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- Confident Eye Examination
- The Red Eye
- The Eye in Systemic Diseases
- Excimer Laser Treatment

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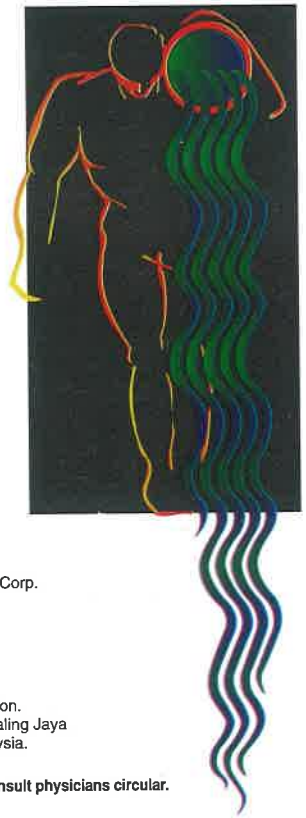

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1. Anderson J T et al: Can Finasteride reverse the progress of BPH? A two year placebo-controlled study. Urology 46(5),1995

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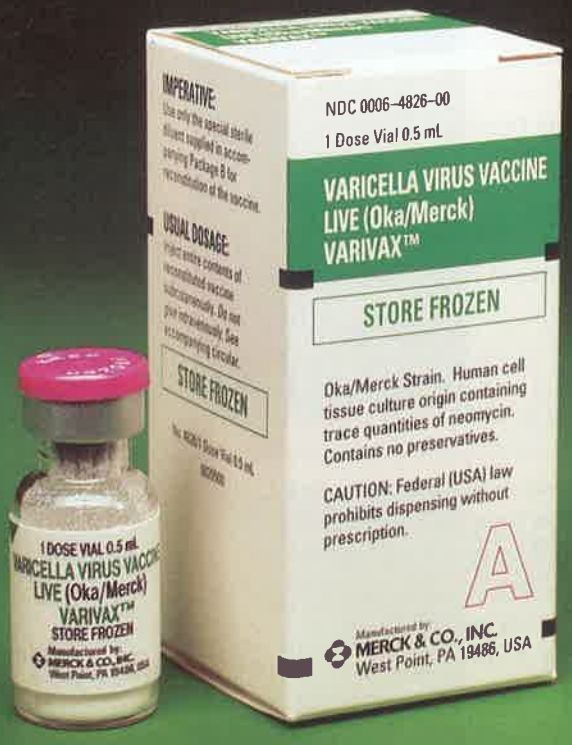

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EDITORIAL

THE EYE IN GENERAL PRACTICE

Eye problems occur at any age, are common, and are usually first presented to the general practitioner/family doctor. In Australia, it is estimated that 2.5% of patients that present to a GP do so with an eye complaint. Around one third are thought to be referred[1]. Local comparative figures are not yet available. It is therefore one of the topics that can be researched upon.

The family doctor should be involved in managing common eye problems. Many such problems are within his ability to diagnose and treat. Short eye courses are invaluable as refresher courses to update and hone the clinical knowledge and skills of diagnosis, treatment as well as decision making to refer or not to refer the patient.

Eye problems presenting to the family doctor range from refractive errors, congenital problems, acute problems of infection and injury, loss of sight and potential or actual complications of systemic disease. The four papers in this edition of the journal from our specialist colleagues address some of these eye problems.

INVITED ARTICLES

Confident eye examination

Confident history taking and eye examination are the cornerstones of correct diagnosis and appropriate management of eye problems. The paper by Dr Caroline Chee addresses this consultation skill.

The Eye in Systemic Disease

The eye may be involved in a very wide spectrum of systemic disorders. A knowledge of the eye involvement that may occur helps the family doctor to anticipate and detect such complications early. A case in point is diabetic retinopathy. At diagnosis, some 6-7% of NIDDM patients will already have diabetic retinopathy, some of whom may require laser coagulation to save their sight. Ocular signs may sometimes be the presenting feature of a systemic disease. Thus the presence of ptosis may be an ocular sign of a Pancoast tumour or a berry aneurysm. The paper by Dr Vivian Balakrishnan in this issue discusses ocular manifestations of common systemic conditions that may be seen in the primary care setting.

The Red Eye

The red eye is a common clinical presentation. Although the majority will be due to conjunctivitis, there may be a few surprises to the unwary. Glaucoma, uveitis and keratitis are cases in point. Dr Wee Tze Lin and Dr Paul Chew's paper discusses this topic.

Laser Treatment for Myopia and Astigmatism

Laser treatment for myopia and astigmatism is a topic of current interest. Could the patient's myopia or astigmatism be treated with laser therapy so he or she does not have to wear glasses? Dr Peter Tseng's paper gives the primary care doctor up-to-date information on this subject. It is important to note that this surgery is not for everybody. Firstly, it is only for those who have stable myopia. Thus it is not for the adolescent. The intending patients should have their refraction checked and followed up for a year. There should be no changes over the year. Secondly, they should not have any other eye diseases such as cataract, glaucoma, keratonconus, or connective tissue diseases. Finally, the predictability and safety of the procedure decreases as the degree of myopia increases. It is not a proposition for high myopia worse than 15 dioptres.

OTHER COMMON EYE PROBLEMS

Other common eye problems encountered in general practice include:

Amblyopia – the lazy eye

The commonest causes of amblyopia in children

are strabismus and unilateral or unequal refractive error between the two eyes. The result is that the child's brain is presented with two disparate images. Unlike older people, children under five can cope with the problem by suppressing one eye at the brain level whereas above this age this ability is lost and permanent diplopia develops requiring intervention for cure. If a child with an amblyopic eye is not referred for treatment before the age of five years, the chances of success diminish enormously. Treatment is to patch the good eye. This is a problem for the ophthalmologist. The family doctor's role is to screen and refer those affected early. Any child with a strabismus (always believe the mother) and any child with a family history of strabismus or lazy eye should be referred to the ophthalmologist. Early referral is one of the greatest services a family doctor can provide to parents[2].

Common Ocular Infections

Bacterial and viral infections involving the anterior segment of the eye are seen in general practice, affecting patients of all ages. These include blepharitis, conjunctivitis, ophthalmia neonatorum, and corneal ulcers. Careful assessment is necessary to distinguish between benign causes and those with more serious complications[3]. Whilst blepharitis and conjunctivitis are easily treated, ophthalmia neonatorum and corneal ulcers should require the opinion of the ophthalmologist.

Acute Eye Injuries

Patients with corneal abrasions, foreign bodies, ocular burns and perforating eye injuries often are presented to the family doctor. Appropriate first aid treatment and onward referral to the Accident and Emergency department is necessary in such cases. In ocular burns from chemicals, alkali or heat should be given first aid treatment with copious and continuous irrigation of the eye with water[4]. Initial management and transport of patients with perforating eye injuries require special mention. These can occur as isolated sports or workplace injuries, or in association with facial or multiple trauma. If a perforating eye injury is apparent, no further examination of the eye should be made until the patient is seen by an ophthalmic surgeon. Unnecessary examination increases the risk of loss of ocular contents and infection. Any intraocular or intraorbital foreign bodies should not be removed as this will result in the immediate loss of intraocular contents, rendering a potential salvageable eye inoperable and visually useless. Ointment and drops should not be instilled. Restriction of eye movements is mandatory to provide adequate rest to the injured eye. This is best achieved by sterile eye pads – one over each eye. These should be lightly padded. Avoid any firm pressure over the injured eye.

The Ageing Eye

In the aged, it is important to remember that cataract can severely reduce quality of life. In a local study of 574 cases aged 60 years and older who were invited to come for eye screening, 451 (78.6%) had cataract, either alone or in combination with other eye diseases. Note that 316 persons (55.1%) were previously undiagnosed. This works out to two cases of unknown cataracts to every one that is known. Beyond 75 years, nearly all (94.6%) had cataract[5].

CONCLUSION

The family doctor is likely to be presented with a variety of eye problems. A working knowledge of their clinical features, diagnosis and management should be acquired through postings, attachments and short courses. A research project on disease patterns of eye diseases in Singapore will yield useful descriptive information.

A/Prof Goh Lee Gan

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CONFIDENT EYE EXAMINATION

C K L Chee, MBBS, FRCS (Ed), M Med (Ophth), FRCOphth

SUMMARY

Ophthalmic problems are common in general practice and many common eye conditions can be managed by the general practitioner. Often, examination of the eye is cursory. This may be because of lack of practice or lack of confidence. This article hopes to describe simple clinical examination which can be carried out in general practice to aid in the management of basic eye problems.

INTRODUCTION

As soon as a patient steps into the consultation room, it may be obvious that he or she has an ophthalmic problem since the face and eyes present themselves immediately. Obviously pathology of the skin, lids and conjunctiva can be spotted immediately. Further examination should be conducted in a systematic manner, continuing inward. Good illumination is important; a small penlight with new batteries providing a concentrated beam and a magnifying device such as a jeweller's loupe, magnifying glass, +20 dioptre lens or binocular loupe that magnifies 1.8 to 5 times can be used.

THE EYELIDS

The lids may be swollen due to angioneurotic oedema in which there is no lid inflammation and the conjunctiva is white. Lid swelling associated with sore, red eyes would point to conjunctivitis. Preseptal cellulitis, usually unilateral, presents with inflammation of the lid with or without involvement of the conjunctiva and does not involve the orbital tissue. **Orbital cellulitis** is a far more dangerous condition because of the risk of cavernous sinus thrombosis. In this condition, the lids are inflamed, the conjunctiva is chemosed, the globe may be proptosed and extraocular eye movements are restricted because of swelling of the orbital contents.

Lumps and bumps of the lids are also easily seen. The **chalazion** (abscess of the meibomion gland) or **hordeolum** (abscess of the eyelash follicle, also known as a stye) are probably the commonest lid lumps seen and can be treated either conservatively or with incision and curettage. **Papillomata and xanthelasmas** are also common, but patients seldom complain of their presence. **Basal cell and squamous cell carcinoma** are more common in the lower lid.

Blepharitis may be a cause of ocular discomfort or recurrent conjunctivitis. The base of the eyelashes may have crusty or scaly material adherent to them, which are easily missed if not specifically looked out for. Hot compresses, lid washes with or without topical or systemic antibiotics may be prescribed if this is the source of the symptoms.

Consultant
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and
Department of Ophthalmology
National University Hospital
5 Lower Kent Ridge Road
Singapore 119214

Trichiasis or entropion resulting in eyelashes brushing the cornea are a common cause of discomfort.

Lagophthalmos from inadequate closure of the lids can be detected by asking the patient to close the eyes. It can result from facial nerve weakness, proptosis or lid retraction. If the Bell's reflex is poor, corneal exposure may lead to exposure keratitis. The presence of Bell's reflex and keratitis should be noted, and if keratitis is present, lubricating ointment with or without tarsorrhaphy is required.

THE CONJUNCTIVA

The conjunctiva may show varying amount of hyperaemia in bacterial, viral or allergic conjunctivitis. Hyperaemia, tearing and soreness are the dominant features of viral conjunctivitis. Severe viral conjunctivitis may even have subconjunctival haemorrhages. Purulent discharge with a history of the lids sticking together on waking up in the morning, associated with itchy eyes are the dominant features of bacterial conjunctivitis. Itchy eyes with stringy discharge with a history of previous episodes and atopy suggest allergic conjunctivitis. A bacterial infection may be superimposed on viral or allergic conjunctivitis. Chemosis or swelling of the conjunctiva may appear to be an extra layer of tissue on the surface of the eye, and this may or may not be associated with hyperaemia. Perilimbal injection of the cornea may be due to iridocylitis. **Pterigia or pingueculae** are commonly seen in this part of the world. The conjunctiva can be examined more thoroughly by having the patient look in each direction in order to bring the opposite area of conjunctiva into view.

The upper lid can be everted with or without the help of a glass rod or Q-tip and the palpebral surface of the conjunctiva can be examined for **papillary conjunctivitis**. The patient is instructed to look downwards and the lashes of the upper eyelid are grasped between the thumb and index finger. The cotton tip applicator in placed at the level of the tarsal fold and the eyelid is then folded back while the patient continues to look downward. The papillae may range in size from microscopic, where the help of a magnifying glass or loupe may be required for observation, to giant papillae which are visible with the naked eye. Giant papillae are more common in children with allergic conjunctivitis or in adults who are soft contact lens users. Lids which have papillae usually evert easily. If the lid is extremely difficult to evert, the likelihood is either the patient is very squeamish or there are no papillae present. Before attempting to evert the lid, a clear explanation and reassurance to the patient that the examination is slightly uncomfortable but not painful, contributes to the success of this manoeuvre. Some children will allow their lids to be everted whereas some adults are unable to cooperate for the procedure. Other reasons to evert the lid may include looking for a foreign body including a missing contact lens, or to enable adequate irrigation of the eye in the case of chemical injury where solid particles e.g. cement may be present. In the case of a missing lens, looking in the lower fornix may reveal a crumpled lens.

PROPTOSIS

Proptosis is examined by standing behind the patient and inspecting the prominence of the eyes, looking over the brow from above and behind, elevating the upper lids. The commonest cause of proptosis, whether unilateral or bilateral, is dysthyroid eye disease; but if unilateral, orbital tumours and high myopia should also be borne in mind.

VISUAL FUNCTION TESTS

Testing of visual function is an important part of ocular assessment, and the basic tests, namely visual acuity and colour vision, can be performed by a trained medical assistant.

Visual Acuity

Visual acuity is tested with a Snellen chart with the patient placed 6 m away. Spectacle wearers should be wearing spectacles for distance. Each eye is tested separately taking care to occlude the fellow eye with a piece of paper or occluder, ensuring that the patient is not peeping through his/her fingers or around the occluding object. If vision obtained is poorer than $^{6}/_{12}$, a pinhole should be used to access whether the vision can be improved with refraction, in which case a change of glasses may be all the patient needs. After the distance vision is tested, reading vision should be tested with near vision charts, with reading glasses if the patient is presbyopic (most patients over 40 years old). If the patient is

mildly or moderately short-sighted, reading may be helped by removing the spectacles.

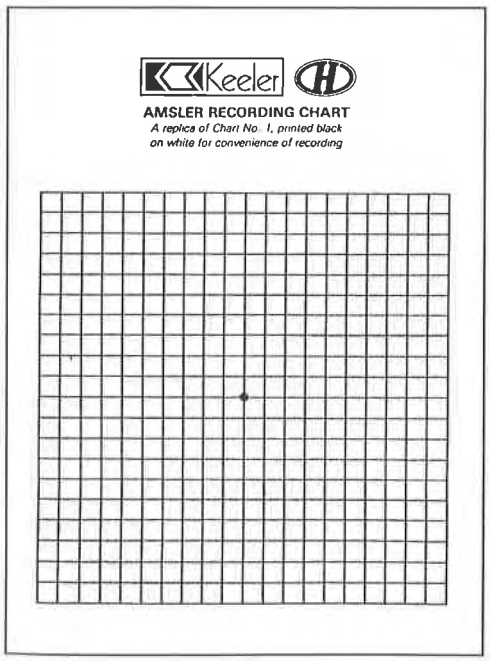
Colour Vision

Colour vision is easily tested with the Ishihara chart, which tests primarily congenital red-green colour blindness, which is present in about 8% of the male population.

