocytes which are infected by the virus 30 ml of heparinised blood is mixed with a chemical and the blood is centrifuged at a speed of 1500 to 2000 rpm. Depending on the density of the cells, bands of cells are formed, the red cells being heavier will settle to the bottom of the tube while the lumphocytes being lighter the cells will form a band right at the top of the gradient.

These lymphocytes are pipetted out washed 3 times and inoculated into special cell lines called the Human T cell line for the growth of the virus. After incubating the cell line for 3 to 4 days a drop of the culture fluid is fixed on a slide and an immunofluroescence test is performed. The virus can be isolated from 80 to 90% of the patients with AIDS or ARC, and 60 to 80% of the asymptomatic patients can yield the virus.

Isolation of the virus is not done in laboratories that do routine tests but done only in research laboratories.

Other markers which are used in diagnosing HIV infection is by determining the lymphocyte subsets, one of the earliest observations made in AIDS patients. The ratio of T4 helper cells to suppressor T8 cells is markedly reduced as the disease progresses and the ratio becomes <1.

Repeated measurement of T cell subsets have prognostic value. These tests are routinely done in many laboratories. It must be borne in mind that other infections and diseases can give a similar result therefore clinical interpretation is necessary.

With the increase in the number of patients screened and the incidence of AIDS on the rise, confirmatory diagnostic methods which are specific and sensitive are required to be done on patients with confusing serological profile.

A new technique available is known as the polymerase chain reaction (PCR) technique. This is an automated test whereby the DNA is extracted from the infected lymphocytes and amplified. One single infected gene could be amplified a million times thus making the test extremely sensitive.[4] The molecular technique, based on DNA amplification is useful in clarifying confusing serological test. The test kits are not marketed as yet and should be available in the near future, for use in the laboratories as a routine test.

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MEDICO-SOCIAL ASPECTS OF FRACTURES IN THE ELDERLY

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INTRODUCTION

As life expectancy increases, fractures in the elderly are becoming increasingly common and constitutes major medico-social problems for the medical community and social workers. These are further aggravated by break-up of the traditional extended family which would otherwise be able to look after these elderly folks during their rehabilitation. In addition, in our haste to get on with our economic development, insufficient attention has been paid to our physical environment to make them more friendly towards the elderly. The stresses of living in an urban environment and increasing life expectancy has exposed this group of patients to cardiovascular diseases, diabetes mellitis and neurological problems.

MEDICAL FACTORS

Three major medical factors are responsible for the majority of fractures in the elderly:

a. Osteoporosis

This is an ever increasing problem as our population ages, in particular females in the post-menopausal age. It is estimated that up to one-third of all fractures admitted to the orthopaedic wards in our hospital are in one way or another connected with osteoporosis.

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b. Muscular Weakness and Inco-ordination

With age, muscles atrophy and inco-ordination results. This will result in increasing falls and accidents, especially in a physical environment that is elderly-unfriendly. Majority of the fractures in the elderly are sustained at home.

c. Poor Vision

Poor eye-sight makes these people more susceptible to falls and injuries. Cataracts and other eye problems are particularly common in the elderly.

COMMON SITES OF FRACTURES

Some of the common sites of fractures in the elderly are:

Vertebral bodies
Neck of femur
Intertrochanteric region of femur
Lower end of the radius
Ribs
Shafts of long bone

Some studies in Western countries have shown that high morbidity and mortality are associated with fractures around the hips. Mortality within the first year of these fractures is reported to be as high as 40%. Majority of these patients in such countries end up in nursing homes after the initial fracture.

What is responsible for the high morbidity and mortality in such patients? This is because many chronic degenerative diseases are associated with the elderly. Conditions like diabetes mellitis,

hypertension, cardiovascular disease, chronic respiratory diseases, malnutrition and chronic urological problems all help to push these patients to greater morbidity and mortality. Good control or prevention of such condition certainly will go a long way towards rehabilitation to an independent life.

Treating such fractures within the hospital does not constitute much of a problem. It is what happens after the patient goes home that remains a major problem and continues to be the bugbear of an ideal rehabilitation and complete recovery.

HOME ENVIRONMENT

It is increasingly common to see elderly patients live by themselves, apart from the grown-up children. This is a price we pay for economic progress and westernization. Often such patients are not independent enough (not at least for 6 months) to live by themselves after a major hip operation. Even if they were to stay with their working children, they will still be unable to fend for themselves during the day when everyone is out.

The home environment is often not conducive for a weakened elderly patient to stay by himself or herself. Kitchen, toilet and other facilities are out of reach and can pose tremendous hazards to them.

SOCIO-ECONOMIC FACTORS

Financial problems is still a very real problem and one must not be blinded by our exterior of affluence to think that they do not exist especially among the economically inactive senior citizens. Loneliness and depression are grave social problems and especially when they are no longer able to get out of the house to mix around with their own friends. Suicides are increasingly common among this group of patients.

CONCLUSION

As we progress to our next stage of development in our society, we must now take stock of our present situation. The problem of the elderly, especially those recovering from a major operation following fractures, are very real. The medical problem may be complex but are surmountable. The social problems are the ones that require urgent attention. Building more nursing homes and homes for the aged are only partial solutions.

Preservation of the extended family and building an elderly-friendly environment, both in public places and at home are desirables and should be encouraged. An independent fully rehabilitated patient which is achievable in many instances, is more cost-effective than nursing homes.

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KNEE PAIN IN THE ELDERLY

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INTRODUCTION

Knee pain is among the more common of the musculoskeletial complaints noted in our elderly population. This is due to the disproportionately high incidence of degenerative joint disease of the knee. The basis of this may be the minor anatomical anomaly, the varus knee, which we so often observe in our younger population. When patients seek medical consultation they do so because of a chronic knee pain that has become intolerable, an acute exacerbation of a chronic knee pain, or a sudden onset of acute knee pain where none existed before.

CHRONIC KNEE PAIN

Almost 80 to 90% of chronic knee pain and acute on chronic knee pain presentations are due to either primary or secondary osteoarthritis (OA) of the knee. Such is the magnitude of the problem of osteoarthritis of the knee in our population.

Osteoarthritis

Patients with knee OA complain of knee pain that is aggravated by activity, and stiffness of the knee following periods of rest. This stiffness, although lasting only 5 to 10 minutes, causes considerable discomfort. They may have obvious deformity, more often a varus knee, jointline tenderness and restriction of joint movement.

X-rays of the knee should be taken while the patient is weight bearing. These will show the classic features of jointline narrowing or obliteration and osteophyte formation. Gross specimens of the knee at surgery demonstrate complete erosion of the articular cartilage and exposure of the subchondral bone that is eburnated. Initial treatment is largely with NSAIDs (non-steroidal anti-inflammatory drugs) to relieve the pain. Physical therapy to preserve mobility of the joint, hot compress during the chronic phase and cold compress during acute exacerbation may be useful. Our patients normally attach a stigma to a walking aid and often prefer not to use them. Attempts at weight reduction are often futile, but should nevertheless be encouraged.

If these measures do not alleviate the patient's pain, surgical intervention is necessary. One must then consider whether one is dealing with isolated patello-femoral osteoarthritis (less common) or tibiofemoral osteoarthritis. Patello-femoral osteoarthritis is characterized by anterior knee that bothers the patient when going up or down stairs, squatting or getting up from a squatting position. Special patello-femoral axial films will demonstrate narrowing of the patello-femoral joint space. When patello-femoral osteoarthritis exists in isolation surgical intervention will take the form of patelloplasty, lateral release and tibial tubercle elevation.

Patients with tibiofemoral OA with or without patello-femoral OA who need surgical intervention because of failed medical treatment have a choice of having either debridement of the joint, osteotomy to correct the alignment or total knee replacement. The choice of the procedure will depend on prevailing conditions but in the elderly patient who leads a sedentary lifestyle, total knee replacement (Fig 1) is by far the most promising.[1]

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Figure 1: Intraoperative picture showing total knee-metallic femoral and high density polyethylene tibial components in place.

TOTAL KNEE REPLACEMENT (TKR)

Total knee replacement (TKR) has a predictable outcome and provides almost complete relief of pain. Today it is a success story that exceeds the successes of all other forms of joint replacement. Total hip replacement, which was started before TKR, begins to give problems and the survivorship begins to fall off after the 7th year. By the 10th year survivorship approached 70% whereas the total knees available today remain in the 92nd percentile at 10 years.[2] There are limitations, however to TKRs. Joint movement is restricted to 100° of flexion. Our Muslim community may be reluctant to accept this because of their need to pray in the Mohammedan position. Going to the toilet should not be a problem as a sitting commode could be installed to replace a squatting one. The morbidity of failure, in particular of infection is nightmarish, but fortunately the risk of infection is low, in the region of 1%.

Hydrocortisone and lignocaine (H&L) injections are occasionally used to secure pain relief. This must be used sparingly. It should be restricted to cases not responding to medical management and cases not suitable for surgical intervention because of prevailing medical conditions. Even so it should not be repeated more than twice. Figure 2 shows a man who could hardly walk because of an iatrogenically induced neuropathic joint following multiple H&L injections.[3]

ACUTE KNEE PAIN

Despite investigations in a significant number of acute knee pain presentation, approximately half, of acute knee pain presentation there will be no obvious diagnosis forthcoming. These patients must be considered probably to have subclinical OA, symptoms of which are precipitated for the first time by a sudden surge in activity level.

Figure 2: Clinical picture showing iatrogenically induced neuropathic knee joint. Inset shows close up of knee demonstrating multiple punctate depigmented spots representing sites of numerous hydrocortisone injection.

The other conditions responsible for acute knee pain include gout, psuedogout, rheumatoid arthritis, infection, meniscal injuries, osteonecrosis and stress fractures. In my experience osteonecrosis, stress fractures, gout and pseudogout and degenearative meniscal injuries form the majority of known causes of acute knee pain. Of these oestonecrosis, stress fractures and gout are among the most commonly missed diagnoses and thus warrant some discussion on their diagnosis and management.

Osteonecrosis

Osteonecrosis is due to bone infarct. It causes severe unremitting pain and NSAIDs do not completely relieve the pain.[4] Unless the patient is made aware of the nature of this condition he is going to doctor-hop to try to get relief for the pain. The initial x-rays will be normal and the condition may resolve after 3 to 4 months without

any apparent change or it may progress to secondary OA and become radiologically demonstrable.

Pathologically, the infract either heals with resolution or a subchondral fracture occurs and an area of subchondral bone and cartilage breaks free and sets the stage for degenerative OA.

Clinically, the patient's pain resolves after a period of a few months, and he remains well, or if the healing is incomplete, it shows gradual progression to secondary OA with increasing deformity and stiffness. Figure 3 shows a clinical specimen of osteonecrosis and Figure 4 a radiological appearance of osteonecrosis.

Figure 3: Intraoperative picture showing separation of osteochondral fragment following femoral condyle osteonecrosis.

Figure 4: X-rays of knee showing classical osteonecrotic lesion in medial femoral condyle (arrow).

STRESS FRACTURES

Less common than osteonecrosis and estimated to be responsible for up to 10% of acute knee pain in the elderly are stress fracture.[5] These stress fractures occur not solely because of unaccustomed activity as seen in the young, but because of concomitant osteoporisis and weakening of the bone in the elderly. They are benign and with appropriate weight relieving measures and analgesics, heal completely. But if missed they can give rise to unfortunate outcomes such as collapse of the affected condyle following completion of the fracture.[6] Figure 5 shows the x-rays of an elderly lady presenting with knee pain due to stress fracture.

Figure 5: X-rays of knee showing strees fracture in medial tibial condyle.

