THE COLLEGE OF FAMILY PHYSICIANS SINGAPORE

The SINGAPORE FAMILY PHYSICIAN

FAMILY PRACTICE RHEUMATOLOGY

- Low Back Pain
- Osteoarthritis
- Osteoporosis
- DMARDs

ISSN 0377-5305

Vol. XX NO. 1
JAN/MAR 1994

NICOTINELL® TTS

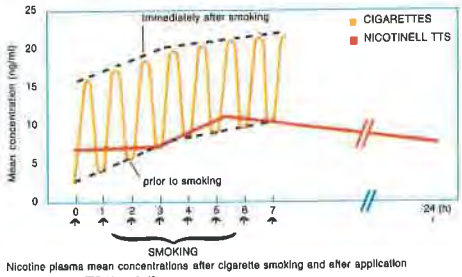
NICOTINE

The first nicotine patch treatment designed to overcome the problems of tobacco withdrawal.

►► ***Unique*** *patch administration of low dose nicotine, to help smokers overcome the agony of tobacco withdrawal.*

►► ***Discreet*** *and easy to use with once daily application which helps to counteract the often automatic search for a nicotine source.*

►► ***Controlled*** *continuous release of low dose nicotine avoids the peaks and troughs seen with cigarette smoking.*



Nicotine plasma mean concentrations after cigarette smoking and after application of NICOTINELL TTS (day 10) (3)

►► ***Impressive*** *abstinence rates both with and without a specialist psychological support programme.*[1,2]

►► ***Simple*** *3 month step-by-step treatment plan with 3 different patch strengths so you can individualise the dose to each smoker's needs.*

►► ***Supportive*** NICOTINELL ***Stop Smoking Programme*** *offering extra support for smokers who want to kick the habit.*

Presentation: Transdermal Therapeutic System containing nicotine, available in 3 strengths (Nicotinell TTS 10, Nicotinell TTS 20, Nicotinell TTS 30) releasing approximately 0.7 mg/cm^2/24 hours. **Indications:** Treatment of nicotine dependence, as an aid to smoking cessation. **Dosage:** The subject should stop smoking completely when starting treatment with Nicotinell TTS. Treatment should be initiated with Nicotinell TTS 30 cm^2 or 20 cm^2 depending on the number of cigarettes smoked per day. For those smoking more than 20 cigarettes a day it is recommended to start treatment with Nicotinell TTS 30 cm^2 once daily. Those smoking less could start with Nicotinell TTS 20 cm^2. Sizes of 30 cm^2, 20 cm^2, and 10 cm^2 are available to permit gradual withdrawal of nicotine replacement using treatment periods of 3–4 weeks. Total treatment periods of more than 3 months and doses above 30 cm^2 have not been evaluated. **Contraindications:** Non-smokers, children, and occasional smokers. As with smoking, it is contraindicated in pregnant and breast-feeding women, acute myocardial infarction, unstable or worsening angina pectoris, severe cardiac arrhythmias, recent cerebrovascular accident, diseases of the skin, and known hypersensitivity to nicotine. **Precautions:** Hypertension, stable angina pectoris, cerebrovascular disease, occlusive peripheral arterial disease, heart failure, hyperthyroidism or diabetes mellitus, peptic ulcer, renal or hepatic impairment. Persistent skin reaction to the patch. To be kept out of the reach of children at all times. **Adverse reactions:** Smoking cessation is associated with withdrawal symptoms. The most frequently reported adverse events in controlled clinical trials regardless of any causal association with study drug were: reaction at application site (usually erythema or pruritus), headache, cold and flu-like symptoms, insomnia, nausea, myalgia, and dizziness. Less common: blood pressure changes, other central nervous system effects, and gastrointestinal disturbances. See full prescribing information. **Packs:** 28 systems. **Forensic Classification:** Prescription-Only-Medicine. Full prescribing information is available on request. **CIBA-GEIGY S.E. ASIA (Pte) Ltd,** 4 Fourth Lok Yang Road, Singapore 2262 Toll-Free Nos. 2664285/6

The College of Family Physicians Singapore

14th COUNCIL 1993/1995

Acting President	Dr Alfred W T Loh
Censor-in-Chief	Dr Goh Lee Gan
Hon Secretary	Dr Arthur Tan Chin Lock
Hon Treasurer	Dr Soh Cheow Beng
Council Members	Dr Choo Kay Wee
	Dr Huan Meng Wah
	Dr Lim Lean Huat
	Dr Richard Ng Hong Hoo
	Dr Wong Song Ung
Hon Editor College Journal	Dr Moti H Vaswani