Scotomata

The **Amsler grid** (fig 1) is a simple and useful test for central and paracentral scotomata, which may be present in age-related macular degeneration, myopic macular degeneration, central serous retinopathy, or optic nerve disease. The Amsler grid is placed 30 cm from the patient's eyes with the patient wearing the appropriate correction for reading, and each eye is tested separately. The patient is asked the following questions: Can you see the dot in the centre? While fixing your gaze on the dot, are all the lines straight with no areas where the lines are wavy or crooked? Are there any dark patches or missing patches in the grid.? Are the lines equally dark in all areas? Dark patches (positive scotomata) or metamorphopsia (wavy lines) usually result from macular disease whereas missing patches (negative scotomata) usually arise from optic nerve disease.

Figure 1: Amsler chart to test central, paracentral scotomata or metamorphosia. The patient can draw the area of abnomaility



Visual Fields

Testing visual fields by confrontation can help pick up gross visual field defects such as bitemporal hemianopia, homonymous hemianopia or constricted fields in glaucoma or retinitis pigmentosa.

THE CORNEA

Corneal Clarity

Corneal clarity, an intact epithelium and the absence of corneal vascularisation are signs of a healthy cornea. Corneal clarity is demonstrated by a smooth, regular, mirror-like anterior surface, with the iris and pupil clearly seen. An oedematous cornea has a ground glass appearance and obscures the anterior chamber details. The other eye, if normal, can be used as a comparison for corneal clarity. Corneal opacities or foreign bodies are usually obvious with some magnification. Vessels on the cornea are more easily missed. In corneal abrasions, corneal ulcers and punctuate epithelial erosions, there is an absence of the epithelium, and this can be demonstrated with a small amount 2% fluorescein solution instilled into the fornix which stains the underlying exposed stroma and can be seen as a fluorescent green colour when using the blue light from the direct ophthalmoscope. Rose bengal 2% can be used to stain devitalised epithelium in severe dry eyes or herpes simplex keratitis. Only a small amount of rose bengal, instilled as a drop at the tip of a glass rod, should be used after a drop of topical anaesthetic as this dye stings and looks messy if too much is instilled. Both fluorescein and rose bengal stain soft contact lens and should not be used while the lenses are worn.

Corneal Sensitivity

Corneal sensitivity can be tested using a wisp of cotton twisted to a point to touch the patient's cornea with the patient asked to look straight ahead. The patient will blink or close the eye immediately if corneal innervation is normal. The cotton should not touch the lashes or lid margin or be seen by the patient as it is coming toward the eye in order not to stimulate lid

closure from anything other than corneal touch. Conditions which reduce corneal sensi-tivity are herpes simplex and herpes zoster (in-volving nasociliary nerve) infections, adenoviral keratitis and trigeminal nerve lesions.

THE PUPILS

Pupillary size, shape, symmetry and reaction to light and accommodation should be observed. The pupils should be equal in size and regularly circular in shape. Irregular pupils may be due to posterior synechia, adherent leukoma (iris stuck to the lens due to iritis or to the cornea from previous corneal perforation respectively), or iris incarcerated in a penetrating injury.

Pupillary Light Reflex

Testing of pupillary light reflex is an essential part of an eye examination. The room should be darkened to allow the pupils to dilate, and the patient asked to look at a distant object to prevent pupillary constriction from the near reaction. The torch should be bright otherwise the batteries should be changed, and then directed to the pupils from below, again to prevent accommodation. The light is directed into the eye for one second, and the pupillary reaction observed. The beam is then directed into the fellow eye. The pupillary reactions should be brisk and equal.

When testing for a relative afferent pupillary defect (commonly called the Marcus-Gunn pupil), light is directed into one eye for 3 seconds and the pupillary reaction observed (fig 2a). The light is then swung to the fellow eye which should be already constricted due to the consensual light reflex. In a normal fellow eye, the pupil will remain constricted as the direct light reflex is intact. If there is a defect in the afferent pathway, the pupil of this eye will dilate when the light is directed into the eye from the normal eye (fig 2b) This is because the consensual light reflex is stronger than the direct light reflex in this eye. Remember that the direct light reflex is often present in this eye, and the pupil will constrict when light is directed in without being swung from the normal eye. The sign to look out for is the dilation when swinging the torchlight from the normal eye into the affected eye. This test is also called the swinging torchlight test.

Even mild optic nerve disease, such as optic neuritis with vision of $^{6}/_{6}$ and only subjective visual disturbance, will result in a relative afferent pupillary defect. However, retinal disease must be quite extensive, e.g. total retinal detach-

Figure 2a: Swinging torchlight test for relative afferent pupillary defect (RAPD): light shone into normal pupil

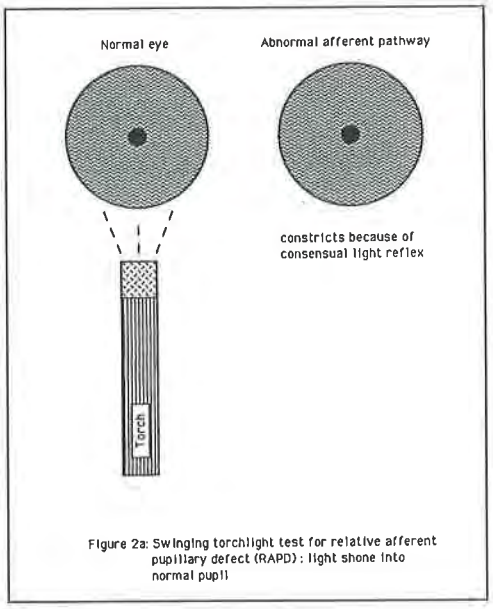


Figure 2a: Swinging torchlight test for relative afferent pupillary defect (RAPD) : light shone into normal pupil

Figure 2b: Light swung from normal pupil to abnormal pupil

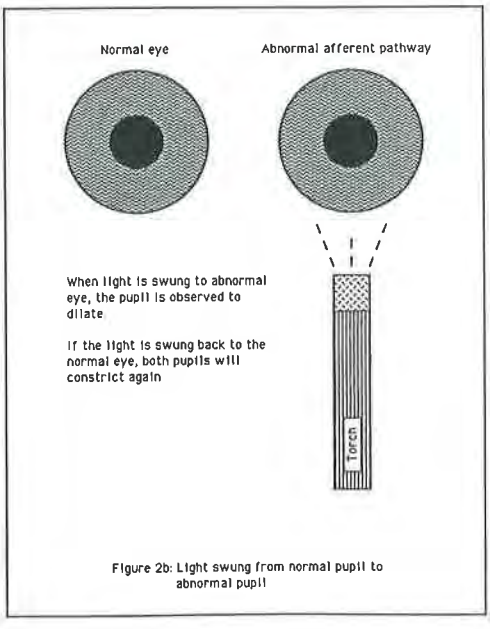


Figure 2b: Light swung from normal pupil to abnormal pupil

ment, before there is a relative afferent pupillary defect. The afferent pathway should be tested in all cases of reduced vision in one or both eye. It is never present if visual loss is due only to cataract, thus if it is positive in the presence of a cataract, there must be additional pathology present e.g. total retinal detachment or optic atrophy, and prognosis after cataract extraction is guarded.

THE RED REFLEX

The red reflex is a simple and useful sign to observe. The red reflex is reduced in media opacities, e.g. cataract, vitreous haemorrhage or vitreous inflammation, and in retinal abnormalities e.g. retinal detachment or retinal tumour especially retinoblastoma where the presenting feature may be a white or cat's eye reflex in an infant. The red reflex is easily observed by using a direct ophthalmoscope held about 30 cm away from the patient's eye, focusing it on the pupil. The type of a cataract may be observed e.g. central opacity, cortical spokes or generalised reduction of light reflex in a nuclearsclerotic cataract. A dense, mature cataract can be seen as a white or brown opacity at the pupil. In older patients miosed pupils may make it difficult to assess the red reflex, and pupillary dilation is useful in these cases. The red reflex takes only one second to observe, and if it is performed routinely, the observer will become familiar with the differences between a normal and abnormal appearance.

INTRAOCULAR PRESSURE

Intraocular pressure can be estimated by gentle digital pressure on the globe through a closed eyelid. Whilst precise intraocular pressure is difficult to estimate, if one side is obviously harder than the other, or if both sides feel much harder than the examiner's own eyes, the intraocular pressure is likely to be abnormally high. In acute angle closure glaucoma, only gentle pressure should be used as the globe will be tender.

EXAMINATION OF THE FUNDUS

Examination of the fundus is much easier if the pupils are dilated. The risk of pupillary dilation is precipitating an acute angle closure glaucoma attack in susceptible individuals. However, there is an argument that it may be better to bring on an attack after the patient is warned of the symptoms and can return for prompt treatment rather than develop an attack at an inopportune time. The patient should be warned to return immediately if symptoms of pain, severe blurring of vision or seeing haloes around lights develop within a few hours after pupillary dilation. Constricting the pupil after dilation does not prevent the development of an acute angle closure attack. In adults, tropicamide 1% (Mydriacyl) is used. If not dilating after 10-15 minutes, additional drops can be instilled every 10-15 minutes for an hour. In addition, phenylephrine 5-10% can be instilled after checking that the patient's blood pressure and heart rate is not raised. Elderly patients and diabetic patient's tend to have miosed pupils which may take longer to dilate.

When using the direct ophthalmoscope to examine the fundi, first observe the red reflex. If there is a poor red reflex, media opacity may be present which would reduce the fundus view. Examine the fundus in a systematic fashion starting with the disc by looking about 15 degrees nasally. This corresponds to the blind spot and the patient will experience less glare when this is being examined. Note the colour of the disc, definition of the disc margins and the cup-disc ratio. Next, follow the retinal vessels away from the disc and note the calibre and whether there are abnormalities of the surrounding retina. Finally, examine the macula by looking slightly temporal to the disc or ask the patient to look directly at the light. Diabetic maculopathy or age-related macular degeneration are conditions to look out for.

CONCLUSION

Examination of the eye is not difficult if it is performed in a systematic fashion on a routine basis.

THE EYE IN SYSTEMIC DISEASES

V Balakrishnan, MBBS, M Med (Ophth), FRCSE, FRCOphth

SUMMARY

The eye may be involved in a very wide spectrum of systemic disorders. Ocular signs may sometimes be the presenting feature in a systemic disease. Conversely, visual function may be compromised by the underlying disease process.

This paper will discuss ocular manifestations of common systemic conditions. It will focus on how the pathophysiology of the systemic disorder leads to characteristic changes is specific ocular tissues, which can be detected in a primary care context. Primary care physicians play an essential role in the prompt recognition of ocular complications and instituting appropriate treatment for their patients. Visual function testing and a directed search for relevant ocular signs should be performed in this group of patients.

Keywords: *Diabetes mellitus, dysthyroid eye disease, rheumatoid arthritis, uveitis, cataract, retinitis.*

INTRODUCTION

The eyes have often been described as windows of the soul; and in a parallel fashion, they often reflect disease processes in other parts of the body. The eye is a direct extension of the central nervous system (optic nerve and retina), richly endowed by a unique vascular system, surfaced anteriorly by mucous membranes (cornea and conjunctiva), and protected by mobile folds of skin (the lids). It is therefore not surprising that the ophthalmic system is so often involved in systemic disorders.

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It is the only part of the central nervous system which is directly visible (via the ophthalmoscope). Swelling, hyperaemia or atrophy of the optic nerve head are clear signs of probable intracranial pathology.

Nine of the twelve cranial nerves are connected with the ocular apparatus directly or indirectly. No neurological examination is complete without an ophthalmic review.

Retinal vessels can be inflamed in autoimmune conditions, dilated and bleeding in diabetes mellitus or sclerosed in hypertension and atherosclerosis. Emboli, both septic or aseptic, are visible signs of vascular anomalies. Endogenous infections like cytomegalovirus or toxoplasmosis or candidal infections present with chorioretinitis.

The mucous membranes of the eyes (the cornea and conjunctiva) may be affected by a number of skin and mucous membrane disorders, e.g. Steven-Johnson's syndrome.

The breadth and depth of this topic precludes a comprehensive review in a paper of this length.

Nevertheless, I will present a systematic review of typical ocular signs which may indicate the presence of systemic diseases in a general practice context.

THE EYELIDS

There are two signs of clinical significance, namely, ptosis and lid retraction.

The presence of ptosis always merits further investigation. Local causes like tumours, cysts and allergy can be easily excluded by simple inspection and history. The two main differential diagnosis are either Horner's syndrome or a third nerve palsy. These can easily be differentiated on the basis of the pupil examination. The pupil will be relatively miosed in Horner's syndrome, and usually dilated in a third nerve palsy.

Lid retraction, lid lag and exophthalmos are classical eye signs of dysthyroid eye disease. Lid retraction (Dalrymple's sign) occurs in 37% to 92% of patients with this condition[1]. Dysthyroid eye disease is characterised by inflammation, enlargement and fibrosis of orbital tissues, particularly the extraocular muscles. It is presumed to be an autoimmune disorder which overlaps with Graves hyperthyroidism. It is noteworthy that the eye signs may be independent of the extent of hyperthyroidism, and may in fact be worse during the hypothyroid phase of the disease.

There are four major pathways in which dysthyroid eye disease affect the vision of a patient.

Firstly, the oedema and swelling within the orbit can cause compressive optic neuropathy which may lead to blindness. In fact, the eye which is least proptotic may actually be at a higher risk, because proptosis or forward displacement of the eye may lower the intraorbital pressure.

Secondly, these patients may develop increased intraocular pressure due to the restrictive myopathy[2] or obstruction of the orbital veins with elevation of episcleral venous pressure.

Thirdly, the swelling and fibrosis of the extraocular muscles often causes diplopia due to a restrictive myopathy. The most common muscles affected are inferior rectus (causing limitation of upgaze) and medial rectus (causing a convergent squint).

The cornea may develop exposure keratopathy due to inability to adequately close the eye and the paucity of blinking.

Consequently, all patients with dysthyroid eye disease require evaluation of visual function, (including colour vision and perimetry), examination of ocular motility, and a CT scan.