Gout/Pseudogout

Gout is not uncommon. It is often missed and may be labelled as septic arthritis. Patients present with sudden onset of acute knee pain and swelling, and other signs of inflammation. The aspirate is often turbid and can be easily passed off as purulent fluid. This is the basis of the misdiagnosis. Although the serum uric acid is often raised it is not necessarily raised all the time. The aspirate must be routinely sent for examination for crystals in addition to culture and sensitivity and the identification of negatively birefrigent crystals will confirm the diagnosis. Once confirmed, the treatment is standard – NSAIDs to relieve the acute knee pain, dietary counselling and allopurinol or probenecid to lower the serum uric acid level to prevent recurrence.

Of course one must not miss the diagnosis of septic arthritis which although rare has a very similar clinical presentation. In the absence of

facilities to investigate these patients with acute knee pain, swelling and evidence of inflammation it is preferable that they be referred to hospital for an immediate workout. Septic arthritis usually follows invasive procedure like acupuncture or aspiration of the knee or injections of hydrocortisone and lignocaine when sterility is breached.

Algorithm for Management of Acute Knee Pain

My routine for patients presenting with acute knee pain in the absence of knee swelling and radiological changes is to treat them symptomatically and with weight relieving measures. In most instances they will fall into the categories of acute exacerbation of OA, stress fractures, osteonecrosis or degenerate meniscal tears. I will caution the patient that he needs to be prudent and use a walking aid to relieve the stress on the affected knee. I will repeat the x-rays in six weeks to see if there is any change to suggest osteonecrotic lesions or stress fractures. If none is present I will continue to treat him symptomatically as for osteoarthritis, reassured that this is not osteonecrosis or stress fracture. If degenerate menicus cannot be excluded artrhoscopic examination may be indicated. When patients can afford specialised investigations I work them up at presentation with MRI or bone scan to establish or exclude the diagnosis of osteonecrosis or stress fracture. MRI will be useful for the diagnosis of degenerate meinscal tears as well.

Patients who present with acute knee pain and moderate or severe knee swelling with evidence of inflammation require investigations to rule out sepsis and gouty arthritis although OA is more likely. If the knee aspirate is golden in colour and clear it can be presumed that it is secondary to OA. In the presence of turbid fluid, the aspirate should be evaluated rapidly for crystals gram stain and culture and if this is not possible in the clinic, they should be referred to the hospital for rapid evaluation. In the absence of crystals or organism a diagnosis or rheumatoid arthritis must be pursued.

CONCLUSION

The focus of this paper has been on the knee itself. However the patient complaining of knee pain with no obvious knee pathology may well have his pathology in the hip or the spine and hence examination of these regions may be necessary when the knee appears normal.

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OFFICE PROCEDURES TO ALLEVIATE SHOULDER PAIN IN THE ELDERLY

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INTRODUCTION

Shoulder pain in the elderly may arise from disease of the shoulder joint per se, the cervical spine, or less frequently the viscera. Mechanical and degenerative conditions of the shoulder e.g. rotator cuff disorders, degeneration of the acromioclavicular joint, biceps tendon disorders and frozen shoulder rank as the most frequent local diseases in the shoulder that may give rise to shoulder pain. Metastatic lesions and infections of the shoulder, although uncommon have also to be kept in mind. Cervical spine disorders such as cervical spondylosis is a common pathology that causes pain to radiate from the neck to the point of the shoulder and presents as pain in the shoulder occasionally. Angina pectoris may be felt as pain in the shoulder and inner side of the left upper limb. Basal pneumonia or pleurisy may also present with shoulder pain. Intraabdominal pathology such as chronic gall bladder disease or even peptic ulcer disease may be referred to the shoulder.

HISTORY & CLINICAL EXAMINATION

The history and clinical examination should therefore be directed not only to determine the nature of possible pathologies in the shoulder but also to exclude sources of referred pain to the shoulder region especially in the absence of definite pathology in the shoulder as evidenced by a full range of shoulder movement and lack of local signs in the shoulder.

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MANAGEMENT: SYMPTOMATIC RELIEF

Once it is established that the pathology is in the shoulder and its nature identified, it may then be possible to alleviate the patient's symptoms by a local injection of steroid and local anaesthetic. The conditions most amenable to this form of treatment include:-

1. acromioclavicular osteoarthritis
2. rotator cuff disease and
3. biceps tendon disorders

The steroid used for the injection is Triamcinolone acetonide in concentrations of 10mg per cc. It is combined with 1% lignocaine in the volume ratio of 2:1. The lignocaine is used to ascertain that the deposition of the steroid is at the right spot, as with correct localisation of the injection the pain is immediately relieved by the lignocaine. This initial relief is temporary but the steroid continues to act over the next few days and brings a more lasting relief of the patient's symptoms. Absolute sterility has to be observed during the injection. If relief lasting for a few weeks or more is obtained, the injection may be repeated up to two to three times when symptoms recur but no more frequently than at 6 weekly intervals. If no relief is obtained further investigations are indicated. If relief is obtained but recurs after each injection surgical management is indicated.

ACROMIOCLAVICULAR OSTEOARTHRITIS [3,8]

Degeneration of the articulation between the lateral end of the clavicle and the acromion is an underestimated cause of shoulder pain in the middle age and elderly patient.[8,5] Pain may be felt

over the point of the shoulder and may occasionally radiate to the base of the neck and angle of the jaw. The physical signs include pain at the extremes of abduction and adduction of the arm across the chest. Local tenderness on balloting the clavicle on the acromion is also diagnostic.

A steroid and lignocaine mixture may be delivered into the joint by palpating for a gap between the lateral end of the clavicle and the acromion (Fig 1). A "give" is felt as soon as the joint is entered. Half to 1 cc of the mixture may be introduced into the joint proper. Another 2 cc may be infiltrated around the joint. Relief of symptoms is immediate (from the action of the local anaesthetic). Pain may recur in an hour or so and the patient must be warned. The steroid acts over the next few days with gradual relief of pain. In the meantime icing the shoulder and NSAIDs will help in the relief of symptoms.

Figure 1: Steroid-anaesthetic mixture introduced into gap between lateral end of clavicle and acromion in patient with acromioclavicular osteoarthritis.

ROTATOR CUFF DISEASE

This includes tendinitis and partial or complete tears of the cuff.[6,7] Symptoms include shoulder pain with an impingement arc of between 60° and 120° of sideways abduction.[4] Acute calcific tendinitis presents with severe agonising pain and all movements of the shoulder are restricted.[1] Weakness in the shoulder especially with sideways abduction may denote a cuff tear.[6,7]

Relief in these situations may be obtained with steroid and lignocaine infiltrations into the subacromial bursa which is often inflamed and responsible for the pain. Entry into the bursa is effected by puncturing the skin 2 to 3 cm, below the easily palpable postero-lateral corner of the acromion. The needle is directed to the undersurface of the acromion (Fig 2) 5cc of the steroid and lignocaine mixture may be introduced. Placement in the bursa is confirmed by easy entry of the injected mixture. Pain relief is again immediate.

Figure 2: Steroid-anaesthetic mixture directed into under surface of acromion to reach the subacromial bursa in patient with rotator cuff tendinitis.

Again icing and NSAIDs are important in the immediate post injection period when pain recurs after the effect of the lignocaine wears out.

BICEPS TENDINITIS [2]

Biceps tendinitis is an uncommon cause of shoulder pain. Discomfort is experienced over the front of the shoulder and may radiate down the front of the arm. Tensing the biceps muscle reproduces the pain. Local tenderness over the long head of the biceps tendon in the bicipital groove may also be elicited.

The steroid and lignocaine mixture is introduced into the tendon sheath of the long head of the biceps brachii. This will be located approximately 2 cm from the anterior edge of

the acromion directly anterior with the shoulder internally rotated 15° in the neutral position. The needle is directed to the floor of the biciptal groove, withdrawn slightly and infiltration of the mixture is effected into the bicipital sheath. 3 to 5 cc of the mixture may be introduced. Post injection, icing and NSAIDs are required as for the previous two conditions.

CONCLUSION

In conclusion, steroid and lignocaine infiltration in painful conditions of the shoulder are not only therapeutic but also diagnostic and help pin point the exact pathology in the joint. In cases of doubt lignocaine alone is infiltrated into the tender spot. If relief is obtained the steroid is introduced. With adequate care they should provide lasting pain relief for the common causes of shoulder pain in the elderly.

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SPINAL DISORDERS IN THE ELDERLY

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Backache is probably second only to upper respiratory tract infection among the common disease entities affecting our general population. It affects young and old. In younger patients, the attacks are of shorter duration and are less disabling. In the elderly, the attacks tend to be more severe and persist longer.

There are many causes of backache which are listed as follows:-

1) Trauma : Backstrain, compression fracture.
2) Deformity : Kyphosis, scoliosis.
3) Inflammation : Infective, non-infective.
4) Degeneration : Cervical or lumbar spondylosis.
5) Mechanical derangement : PID, Spondylolysis, Spondylolisthesis.
6) Neoplasm: Secondary, Primary, Multiple myeloma.
7) Miscellaneous.

Backache in the young patients is usually related to their jobs and daily physical activities. Whereas in the old, osteoporosis by far is the most common cause of backache especially in women. Compression fracture is liable to occur from only trivial injury. As our society becomes more affluent, the infection of the spine has become a rarity. We are now treating more and more degenerative disorders of the spines.

History taking is of utmost importance. We should first establish whether the pain is acute or chronic, whether it is associated with numbness, weakness or bowel and bladder dysfunction. Is the pain related to recumbency, posture of ambulatory activity? Multiple joints involvement may signify rheumatoid arthritis or ankylosing spondylitis. Sciatica pain always goes below the knee. One must differentiate neurogenic claudication from the vascular claudication. The symptoms of neurogenic claudication are consistently relieved by sitting, squatting or leaning forwards to flex the spines, whereas the symptoms of vascular claudication are relieved by standing. As the disease progresses, the patients experience more radicular pain.

Intractable, constant and night pain should not be taken lightly. As it may denote secondary to the spines and multiple myeloma. The neoplasm of the spine is often missed in the elderly until very late stage. One should be vigilant on the look out for the condition. A sixty-four year old Malay who presented to many general practitioners with intractable and night backache for three months. The backache was not relieved by analgesics. He was later found to have renal cell carcinoma with secondaries to lumbar and scaral spines. He underwent a decompression laminectomy and followed with radiotherapy. He had good pain relief after the surgery and radiotherapy.

Another patient, a seventy-six year old Chinese lady was initially treated for senile osteoporosis and compression fracture T12 and L1 vertebral with calcitonin. She developed progressive weakness and paraesthesia. Finally she ended up with paraparesis which rendered her bedridden. The lumbar myelogram showed a complete block at T12 and L1 level. She underwent a decompression laminectomy and Luques' rodding with spinal fusion. Post

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operatively she recovered very well. When she was last seen she was walking without any aid. The lesson we learn from this case is that we should not underestimate senile osteoporisis and compression fractures which may develop quite a severe neurological deficit.

The physical examination must include the examination of hips, abdominal palpation and a thorough neurological examination. In the presence of a definite neuroglogical deficit it is likely that there is nerve entrapment. Marked limitation of straight leg raising, a crossed lasegue test, a positive bowstring test over the posterior tribial or lateral popliteal nerves and tenderness in the sciatic notch are strongly suggestive of nerve compression or irritation. With careful examination, one can always pick up muscles wasting, motor weakness and sensory loss. The dorsalis pedis and femoral pulses must be felt routinely. Back symptoms may be no more than a focal manifestation of a generalised skeletal disease.