BOARD OF CENSORS

Censor-in-Chief	Dr Goh Lee Gan
Censors	Dr James Chang Ming Yu
	Dr Lim Kim Leong

CONTINUING MEDICAL EDUCATION COMMITTEE

Chairman	Dr Richard Ng Mong Hoo
Secretary	Dr Huan Meng Wah
Ex-Officio	Dr Soh Cheow Beng
Members	Dr Goh Lee Gan
	Dr Hia Kwee Yang
	Dr Omar bin Saleh Talib
Library	Dr Chan Cheow Ju
	Dr Chong Hoi Leong
	Dr Huan Meng Wah

RESEARCH COMMITTEE

Chairman	Dr Choo Kay Wee
Secretary	Dr Bina Kurup
Ex-Officio	Dr Koh Eng Kheng
Members	Dr Paul Chan Swee Mong
	Dr Shanta C Emmanuel
	Dr Goh Lee Gan
	Dr Hong Ching Ye
	Dr Kevin Koh
	Dr Lee Pheng Soon
	Dr Alfred Loh Wee Tiong
	Dr Wong Song Ung

UNDERGRADUATE TEACHING COMMITTEE

Chairman	Dr Lim Lean Huat
Secretary	Dr Kevin Koh
Ex-Officio	Dr Koh Eng Kheng
Members	Dr Goh Lee Gan
	Dr Richard Ng Hong Hoo
	Dr Wong Song Ung

PRACTICE MANAGEMENT COMMITTEE

Chairman	Dr Huang Meng Wah
Secretary	Dr Goh Lee Gan
Ex-Officio	Dr Koh Eng Kheng
Members	Dr Ganesh Balasundram
	Dr Choo Kay Wee
	Dr Tan Chek Wee

PUBLICATIONS COMMITTEE

Chairman	Dr Moti H Vaswani
Secretary	Dr Goh Lee Gan
Ex-Officio	Dr Alfred W T Loh
Members	Dr Choo Kay Wee
	Dr Huan Meng Wah
	Dr Arthur Tan Chin Lock

FINANCE COMMITTEE

Chairman	Dr Soh Cheow Beng
Secretary	Dr Lim Leah Huat
Ex-Officio	Dr Koh Eng Kheng
Members	Dr Paul Chan Swee Mong
	Dr Leong Vie Chung
	Dr Frederick Samuel
	Dr Wong Heck Sing

SECRETARIAT

Administrative Secretary	Ms Sonia Fam
Asst Admin Secretary	Ms Sandy Ler
Chief Clerk	Ms Rose Hoon
Clerk	Ms Najmunisa

EDITORIAL BOARD

Hon. Editor	Dr Moti H Vaswani
Members	Dr Choo Kay Wee
	Dr Goh Lee Gan
	Dr Huan Meng Wah