PROPTOSIS

Although dysthyroid eye disease as described above is the commonest cause[3] of proptosis, it is important to recall the other differentials of proptosis. Inflammatory pseudotumour or infectious orbital cellulitis will present with proptosis, and require treatment with steroids and antibiotics (and perhaps surgery) respectively. It must also be borne in mind that lymphomas, neoplasms (especially breast carcinoma), sarcoidosis, amyloidosis, Wegener's granulomatosis and vasculitis may also cause proptosis. The CT scan and selected serological markers, e.g. ANCA etc., play an important role is sorting these differentials.

CONJUNCTIVA AND CORNEA

The conjunctiva is commonly involved in mucosal and dermatological disorders, e.g. seborrhoeic dermatitis, erythema multiforme (Steven Johnson's syndrome) toxic epidermal necrolysis, acne rosacea, pemphigus, pemphigoid etc. The conjunctiva is inflamed (injected or chemotic) initially and may be fibrotic or scarred subsequently. These are non-specific changes and not necessarily diagnostic. Consequently, specific diagnosis will be made on the basis of systemic findings and relevant investigations. However, the physician will have to provide supportive therapy to the eyes in the form of lubricants, antibiotic prophylaxis and topical steroids in selected cases under ophthalmic supervision.

Corneal involvement in these conditions represent an ophthalmic emergency, and immediate ophthalmic referral is mandatory. In acute stages, corneal melts or perforation may occur, with loss of the eye. In later stages corneal scarring will compromise acuity and corneal grafts may be required in selected cases.

Kayser-Fleisher rings are brown or brownish blue deposits in Descemet's membrane of the cornea. They occur in 100% of patients with

neurological manifestations of Wilson's disease[4], but unfortunately are often only visible on slit lamp examination, thus precluding its usefulness in a general practice setting.

Jaundice, classically recognised by the yellow discolouration due to deposition of bilirubin in the conjunctiva, always requires further investigation.

Dry eyes or keratoconjunctivitis sicca[5] may occur with rheumatoid arthritis (15% to 25%), primary Sjogren's syndrome, systemic lupus erythematosis, systemic sclerosis, polyarteritis nodosa or polymyositis. Although most cases of dry eyes are idiopathic, a high index of suspicion is necessary to exclude these systemic conditions. Treatment is usually symptomatic and consists of artificial tear preparations, acetylcysteine drops or punctal occlusion.

The more sinister manifestations of rheumatoid arthritis include corneal stromal keratitis (with corneal opacities visible), peripheral ulcerative keratitis and scleritis (recognised by deep boring pain and inflammation of the sclera).

UVEITIS

Uveitis is an autoimmune process characterised by intraocular inflammation. Clinically, patients may present with photophobia (discomfort on exposure to light), redness of the eyes, ocular pain and blurring of vision. The ocular signs are often subtle and difficult to diagnose without a slit lamp. However, ciliary injection (redness around the limbus of the cornea), an irregular small pupil, or hypopyon may be evident on examination with a torchlight. It is essential to ask for a history of joint pains, backache, and bowel disorders. The differential diagnoses include HLA B27 positive spondyloarthropathies like ankylosing spondylitis, Reiters syndrome (polyarthritis, urethritis, and conjunctivitis), psoriasis and inflammatory bowel disease.

A sterile hypopyon (pus in the anterior chamber) is a typical finding in Bechet's disease, which is a relapsing and remitting systemic vasculitis accompanied by oral and genital ulcers. Other ophthalmic complications include retinal vasculitis and retinitis. Prompt recognition is essential in order to avoid complications which include blindness and even death.

It is noteworthy that up to 6% of cases of uveitis occur in children[6,7]. It is must be borne in mind that children with juvenile rheumatoid arthritis may be completely asymptomatic as far as their eyes are concerned in the early stages of the iritis.

However, they may develop blinding complications like cataracts, band keratopathy, posterior synechiae, glaucoma, optic nerve oedema and macular oedema later on. Consequently, it is essential that regular ophthalmic reviews be performed before the onset of visual symptoms.

CATARACTS

The vast majority of senile cataracts are idiopathic. Diabetics may develop a premature onset of senile cataracts, and 15% to 20% of patients with Wilson's disease may develop a sunflower cataract[8]. Nevertheless, these constitute an extremely small minority, and senile cataracts usually do not undergo extensive investigations as far as the aetiology is concerned.

Steroid therapy also causes posterior subcapsular lenticular opacification in a dose and duration related effect. Younger children are particularly prone to this development, and require regular screening for the duration of the treatment.

60% of bilateral paediatric cataracts[9] are idiopathic, with a further 30% being hereditary. However, 5% are due to genetic, metabolic and systemic diseases and 3% due to intrauterine infections. Consequently, a systematic search for an aetiology diagnosis in paediatric cataracts is mandatory because it will identify other organ systems at risk (e.g. rubella) and specific treatment may be available, e.g. withdrawal of galactose in galactosaemia.

A family history is essential. Routine investigation include a TORCH (toxoplasmosis, rubella, cytomegalovirus, herpes, syphilis) screen for intrauterine infections, urine for reducing sugars (galactosaemia), urine amino acids (Lowe's oculocerebrorenal syndrome), blood calcium and phosphorus levels.

RETINA

Diabetes mellitus is by far the most important disease affecting the eye, as it is a leading cause of irreversible blindness in developed coun-

tries[10,11,12]. Up to 90% of patients with Type I (IDDM) diabetes will develop some form of retinopathy by 15 years post diagnosis, and 50% may develop potentially blinding proliferative retinopathy by 20 years post diagnosis. However, they usually do not develop retinopathy during the initial 5 years of diagnosis. In patients with Type II (NIDDM) diabetes 3% may have visually threatening macular oedema at the point of diagnosis. This difference in timing probably relates to the lag period between onset of NIDDM and diagnosis[13,14].

Diabetic retinopathy is a microangiopathy that has two basic pathophysiologic mechanisms, namely, leakage and occlusion.

Loss of pericytes of the capillaries leads to the formation of microaneurysms and increased permeability. The increased permeability causes a breakdown of the usual blood ocular barrier, and leakage of serum components into the retina. This becomes visible as yellowish-white glistening well-defined hard exudates and diffuse retinal oedema. If retinal oedema occurs in the central macular area (which is the most sensitive part of the retina) visual acuity will be compromised. The microaneurysms and associated haemorrhages are visible as dot haemorrhages if the haemorrhage is in the deeper layers of the retina, and as flame haemorrhages if they occur in the more superficial layers of the retina.

In addition, progressive occlusion of retinal capillaries occurs, due to a combination of thickening of the basement membrane, endothelial damage and proliferation, and increased platelet aggregation. Capillary occlusion causes nerve fibre infarcts which are visible as greyish white feathery edged cotton wool spots (formerly called soft exudates).

Progressive capillary closure causes larger areas of ischaemic retina to produce presumed angioproliferative factors which lead to the development of new vessels. These new vessels may leak (causing further oedema) and bleed (causing vitreous haemorrhage). Ultimately, proliferation of new vessels is accompanied by fibroblast proliferation which may cause tractional retinal detachments. In addition, proliferation of new vessels on the iris (rubeosis) causes often intractable glaucoma which leads inexorably to blindness.

In summary, diabetic retinopathy may compromise vision by causing macular oedema, macular ischaemia, vitreous haemorrhage, tractional retinal detachment, and rubeotic glaucoma.

The key to managing this condition is early detection and prompt institution of argon laser photocoagulation which has been shown to be effective in treating oedema and causing regression of new vessels[15].

All newly diagnosed NIDDM patients need immediate ophthalmic screening, in contrast to IDDM who should usually have their first screen within the first 5 years of diagnosis. Inspection of the retina using a direct ophthalmoscope through an undilated pupil is dangerously useless. The minimum is a Polaroid fundal photograph revealing at least 30° field of view. There is currently a scheme in Singapore where diabetics can be referred to polyclinics for fundal photography at a nominal fee. The photographs are interpreted by an ophthalmologist and the reports sent to the primary care physician. This programme has been running very successfully since its inception in July 1991.

Apart from diabetes, the retina may also be involved in other systemic conditions. For example, autoimmune vasculitis, like systemic lupus erythematosis may present with retinal vasculitis. This presents with cotton wool spots, retinal haemorrhages and exudates and disc changes. Atherosclerosis and hypertension predispose to retinal vascular disorders like central retinal vein or artery occlusion.

The emergence of AIDS has also led to a dramatic increase in the number of patients with retinitis due to opportunistic cytomegalovirus (CMV) reactivation. Up to 25% of patients with AIDS will develop CMV retinitis, and it may occasionally be one of the first clinical signs of HIV infection. The median survival after diagnosis of CMV retinitis in some series of AIDS patients has been only 7-10 months[16]. Consequently, it is a sinister development, and any apparently healthy patient presenting with opaque retinal lesions and blotchy retinal haemorrhage and vasculitis requires a comprehensive workup.

More recently, there has also been a series of local children who developed CMV retinitis

after organ transplants due to immunosuppression[17].

CONCLUSION

The eyes are affected in a very wide spectrum of systemic diseases. The primary care physician should always be aware that eyes may sometimes pro-vide the first signs of a systemic disease or may be compromised by the systemic disease process itself. Visual acuity testing, and a quick exclu-sionary search for relevant eye signs should form part of the assessment of any patient with a systemic disorder.

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THE RED EYE

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INTRODUCTION

Complaints of red eyes are among the most common of ophthalmic presentations. They range from innocuous conditions like dry eyes to blinding ophthalmic conditions such as acute glaucoma. As such, the general practitioner, being the first line of medical consults, must be able to distinguish the various pathologies with some degree of accuracy and competency and thereafter to make the necessary referral for continued ophthalmic care. The authors hope the following article will be of value and help guide general practitioners in this regard.

CAUSES OF RED EYE

Red eyes can be due to:

Blinding Pathologies:

- Acute glaucoma
- Corneal ulcers
- Endophthalmitis
- Some cases of trauma
- Uveitis

These conditions must be treated with urgency and referred to an ophthalmologist immediately. Delay in treatment may result in permanent loss of vision.

Others:

- Infective conditions e.g. conjunctivitis
- Allergic eye diseases
- Inflammatory disorders
- Surface disorders
- Some cases of trauma
- Subconjunctival haemorrhage

These conditions may be dealt with more electively. Some of these conditions can be managed by the general practitioner.

Acute Glaucoma

A significant acute rise in intraocular pressure, whatever the cause, will give rise to corneal edema and haziness, eye pain, unilateral headaches, blurring of vision as well as red eye. This is important to diagnose as sustained high intraocular pressures will lead to retinal vasculature and even to optic nerve head infarction, leading to irrecoverable loss of vision.

Several pathological mechanisms may lead to this:

- Acute angle closure glaucoma
- Secondary angle closure glaucoma
- Secondary open angle glaucoma
- Lens-induced glaucoma

Infective Causes

Infective conditions involving the conjunctiva, cornea or intraocular structures will give rise to a red eye. These include the following:

- Conjunctivitis, usually viral or bacterial
- Corneal ulcers
- Endophthalmitis

Allergic or Inflammatory Causes

- Allergic eye disease, ranging from Giant Papillary Conjunctivitis to Vernal Keratoconjunctivitis

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- Uveitis
- Episcleritis
- Scleritis
- Blepharitis with or without secondary involvement

Surface Disorders

Surface disorders can give rise to red eyes. These include:

- Pterygia and/or pingueculum
- Dry eyes
- Epithelial defects
- Foreign bodies including corneal and subtarsal foreign bodies
- Epithelial malignancies e.g. carcinoma-in-situ of conjunctival epithelium

Trauma

Trauma including chemical injury will result in red eyes. This is usually evident in the history and may be accompanied by blurring of vision. Clean corneal abrasions can be managed by the general practitioner by padding up the eye after an antibiotic ointment has been applied. Close review is recommended. Cases which get infected must be referred urgently to the ophthalmologist.

Subconjunctival Haemorrhage

This may be spontaneous or it may be secondary to trauma or to episodes of raised thoracic pressure e.g. bouts of sneezing, coughing, vomiting etc.

MANAGING THE RED EYE

History

Itch

Itch is a useful symptom to ask for. Significant itch points towards the direction of an allergic phenomenon. Viral conjunctivitis may also be accompanied by symptom of itch.

Discharge

Discharge forms a predominant symptom in conjunctivitis. Patients with bacterial conjunctivitis wake up in the mornings with upper and lower eyelids adherent to each other. In viral conjunctivitis, discharge is often watery. Purulent discharge especially in copious amounts may indicate Gonococcus as the offending agent. This is important to distinguish as Gonococcal conjunctivitis can cause corneal perforation within 48 hours if not treated.

Pain

Pain would raise a suspicion of acute glaucoma, corneal ulcers or endophthalmitis. The latter is a feared complication following ocular surgery and must be considered in patients with recent ocular surgery or history of previous glaucoma operations. The cornea in these cases may be cloudy, with or without a hypopyon. Corneal ulcers must be kept in mind especially in contact lens wearers. A corneal opacity may be seen. Acute glaucoma presents with eye pain, headaches with or without vomiting, decrease in vision in addition to redness, and must always be borne in mind as it is a treatable blinding condition.

Vision

Significant drop in vision in conjunction with a red eye may be related to acute glaucoma, intraocular inflammation e.g. uveitis, or endophthalmitis. Corneal pathologies involving the visual axis e.g. corneal ulcers, epithelial abrasions etc. will understandably also affect vision. Conjunctivitis by and large does not affect visual acuity. However it must be borne in mind that those due to adenoviral strains may lead to keratitis and this may cause a drop in vision. If so, this usually occurs at about 10 days from the onset of conjunctivitis, when the injection and discharge is tailing off. Trauma with hyphaema either in the macroscopic or microscopic form will also affect vision.

Examination

In corneal abrasion, conjunctivitis etc., there may be some degree of pain or discomfort that precludes a proper ocular exam. In such instances, after confirming that the globe is intact, a drop of Amethocaine may be instilled for pain relief and the eye examined about five minutes after the instillation of the eyedrop.

Visual Acuity

This serves as an important guide to the ocular pathology and is useful as a baseline for future consultations.

Pattern of Injection

Forniceal injection points to a conjunctival pathology. Limbal injection indicates a corneal or intraocular pathology. Nasal or temporal injection may be related to pterygia and/or pingueculum.

Occasionally, 10% phenylepinephrine may be useful to distinguish the level of vessels involved in the injection. In episcelritis, 10% phenylephrine will constrict the superficial vessels and reduce the injection. In deeper inflammations e.g. scleritis, the vessels are not blanched.

Cornea

Corneas must be clear. Focal opacities should alert the physician to the possibility of a corneal ulcer. Diffuse haziness of the cornea may be a sign of acute glaucoma or severe intraocular inflammation.

Pupil

If a fixed and dilated pupil is noted in the red eye, acute glaucoma must be excluded. A small pupil with injection is consistent with anterior uveitis.