The treatment of backache in the elderly has to begin with establishing the specific diagnosis. The conservative treatment modalities for spinal disorders encompass the following:-

1) Drugs:
   Analgesics
   Antispasmodics
   Anti-inflammatories

2) Physiotherapy:
   Weight loss
   Bed rest
   Exercises
   Bracing
   Traction
   Heat, cold and massage
   Ultrasound
   Transcutaneous electrical stimulation

3) Procedures:
   Acupuncture
   Local nerve blocks and injections
   Epidural steroids, analgesics and narcotics

4) Education:
   Back School
   Body mechanics and dynamics
   Instructions
   Audiovisual aids

Most of the elderly patients respond to conservative treatment. Only a small proportion of them end up surgery. The chief essential of any operation is that it should not make the patient worse than he was before he submitted for it.

The operative treatment consists of laminectomy. Laminectomy anterior discectomy with fusion and posterior spinal fusion. The indication of surgery, choice of the most appropriate operation, the technical aspect and the post operative management are matters of highest importance. It is impossible to cover all of them in this lecture.

Old folks are prone to develop contracture and stiffness after a bout of backache. A proper back rehabilitation programme should be initiated as early as possible. The programme includes postural training, lifting exercises, alignment, conditioning exercises and activity precautions. Prolonged usage of analgesics in the elderly can cause severe gastrointestinal bleeding. One should not overlook the pathology that needs immediate treatment such as cauda equina syndrome, infection or neoplasm. The success of treatment for the elderly with spinal disorder will depend on the cooperation of the patient, the understanding of actual pathology and the tender loving care given by the physician.

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FITNESS PROGRAMME FOR THE ELDERLY

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INTRODUCTION

The elderly population in Singapore is increasing. By the year 2000, there are expected to be about 300,000 people aged 60 years and above.[1] To enable the elderly to be less dependent on others, as well as be an important source of manpower, they would need to be sufficiently healthy and fit – physically and mentally. It is a known fact that the body's functions tend to decline with age. What is often not obvious is that age only accounts for about 50% of this decline. The other 50% is often the result of the reduction in physical activity.[2] Regular physical activity can help to preserve physical and mental health, with resulting independence to the greatest extent and as late in life as possible. It would also help to improve the quality of life and lengthen the duration and improve the enjoyment of those 'good years', by maximising the remaining capacity of the body. Whether it prolongs life expectancy in the elderly is not known, although it has been estimated that physically active men would live 1 to 2 years more than the inactive.[3]

PHYSICAL ACTIVITY FOR THE ELDERLY

It is therefore important for the elderly to be involved in regular physical activity. The following are some important points to consider when introducing exercise to the elderly:

(1) Convince the elderly on the need for regular physical activity.

(2) Provide the facilities and programmes to enable the elderly to exercise regularly.

(3) Provide advice and training on exercises to keep healthy and fit.

(4) Increases in intensity and duration of physical activities must be slow and gradual.

(5) Regular checkups, monitoring and reminders would help to keep the elderly motivated to exercise regularly.

Exercise, sports, games and other physical activities can very broadly be subdivided into aerobic, callisthenic, relaxation and high resistance anaerobic exercises.[4]

The "FITT" formula guidelines for exercise in the elderly are as follows:-

F = Frequency	:	3 to 5 times a week, evenly spaced out over the week. Daily exercise is possible if the exercise is not too intense or prolonged.
I = Intensity	:	40 to 70% of maximum functional heart rate initially, building up to 60 to 85% of actual or age predicted maximum heart rate if possible and if required. For those elderly who are already exercising and used to doing so fairly intensely, there is normally no need to reduce on intensity.
T = Time or duration	:	15 to 60 minutes of fairly continuous exercise during

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each session, with about 3 to 5 minutes of warm-up and 3 to 5 minutes of cool-down light exercises if the exercise is fairly vigorous.

T = Type of activities	:	A combination of light aerobic and light callisthenic exercises is recommended.

Aerobic exercises are those which improve the fitness of the heart, lungs and blood circulation, which is generally considered to be the most important component of fitness for all, irrespective of age, sex, health or socio-economic status. Such exercises use the larger muscle groups like the lower limb muscles and can be done fairly continuously at a moderate to reasonably high intensity. Examples of aerobic exercises suitable for the elderly, which have lower risk of injuries, are brisk walking, swimming, stationary cycling, and low-impact aerobic dance. These activities should be done at an intensity which results in sweating and deep breathing, but without developing breathlessness or other medical problems.

Light callisthenic exercise like arm-swinging, trunk-twisting and sit-ups help to improve muscle tone, muscle endurance and flexibility of joints and muscles. Some callisthenic exercises suitable for the elderly are attached (Appendix A).

The types of activities recommended must also take into account the biological age, social situation and any medical limitations. Problems of poor vision or balance and postral hypotension would limit participation in activities where there is a risk of collision, falling or drowning.

GUIDELINES AND RECOMMENDATIONS FOR MAXIMUM BENEFITS FROM REGULAR EXERCISE

a. Those who have not been exercising regularly for some time and are uncertain of their state of health and fitness, or who already have significant medical or fitness problems, for example, heart disease, high blood pressure, diabetes, are advised to consult a medical doctor before embarking on any vigorous or moderately vigorous exercise programme.

b. The exercise programme chosen should be sufficiently beneficial, safe, appropriate to needs as well as health and fitness status. Always begin slowly, then gradually building up to the required and desired amount of exercise. Remember to "train, but don't overstrain".

   A 4 to 6 week gradual conditioning programme of mild to moderate physical activity (for example, slow walking and light callisthenics) is recommended before more vigorous activities (for example, brisk walking, low impact aerobic dancing, racket and ball games) are indulged in. This is particularly important for the unfit and those who have not been exercising regularly for some time.

c. Do not exercise when unwell, particularly when suffering from a significant medical problem including injury or acute infection (for example, flu or chest infection). Serious complications may result. Resume only when totally well and gradually build up to the previously desired or required amount of exercise. Consult the doctor if necessary.

d. Any time, except the 1 to 2 hours immediately after a meal, is suitable for exercising. A person's best time is that which is sufficiently convenient and conducive enough for him to be able to exercise regularly and judiciously. Avoid the heat of the day until you are well acclimatised.

e. Before, during and after prolonged physical activities, especially under our hot and humid environmental conditions, prepare for and replace excessive sweat loss through liberal intake of fluids. This would reduce the chances of developing problems like heat cramps, heat fatigue, heat exhaustion or heat stroke. It is advisable to exercise in shorts and T-shirts rather than long pants, long-sleeved shirts or track suits. Attire made of plastic or rubber (for example, ponchos, windbreakers, raincoats) in particular should not be worn. The elderly are not able to tolerate heat well, partly because of medical conditions eg. hypertension, diabetes which they may have. It is therefore especially

important for them to heed this recommendation.

f. Abstinence from unhealthy practices like smoking and overindulgence in alcohol, would further minimise the risks and enhance the benefits of exercise. In most cases of obesity, exercise does not eliminate the need to diet judiciously.

g. Allow the blood in the lower limbs to be circulated back to the heart by not standing still immediately after vigorous exercise. Failure to do so may result in giddiness, fainting or more serious complications. Cool down by walking slowly for at least 3-5 minutes. If very tired, lie flat (on back) with feet slightly raised.

CONCLUSION

Hippocrates wrote some 2,500 years ago that "all parts of the body which have a function, if used in moderation, and exercised in labours to which each is accustomed, become thereby healthy and well developed, and age slowly; but if unused and left idle, they become liable to disease, defective in growth and age quickly ...". For the elderly, as well as everyone else, it is well worth recommending regular exercise, three to five times a week, 15 to 60 minutes each time, combining aerobic and callisthenic exercises done at a moderately vigorous intensity, to "add more life to your years" and hopefully "more years to your life".

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APPENDIX A

LIGHT CALLISTHENIC EXERCISES SUITABLE FOR SENIOR CITIZENS

Compiled by: Sports Medicine & Research Centre
Singapore Sports Council
National Stadium, Kallang
Singapore 1439

EXERCISES IN SITTING POSITION (ON A CHAIR)
在椅子上坐着运动

1a Slowly turn head as far as possible to one side and hold (no jerking).

Repeat to the other side x 5 times.

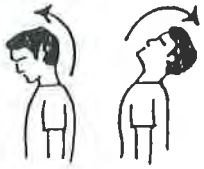
慢慢地将头部尽可能的转向左边，停一会，回来原有位置，共做五次。

然后用同样的做法，但把头转向右边，也是共作五次。

b Bend neck forward dropping the chin towards the chest and hold, and then backwards as far as possible and hold x 5 times.

将颈部向前弯下，停一会，回来原有位置。共作五次。

用同样的做法，但颈部向背后弯下，也共作五次。



c Bend neck to one side moving the ear towards the shoulder (do not lift the shoulder).

Hold and repeat to the opposite side x 5 times.

将颈部向左边弯下，使耳朵倾向肩膀（不要提升肩膀），停一会，重回原有位置，共作五次。

同样的做法，但颈部向右边弯下，也共作五次。

2a Lift arms alternately forward so that one is going up while the other is coming down x 5-10 times.

左臂由前面向上提升。然后当左臂向下降时，右臂向上提升，共作五次。

b Lift both arms forward and up, keeping elbows straight, then return to starting position x 5-10 times.

将双臂由前面向上提升，双肘成直线，重回原有位置，共作五次。

3a With both hands on shoulders, slowly move both arms through full circles, repeat in the opposite direction x 5-10 times.

两手放在肩膀，慢慢地作圆圈运动，共作５—１０次。

b With both hands on shoulders, pull elbows together, then separate them x 5-10 times.

两手放在肩膀，把双肘拉近，然后双肘又分开，共作５—１０次。

c Clasp the hands behind the head and push elbows back as far as possible and hold x 5-10 times.
两手在头的后部捉着，把双肘推向背后，停一会，再做，共作5—10次。

4 Clasp the hands overhead with the palms facing up and stretch as high as possible and hold x 5-10 times.
两手在头的上部捉着，掌心向上的伸张，停一会，重回原有位置，共作5—10次。

5 Place one hand on opposite shoulder so that the elbow points forward.
Pull the elbow towards the body with the other hand and hold (5 counts).
Repeat on the opposite side x 5 times.
一手放在左边的肩膀，肘部指向前面伸出，用另一只手把肘部推向身体背后。
停一会，再做，共五次。
相反方向也同样做五次。

6 Place the palm of one hand between the shoulder blades so that the elbow points upward.
Push down on the elbow with the other hand.
Hold and repeat on the opposite side x 5 times.
放左手掌在背后肩胳之间，使肘部向上。
用右手推肘部向下，停一下，重做，共作五次。
同样地，相反方向也作五次。

7 Hold the arms out at shoulder level with palms facing forward keeping elbow straight.
Stretch arms back as far as possible and hold x 5-10 times.
两臂向外伸张与肩膀平齐，肘要直，掌心向前，然后尽量向后伸张，停一会，重做。这样共作5—10次。

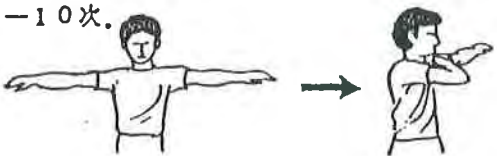
8 With one arm overhead and the other hanging beside the chair, lean as far as possible in the direction of the hanging arm (do not lift seat off the chair).
Hold and repeat to the other side x 5-10 times.
左手臂在头上部，右手在椅子旁，左手与身斜向右边，停一会，重做。（不要离开座位）。
相反方向，亦作同样运动。做5—10次。

9 Hold both arms out to the side with the palms facing down.

Gently twist the trunk and turn the head to one side while bringing the opposite arm across the chest.

Hold and repeat to the other side x 5 times.
(There are variations to this exercise).

掌心向下，双臂向前伸出，然后沿着胸前向左边伸出，又向右边伸出，共5—10次。



10 Slowly rotate the wrists through complete circles.

Repeat in the opposite direction x 5-10 times.

手腕慢慢地向左旋转，然后向右旋转，共作5—10次。

11 With the arm straight, bend the wrist so the fingers point up.

Gently pull the hand back with the other hand and hold.

Repeat with the other side x 5 times.

左手臂向前伸直，手腕向上弯而使手指向上指。轻轻地用右手把左手拉回身边，停一会，重做。共作5次。

另一只手，也作五次。

12 Bend the wrist so the fingers point down.

Gently pull hand back with the other hand and hold.

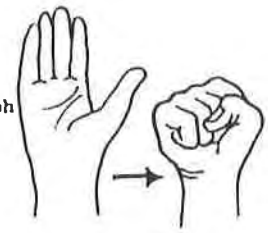
Repeat with the other side x 5 times.

把左手腕向下弯，而使手指向下，轻轻地用右手把左手拉回身边。停一边，重做，共做五次。

另一只手，也作五次。

13 With arms raised forwards to shoulder level, slowly clench the fists, then relax, x 5-10 times.

两手臂向前伸起至肩膀平高，慢慢地提紧拳头，然后又放松拳头，共作5—10次。



14 Sit up straight and bend one knee, bringing it up to the chest with both hands and hold.

Repeat with the other leg x 5-10 times.

直立坐着，左膝弯曲，並用两手提升至胸部，停一会，重做。共作五次。

另一只腿，亦作五次。

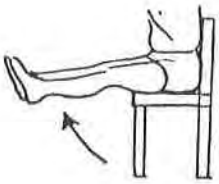
15a Lift one leg till parallel to the floor (leg should be straight) and hold for two to six seconds.

Return to starting position and repeat with the opposite leg x 5-10 times.

提升左腿至腿部与地板平行。（腿部应该要直）。停约两到六秒钟，重回原有位置。重作，共作五次。
另一只腿，亦做五次。

b Same as 15a, but lifting both legs at the same time.

与（15a）相同，不同的只是把双腿一起提升。



16 Bend forwards at the hips, keeping head and back straight, then return to upright sitting position x 5-10 times.

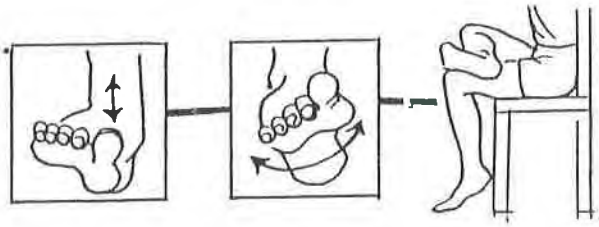
头部和背部要保持直，把臀部向前弯，后重回原有位置，重作，前后共十次。

17 Cross one leg over the other and rotate the ankle in a clockwise direction and then anti-clockwise direction.

Follow with pointing of toes down as far as possible then up and backwards as far as possible x 5-10 times.

Repeat with opposite leg.

把左腿放在右腿上成交叉状，然后左脚跟向顺时针方向旋转五次。反时针方向也作五次旋转。
脚脂向上，向下运动，各五次。
同样地，另一只脚也作五次。

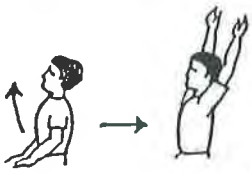


18 Place both feet flat on the floor and try gripping the carpet or floor x 5-10 times.

放双脚平贴着地板，並好像紧挺着地板或地毯。作各五次。

19 Deep breathing exercise x 5 times.

深呼吸运动，共五次。



EXERCISES IN STANDING POSITION
站 立 式 运 动

Get up from chair by leaning forwards at the hips, without using the arms if possible.
从椅子上站起来，臀部倾向前。可能的话，不要用双臂。

Stand with feet apart with both arms stretched out horizontally to the sides.

Circle the arms forwards, then backwards x 5-10 times.
两脚分开站立，两手臂平行的向左右伸张出去，然后向前作旋转圆圈运动五次，然后向后作旋转圆圈运动五次。

2 Keeping the elbows up, slowly press both arms back as far as possible, hold for 2 counts and return to starting position and repeat x 5-10 times.
提升两肘，慢慢的将两臂向后压，停一会，重回原来位置。重做，共五次。

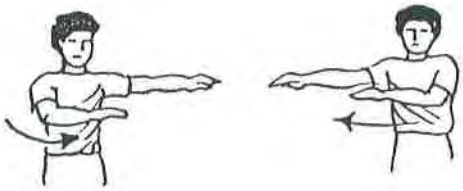
3 Stand with feet apart, raise one arm to the side at shoulder height, bend the elbow to place the hand behind the back, reaching down as far as possible, return and repeat with the other arm x 5 times.

两脚分开站立，左臂向左边提升至与肩膀高，把肘弯向背后，尽可能弯下，重回原有位置。重做，共五次。
相反用右臂，也做五次。

4 Raise one arm above head so that it is in a straight line with the side of the body, plam facing the ceiling, hold the stretch a few seconds, then change arm x 5-10 times.
向上举起左手，使与身体成直线，掌心向上的伸张，停几秒钟，做五次。
相反地，用右手，也共做五次。

5a Swing both arms together at waist level to the (L) side so they wrap around the body. Maintain the position with face looking to the (L) side.

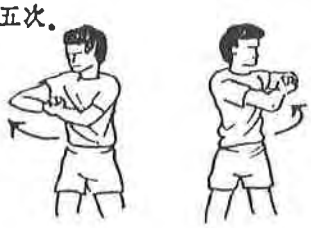
Return to starting position and repeat to the (R) side x 5-10 times.
提高双臂至腰部，然后双臂一齐摇向左边，使两臂能围绕身体，双眼也一齐向左看。停一会，重回原来位置。重做五次。相反方向，也作五次。



b Stand with feet wide apart.

Arms bent with hands in fists together in front of chest, twist arms to the (L) side as far as possible, at the same turn head to (L) side to look at far elbow, return to starting position and repeat on (R) side 5-10 times.

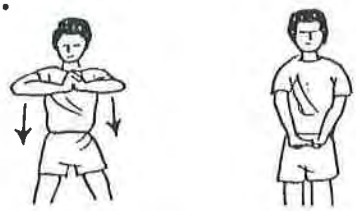
两脚分开站立。双臂弯曲使两手在胸前相扭着，双臂一起扭向左边，双眼向左边看着左肘，回复原来位置，重做五次。
相反地，向右边，也作五次。



6 Stand with feet wide apart.

Hand clasped in front of chest, elbows out to the sides, turn the palms out and straighten the arms to the front, return and repeat x 5-10 times.

两脚分立站着。两手在胸前相捉着，使两肘伸向外，掌心向外，並把双臂向前面伸直，又收回来，共做五次。



7a Stand with feet wide apart.

(L) hand on the hip, (R) arm in an arc overhead with knees lookod, bend to the (L) side from the waist, pressing the (R) arm to the (L).

Return to starting position, repeat on each side x 5-10 times (increasing difficulty by increasing speed of exercise).

两脚两开站着。左手放在臀部，右臂作弧形向上弯过头部，弯向左边，腰部亦作弯状，向左边压右臂。重回原有位置。重做五次。
相反地，右手也做五次。

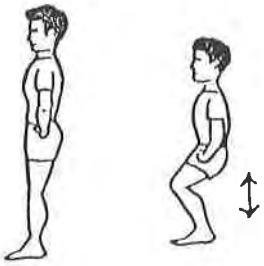
b Stand with feet shoulder-width apart and stretch arms above head. Bend body to right, then upright, and then to the left side x 5-10 times.

两脚两开站立，两臂向上伸张，使身体弯向右边，然后直立，重做五次。
相反地，两臂向上伸张，使身体弯向左边，然后直立，作五次。

8 Stand with feet apart.

Bend the knees slightly about 30° and return to starting position x 5–10 times (increasing intensity by holding longer in bent position).

两脚分开站着，两手插在臀部，向下使膝部作３０° 弯，重回原有位置，作五次。



9 With hands on hips, raise left foot off the floor, hold for 2–5 seconds, then back to starting position, and repeat with other leg x 5–10 times.

两手插在臀部，提起左脚，停约２—５秒钟，回复原来位置，重做五次。

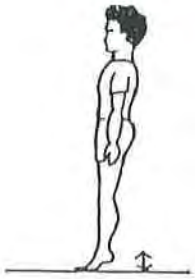
相反地，右脚也做五次。

10 Stand with hands on hips in a lunge position.

Keep both heels on the ground, with toes pointing forwards, and lean forwards slowly to stretch the calf muscles of the back leg.

Hold for 5–10 seconds. Repeat with different leg forward x 5–10 times.

两手插在臀部作要向前冲的姿势。保持脚跟贴地，脚指向前压地，身体倾向前，慢慢地伸张腿部肌肉停约５—１０分秒钟，然后身体倾向后，亦做五次。



11 Stand with feet slightly apart, hands on the hips.

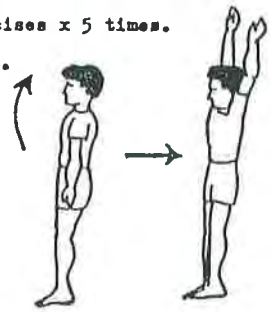
Raise body up on the toes, and then relax back onto the heels.

Repeat x 5–10 times (increase difficulty by holding longer in the raised position). (Variation with arms held forward at shoulder level).

两脚稍为分开站立，两手插在臀部。用脚指支持，提升身体，后又用脚跟支持站着。共做五次。（你可试当你站起来，用脚指支持时，站久一会儿）。

12 Deep breathing exercises x 5 times.

深呼吸运动，共五次。



TREATMENT OF CHRONIC HEART FAILURE

Omar B S T, MBBS (S'pore), MCGP (S'pore), FRACGP

INTRODUCTION

Heart failure, a commonly encountered problem in general practice, is defined as the inability of the heart to pump adequate amounts of blood to meet the requirements of the tissues. The past decade has seen considerable advances in the management of heart failure due to improved understanding of pathophysiology, better methods of assessment and the introduction of potent new therapeutic agents, particularly angiotensin-converting enzyme inhibitors (ACEIs). Application of modern medical treatment may now improve patients' comfort and mobility and also improve the poor prognosis of this condition.

ASSESSMENT AND MANAGEMENT

In general practice, the assessment of patients with heart failure is based on clinical criteria. A useful classification is that devised by the New York Heart Association (NYHA), which characterizes patients as having mild, moderate or severe heart failure according to their symptoms (Figure 1). The classification is helpful both in the assessment of the patient's response to treatment as well as indicating the likely prognosis of untreated disease. Thus, untreated NYHA class II and IV is associated with a mortality of approximately 20 and 50% per annum respectively.

Although there are many causes of heart failure (Figure 2), the principles of management are basically similar. Nevertheless treatment should be individualized according to response, periods of decompensation and progression of the disease. Optimum management demands: 1) removing the underlying cause where possible, 2) excluding, avoiding and correcting precipitating factors, 3) improving the cardiac performance and reducing the workload on the heart, and 4) eliminating salt and water retention. The major endpoints to be considered are : a) improvement of symptoms namely, fatigue, dyspnoea and oedema, and b) improvement in prognosis and therefore reduced mortality.

General advice is important and essential. Patients should be advised to maintain an optimal weight, avoid excessive salt intake and stop smoking. High alcohol intake should be avoided. Simple measures such as raising the head of the bed, and taking exercise more slowly and steadily, will help. It is important to remember that heart failure can be made worse by the effects of certain drugs. For example, corticosteroids, NSAIDs and liquorise derivatives cause sodium and water retention, beta-blockers, verapamil and disopyramide are negative inotropic drugs and they reduce cardiac output.