Florescein

Florescein is very useful to delineate epithelial defects. Filamentary keratitis will also be highlighted by florescein.

Eyelids

In cases of foreign bodies, eyelids must be everted and swept to see if any foreign body is present. Severe cases of allergic conjunctivitis e.g. Vernal's keratoconjunctivitis or Giant Papillary Conjunctivitis may also manifest papillae especially on the upper tarsal conjunctiva. Blepharitis is characterised by the presence of squames and debris on the eyelashes.

SUMMARY

In conclusion, the general practitioner will be faced with the red eye as it is one of the most common ophthalmic presentations. The underlying pathology may be potentially blinding if not recognized and treated promptly. It is therefore imperative that the GP being the first line of consult in many of these cases be able to distinguish those that require urgent treatment and attention and manage accordingly. In addition, cases with chronic injection and those who do not respond as one would expect based on the diagnosis made must also be referred to the attending ophthalmologist.

EXCIMER LASER TREATMENT FOR MYOPIA AND ASTIGMATISM

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INTRODUCTION

There has been an increasing demand for the correction of myopia and astigmatism by Excimer Laser Photorefractive Keratectomy. General practitioners are often faced with patients who want to know more about the procedure before deciding if they want to be referred to an ophthalmologist. This article hopes to give the general practitioner a better understanding of the current situation with regard to this procedure. Should patients be encouraged or discouraged or should they be told to wait a little longer? What is the difference between LASIK and PRK? These are the questions which are often asked and it is sometimes difficult to give a straight answer.

RETURN TO BASICS

Let's begin by returning to some basic optics. Firstly, myopia is a condition where there is an abnormal elongation of the eyeball and therefore light rays from a distant object gets focused in front of the retina. Near objects are seen clearer than those at a distant. There is a very high incidence of myopia in Singapore. This is especially so amongst the Chinese.

Hypermertropia is the reverse situation where the eyeball is smaller than normal and so light from a distant object gets focused behind the retina. Distant objects are therefore clearer than near objects.

Astigmatism is a condition where the cornea is not equally round like the surface of a football but instead has a curved surface similar to a rugby ball. Hence there are two points of foci. Both distant and near objects are blur because they cannot be properly focused onto the retina. Most patients that have myopia also have a certain degree of astigmatism.

Presbyopia is a result of aging and this occurs when the eye gradually loses its ability to focus on near objects. This occurs because of the gradual hardening of the lens. This condition often begins around the age of 40 when patients start to have difficulty reading.

METHODS OF CORRECTION OF MYOPIA

Myopia can be corrected by several means and the most common ways available are by the use of spectacles and contact lenses. These remain the most popular methods of correcting short sightedness. There are however some problems which patients complain about such as distorted peripheral vision especially in patients with high myopia and thick glasses as well as discomfort around the nose bridge and ears. Contact lenses tend to be inconvenient at times and long term wearers often develop problems of allergy and sensitivity. Corneal infection is a potential danger to all contact lens wearers.

Refractive surgery is an option made available over the past decade. This began with radial keratotomy, an operation which involved making deep radial incisions on the cornea to allow

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flattening of the central cornea. This procedure is still being done in certain countries and has shown to be quite successful especially for the lower degrees of myopia. It is currently not practised in Singapore.

Photorefractive Keratectomy (PRK) uses the excimer laser to sculpt and flatten the central corneal surface to correct both myopia and astigmatism (Fig. 1). This procedure has gained much popularity and is the most commonly accepted surgical method of correcting refractive errors worldwide today.

Laser In-situ Keratomileusis (LASIK) is a modification of PRK. In this procedure an instrument known as the microkeratome is used to cut a thin corneal flap about 160 microns thick. This flap is then folded back and the exposed corneal bed is then treated with the excimer laser. The corneal flap is then replaced (Fig. 2). There are several advantages to this procedure and the most important one is the decreased incidence of haze especially in patients treated with higher degrees of myopia. There are however certain complications that have been reported with the use of the microkeratome itself.

Figure 1: Photorefractive keratectomy (PRK) for myopia
The excimer laser sculpts the cornea to flatten it, thereby allowing images to be focused clearly onto the retina.

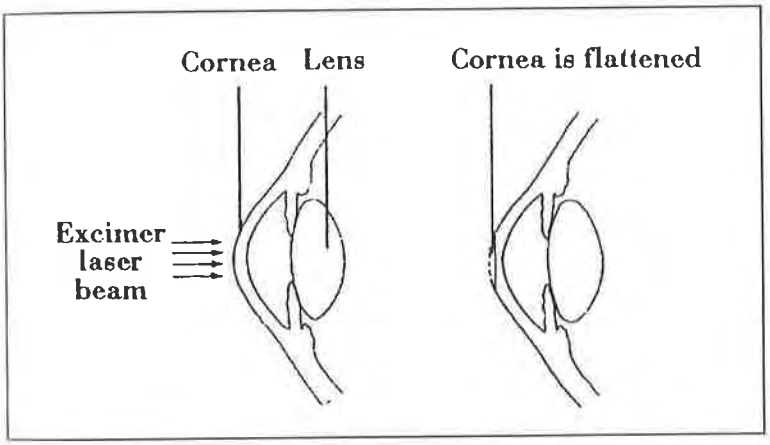
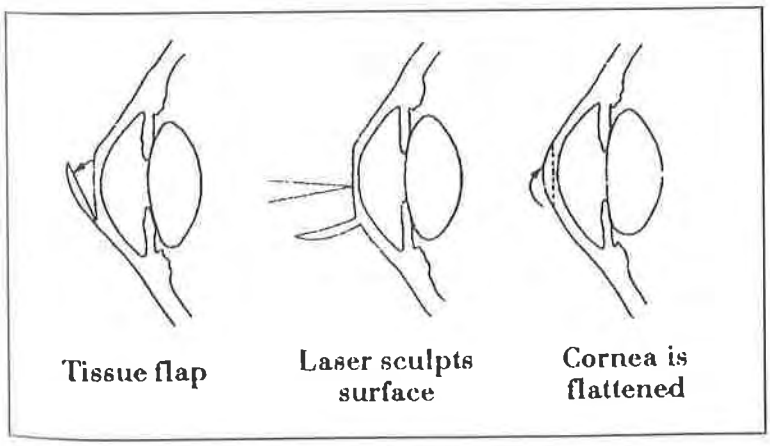


Figure 2: Laser-In-Situ Keratomileusis (LASIK)
Tissue flap is first made with a microkeratome. Excimer laser then sculpts corneal surface to flatten it and the corneal flap is then replaced.



ELIGIBILITY FOR SURGERY

In general we often require patients to sign their own consent form and so they should be 21 years and above. Another reason is that at this age the myopia should be more or less stable. They should therefore not have progressive myopia and their refraction should not have changed over the past year. They should not have any other eye diseases such as cataract, glaucoma, keratoconus, or connective tissue diseases.

Myopia up to 15 dioptres can be treated. The predictability and safety of the procedure decreases as the degree of myopia increases. Astigmatism up to 5 dioptres can be treated. Certain excimer lasers are able to treat hypermetropia up to a certain degree but presbyopia cannot be treated with the excimer laser. All patients will expect to require reading glasses as part of the normal aging process.

METHOD

A preoperative assessment of the patient is done to determine the suitability for treatment. It is important that contact lens wearers stop contact lens wear about 2 weeks prior to the consultation. During the visit, a comprehensive eye examination is performed, a refraction to determine the degree of myopia and astigmatism and a computerized mapping of the corneal surface is done. An informed consent is signed.

The treatment itself is relatively short. It requires the patient to lie on an operating chair which positions the eye under the laser machine. Only topical anaesthetic eyedrops are required. There is no pain during the procedure. A speculum is inserted to keep the eyelids open during the operation. The corneal epithelium is removed first either mechanically or with the use of the laser. The patient fixates on a blinking red target light throughout the procedure. The laser treatment itself usually takes about 15 to 60 seconds depending on the degree of myopia to be corrected. The eye is then patched to allow the corneal epithelium to heal. This usually takes about 2 to 3 days. During this time the patient is given analgesics and sleeping tablets to help in the pain relief. Once the epithelium has healed over the patch is no longer required and the patient begins to use eyedrops. The vision may vary significantly for the first few weeks but it should gradually improve and stabilize in about one to three months.

RESULTS

At the Singapore National Eye Centre, PRK has been the main form of corrective surgery for myopia since 1992. Initially, only myopia up to 10 dioptres was treatable. There was no effective method of correcting astigmatism. Today we have upgraded to an excimer laser machine that allows treatment of myopia up to 20 dioptres and astigmatism up to 5 dioptres cylinder. In general up to 90% of patients treated for mild to moderate myopia can expect to attain an unaided vision of $^{6}/_{12}$. This is vision adequate to qualify for a driver's licence with the Land and Transport Authority of Singapore. These results are equal to that obtained from most centres around the world.

COMPLICATIONS

There have been great concerns as to how safe is excimer laser treatment. The greatest fear is for the eye to go blind. There has been no major complication leading to blindness since we began treatment in 1992. Common complaints include light sensitivity, glare, haloes and corneal haze in the initial few months. A small number of patients will still be slightly myopic (undercorrection) or will become hypermetropic (overcorrected). Patients who are undercorrected may have enhancement surgery six months later. However not all patients can be re-treated and some may require spectacles or contact lenses to achieve satisfactory vision.

Corneal haze or scarring occurs in a small percentage of patients and this causes a decrease in the best corrected visual acuity. The incidence of haze increases with the severity of myopia corrected. Management of haze and regression with steroid eyedrops has proved to be successful in many cases and a majority of patients return to good vision after treatment.

Rare complications include delayed epithelial wound healing, elevated intraocular pressure, cataract formation, induced astigmatism, decentered treatment, and drooping eyelids.

Complications associated with the LASIK procedure are mainly due to the use of the microkeratome and these include damaged corneal flaps, lost flaps, and even damaged anterior chambers of the eyes. It has been noted that a small percentage of patients develop irregular corneal astigmatism after the LASIK procedure.

PRK vs LASIK

There is now a frequently asked question as to whether PRK or LASIK is better. Many surgeons have contemplated answering this question and the most sensible approach is to examine each procedure and determine the complication rates of each.

A common view is that with PRK there is an increasing problem of haze together with loss of best corrected vision with higher attempted myopic corrections. With LASIK there is the problem of irregular astigmatism which is

related to the use of the microkeratome itself. If one has to compare the percentage incidence of the complications of each procedure with the degree of attempted myopic correction, then there is a point at about -6.00 dioptres and above that the LASIK becomes a relatively safer procedure than PRK (Fig. 3). Hence one can claim that for lower myopic corrections below -6 dioptres PRK should be recommended, whereas above -6.00 dioptres LASIK can be performed with relative safety. It is however, still important for the patient to discuss both procedures with their ophthalmologists (Table 1).

CONCLUSION

Excimer laser surgery for the correction of both myopia and astigmatism has been found to be not only safe but also predictable and stable over the long term. It must be remembered however that this form of surgery is still considered cosmetic in nature and as such should be viewed upon with much consideration of the possible side effects and complications. The incidence of complications is low but it does happen and it makes for some unhappy patients. Excimer laser surgery is not for everyone but there are many

Figure 3: Haze vs Irregular Astigmatism

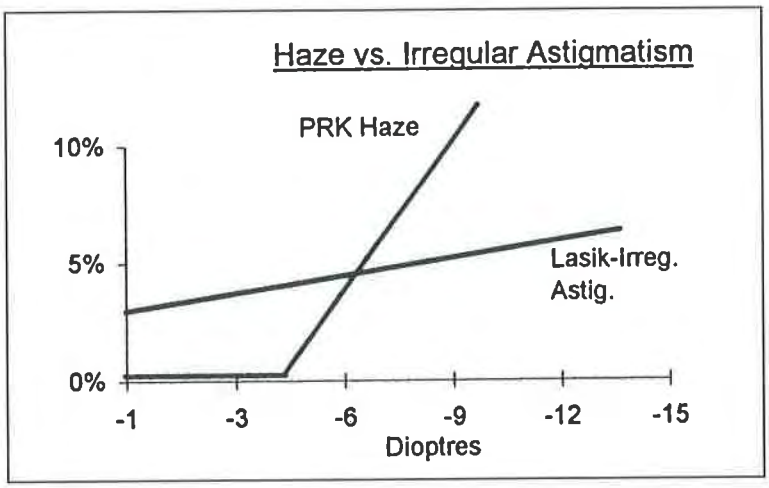


Table 1: The Correction of Myopia: PRK vs LASKI

	PRK	LASIK
Accuracy	=	=
Stability	=	=
Initial Healing Time (Days)	3 – 4	1
Post-Operative Pain	Mild	Very Mild
Infection Rate	Rare	Rare
Corneal Haze Rate		
- 1.00D to - 7.50D	$^{1}/_{100}$	0
- 7.75D to -10.00D	$^{5}/_{100}$	0
-10.25D to -15.00D	$^{25}/_{100}$	0
Corneal Irregular Astigmatism		
- 1.00D to - 7.50D	$^{1}/_{100}$	$^{3\text{-}10}/_{100}$

thousands of very happy patients who have lost their dependence on glasses and contact lenses and have found a fresh view of life.

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SUBDERMAL IMPLANTS AS A METHOD OF CONTRACEPTION

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INTRODUCTION

Segal and Croxatto in 1967 proposed the concept of a reversible long acting contraceptive method using a silastic capsule which is placed under the skin and which has a slow and constant release rate of contraceptive steroids. The task ahead was to identify the most suitable contraceptive compound and to establish the surface and wall thickness of the medical grade polydimethysiloxane needed to achieve a daily release rate and blood level of steroid that would result in desirable measures and effectiveness[1]. By 1975 levonorgestral, norgestrienone and megestrol acetate were identified by the International Committee for Contraceptive Research (ICCR) as the most promising progestins for further evaluation as subdermal implants[2]. In the early stages of the international study, megestrol acetate was withdrawn from further evaluation because it was found to produce breast nodules in beagle dogs. Based on the higher contraceptive efficacy and estimated longer duration of action of levonorgestrel implants than norgestrienone implants, the ICCR selected levonorgestrel as a drug with which the silastic subdermal implants would be developed further. Thus the NORPLANT system was developed[3].

Finland was the first country to give regulatory approval in 1984. In 1985, the International Planned Parenthood Federation (United Kingdom) included NORPLANT as the list of contraceptives made available to IPPF affiliates throughout the world. In 1985 also, preintroduction trials began in Singapore. To-date, NORPLANT is being used by over two million women in 46 countries in Europe, North and South America, Africa and Asia[4].

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MODE OF ACTION

As mentioned earlier, the NORPLANT system consists of six silastic capsules, each containing 36 mg of levonogrestrel in the dry powder form and having a length of 3.4 cm and a diameter of 2.4 mm.