DRUG THERAPY

Drug treatment is aimed at the reversal of the three main pathophysiology abnormalities in heart failure: a) fluid retention b) pump failure, and c) Vasoconstriction (Figure 3). The main groups of drugs used in heart failure are: 1) diuretics 2) vasodilators, particularly ACEIs, and 3) positive inotropic agents.

Diuretics (Figure 4)

In spite of the advent of newer, more sophisticated drugs, diuretics remain the main

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Singapore 1646

treatment of heart failure. They are the first drugs to be used in all grades of heart failure. In milder disease, **thiazides** may be appropriate, but with increasing symptoms, more potent agents – the **loop diuretics** will be required. The promotion of salt and water excretion reduces plasma volume and intracardiac pressures (preload), correcting systemic and pulmonary congestion and improving exercise tolerance and peripheral oedema.

Thiazides are effective, and because of their slow action, work for longer, and with less dramatic effects than the loop diuretics. The latter are more powerful, but because they have a short intense period of action, they often cause problems. The elderly may find themselves prevented from leaving the house while the diuresis is working, or may even be 'caught short' if they are not near a lavatory. It is important, where possible, to modify the dose schedule to suit the patient's activities.

Thiazide and loop diuretics causes a fall in serum potassium levels. However, for most of the population, whose intake of potassium is adequate, the hypokalaemia may be more of an incidental laboratory finding than an important factor in treatment. But oral potassium supplements should be given to those patients in whom even mild hypokalaemia could be dangerous, e.g. those with chronic liver disease or those being treated with digoxin, or drugs known to cause potassium loss e.g. corticosteroids and liquorice derivatives (carbenoxolone).

Heart failure which is refractory to increasing dosages of loop diuretics may improve with the addition of a **potassium-sparing diuretics** such as amiloride of triamterene. Although weak diuretics in their own right, they are useful alternative to oral potassium supplements which could otherwise be necessary. Spironolactone, another potassium-sparing diuretic, antagonizes adosterone and is particularly useful when features of right heart failure prevail. **Combination diuretics** containing a potassium-sparing agent with a thiazide (e.g. amiloride with hydorchlorthiazide – Moduretic®, triamterene with hydrochlorthiazide – Dyaside®) or with a loop diuretic (e.g. amiloride with frusemide – Frumil®) are useful in heart failure where hypokalaemia is particularly likely to induce arrhythmias. Combination tablets may improve compliance but as with all diuretics serum urea and electrolytes should be carefully monitored. When there is renal impairment potassium-sparing diuretics are best avoided.

In chronic usage, diuretics do not improve cardiac output and may in high doses reduce output by causing excessive shrinkage of the vascular volume and reduction of venous return (preload). This overdiuresis must be avoided, especially in the elderly, as it may cause progressive renal impairment and further stimulation of the renin - angiotensin system, with loss of effectiveness of the diuretic.

Monitoring of response is important and can be difficult on an outpatient basis. However, examination of weight records, the jugular venous pulse, lung bases, the hepatojugular reflex, the periphery and renal function provide valuable information and can be used as a guide in adjusting dosage to achieve maximum benefit without causing a fall in cardiac output.

Vasodilators (Figure 5)

Vasolidator agents achieve some of the aims of drug therapy in congestive heart failure by relaxing vascular smooth muscle. This leads to arteriolar or venous dilatation, or both, thereby improving symptoms of congestion by reducing preload. They also improve cardiac function by reducing afterload and impedance, resulting in improved ventricular emptying and cardiac output.

Vasodilators are potentially beneficial in heart failure due to aortic or mitral regurgitation ischaemic heart disease, cardiomyopathy and ventricular septal defect. They are of little benefit, and may be deleterious, in valvular stenosis unless there is associated severe ventricular dysfunction with dilatation.

Long-acting **nitrates** (e.g. isosorbide dinitrate) reduce effort dyspnoea and may be useful in heart failure secondary to ischaemic heart disease. However, clinical benefit is modest and other treatment is usually necessary. Long term therapy may be limited by tolerance but this is more common with **hydralazine** and **prazosin** (both of which have declining roles in the management of

heart failure). When used alone, hydralazine has not been demonstrated to increase exercise test over placebo. But combination of hydralazine with nitrates has been demonstrated to prolong life in patients with moderate heart failure. Prazosin has been shown to have beneficial effects in the acute phase of congestive heart failure. **Calcium-channel blockers** with systematic vasolidator properties (e.g. nifidipine) may be beneficial in congestive heart failure particularly due to ischaemic or myopathic process. However, clinical benefit usually does not persist for more than a few days.

ACEIs

ACEIs(e.g. captopril, analapril, lisnopril, quinapril, perindopril), offer the best orally effective combination of venous and arteriolar dilation. Their mode of action is by inhibition of formation of angiotensin, a potent vascontrictor produced in excessive amounts in heart failure. In addition, ACE inhibition reduces the secondary overproduction of aldosterone, thus reducing salt and water retention. Initially, they were used only for the severe forms of heart failure (NYHA IV), but with increasing experience they are now advocated in patients with moderate disease (NYHA II-III). Large trials are currently being undertaken to determine the efficacy of ACEIs in mild or very early heart failure (NYHA I).

ACEIs form the second line of treatment when diuretics are insufficient to control symptoms. Addition of ACEIs should be considered in patients who are still symptomatic on the equivalent of 80mg of frusemide or more, before evidence of dehydration or impaired renal function is apparent. ACEIs relieve shortness of breath in heart failure and increase exercise and functional capacity. The improvement in haemodynamics remains for longer than during treatment with conventional vasodilators such as prazosin – probably because volume expansion is prevented. In addition, ACEIs have been shown to prolong survival in patients with severe heart failure (NYHA IV). It has been suggested that ACEIs might delay the onset of severe symptoms if used much earlier in patients with cardiac muscle disease. Very little evidence supports this suggestion, and they are certainly no substitute for diuretics in the management of symptomatic patients.

ACEIs can exacerbate renal impairment and plasma potassium can increase. Thus potassium supplements should be stopped and potassium-sparing diuretics should not be used in conjunction with ACEIs. In some patients, the first dose may lower the cardiac output dramatically causing fainting. In severely diuresed patients, the drug should be started in low doses, omitting the morning dose of diuretics. The dose of ACEI can be increased slowly over the following days.

Hypotension following the first dose of ACEI is more common in patients with heart failure than in those with hypertension. It cannot be predicted who will develop this reaction, but hyponatraemic patients, those with renal insufficiency, the elderly, and those who have been receiving large doses of diuretics are particularly liable. Patients with heart failure, due to valvular steriosis and those with a systolic blood pressure below 100 mm Hg should not be prescribed ACEIs. Patients with cor pulmonale often respond poorly to ACEIs.

Positive inotropic agents

These drugs would obviously be useful in heart failure, but until recently only **digoxin** has been available for chronic oral therapy in heart failure, but the long term effectiveness of digoxin has been disputed. Clinical trials, which have either studied prospectively the effects of digoxin versus placebo or evaluated withdrawal of the drugs from chronically treated patients in general practice, have failed to substantiate a longer term beneficial effect in heart failure patients in sinus rhythm. However, the importance of digoxin in the control of atrial fibrillation and associated heart failure remains unchallenged. It should be noted that digoxin is generally not recommended for heart failure due to acute myocardial infraction, cor pulmonale, mitral stenosis with sinus rhythm, and hypertropic obstructive cardiomyopathy.

Digoxin has a very low therapeutic index and great care must be taken to find the correct dose for the individual patient. Dosage may have to be reduced considerably in the elderly or if there is significant renal impairment. The narrow therapeutic - toxic ratio should also be noted and considered when prescribing other medication,

e.g. quinidine, verapamil, amiodarone and NSAIDs, which have been known to increase serum digoxin levels and predispose to toxicity.

The classical adverse effects of digoxin are well known (Figure 6). However, it must be remembered that the most common warning sign of impending toxicity remains nausea and vomiting. The adverse effects of digoxin are enhanced by hypokalaemia, hypoxia and acidosis, and hypothyroidism, and they occur more commonly in the elderly.

Recently, a non-glycoside, orally effective positive inotropic agent has become available. **Xamoterol** is a beta-selective partial agonist – i.e. a combined agonist/antagonist. As such it improves both contraction and relaxation of the heart without affecting the peripheral circultation or producing tachycardia, with consequent improvement in exercise tolerance and fatigue in heart failure patients.

However, as a partial agonist, it will also have antogonist properties in situations of high sympathetic drive, for example, in more severe heart failure (NYHA III, IV), and under these circumstances may make failure worse. Early clinical experience in patients with mild heart failure is, however, promising, and it may represent a useful alternative to increasing diuretic dose or adding a vasodilating agent in this group of patients. Xamoterol is contraindicated in patients with moderate to severe heart failure.

FOLLOW-UP

As heart failure is a condition that will probably worsen over time, regular observation of the patients is important. Follow-up should be done monthly, reducing to 3-monthly when the patient's condition is stable. Symptoms, weight and renal function should all be checked, significant deteriorations noted and medications changed accordingly. Life-style monitoring, patients education, and discussion of the patient's fears and anxieties should be carried out during follow-ups.

REFERRAL TO HOSPITAL

For many patients with heart failure, the diagnosis and treatment of their condition can be carried out effectively in general practice. Indeed the rapport that is established between a general practitioner and his or her patient enables a continuity of care that can never be mimicked in the hospital. The threshold for referral of the adult patient to hospital depends on the age of the patient, the underlying pathology, the severity of symptoms and the response to conventional treatment.

The younger patient should undergo thorough investigation to provide an accurate prognosis and to exclude any potentially remediable cause or aggravating factors. If valve disease is suspected, referral must be considered early even in elderly patients, since in otherwise fit individuals a good result may be anticipated from valve surgery. Another indication for referral is when specialised investigations (e.g. echocardiography may be required to elucidate the underlying pathology. Refractory heart failure, in spite of what appears to be adequate therapy, is yet another reason for referral.

CARDIAC TRANSPLANTATION

Patients with advanced cardiac failure have a poor prognosis and their quality of life is diminished. Cardiac transplantation is rapidly becoming established as an effective treatment for these individuals and probably offers the only realistic hope for an improved prognosis.

CONCLUSION

Congestive heart failure is a common clinical syndrome and a pathophysiologic entity of considerable heterogeneity. Reflection on basic mechanisms in cardiac failure gives an insight into the modes of therapy available in management. Therapeutic options in the past decade expanded markedly with enhanced quality and quantity of life. With end stage disease the option of a heart transplant is providing quality of life to increasing numbers of selected patients.

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MULTIPLE CHOICE QUESTIONS

1. Digoxin:
   A. intestinal absorption is impaired by kaolin-pectin mixtures.
   B. produces a diminishing response when given over a long period.
   C. toxicity is more likely in patients with renal impairment.
   D. gastrointestinal upset may be an early warning symptom of toxicity
   E. renal excretion may be impaired by NSAIDs.

2. Which of the following statements about diuretics is/are correct?
   A. therapeutic drug level monitoring is useful.
   B. frusemide is potent long-acting diuretic.
   C. spirolonactone antagonizes the effect of frusemide if the two drugs are used in combination.
   D. thiazides may precipitate gout.
   E. potassium supplements should always be given with thiazedes.

3. Spironolactone:
   A. is a weaker diuretic than hydrochlorothiazide.
   B. is a competitive aldosterone antagonist.
   C. is particularly useful in combination with ACE inhibitors.
   D. has a slow onset but prolonged action.
   E. is well recognised as a precipitant of diabetes mellitus.

4. The following are correct observations about the use of vasodilators in heart failure:
   A. nitrates act on the arterial system
   B. venodilators reduce venous return to the heart and thus reduce cardiac preload
   C. addition of ACE inhibitors should be considered in patients who are still symptomatic on the equivalent of 80 mg of frusemide or more.
   D. vasodilator therapy is not useful if the patients is already receiving digoxin.
   E. ACE inhibitors have useful vasodilator effects on both arteries and veins.

5. In the treatment of heart failure:
   A. diuretics are not useful in severe cases
   B. ACE inhibitors are not to be used in combination with thizadies.
   C. potassium supplements are necessary when ACE inhibitors are used.
   D. digoxin is contraindicated in the elderly.
   E. chronic use of diuretics in high doses may reduce cardiac output.

ANSWERS

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

Figure 1 New York Heart Association functional classification of heart failure

Class I
Patient with cardiac disease but without limitation of normal physical activity (i.e. no dyspnoea, fatigue or chest pain)

Class II
Slight limitation of activity; comfortable at rest but normal physical activity results in fatigue, dyspnoea or chest pain.

Class III
Marked limitation of activity; comfortable at rest but fatigue, dyspnoea or chest pain induced by less than normal level of exertion.

Class IV
Symptoms of dyspnoea or fatigue present at rest, with marked exacerbation by any degree of physical activity.

Figure 2 Causes of heart failure

Low or normal cardiac output
Ischaemic heart disease
Hypertension heart disease
Valvular heart disease
Cardiomyopathy
Congenital heart disease
Pericardial disease

Normal or high cardiac output
Thyrotoxicosis
Severe, anaemia
Paget's disease

Figure 3 Pathophysiology of heart failure, and sites of action of the commonly used drugs. (After Grahame-Smith and Aronson. Oxford textbook of clinical pharmacology and drug therapy. Oxford University Press, 1984)

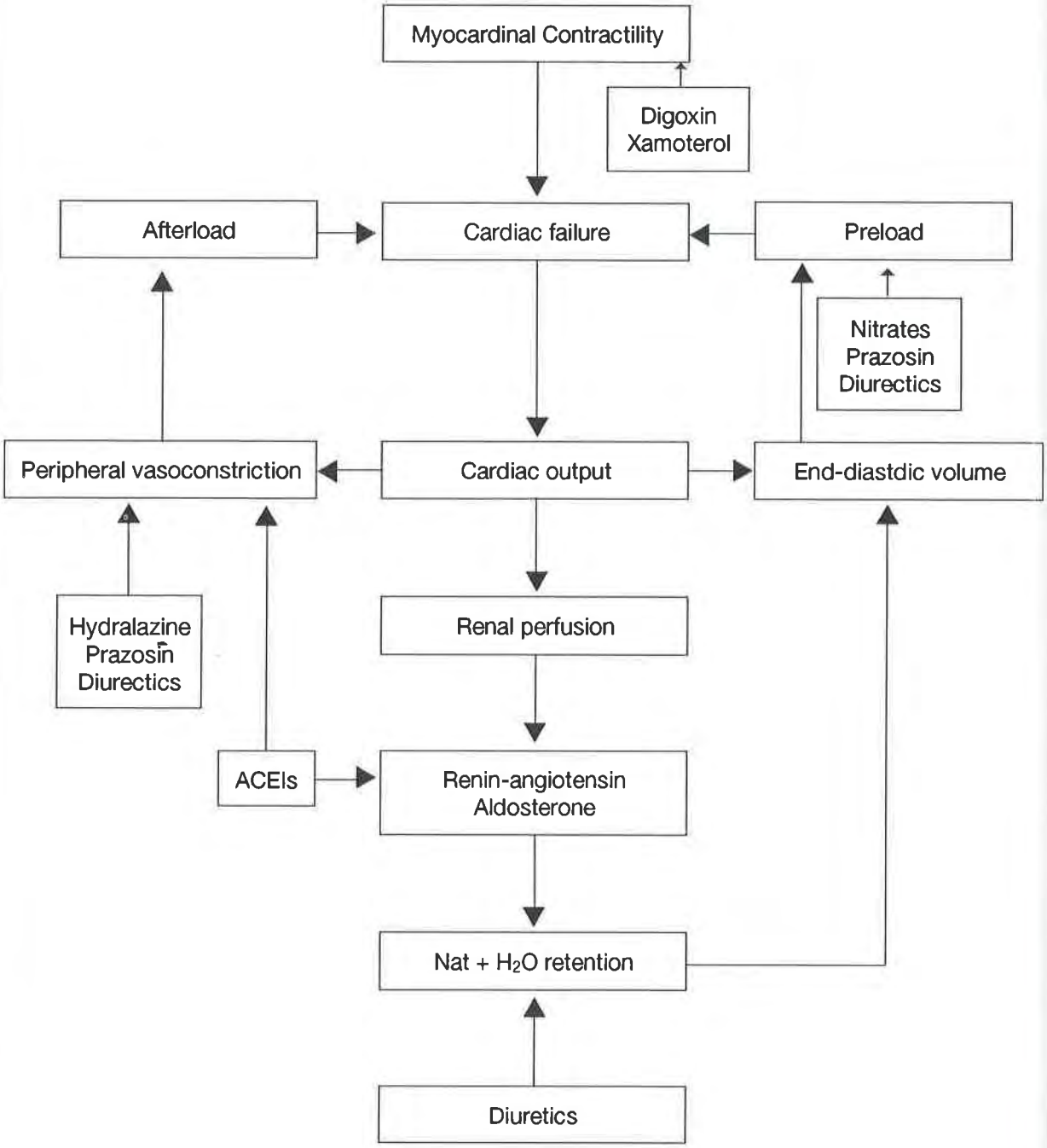


Figure 4 Characteristics of commonly used diuretics in congestive heart failure.

Drug	Site of action	Onset of effect (hours)	Peak effect (hours	Duration (hours)	Dosage range (mg/day)
Loop diuretics (potent)					
Frusemide	Loop of Henle	oral 1 IV 5 min	1–2 30 min	6 3	20–160
Bumetanide	Loop of Henle	oral 30 min IV 5 min	1–2 30 min	2–6 2	0.5–3
Thiazides (moderate potency)					
Chlorothiaziade	Distal tubule	1	4	6–12	500–1000
Hydrochlorothiazide	Distal tubule	1	4–8	12–18	25–100
Potassium-sparing diuretics (weak)					
Amiloride	Distal tubule	2	4–8	12–24	5–10
Triamterene	Distal tubule	2	6–8	12–16	50–200
Spironolactone	Distal tubule	gradual	1–2 days after initiation	2–3 days after cessation	25–200

Figure 5 Some oral vasodilators used in congestive heart failure.

Drug	Predominant effect	Mechanism of action
ACEIs (eg. captopril, enalapril, lisnopril quinapril)	Arteriolar & venous dilation	Interruption of renin-angiotensin-aldosterone system
Nitrates (eg. isos-orbride, dinitrate	Venous dilation	Unknown
Calcium-channel blockers (nifidipine)	Arteriolar dilation	Calcium entry blocker
Prazosin	Arteroilar & venous dilation	alphai-adrenoreceptor antagonist
Hydralazine	Arteriolar dilation	Direct smooth muscle

Figure 6 Major adverse effects of drugs used in heart failure.

DIURETICS

Thiazide and loop diuretics

dehydration, hypokalaemia, impaired glucose tolerance, hyperuricaemia

Potassium-sparing diuretics

hyperkalaemia, renal impairment, gynaecomastia (with spirolonactone)

VASODILATORS

Nitrates

hypotension, headaches

Calcium-channel blockers

hypotension, headaches, ankle oedema

Hydralazine

lupus-like syndrome

Prazosin

hypotension (especially after the first dose)

Angiotensin-converting enzyme inhibitors

first dose hypotension, angioneurotic oedema, cough, skin rashes, proteinuira, neutropenia, taste distrubances

INOTROPIC AGENTS

Digoxin

anorexia, nausea, vomiting diarrhoea, confusion, visual distrubances, cardiac arrhythmias, heart block

Xamoterol

GIT disturbances, headaches, dizziness, chest pain, palpitations, muscle-cramps, rashes

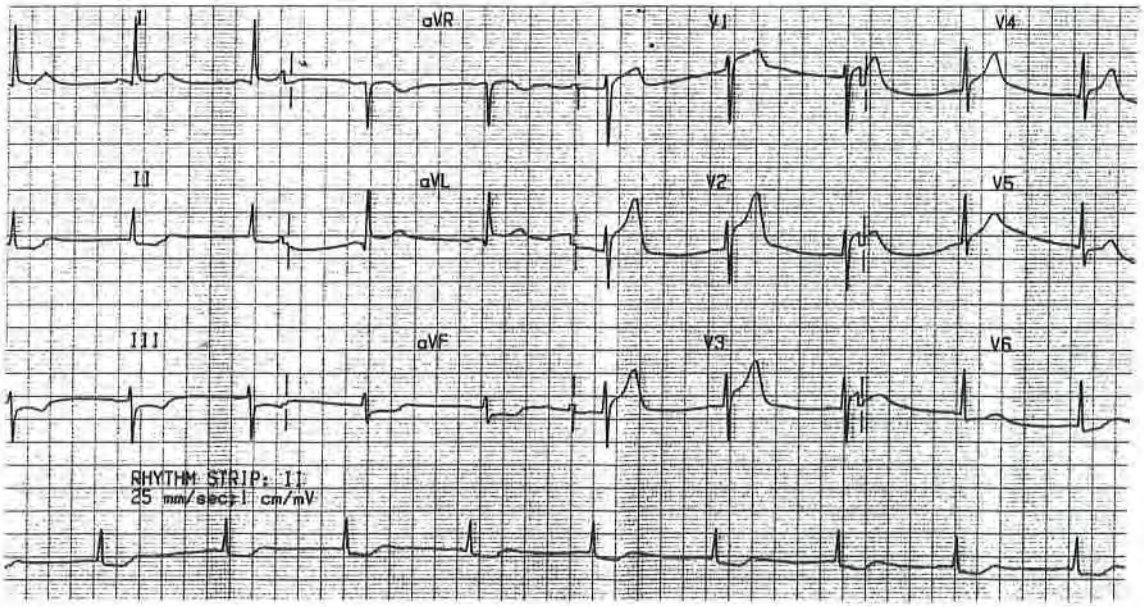
ECG QUIZ

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

The ECG shown below belongs to a 56 year old Chinese male who presented with retrosternal pain for 1/2 hr. When seen in the emergency room he was in severe pain and very diaphoretic.

(1) What are the ECG abnormalities and what do you think is the clinical diagnosis?

(2) How would you manage this paitent?



ANSWERS TO ECG QUIZ

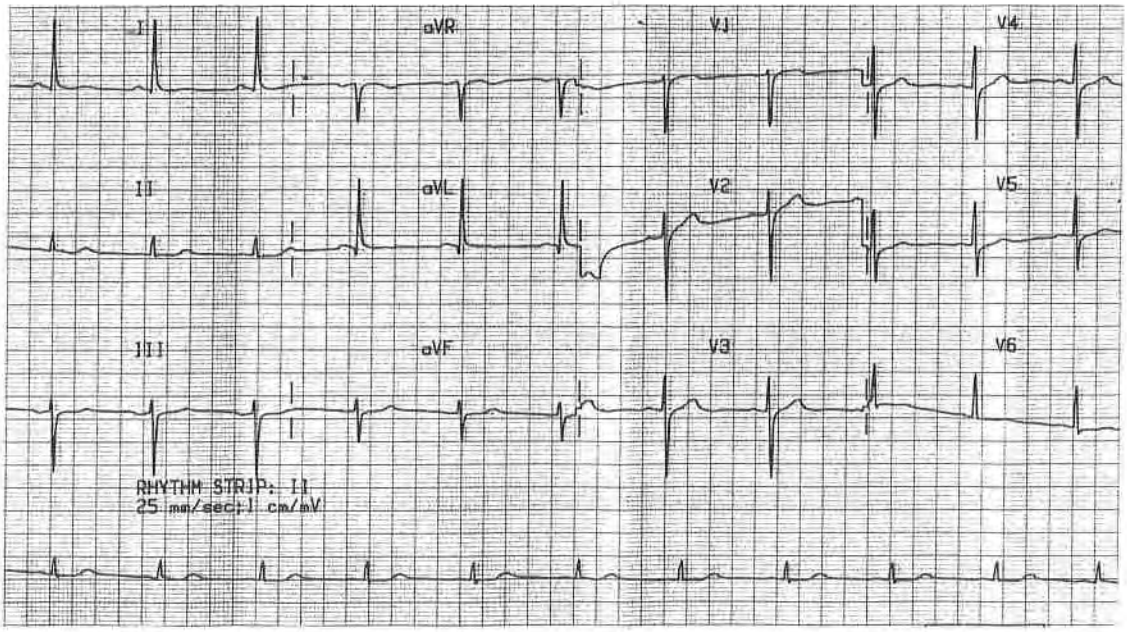
(1) The ECG shows ST elevation in Leads I, aVL and VI to V4. Reciprocal ST depression is seen in II, III, aVF. These are hyperacute changes of an involving acute myocardial infarction.