The capsules are inserted subdermally, usually in the inner aspect of the upper arm by means of a minor surgical procedure under local anaesthesia. The insertion usually takes less than 10 minutes. The removal process is also done under local anaesthesia with removal time averaging 10 to 20 minutes. Sutures are not required to close the small incision[5].

Following placement of the implants, levonorgestrel diffuses slowly and continuously through the silastic capsule. Effective blood levels of levonongestrel are reached within 24 hours and can last for five years[6]. The initial release rate is about 85 micrograms per day. This gradually declines to 35 micrograms per day by 18 months[7].

The main mode of action of NORPLANT is changes in the cervical mucous and endometrial changes. The cervical mucous remains scanty, thick and impenetrable to sperm. Furthermore, changes in the endometrium prevent implantation from taking place. There is ovulation suppression in more than 50% of users' cycles[8].

EFFECTIVENESS

The effectiveness rate of NORPLANT over the first three years of use is comparable to surgical sterilization with an annul pregnancy rate of 0.1 percent per year. The rate increases to 0.7 and 1.3 for years 4 and 5 respectively. The cumulative five-year pregnancy rate is approximately 1.3%[9].

High continuation rates have been reported in many studies. After 5 years, a rate of 66% is reported by WHO and local studies in Singapore have also reported a continuation rate of 60% after 5 years[10].

RETURN OF FERTILITY

After removal of implants, levonogestrel becomes immeasurable in blood samples within a few days and the woman's fertility is restored without a time lag. Studies both internationally and locally have shown that women who remove NORPLANT in order to have a planned pregnancy are able to conceive without difficulty[10,11]. Moreover there has been no report of untoward incidence of ectopic pregnancy, spontaneous abortion, still birth or congenital malformation.

SIDE EFFECTS

Menstrual disturbances

The main side effect is the disruption of the woman's normal pattern of mensuration. There is an increase in the number of bleeding and spotting days. The changes in mensuration occur most frequently during the first few months after beginning to use the method. Studies in the United States reveal that during the first year, nine women out of a hundred will discontinue use because of dissatisfaction with the adverse effect on menstrual irregularity and excessive number of bleeding days. By the fifth year of use this percentage has fallen to about three percent[12]. Locally too, there was a nett termination of 12% for menstrual problems and this occurred in the first three years of use. The majority (>90%) of the removals were for prolonged bleeding or spotting of more than 10 days or frequent bleeding. There was only one removal in the local study for prolongel amenorrhoea of 400 days. None of the menstrual disturbances was noted in the fourth and fifth years of use[10].

Other Medical Reasons

Headache is the main medical reason unrelated to menstrual bleeding for discontinuing use of NORPLANT implants. Headaches occur in around 25 – 50 percent of women using NORPLANT[13,14]. However in one study only 1.3% of 2,358 stopped NORPLANT because of headaches[15]. Locally there were no removals because of headaches. Other reported complaints include weight changes (both gain and loss), mood changes and depression. However these have not been reported in our local studies.

There is no evidence that NORPLANT use causes an elevation of blood pressure. This is the case even in studies carried out in Asian countries like China[16].

Complications of Insertion and Removal

The most uncomfortable part of the insertion and removal procedure is reported to be the sharp stinging from the local anaesthetic. This has never been reported locally in Singapore. If this is a problem, the anaesthetic can be buffered with 1mEq of sodium bicarbonate in each 10 ml of local anaesthetic[17].

Other complications such as bruising, irritation or infections are frequent. Superficial horizontal placement of implants in a single plane under the skin is essential and appears to be the most important factor for later ease of removal. When implants are improperly placed initially, the removal process can become difficult and should be done only by health care providers well trained and skilled in NORPLANT insertion and removal. In cases where the implants have been placed too deeply, the women can suffer bruising, pain and unnecessary scarring if removal is attempted by someone without adequate training.

Changes in Glucose and Lipid Metabolism

Studies performed to-date indicate that the NORPLANT system does not markedly alter carbohydrate metabolism[18-23].

With regard to lipid metabolism, a decrease in triglycerides and total cholesterol, increased LDL cholesterol and a transient increase in HDL cholesterol have been reported. Despite these changes, there is no shift in the ratio of HDL

cholesterol to LDL cholesterol[21,23-26]. There are no clinically significant changes in liver, kidney, pancreas, adrenal or thyroid function.

Changes in Haemostatic Function

The decreased synthesis of vitamin K dependent factors II, V and VII by the liver and the lower fibrinolytic activity observed in Singapore studies indicate that prolonged use of NORPLANT implants does not enhance a state of hypercoagulation or an increased state of activation of the coagulation system seen with oral contraceptive use[26]. The increased platelet count and enhanced platelet aggregation seen locally need further evaluation.

In our local studies, the haemoglobin concentration was noted to increase at the end of 5 years of NORPLANT use[26]. This is also reported in other studies where, despite experiencing extra days of menstrual bleeding and spotting, blood iron and haemoglobin levels increase after NORPLANT use because the overall blood loss is less than that of normal menstruation[27].

CONCLUSION AND THE FUTURE

The above review clearly demonstrates the effectiveness, acceptability, reversibility and comparative medical safety of the NORPLANT subdermal contra-ceptive implant system. This new delivery system has potential and is considered suitable for use in fertility regulation programmes along with other currently available contraceptive preparations and devices.

Acceptance of the subdermal implant methodology by women around the world has encouraged further research into the development of additional contraceptive implants. Leiras Pharmaceuticals in Finland is prepared to introduce NORPLANT II rods, the most advanced second generation implants. It consists of only 2 rods and this would result in easier insertion and removal procedures and reduce the incidence of any complications associated with these procedures. Furthermore, there are two implant methods under development that attempt to simplify even further the insertion and removal process by reducing the number of implants to one.

Organon International located in Netherlands is sponsoring research on IMPLANON, an implant that would last for three years and Theramex of Monaco is looking into the introduction of a one year implant method – UNIPLANT.

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DISCRIMINANT ANALYSIS OF VARIABLES ASSOCIATED WITH DENGUE INFECTIONS TO OBTAIN PREDICTOR VARIABLES TO PREDICT DENGUE SHOCK SYNDROME

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Summary

Variables associated with dengue fever (51 cases), dengue haemorrhagic fever grades 1 & 2 (57 cases) and dengue shock syndrome (54 cases) were subjected to discriminant analysis to choose predictor variables for dengue shock syndrome. The cases were those who were admitted to Yangon Children's Hospital with the provisional diagnosis of dengue haemorrhagic fever and had virological evidence of recent dengue infection. The analysis revealed that drowsiness, vomiting, liver enlargement and pulse rate (with or without packed cell volume and platelet count determinations) are good predictor variables for dengue shock syndrome.

INTRODUCTION

The first outbreak of dengue haemorrhagic fever (DHF) occurred in Myanmar in 1970. Since then DHF outbreaks occurred yearly in Yangon (capital city of Myanmar) and some parts of Myanmar[1].

Although mortality is rare in dengue fever (DF) and DHF grade 1 & 2, it is considerable in dengue shock syndrome (DSS)[2]. Thus it is important to predict DSS in dengue infections to be able to provide timely clinical management. In the present study, variables associated with DF, DHF grades 1 & 2 and DSS were subjected to discriminate analysis to obtain predictor variables for DSS.

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MATERIALS AND METHODS

Study population

Cases that were admitted to Yangon Children's Hospital in 1987, with a clinical diagnosis of DHF, who had complete clinical records, and had evidence of recent dengue infection by viral serology (by haemagglutination inhibition test) were studied. There were 51 DF cases, 57 DHF grades 1 & 2 cases and 54 DSS cases. The clinical record sheets of these cases were reviewed and the variables associated with these cases noted. A variable was regarded as present if it was observed at any time during the stay in hospital for DF and DHF grades 1 & 2 cases. As for

DSS cases, a variable was regarded as present if it was observed before the onset of shock or at the earliest moment of occurrence of shock. Gradations of severity of these cases were according to those of World Health Organization guidelines[2].

Laboratory Investigations

Haemagglutination inhibition test was performed according to the method of Clarke & Casals[3] with modification to microtitre technique[4]. Classification of antibody responses as primary or secondary were as described in World Health Organization guidelines[2].

Table 1: Characteristics of DF, DHF grades 1 & 2 and DSS cases included in the study

Characteristics	Dengue Fever	DHF Grades 1 & 2	DSS
Number	51	57	54
Age (yrs)			
– mean	5.4	5.5	4.9
– SD	2.8	3.0	2.6
– range	1-11	0-11	0-12
Sex			
– male	21	24	23
– female	30	33	31
Seroresponse pattern			
– primary	12	4	0
– secondary	39	53	57

Table 2: Pulse rate (centile values) of DF, DHF grades 1 & 2 and DSS cases included in the study

Group	Centile Values			
	25th	50th	75th	95th
DF	108	116	120	132
DHF gr 1 & 2	100	108	120	140
DSS	110	120	130	149

Packed cell volume (PCV) was determined using micro-haematocrit capillary tubes. Platelet count (Plct) was determined as described in WHO guidelines[2].

Discriminant Analysis

As the prime interest was to obtain predictor variables to differentiate shock and non-shock dengue cases, the DF cases and DHF grades 1 & 2 cases were combined as non-shock (NS) group and DSS cases as shock group. Two-group discriminant analysis was then performed by computer aided analysis of the variables associated with these two groups utilising SPSS software. The variables that were included were: drowsiness, vomiting, liver enlargement, pulse rate, abdominal pain, lymphadenopathy, PCV and Plct.

The variables pulse rate, PCV and Plct were converted to dichotomous variables after obtaining biological cut-off values for these for the two groups. To obtain cut-off values, 25th, 50th, 75th and 95th centile values of PCV, pulse rate and Plct of the DF, DHF grades 1 & 2 and DSS cases were calculated first. Then, for PCV and pulse rate, a value of each where approximately 75% of DSS cases will have that value and above and 75% of non-shock cases will have lower than that value were taken as cut-off points. As for Plct, the value where approximately 75% of DSS cases will have that value and below and 75% of non-shock cases will have higher than that value was chosen as cut-off point.

Results

The age, sex and serological response patterns of the studied cases were as shown in Table 1.

The 25th, 50th, 75th and 95th centile values of pulse rate, Plct and PCV of DF, DHF grades 1 & 2 and DSS cases are provided in Tables 2, 3 & 4. Based on these observations, the cut-off values for pulse rate, PCV and Plct were chosen as 115/minute, 42% and 50,000/cu. mm respectively.

Table 5 shows the prevalence (in percentage) of raised pulse rate, presence of drowsiness, presence of liver enlargement, presence of vomiting, presence of abdominal pain and presence of lymphadenopathy in non-shock (NS) group and DSS group.

When the variables drowsiness, liver enlarge-

ment, vomiting, abdominal pain, pulse rate and lymphadenopathy were subjected to discriminate analysis, it was observed that only drowsiness, vomiting, liver enlargement and pulse rate were good predictor variables to differentiate non-shock (NS) group and DSS group. Table 6 shows the classification results of NS and DSS groups, based on discriminant scores, utilising the variables drowsiness, vomiting, liver enlargement and pulse rate. It will be noted that 87% of NS group and 74% of DSS group were correctly classified.

When the variable Plct was included in the above clinical parameters (drowsiness, vomiting, liver enlargement and pulse rate) in the discriminant analysis, it was observed that 90% of NS group and 80% of DSS group could be correctly classified (Table 7).

Table 3: Platelet count (centile values) of DF, DHF grades 1 & 2 and DSS cases included in the study

Group	Centile Values			
	25th	50th	75th	95th
DF	*156	208	250	302
DHF gr 1 & 2	40	58	70	92
DSS	26	36	54	78

* x1000/cu mm

Table 4: PCV (centile values) of DF, DHF grades 1 & 2 and DSS cases included in the study

Group	Centile Values			
	25th	50th	75th	95th
DF	34%	37%	40%	43%
DHF gr 1 & 2	35%	39%	42%	46%
DSS	42%	45%	49%	55%

Table 5: Prevalence (in percentage) of variables studied in NS group and DSS group

Group	Variables					
	Drowsiness	Vomiting	Liver enlargement	Raised pulse rate	Abdominal pain	Lymphadenopathy
NS	10%	81%	30%	48%	38%	85%
DSS	66%	70%	70%	75%	48%	85%

Table 6: Classification results using variables drowsiness, vomiting, liver enlargement and pulse rate

Actual group	No of cases	Predicted group membership	
		NS group	DSS group
NS group	108	94 (87.0%)	14 (13.0%)
DSS group	54	14 (25.9%)	40 (74.1%)

Table 7: Classification results using variables drowsiness, vomiting, liver enlargement, pulse rate and Plct

Actual group	No of cases	Predicted group membership	
		NS group	DSS group
NS group	108	97 (89.8%)	11 (10.2%)
DSS group	54	11 (20.4%)	43 (79.6%)

When the variable PCV was included in the above clinical parameters (drowsiness, vomiting, liver enlargement and pulse rate) in the discriminant analysis, it was observed that 90% of NS group and 93% of DSS group could be correctly classified (Table 8).

Table 8: Classification results using variables drowsiness, vomiting, liver enlargement, pulse rate and PCV

		Predicted group membership	
Actual group	No of cases	NS group	DSS group
NS group	108	96 (88.9%)	12 (11.1%)
DSS group	54	4 (7.4%)	50 (92.6%)

DISCUSSION

The aim of the study was to obtain predictor variables for dengue shock syndrome which has considerable mortality. As the laboratory parameters may not be available in some areas, only the clinical parameters associated with dengue NS group and DSS group were subjected to discriminant analysis as a first step. As shown in Table 6, by observing whether a dengue suspected case has drowsiness or not, vomiting or not, liver enlargement or not and raised pulse rate or not, it will be possible to identify potential DSS cases in 70%. Although a plethora of combination of variables stated above could be presented, on scrutinizing the classification results (which is based on discriminant scores), it can be stated: "If a dengue suspected child has drowsiness associated with vomiting and/or liver enlargement, and/or raised pulse rate, there is the possibility that the child will develop shock. Even if the child is not drowsy, if that child has enlarged liver and raised pulse rate, there is still the possibility that the child will develop shock".

As a second step, the laboratory parameters Plct and PCV were included in the clinical parameters and subjected to discriminant analysis. As shown in Table 7, when Plct was included, it was possible to correctly classify 79.6% of DSS cases. As shown in Table 8, when PCV was included, it was possible to correctly classify 92.6% of DSS cases. On scrutinizing the classification results, it was observed that the correctly classified rates were increased (when Plct and PCV were included in the discriminant analysis) for the following reasons: "If a dengue suspected child is drowsy and has raised PCV or reduced Plct, even without vomiting or liver enlargement or raised pulse rate, there is the possibility that the child will develop shock. Even if the child is not drowsy but associated with reduced Plct, and if the child has liver enlargement or raised pulse rate, there is the possibility that the child will develop shock. Also, even if the child is not drowsy and has no enlargement of liver nor raised pulse rate, but the child has raised PCV, then there is the possibility that the child will develop shock".