This patient smoked 20 cig./day but had no other coronary risk factors.

(2) The patient was assessed very quickly and contraindications to thrombolytic therapy excluded. He was started on an infusion of 1.5 million units of Streptokinase and admitted to the CCU. He was given IV fluids, sublingual GTN and IV Morphine sulphate in addition to other medication. The paitent became pain free soon after completion of Streptokinase and the ECG changes reverted to a near normal state. (See tracing below.)

He was maintained on IV Herapin for 48 hr and had coronary angiography within a few days. This revealed a long 95% proximal left anterior descending artery stenosis. He also had other less critical lesions in the Left circumflex and Rt. coronary arteries. Coronary artery bypass graft surgery was subsequently done.

This case illustrates the adage that "Time is muscle". Early thromboloytic therapy made a significant contribution to saving myoccardium and thereby significantly influencing the final outcome.



WORLD HEALTH ORGANIZATION PUBLICATIONS

NEW BOOK ANNOUNCEMENT

DIET, NUTRITION, AND THE PREVENTION OF CHRONIC DISEASES

This book records the consensus reached by a group of experts commissioned to examine the strength of evidence linking dietary factors to the development of several chronic diseases, including coronary heart disease, hypertension, stroke, cancer, diabetes, and osteoporosis. The experts were also asked to issue advice on prevention consistent with both the strength of scientific evidence and the magnitude of health problems associated with dietary factors. Although emphasis is placed on diet-related chronic diseases, the persistent problem of nutrient deficiencies is also considered.

The opening sections assess epidemiologial data linking changing patterns of disease to changes in diet and summarize what is known about the place of specific nutrients and dietary factors in the etiology of chronic diseases. The experts conclude that repeated and consistent findings of an association between specific dietary factors and a disease suggest that such associations are "real and indicative of a cause-and-effect relationship". The report also sounds the alarm concerning the consequences of dietary changes in developing countries, which are now experiencing a universal and spontaneous shift towards the "affluent" diet. By the end of this century, the report concludes, cardiovascular disease and cancer will be established as major health problems in virtually every country in the world.

Having confirmed the link between dietary factors and disease and assessed the magnitude of the problem, the report turns to the question of prevention.

In one of its key achievements, the report issues a series of "population nutrient goals", put forward as a universal guide to the nutrient intakes needed to prevent all diet-related diseases and appropriate for application in all countries throughout the world. Lower and upper intakes are set for each of the main nutrient groups, including total fat, saturated fatty acids, polyunsaturated fatty acids, protein, total carbohydrates, complex carbohydrates, and free sugars. Expressed as a proportion of total energy, this recommended "safe" range of intakes specifies the minimum intake of a nutrient needed to prevent deficiency diseases and the maximum intake that should not be exceeded in the interest of preventing chronic diseases. Recommended daily intakes, expressed in grams, are also issued for salt, dietary fibre, dietary cholesterol, and fruits and vegetables.

The report is explicit in its insistence on the need for a population-wide, as opposed to individualized, approach to the prevention of diet-related chronic diseases, arguing that the entire population of most affluent countries shows a high risk profile and that intervention on a mass scale is needed to shift dietary patterns closer to the "safe" range of intakes specified in the report.

The concluding sections, devoted to food policies, explain why current policies governing food production and supply are essentially agricultural policies driven by the economic and political issues of food availability, food security, and the security of food producers. A review of the evolution of these policies, most of which were formulated in the 1940s, reveals roots in nutritional concepts based on the need to prevent deficiency diseases and thus ill-equipped to protect populations from the many diseases now linked to nutrient excesses. Although extensive advice on the adaptation of these policies is given, the report notes that the new nutritional objectives of preventing both the deficiency diseases and the chronic diseases will have immense implications for the economics of farming, for government, industrial and social policies, and for international trade, and can thus be expected to meet with considerable opposition.

Diet, Nutrition and the Prevention of Chronic Diseases
Technical Report Series, No. 797
1990, 203 pages (available in English and French; Spanish edition in preparation)
ISBN 92 4 120797 3
Sw.fr. 26.–/US $20.80; in developing countries: Sw.fr. 18.20
Order no. 1100797

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NEW BOOK ANNOUNCEMENT

ACHIEVING HEALTH FOR ALL BY THE YEAR 2000

MIDWAY REPORTS OF COUNTRY EXPERIENCES

edited by *E. Tarimo* and *A. Creese*
1990, v + 262 pages (available in English; French and Spanish in preparation)
ISBN 92 4 156132 7
Sw.fr. 46.–/US $36.80
In developing countries: Sw.fr. 32.20
Order no. 1150341

This book explores the extent to which the principles of primary health care, mapped out at the Alma-Ata Conference in 1978, have had a real impact on health care in different parts of the world. While acknowledging the importance of changes in health policy and the structure of services, the book makes a special effort to uncover what has actually happened in the human setting where both the ambitions of primary health care and the true measures of success reside.

The book consists of 15 country reports authored by public health experts having first-hand knowledge of experiences in the country. Written in a spirit of frank self-assessment, these country profiles illustrate the diversity of ways in which the principles of primary health care have been put to work in rich and poor countries alike. Reports from Canada, Finland, Hungary, and the Netherlands demonstrate the applicability of primary health care in industrialized countries, whether to contain health costs, reach the underserved, reduce dependence on the hospital system, or reap the social and economic benefits of preventive medicine. These reports also identify some of the mechanisms by which the impetus for better health care can move from public pressure to the political agenda and finally to legislative action.

A completely different set of challenges emerges in reports from Burkina Faso, Ethiopia, Mozambique, Nigeria, and Papua New Guinea, where priorities remain defined by the need for essential health care and obstacles range from high rates of illiteracy and severe poverty to terrorist attacks on health posts and the persistence of life-threatening traditional remedies. Under such conditions, documented improvements in health take on particular significance as lessons in the use of simple, inexpensive technologies to stretch resources and extend health coverage despite highly unfavourable conditions. These reports also confirm the effectiveness of initiatives at the district level, though the reformulation of national health policies and priorities has also been important.

A frank documentation of setbacks and mistakes characterizes the report from Egypt, where efforts to improve public health are thwarted by a severe financial crisis, shrinking health budget, unbridled population growth, fragmented health services, weak management, and a chanelling of scarce funds into the construction of new hospitals. The most heartening reports come from China, Indonesia, Malaysia, Sri Lanka, and Thailand. Facts and figures reveal dramatic improvements in health, while illustrative examples and anecdotes are used to analyse what has happened in the human setting and why. These reports confirm the power of the primary health care approach to achieve better health, whether measured in terms of extended life expectancy or a simple increase in the number of telephones and vehicles in the rural health network. These reports are equally a record of the power of simple innovations — a cooking competition to teach mothers about nutrition, a share-holding scheme for funding essential drugs — to reach people and make a life-and-death difference in the world.

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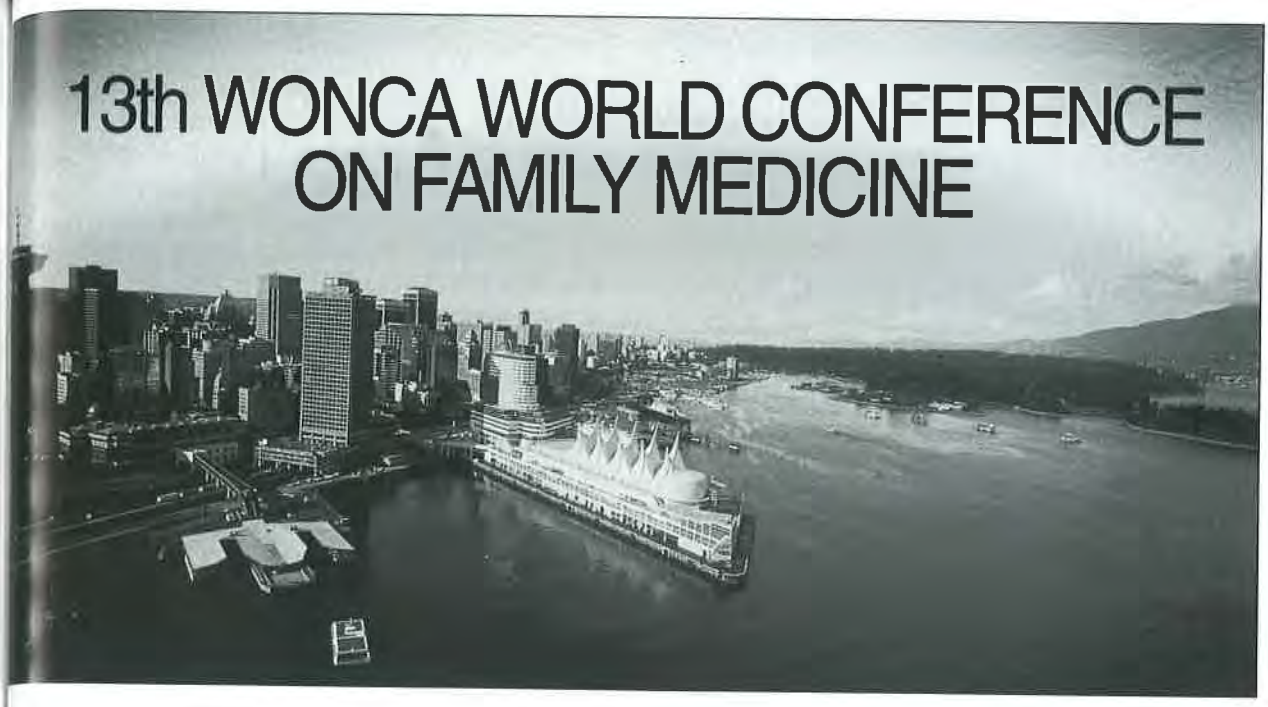

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  Type throughout in upper and lower case, using double spacing, with three centimetre margins all round. Number every page on the upper right hand corner, beginning with the title page as 1. Make all necessary corrections before submitting the final typescript.

  Headings and subheadings may be used in the text. Indicate the former by capitals, the latter in upper and lower case underlined.

  Arrange the manuscript in this order: (1) title page, (2) summary, (3) text, (4) references (5) tables, and (6) illustrations.
* Send three copies of all elements of the article: summary, text, references, tables and illustrations. The author should retain a personal copy.

The title page

* The title should be short and clear.
* Include on the title page first name, qualifications, present appointments, type and place of practice of each contributor.
* Include name, address and telephone number of the author to whom correspondence should be sent.
* Insert at the bottom: name and address of institution from which the work originated.