In conclusion, if a clinically suspected DHF child presented with 'drowsiness' and one of the following signs/symptoms – vomiting, liver enlargement (2 cm or more), pulse rate 115 minute or more, PCV 42% or more and platelet count 50,000/cu mm or less, then there is the possibility that the child will go into shock. Even if the child is not 'drowsy' but associated with any two of the following signs/symptoms - liver enlargement, pulse rate 115/minute or more, PCV 42% or more and platelet count 50,000/cu mm or less, then there is the possibility that the child will go into shock.

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EVALUATION OF THE USEFULNESS OF PREDICTOR VARIABLES FOR THE PREDICTION OF SHOCK IN DHF CASES

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Summary

Predictor variables for DSS that had been identified by discriminant analysis technique were evaluated for their usefulness in clinical practice. Fifty-two cases of DHF that were admitted to the Yangon Children's Hospital in 1994 were studied. The presence of predictor variables on admission and during the clinical course were noted (i.e. a combination of variables – drowsiness, vomiting, liver enlargement, raised pulse rate, reduced platelet count and raised PCV) and the outcome of these cases were observed. Analysis of the data revealed that the predictor variables for the prediction of shock in DHF have positive predictive value of 36% and negative predictive value of 100%.

INTRODUCTION

Dengue Haemorrhagic Fever (DHF) is one of the major public health problems in Myanmar. Approximately 2,000 cases occur every year in Myanmar and approximately half of these cases occur in Yangon. Although there is no fatality in mild cases of DHF it is considerable in dengue shock syndrome (DSS) cases. Approximately 100 cases die of DSS every year in Myanmar[1]. It is thus evident that it will be of help if predictor variables for the prediction of DSS in DHF cases can be identified. Accordingly, using discriminant analysis technique, an attempt has been made to identify predictor variables for the prediction of DSS in DHF cases[2]. The present study was undertaken to evaluate the usefulness of the identified predictor variables in the prediction of shock in DHF cases in clinical practice.

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MATERIALS AND METHODS

Clinical Studies

90 cases of clinically suspected DHF who were not in the state of shock, admitted to Yangon Children's Hospital between May and October 1994 were observed daily for the presence of the predictor variables until the patient developed shock or was discharged. The predictor variables that were noted were: presence of drowsiness, liver enlargement (two cm or more below the right costal margin), raised pulse rate (115 beats/min or more), raised packed cell volume (PCV) (42% or above) and reduced platelet count (Plct) (50,000/cu mm or less). If the case fulfilled the following criteria the case was regarded as a potential shock case.

Criteria:

1. The child is drowsy and is associated with any of the following:
   - vomiting
   - liver enlargement (two cm or more below the right costal margin)
   - raised pulse rate (115 beats/min or more)
   - raised PCV (42% or above)
   - reduced Plct (50,000/cu mm or less)
2. The child is not drowsy but is associated with any two of the following:
   - liver enlargement (two cm or more below the right costal margin)
   - raised pulse rate (115 beats/min or more)
   - raised PCV (42% or above)
   - reduced Plct (50,000/cu mm or less)

Only those children who had virological evidence of recent dengue infection were analysed. Fifty two cases were available for analysis.

Table 1: Predicted and observed shock and non-shock cases when all the predictor variables were utilised

	Observed		
Predicted	Shock	Non-Shock	Total
Shock	16	28	44
Non-Shock	0	8	8
Total	16	36	52

Table 2: Predicted and observed shock and non-shock cases when all the predictor variables (without platelet count) were utilised

	Observed		
Predicted	Shock	Non-Shock	Total
Shock	15	25	40
Non-Shock	1	11	12
Total	16	36	52

Table 3: Predicted and observed shock and non-shock cases when all the predictor variables (without PCV) were utilised

	Observed		
Predicted	Shock	Non-Shock	Total
Shock	13	22	35
Non-Shock	3	14	17
Total	16	36	52

Laboratory Studies

Haemagglutination inhibition test was performed according to the method of Clarke & Casals[3] with modification to microtitre technique[4]. Grading of severity of DHF and classification of antibody responses as primary or secondary were as described in World Health Organization guidelines[5].

PCV was determined using micro-haematocrit capillary tubes. Plct was determined as described in WHO guidelines[5].

RESULTS

Table 1 shows the predicted and observed shock and non-shock cases when all the predictor variables were utilized. All 16 shock cases were correctly predicted but 28 non-shock cases were also predicted as potential shock cases. None of 8 cases that did not have the predictor variables developed shock.

Table 2 shows the predicted and observed shock and non-shock cases when all the predictor variables (without platelet count) were utilized for the prediction of shock. Only one shock case was missed and 15 shock cases were correctly identified. Twenty five non-shock cases were overdiagnosed as potential shock.

Table 3 shows the predicted and observed shock and non-shock cases when all the predictor

variables (except PCV) were utilized for the prediction of shock. Only 13 shock cases were correctly predicted but 3 shock cases were missed. Twenty-two non-shock cases were also predicted as potential shock cases.

Table 4 shows the predicted and observed shock and non-shock cases when all the predictor variables (except PCV and Plct) were utilized for the prediction of shock. Only 10 shock cases were correctly predicted but 19 non-shock cases were also predicted as potential shock cases. Six of 23 cases that did not have the predictor variables developed shock.

Table 4: Predicted and observed shock and non-shock cases when all the predictor variables (without PCN & PLCT) were utilised

	Observed		
Predicted	Shock	Non-Shock	Total
Shock	10	19	29
Non-Shock	6	17	23
Total	16	36	52

DISCUSSION

Fifty two cases of DHF that were admitted to YCH (in 1994) were observed for the presence of predictor variables and their outcome evaluated. When all the predictor variables (i.e. the clinical parameters – drowsiness, vomiting, liver enlargement, raised pulse rate; and the laboratory parameters – PCV and Plct) were utilized, it was observed that all 16 shock cases could be correctly predicted (i.e. positive predictive value of 36%) but 28 non-shock cases were also predicted as potential shock cases (see Table 1). In clinical practice it is better to overdiagnose potential shock cases and then treat them energetically than to underdiagnose and miss the potential shock cases. Another important finding is that none of the 8 cases that had no predictor variables developed shock (i.e. negative predictive value of 100%). Thus, clinicians could confidently exclude potential shock cases until the child fulfilled the stated criteria for identifying potential shock.

In some clinical settings, although PCV is available, platelet count may not be available (e.g. during non-office hours).

In such a situation, 15 potential shock cases were correctly identified, but one potential shock case was missed. Twenty five cases were also overdiagnosed as potential shock cases. Thus positive predictive value was calculated to be 37%. Out of 12 cases who did not have the predictor variables, one developed shock. Thus negative predictive value was lowered to 91% (see Table 2).

To determine PCV, micro-haematocrit tubes and a micro-centrifuge would be required, and these may not be available in some hospitals. Thus PCV was excluded from the criteria and the cases were reassessed for identification of potential shock. Without PCV, only 13 shock cases could be correctly predicted and 3 shock cases were missed. Twenty-two non-shock cases were also predicted as potential shock cases (see Table 3). Thus, approximately one third of cases that had predictor variables will develop shock (i.e. positive predictive value of 37%) and one fifth of cases (18%) that did not have the predictor variable will develop shock (i.e. negative predictive value of 82%).

In some remote areas, PCV and platelet determinations may not be possible and the physician may have to rely only on the clinical criteria. In such situations, only 10 shock cases could be correctly predicted but 19 non-shock cases were also predicted as potential shock cases (see Table 4). However, 6 cases that did not have the predictor variables developed shock. Thus, approximately one third of cases that had predictor variables will develop shock (i.e. positive predictive value of 34%) and one fourth of cases (26%) that did not have the predictor variable will develop shock (i.e. negative predictive value of 74%).

In summary, positive predictive values did not change markedly whether all the predictor variables or only the clinical parameters were utilized, in the prediction of shock. It ranged between 34% to 37%. However, if all the predictor variables were utilized, none of the

shock cases will be missed. If platelet count is not included in the criteria, 6% of the potential shock cases could be missed. If PCV is not utilized, 18.7% of the shock cases could be missed. If both PCV and platelet count are not available, and the prediction is based on the clinical parameters only, then 37.5% of the shock cases could be missed.

CONCLUSION

1. Predictor variables identified by discriminant analysis technique could:

   - Select all potential shock cases. However, one should bear in mind that 64% will be overdiagnosed as potential shock cases.

   - Confidently exclude potential shock if a DHF suspected child does not have the stated predictor variables (negative value of 100%).

2. PCV and platelet count are of immense value not only in case management but also in identifying potential shock cases; without these parameters some potential shock cases might be missed.

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INFECTIONS AND INFESTATIONS IN THE SKIN, HAIR AND NAILS

K H Lee, MBBS (Sing), MCGP (Sing), M Med (Fam Med)

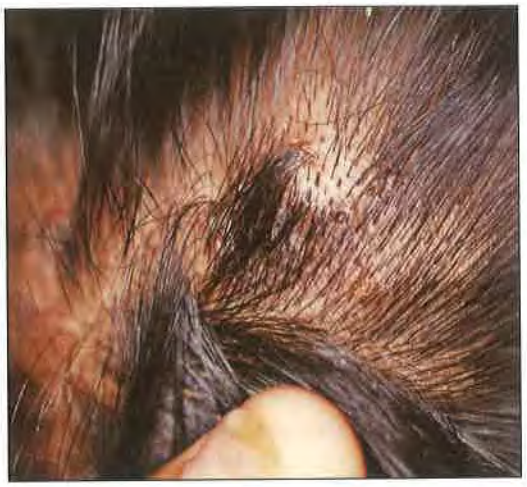
INTRODUCTION

Infections and infestations of the skin, hair and nails are easy to miss in a busy family practice. Symptoms are frequently under-reported or down-played by patients. Many patients ignore or self-medicate for their "rash". The wide array of over-the-counter oils, creams and ointments in the pharmacies and the traditional medical halls attest to this. The photographs presented in this article are more picturesque cases that were seen in an average general practice clinic over a one-month period.

Head Lice

This 12 year old girl presented with a scalp rash. On examination, an excoriated eczematous plaque was seen. Occipital nodes were palpable and closer examination of the hair showed nits that were attached to the hair and close to the scalp. The condition cleared with an application of malathion lotion (Derbac-M) and removal of dead lice and nits with a fine toothed comb. It is important to use the nit comb while the hair is wet.

Pediculosis capitis is more common in school children. It had been found to be 19 times more common in girls than boys[1]. It is no longer a disease associated with poor hygiene. It is not uncommon to have patients who belong to the higher social-economic class who live in clean, modern housing. It is transmitted from person to person (probably hair to hair contact) which occurs in ordinary close-proximity interactions. Unfortunately there is still much prejudice and social stigma associated with this condition. Physicians should be tactful and cautious when considering contact tracing which is frequently rewarding. The aim of treatment is to kill all the insects and their eggs. Re-infection and incorrect application of lotions and shampoos are common causes of treatment failure. Of the many insecticides available, permethin is apparently the most effective while the less effective lindane and natural pyrethines are no longer recommended[2].



Scabies

This 3 year old child was brought by the mother who was concerned that the child was frequently scratching his genitals. What brought her to the clinic was when she found two nodules on the scrotum. Examination showed that they were

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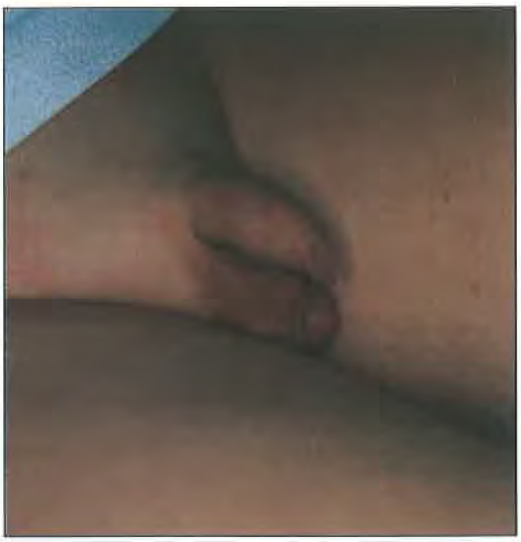
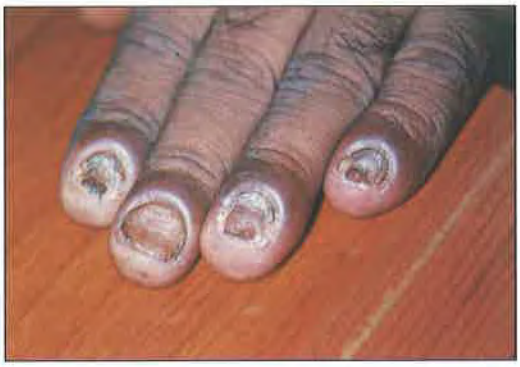


scabetic nodules. The child also had many erythematous papules in the groin area. Similar papules were also seen when the hands were examined. The mother was surprised when these were brought to her attention. She had not noticed the mundane-looking rash. The child was successfully treated with an application of malathion lotion and a sedative antihistamine at night. The nodules persisted and the parents accepted the recommendations that they are best left alone.

The trick in diagnosing scabies is to look for it. It is often missed and complicated by treatment with steroid creams. Diagnosis is even more challenging in a child with atopy. Looking for "burrows" or doing skin scraping is always advocated in textbooks but are of limited practical value in general practice. It is more useful to look for pruritic papules on the hands and groin. Diagnosis is easier if the condition is long-standing and pruritus is more prominent at night. It was often thought that it spares the face. This is not a reliable guide[3]. Involvement of the face does not exclude the diagnosis, especially in children. The challenge in management is in convincing the patients that the rash is caused by an "invisible insect" and not due to allergy or "poison in the blood". It is also difficult to tell them that your treatment will not relieve the itch or make the rash go away immediately. The job is easier in long-standing cases who have tried various remedies when you tell them that your treatment my cure them once and for all. The suffering inflicted by scabies on young children is very high[4]. Such a highly treatable disease should not be missed.

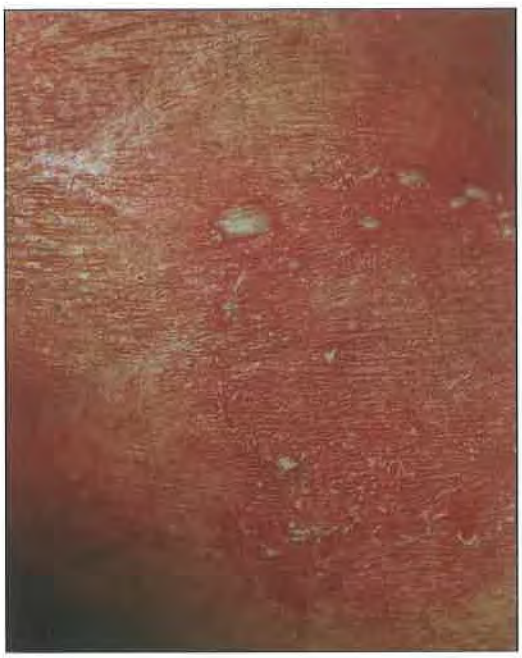
Tinea Unguium



This 49 year old newspaper vendor is a regular patient who is on follow-up for hypertension. His discoloured nails were noticed during blood pressure measurement. All ten fingers were involved but the toes were spared. The nails were dystrophic and crumbling with horizontal ridging. There was also an indolent paronychia. The patient was not bothered by the nails which is not unusual. Patients are either totally nonchalant like this gentleman or terribly upset and embarrassed by their unsightly nails. For patients who are more concerned about their appearance, nails are an important part of their body image. Their complaints should not be trivialised. Nails are also functional. This becomes very obvious when you have an itch or when you try to peel off a plaster. Jewellers, watch-repairman, tailors and even the concert guitarist may find it difficult to work with nails like those shown in the photograph. This gentleman politely refused treatment for his nail condition. The nails may be infected by different types of fungus. It may be a dermatophyte, a mould, Candida spp, or it may even be a mixed infection. Treatment is very difficult. Unlike many common diseases, fungal nail infection is not self-limiting. It spreads from digit to digit and goes on for years. The fungus is well entrenched and the damaged, slow growing nail has no chance of "outgrowing" the fungus. Antifungal lotions are effective only for treating the parony-chia. Newer lacquer applications have not been as effective as hoped for. Pulse

therapy (one week on, three weeks off, for 2 or 3 pulses) with oral antifungal agents such as itraconazole may be preferable especially in mixed infections[5]. Whatever the period, the treatment period is long because nail growth is slow. As a result compliance is difficult to ensure. Cost of the medicine is also prohibitively high for some.

Secondary Bacterial Infection in a Patient with Atopic Eczema



A 55 year old man with a history of atopic eczema presented with an exacerbation of his condition. On closer inspection, some of the lesions were found to have vesicles that were filled with pus. The lesions improved rapidly with a course of erythromycin ethinylsuccinate 800 mg bd together with the use of a mild soap, emollient and steroid cream.

Secondary bacterial infection is increasingly recognised as a cause of treatment failure in the management of eczema. Such lesions are said to be "impetiginised" as the lesions resemble impetigo superimposed upon a background of eczema. The offending organism is usually Staphylococcus aureus or group A beta-haemolytic streptococcus. Mixed infections are also possible. Adding a suitable antibiotic that covers both these organisms would frequently help to clear up the eczema. In the West, there are reports of increasing Staphylococcal resistance to erythromycin. Recent studies have also suggested that Staphylococcus aureus may play the role of a super-antigen and contribute to the disease process of atopic eczema itself[6].

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TEST YOUR EYE-Q (NO. 2)

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AN INCIDENTAL OPHTHALMOSCOPIC FINDING

A 61-year-old woman with a history of non-insulin-dependent diabetes mellitus for five years was screened for diabetic retinopathy. She had no visual or ocular symptoms.

Her visual acuity was normal in both eyes. Her ocular fundus photographs are as shown in Fig 1a & 1b. No other abnormality was detected on ophthalmic and systemic examination.

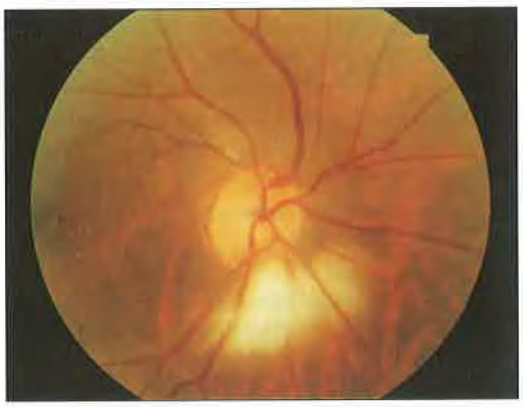

Figure 1a: Right eye

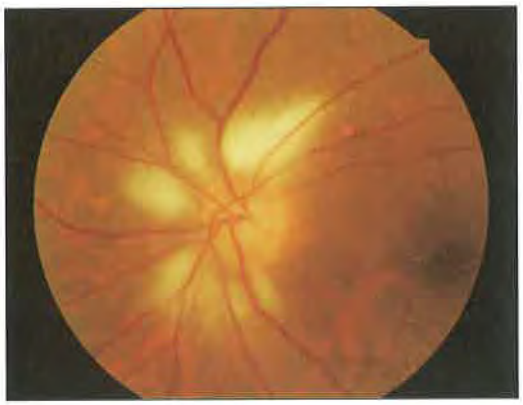

Figure 1b: Left eye

1. What do Fig 1a & 1b show?
2. What is the histopathology of these lesions?
3. What are its differential diagnoses?
4. Are these lesions usually symptomatic?
5. Do these lesions affect visual acuity?

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For answers to Eye-Q, please turn to page 41

ANSWERS TO 'TEST YOUR EYE-Q (NO. 2)

1. The figures show several peripapillary white patches of myelinated (medullated) nerve fibres with frayed and feathered edges in a striate configuration coincident with the retinal nerve fibres. The vessels that pass within the superficial layer of the myelinated nerve fibres are partially obscured by the lesions.

2. Myelinated nerve fibres consist of intraocular investment of the axons of retinal ganglion cells with a myelin sheath formed by oligodendrocytes. Usually, myelination of these axons is limited to the optic nerve. The myelination begins at the lateral geniculate body and ordinarily cease at the lamina cribrosa. Occasionally, however, some of the retinal nerve fibres acquire a myelin sheath in the first month of life.

3. (a) Cotton wool spots (sometimes called soft exudates or cytoid bodies). These are found in diabetic retinopathy, hypertensive retinopathy, acquired immune deficiency syndrome and collagen vascular disorders (dermatomyositis, systemic lupus erythematosus, polyarteritis nodosa and, rarely, scleroderma)

   (b) Swollen optic disc. Myelinated nerve fibres at the optic disc margin may be mistaken for optic disc swelling.

4. No. They are almost invariably noted on routine examination of the ocular fundus.

5. The visual acuity is usually good unless the macula is involved. Relative or absolute scotomas may be present and correspond to the areas of myelination. No treatment is necessary.

ECG QUIZ

C C Koo, MBBCh (Belfast), MRCP (UK), FAMS, FRCP (Edinburgh)

A 47 year old man, who had an extensive anterior myocardial infarction six months ago, walked into the clinic feeling "well" without any dizziness, chest discomfort, palpitations nor undue breathlessness. He could still manage with his hectic schedule. However, his pulse was irregular and fast at 132 bpm. JVP was not raised. The blood pressure was 100/70 mm Hg and the lungs were clear.

(a) What is the cardiac rhythm?

(b) How serious is thus cardiac rhythm, if left untreated?

(c) How would you further manage this patient?

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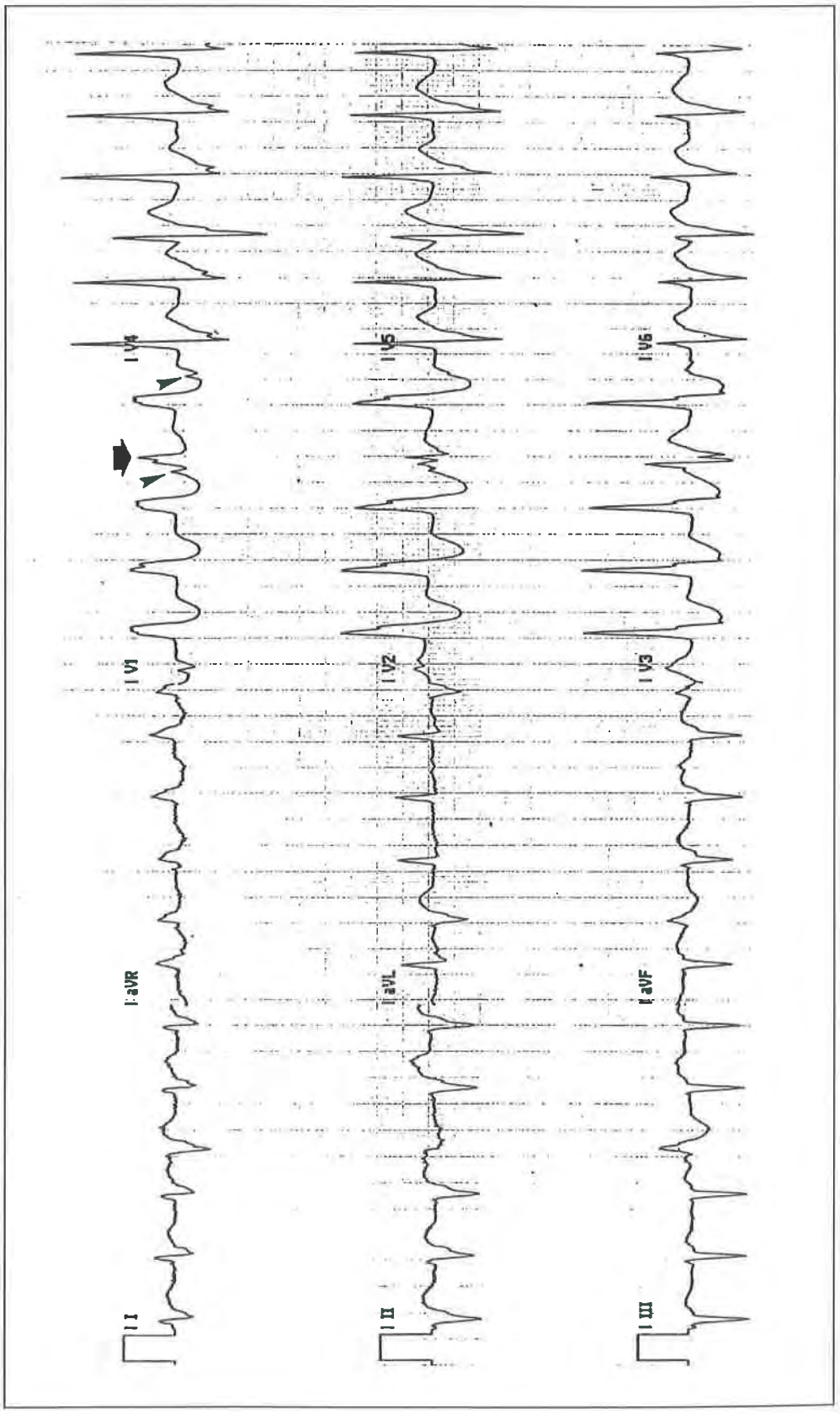
For answers to ECG Quiz, please turn to page 44 & 46

ECG I



ANSWERS

ECG I

(a) The cardiac rhythm is **monomorphic ventricular tachycardia**. Firstly, the heart rate is fast at 136 bpm. **Tachycardia** indicates a heart rate of more than 100 bpm. Secondly, the QRS complexes are broad or wide i.e. equal or more than three "little squares" or ≥120 msec. His QRS complexes are 3 $^{1}/_{2}$ little squares or 140 msec. Most of the QRS complexes are of the same shape or morphology i.e. **monomorphic**. The QRS complexes have right bundle branch block (**RBBB**) pattern. The *differential diagnosis of* ***wide complex tachycardia is supraventricular tachycardia with aberrancy or antegrade conduction via the accessory pathway***. The latter is rare. **Ventricular tachycardia with RBBB pattern** has the following features:

(1) Superior cardiac axis or left axis deviation (see the predominant negative deflection (S wave) in the inferior leads II, III and AVF,

(2) monophasic R wave in V1,

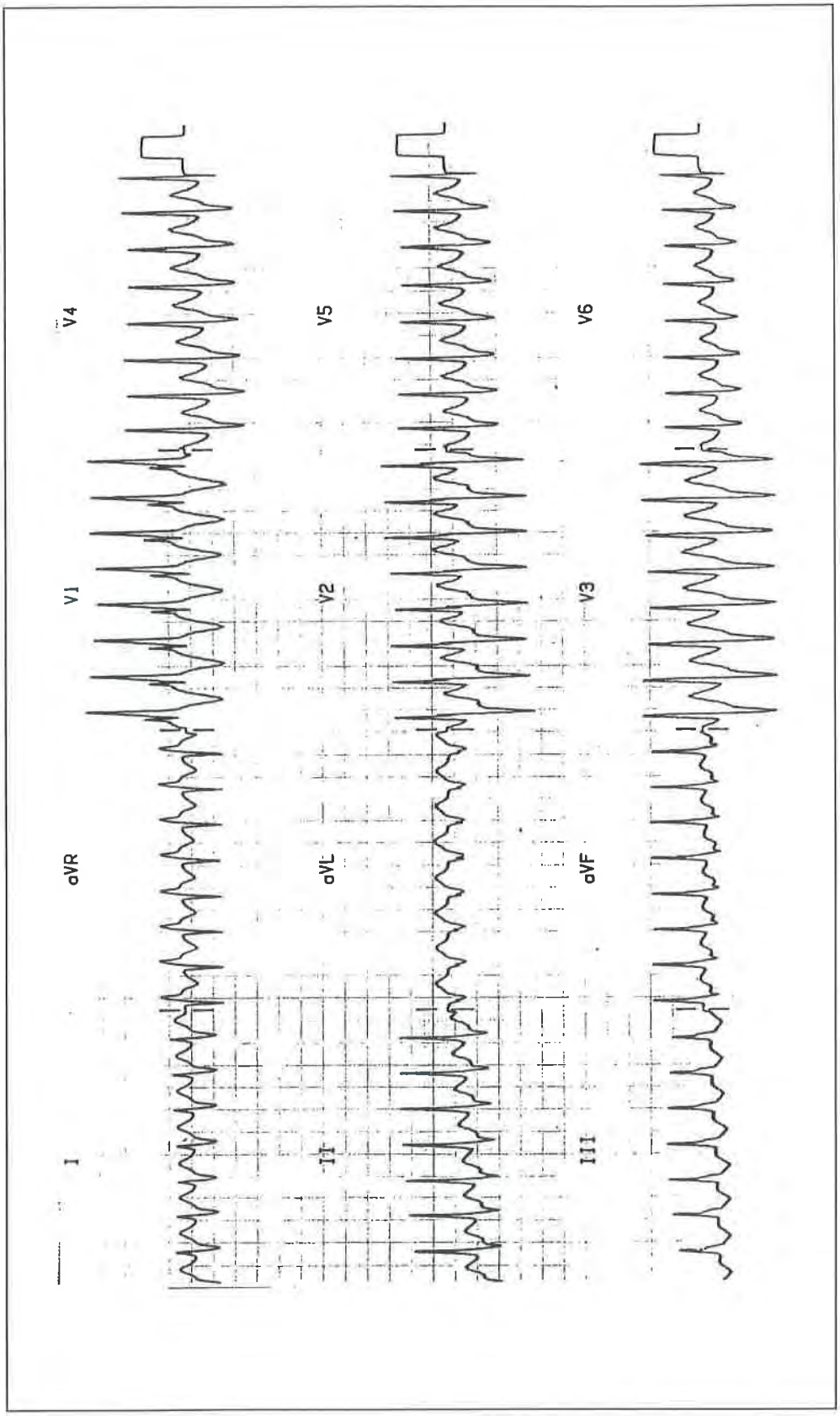
(3) R wave is of smaller amplitude than the corresponding S wave in V6,

(4) AV dissociation (see the "p" waves before the 4th and after the 5th QRS complexes in V1). This is unrelated to the corresponding QRS complexes, and

(5) ventricular fusion beats (see the 4th QRS complex). The latter indicates partial depolarisation of the ventricle by the sinus beat conducting down the normal AV node-His Purkinje pathway and the ventricular tachycardia focus.