The summary

* The summary should describe why the article was written and give the main argument or findings.
* Limit words as follows: 100 words for major articles; 50 words for case reports.
* Add at end of summary: an alphabet listing of up to 8 keywords which are useful for article indexing and retrieval.

The text

The text should have the following sequence:

* Introduction: State clearly the purpose of the article.
* Materials and methods: Describe the selection of the subjects clearly. Give references to established methods, including statistical methods; provide references and brief descriptions of methods that have been published but are not well known. Describe new or substantially modified methods, giving reasons for using them and evaluate their limitations. Include numbers of observations and the statistical significance of the findings were appropriate.

  Drugs must be referred to generically; all the usual trade names may be included in parentheses. Dosages should be quoted in metric units.

  Laboratory values should be in SI units with traditional units in parentheses.

  Do not use patient's names, initials or hospital numbers.

* Results: Present results in logical sequence in the text, tables and illustrations.
* Discussions: Emphasise the new and important aspects of the research and the conclusions that follow from them. Indicate the implications of the findings and limitations. Relate the observations to other relevant studies.

Illustrations

* Diagrams, line drawings, photographs or flow charts are valuable but their use will be subject to editorial policy. Transparencies or prints are acceptable for colour reproduction at the authors' expense.
* Each illustration must carry its appropriate Figure number and the top should be clearly labelled.
* Figure legends, typed (double-spaced) and each on a separate page should be no more than 45 words.

Tables

* Any table must supplement the text without duplicating it.
* Each should be numbered, typed on a separate sheet with an appropriate title.

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References

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References should be double spaced and arranged alphabetically by author. Personal communications are not acceptable as references. Unpublished material should be included only if an address can be given from which a copy of the material cited is available.

Authors are responsible for accuracy of references, which should conform to the Vancouver style (see Further reading). List all authors (include all initials) when there are six or fewer; when seven or more list the first three and add et al. Give the title of the paper cited in full, the title of the journal abbreviated according to Index Medicus (if not listed by Index Medicus spell in full); the year; the volume number and the first and last page number of the article.

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Further reading

1. INTERNATIONAL COMMITTEE OF MEDICAL JOURNAL EDITORS. Uniform requirements for manuscripts submitted to biomedical journals. Ann Intern Med 1988; 108: 258-265.
2. Bailar III JC and Mosteller F. Guidelines for Statistical Reporting in Articles for Medical Journals. Ann Intern Med 1988; 108: 266-273.

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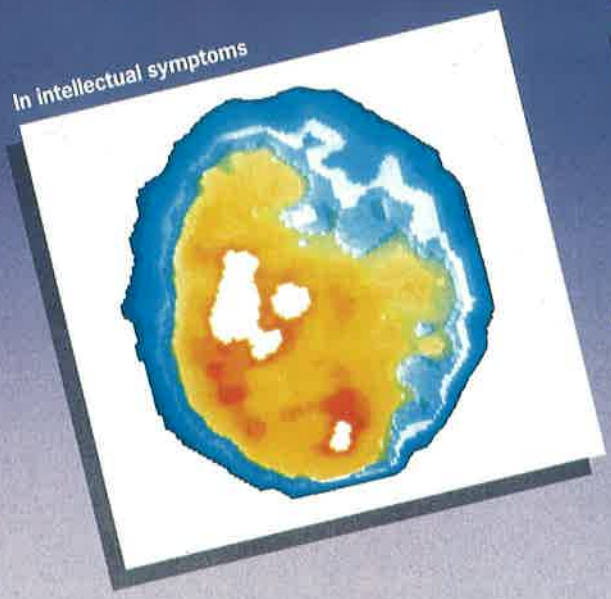
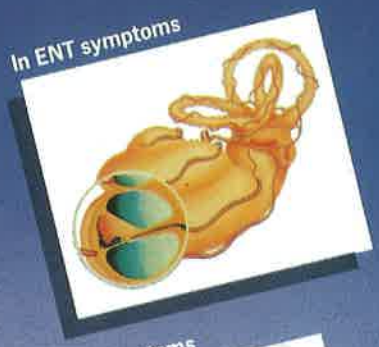
Relief through oxygen





Duxaril®

Almitrine + Raubasine

2 tablets daily

brings more oxygen to the brain and neurosensory tissue

• Duxaril relieves the patient's symptoms

Duxaril markedly relieves the symptoms of cerebral insufficiency:
- Intellectual symptoms: Duxaril significantly improves vigilance, concentration, memory, learning and cognitive thinking (1);
- ENT symptoms: Duxaril rapidly relieves vertigo and tinnitus, and markedly improves hearing (2);
- Visual symptoms: Duxaril significantly improves near and distant visual acuity, the visual field, and colour vision (3).

• Duxaril improves cerebral oxygenation

Duxaril brings more oxygen to the brain and neurosensory organs.
Duxaril increases the arterial oxygen content by improving the function of the alveolocapillary exchange system without affecting the respiratory rate (4).
Duxaril raises arterial oxygen saturation and promotes cerebral aerobic metabolism (4,5). The increase in cerebral oxygenation, measured in the cerebral cortex by Plotkine's method, is of the order of 40 to 50% (5).
Duxaril therefore revitalizes the cells essential to intellectual and neurosensory function.

(1) Berthaux P. Laforestrie R. Presse Med. 1983; 12 (13): 852-855.
(2) Bordes L.R. Cahiers ORL 1983; 18 (5): 403-410.
(3) Bourgeois H., Huray A., Moccatti D., Ophtal Fr. 1983; 39: 23-36
(4) Sancier A., Manelfe C., Gaz Med France 1980; 87, 28 (suppl.): 16-22
(5) Charpentier J. Le Praticien 1980; 372: 15-21.

Composition: almitrine bismesylate 30 mg, raubasine 10 mg, per tablet. **Presentation:** Box of 30 tablets. **Indications:** Cerebral insufficiency, characterized by intellectual impairment (disorders of memory, concentration, etc.). Auditory, vestibular, and visual disorders of ischemic etiology. Used also in functional rehabilitation after cerebrovascular accidents. **Contraindications:** Not to be prescribed in combination with MAOIs. **Precautions:** The use of Duxaril is not recommended in women during pregnancy, and particularly not during the first three months, unless, in the opinion of the doctor, the therapeutic advantages outweigh any potential risk. In no case can Duxaril replace specific antihypertensive therapy in the hypertensive patient. Avoid concomitant use of other drugs containing almitrine. Formication, prickling, or numbness in the lower limbs have been reported during long-term treatments. These manifestations, which are very rare, should lead to treatment withdrawal. **Adverse effects:** Mild gastrointestinal disorders, sleep disturbances, palpitations, anxiety, dizziness. Rarely, loss of weight. **Overdosage:** Signs of massive voluntary or accidental overdosage include: tachycardia, arterial hypotension, polypnea, and respiratory alkalosis. Treatment should consist of gastric lavage and symptomatic supportive measures: the subject should be placed under close cardiorespiratory observation with repeated monitoring of blood gases. *Refer to data sheet for complete prescribing information.*

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Gastrointestinal Motility Regulator

CEREKINON® Tablets

(Trimebutine maleate)

Brand Name

CEREKINON® Tablets

Generic Name

Trimebutine maleate

Chemical Name

(±) 2-Dimethylamino-2-phenylbutyl 3, 4, 5-trimethoxybenzoate hydrogen maleate

Description

	Content of Trimebutine maleate
CEREKINON Tablets	100 mg/tablet

Indication

Gastrointestinal symptoms associated with chronic gastritis (feeling of abdominal distention, abdominal pain, nausea and eructation)
Irritable bowel syndrome

Administration & dosage

For gastrointestinal symptoms due to chronic gastritis:
Usually, for adults daily dose of 300 mg as trimebutine maleate (3 tablets) in 3 divided doses through oral route The dose may be increased or decreased according to the age and symptom of patients
For irritable bowel syndrome:
Usually, for adults daily dose of 300 — 600 mg as trimebutine maleate (3 — 6 tablets) in 3 divided doses through oral route

Clinical application

1. **Clinical effects**
   1) Chronic gastritis
   As the results of clinical trials including two comparative double-blind studies, CEREKINON Tablets showed overall improvement rate of 64 1% (including moderate improvement) in the treatment of gastrointestinal symptoms (feeling of abdominal distention, abdominal pain, nausea, and eructation) due to chronic gastritis (526 cases)
   2) Irritable bowel syndrome
   As the results of clinical trials including two comparative double-blind studies, CEREKINON Tablets showed overall improvement rate of 56 5% (including moderate improvement) in the treatment of abnormal stool and gastrointestinal symptoms due to irritable bowel syndrome (642 cases)
2. **Adverse reactions**
   Adverse reaction episodes of 259 cases (0 4%) were reported out of total 59,801 cases The most often encountered adverse reactions were gastrointestinal symptoms such as diarrhea, constipation and thirst

Precaution

1. **Adverse reactions**
   a) Gastrointestinal system
   Constipation, diarrhea, borborygmus, thirst, numbness in the mouth may occur rarely
   b) Cardiovascular system:
   Palpitation may occur rarely.
   c) Psychoneurotic system:
   Sleepiness, dizziness, lassitude, headache, may occur rarely
   d) Hypersensitivity:
   Hypersensitive symptoms such as eruption may occur infrequently In such case, medication should be discontinued
2. **Medication to pregnant women and nursing mothers**
   The safety of CEREKINON Tablets in pregnancy or nursing women has not been established Therefore, CEREKINON Tablets should not be given to pregnant women, women suspected of being pregnant and nursing women, unless the potential benefits outweigh the possible risks
3. **Medication to children**
   The safety of CEREKINON in children has not been established

Handling

1. **Caution:** Dispense by physician's prescription
   Keep out of reach of children
2. **Storage:** Store in a light-resistant tight container below 30°C
3. **Expiry date:** Indicated on the outer package

How supplied

CEREKINON Tablets: 100 tablets (10 tablets × 10) P.T P.

Further information is available on request.

Distributor:

WALETA SINGAPORE PTE LTD
63 Hillview Avenue #08-12
Lam Soon Industrial Building Singapore 2366
Tel : 7603833 (8 Lines) Fax : (65) 7625036
Tlx : RS 56043 WALETA

TANABE SEIYAKU CO., LTD.
2-10, Dosho-machi 3-chome, Chuo-ku, Osaka, Japan.

P-CEK-893 (H)

ONE-A-DAY
Oruvail 200
Controlled release ketoprofen

The power to bring stiff and painful joints to life

Oruvail Prescribing Information:
Indications: Rheumatological disorders including osteoarthritis, ankylosing spondylitis, acute musculoskeletal conditions, dysmenorrhoea. **Dosage:** Adults: 200mg once daily, with food. Children: Not established. Elderly: Start at 100mg once daily and maintain on lowest effective dose. **Contraindications:** Active peptic ulceration; history of recurrent peptic ulceration or chronic dyspepsia; severe renal dysfunction; known hypersensitivity to ketoprofen or aspirin or other NSAIDs; bronchial asthma or allergic disease. **Precautions:** Use with caution in patients with renal impairment. Pregnancy and lactation; Avoid ketoprofen in pregnancy unless considered essential. Trace amounts are excreted in breast milk, therefore avoid use of ketoprofen unless considered essential. **Interactions:** If used with other protein binding drugs, a dosage reduction of these may be necessary. Aspirin or other NSAIDs should not be administered with ketoprofen. Serious interactions have been recorded after the use of high dose methotrexate with NSAIDs including ketoprofen. **Adverse effects:** Gastrointestinal intolerance, headache, mood change, insomnia, occasional peptic ulceration or haemorrhage or perforation, dizziness, mild confusion, vertigo, drowsiness, haematological reactions including thrombocytopenia, hepatic or renal damage, dermatological reactions, bronchospasm, anaphylaxis.

Further information available on request

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