Whereas, in **supraventricular tachycardia with RBBB pattern** (see **ECG II**), the origin of the rhythm disorder is from the atrium or the AV junction. Firstly, the cardiac axis is either normal or inferior/right. Secondly, the QRS complexes in V1 is triphasic i.e. rSR pattern. Thirdly, the R wave in V6 is taller than the corresponding S wave.

ECG II



ANSWERS

ECG II

(b) Every attempt must be made to convert ventricular tachycardia to sinus rhythm. Untreated, these patients with coexisting impaired LV function will be complicated with clinical left ventricular failure, while others may die suddenly from ventricular fibrillation!! There are three choices of treatment for ventricular tachycardia.

(1) Chemically with medications,

(2) pacing and

(3) electric shock with DC cardioversion.

The choice of treatment depends on whether the patient is haemodynamically stable and the availability of treatment. If the patient is conscious like this case, the first choice is to admit the patient for ECG monitoring and to give the patient a bolus dose of intravenous lignocaine. If this fails and the patient is still haemodynamically stable, the next drug of choice is intravenous procainamide. Beware, as this drug has a significant negative inotropic side effect and the blood pressure may drop significantly! The alternative is to use intravenous amiodarone but this takes a longer time to act. In selected cases, the ventricular tachycardia is paced-terminated by inserting a pacing wire into the right ventricle. This is seldom performed unless there are adequate facili-ties or skilled doctors. Furthermore, pacing is not consistently effective to terminate ventricular tachycardia. It is contraindicated in patients with polymorphic ventricular tachycardia. In 25% of cases, pacing accelerates the ventricular tachycardia with haemodynamic compromise requiring DC cardioversion! Lastly, in patients with ventricular tachycardia with haemo-dynamic compromise, the only logical treatment is DC cardioversion.

(c) Ventricular tachycardia in the presence of structurally abnormal heart i.e. previous myocardial infarction, indicates that the patient is at high risk for future sudden cardiac arrhythmic death. Hence, the *goals of treatment* are to

(1) identify and ablate the focus of ventricular tachycardia (catheter or surgical ablation),

(2) prevent future recurrences of ventricular tachycardia using anti-arrhythmic drugs,

(3) convert ventricular tachycardia to sinus rhythm using anti-arrhythmic drugs or automatic implantable cardioverter and defibrillator (AICD).

In patients with impaired LV function, anti-arrhythmic drugs can be potentially dangerous and lethal. They can accelerate the rate or increase the episodes of ventricular tachycardia!!

REMEMBER:

(1) Patients with ventricular tachycardia can be relatively well as indicated with this case. However, for most patients with ventricular tachycardia, they are *symptomatic* with palpitations associated with sweating, breathlessness, chest discomfort, dizziness and syncope.

(2) It is important to recognise ventricular tachycardia based on ECG criteria. If in doubt, always treat any wide complex tachycardia as ventricular tachycardia until proven otherwise.

(3) Always treat ventricular tachycardia with respect as the next recurrence can be fatal.

(4) Management of ventricular tachycardia post myocardial infarction is often complicated.

RISK FACTORS FOR MYOCARDIAL INFRACTION

M Shah, MBBS (S'pore), M Med (Fam Med)

Upon waking up in the CCU, 2 questions are often asked by the cardiac patient:

1. "Where am I?"
2. "How did I get here?"

The first answer is easy, but the second one is difficult to answer in one breath. This is due to the complex events that precede the acute myocardial infarction (AMI) and due to the fact that the events start in the early years of a person's life.

Casual agents in a person's life cycle include physiological concerns (risk factors) and psychological, sociological and personality factors.

"An understanding of what has occurred before must be reached before understanding what is now."

PHYSIOLOGICAL FACTORS/ RISK FACTORS

Primary: Those capable of being altered or controlled and those which have a major influence on the progression of the atherosclerotic process e.g. hypertension, hyperlipidaemia, smoking.

Secondary: Those which can be controlled and those over which there is no control e.g. obesity, diabetes, hereditary, lack of exercise, age, sex, stress.

Registrar
Primary Health Division
Ministry of Health
Singapore

The above definitions have been made based on their effects on atherosclerosis. Our usual definitions are ***irreversible*** (age, sex, family history, race) and ***reversible*** (obesity, smoking, lack of exercise, hypertension, hyperlipidaemia, diabetes, stress).

1. Hypertension

- Present in about 50% of patients with AMI and 61% of patients with stroke.
- American Heart Committee criteria:

males < 45 years	BP > 130/90
males > 45 years	BP > 140/95
females	BP > 160/95

- WHO criteria: BP > 160/95
- two times increased risk of cardiovascular disease in patients with BP between 140/90 and 160/95, according to the Framingham study.
- Management must include lifestyle modification (reduce obesity, stress, smoking, and increase exercise), diet modification (low salt diet) and drug treatment when necessary.

2. Hyperlipidaemia

- AHA Committee report states that there is a more than 5 times increased risk related to a range of cholesterol levels in the blood, in under 50 year olds.
- *Recommendations:*
  a. restrict caloric intake for the obese,
  b. reduce saturated fat intake to about 8-10% of food energy, and restrict cholesterol to less than 300mg/day,

c. increase intake of foods rich in gel-forming fibre, such as pectin, and

d. have a large proportion of dietary protein derived through vegetable sources.

3. *Smoking*

- "There is really nothing positive that can be said for smoking from a general health, cancer, coronary heart disease, or financial point of view."
- 3 times increased risk of CHD for those smoking more than 20 cigarettes per day
- *Biological mechanisms:*

  a. Nicotine stresses the heart by increasing heart rate and BP.

  b. Carbon oxide (CO) competition with oxygen for haemoglobin (Hb) reduces the heart's ability to respond to stress because there is less oxygen available for myocardial muscles. As a result, the strength of contraction decreases.

  c. CO and nicotine increase platelet stickiness, increasing the tendency for blood clotting +/- occurrence of thrombosis.

  d. Stimulation of red blood cell (RBC) manufacture makes the blood thicker.

  e. Increased tendency for heart to beat arrythmically.

  f. Regular smoking is related to high cholesterol levels in the blood, resulting in increased risk of AMI.

- International Society and Federation of Cardiology (ISFC) Committee reported that once smoking is *ceased*:
  - Risk of fatal reinfarction or sudden death is reduced by 20-50%.
  - Non-fatal reinfarction rates may be reduced.
  - Benefits apparent in first five years after myocardial infarction (MI), and adverse effect of continuing smoking is still does-related.

4. *Hereditary Factors*

- Genetic make-up and predisposition is irreversible, but environmental modification in those predisposed is possible.
- These people require preventive approach early to make positive control of risk factors for coronary heart disease (CHD) a family affair.

5. *Diabetes and Hyperglycaemia*

- Risk of MI is increased 2 times in males and 3 times in females with diabetes.
- Control of diabetes alone is not enough to reduce risk, and control of other risk factors is necessary to reduce risk due to diabetes.

6. *Physical Activity*

- Regular exercise improves functional capacity of cardiovascular system and reduces myocardial oxygen demand.
- Moderate cardiopulmonary exercise (walking, jogging, swimming, cycling)

Table 1: Patients at Increased Risk of Reinfarction and Death

Mortality rate	in first 6 months	16.0%
	in first 3 months	12.8%
Reinfarction rate	during the first year	19.0% in males 34.0% in females

Table2: Cumulative Mortality Rates in Post-MI Patients (%)

Time after AMI	Males	Females
30 days	16	28
1 year	19	34
2 years	22	38
3 years	26	40
4 years	31	48
5 years	36	51
6 years	38	54

Framingham Study

is required, that is 3 to 5 times a week, for half an hour or more.

Sudden death is defined as death within 24 hours of onset of illness/injury (WHO).

Sudden death in CHD is defined as death within first hour of onset of symptoms. (Moss) (i.e. symptoms of cardiac arrest or AMI)

60% of coronary artery deaths occur in the first hour.

Other causes of sudden death in CHD patients include:

a MVP, HOCM
b. Aortic aneurysm
c. Pulmonary thromboembolism
d. Cerebral haemorrhage
e. Dysrhythmias.

Identification of high-risk patients is necessary for secondary prevention.

Reduction of mortality and non-fatal reinfarction can be achieved by special measures, e.g. CABG, PTCA, streptokinase infusion, heart transplant, cardiac defibrillation, new pharmacological agents, programmes geared towards reduction of modifiable risk factors.

Major contributors found as increasing chances of mortality and reinfarction, and their assessment, are as follows:

a. extent of AMI (ECG, coronary angiography, 2D echo: dyskinetic segment),
b. extent of ventricular dysfunction (ventriculography, 2D echo-LVEF),
c. ventricular arrhythmias (ECG, stress ECG),
d. progression of disease (identified by exercise tolerance testing), and
e. modifiable risk factors (smoking, hypertension, hyperlipidaemia, lack of exercise).

These are all associated independently with increased mortality and reinfarction.

SOCIOLOGICAL FACTORS

1. People in higher socioeconomic classes have a higher incidence of CHD.
2. High occupation/status jobs are associated with higher incidence of CHD.
3. Incongruity in social and employment factors leads to higher incidence of CHD e.g.s wife more educated, more than three jobs per 20 years, excessive moving, job expectations greater than educational ability.
4. Dissatisfaction in life increases risk of CHD e.g. job difficulties, job dissatisfaction, lack of recognition by supervisors, lack of leisure and social activities, long-term frustrations, marital stress, monotony of life, prolonged illness or death in family.

Stress

Stress is defined as any potential situation that places special physical or psychological demands on a person, thus destroying the equilibrium.

Stress not only has negative impacts on health, but positive as well, such as improved performance, competitive spirit, and problem solving.

Changes due to stress include anger, rage, frustration, and emotional struggle; and these cause increased blood pressure, heart rate, cholesterol, and insulin/other hormones.

The aim in management is not to remove stress but to control the stressors by modification programmes.

CORONARY PRONE PERSONALITY

"Type A behaviour is an *action-emotion complex* that can be observed in any person who is *aggressively* involved in a chronic incessant struggle *to achieve more and more* in less and less time ... against the opposing efforts of other things or other persons."

Friedman & Rosenman

"Coronary prone behaviour is an overt behaviour syndrome on style of living characterized by extremes of competitiveness, striving for achievement, aggressiveness (sometimes stringently repressed), haste, impatience, restlessness, hyper alertness, explosiveness of speech, tenseness of facial musculature, and feelings of being under pressure of time and under the challenge of responsibility."

- Such persons are deeply committed to their profession/vocation.
- Not all aspects need to be present to be classified as coronary-prone behaviour.

- The pattern is the reaction of a characteristically predisposed person to a situation which challenges him.

Jenkins

Coronary-prone type A behaviour is significantly associated with:

- the incidence of AMI,
- unrecognised MI,
- angina
- recurrent and fatal MI,
- increased deposition of atherosclerotic materials in coronary arteries.

Western Collaborative Group Study

The basis for depression in post-MI patients is the threat of invalidism and a subsequent loss of autonomy and independence. There is no better approach to restoring sense of self-esteem and removing depression than to get the patient active again.

Hackett & Cassem

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* Normally the text should not exceed 2000 words and the number of illustrations should not exceed eight.

  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.

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* 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.

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* Introduction: State clearly the purpose of the article.

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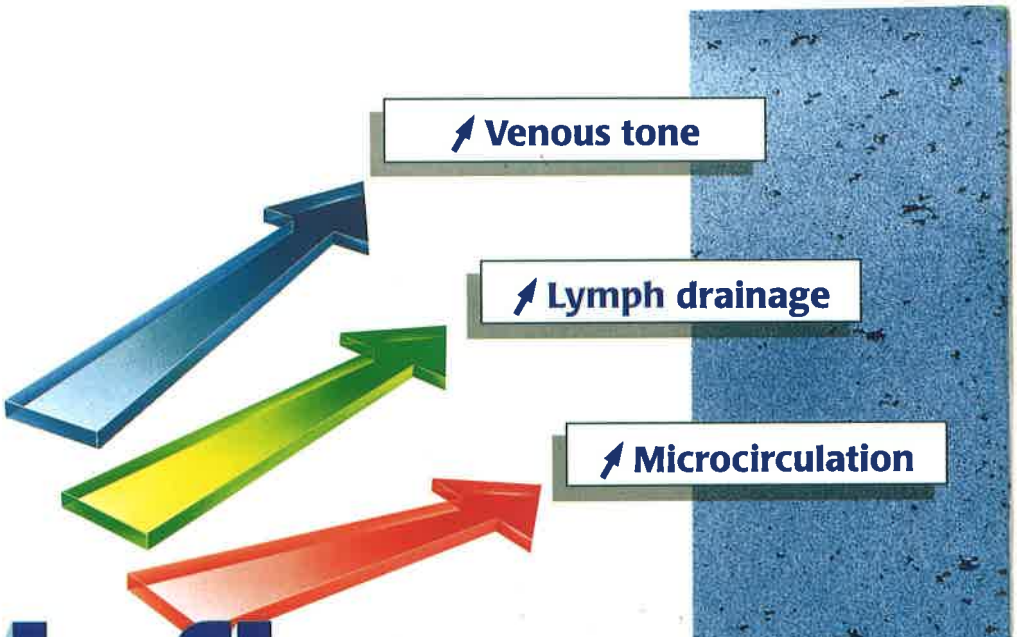
  Drugs must be referred to generically; all the usual trade names may be included in parentheses. Dosages should be quoted in metric units.

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

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

* Results: Present results in logical sequence in the text, tables and illustrations.



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