Improved mobility – improved quality of life

®VOLTAREN

The antirheumatic agent

Presentation: Diclofenac sodium: tablets of 25 mg and 50 mg; sustained-release tablets of 100 mg; suppositories of 12.5 mg, 25 mg, 50 mg, and 100 mg; ampoules of 75 mg/3 ml. **Indications:** Inflammatory and degenerative forms of rheumatism. Acute musculo-skeletal disorders. Acute gout. Post-traumatic and post-operative inflammation and swelling. Painful and/or inflammatory conditions in gynaecology, e.g. dysmenorrhoea. Renal and biliary colic (ampoules). As an adjuvant in severe painful inflammatory infections of the ear, nose, or throat. (Fever alone is not an indication). **Dosage:** Depending on the indication 75-150 mg/day (dysmenorrhoea: up to 200 mg). Ampoules: 1 or at the most 2 per day as initial or acute therapy for not more than 2 days. Children: 0.5-3 mg/kg/day. See full prescribing information. **Contra-indications:** Peptic ulcer, known hypersensitivity to the active substance, acetylsalicyclic acid, or other prostaglandin-synthetase inhibiting drugs. Known hypersensitivity to sodium metabisulphite or other excipients (ampoules). Proctitis (suppositories). **Precautions:** Symptoms/history of gastro-intestinal disease, impaired hepatic, cardiac or renal function. Pregnancy. Porphyria. Cautious use in elderly. Patients with extracellular volume depletion from any cause. Patients on diuretics, anticoagulants, or antidiabetics. During prolonged treatment, periodic monitoring of liver function should be carried out and blood counts are recommended. Possibility of hypersensitivity reactions to sodium metabisulphite particularly in patients with asthma (ampoules). See full prescribing information. **Side effects:** Occasional: gastro-intestinal disorders, headache, dizziness, or vertigo, rash, elevation of SGOT, SGPT. Rare: peptic ulcer, gastro-intestinal bleeding, hepatitis, hypersensitivity reactions. In isolated cases: disturbances of sensation, erythema multiforme, purpura, abnormalities of renal function, blood dyscrasias. See full prescribing information. **Packs:** Voltaren is supplied in packs of 100 and 1000 coated tablets of 25 mg. 100 and 500 coated tablets of 50 mg. 30 and 150 tablets of 100mg (Voltaren SR 100). 5 and 50 ampoules of 75 mg (3ml). 10 suppositories of 12.5 mg; 25 mg and 50 mg. **Forensic Classification:** Prescription-Only-Medicine. Full prescribing information is available on request. **CIBA-GEIGY S.E.ASIA (Pte) Ltd,** 4 Fourth Lok Yang Road, Singapore 2262. Toll-Free Nos. 2664285/6.

®VOLTAREN
Geigy

The Singapore Family Physician

The College of Family Physicians Singapore
College of Medicine Building
16 College Road #01-02, Singapore 0316

Vol XX No. 1 Jan/Mar 1994 M.I.T.A (P) NO. 147/01/94
Price to Non-Members S$7.50

CONTENTS

Page

SKILLS IN FAMILY MEDICINE

Family Medicine has been recognised as a distinct medical discipline in its own right, with its own separate content of knowledge, skills and attitude. The term *Family Medicine* emphasises the special importance and impact of the individual and his illness on his family and community, and the influence of the family and community on him and his illness. The family doctor, who gives personal, primary, comprehensive and continuing care to individuals, families and a practice population, irrespective of age, sex and illness, has to have special skills to fulfill these functions.

Skills in Family Medicine comprise:

1. Behavioural skills
2. Special clinical skills
3. Resource management skills, and
4. Practice management skills.

BEHAVIOURAL SKILLS

These include intellectual skills, appropriate attitudes and manual skills. Knowledge, interpretive and problem-solving skills make up the *cognitive* domain; attitudes, values, communicative and interpersonal skills the *affective* domain; while the *psychomotor* domain is made up of manual and perceptual skills.

Communication, information-gathering and interpersonal skills will allow the family physician to conduct interviews with his patients and their families and obtain relevant information by proper listening and by observing and responding to verbal and nonverbal cues to determine the range of problems present. He must be willing to adapt his skills to conduct a satisfactory interview with all types of patients presenting with all types of problems in the "patient-centred" environment of Family Practice, and be able to explore and elicit the patient's own views, attitudes, concerns or anxieties, and expectations related to his problems, and the impact of these problems on the patient's life and that of his family, tackling problems in emotionally sensitive areas with tact and sympathy.

His interpretive and problem-solving skills will help him take account of the physical, psychological and social factors of the patient's problem(s), and through these skills, he can maintain confidence and trust with his patient and establish rapport and a beneficial therapeutic relationship, so important in enabling him to assist the patient in the resolution (or acceptance when appropriate) of his problems.

Manual skills are necessary for the family doctor to carry out a clinical examination with confidence and consideration, and to perform simple office laboratory tests and operative or manipulative procedures indicated for the presenting problems. Perceptual skills will make it possible for him to use time as a diagnostic, therapeutic and organisational tool, to conduct the whole consultation in a time appropriate to the family practice setting, and to explain to each patient the diagnosis and prognosis of all illnesses, avoiding jargon, complicated medical terms or phrases and a paternalistic approach.

SPECIAL CLINICAL SKILLS

With special clinical skills of importance to Family Medicine, the family physician can make an initial and appropriate decision about every problem which is presented to him as a doctor, practice preventive medicine, conduct counselling and health education, and undertake the continuing management of the health problems of all age groups and of patients with chronic, recurrent or terminal illnesses.

Problem definition, diagnosis and solution

To be able to recognise, analyse, define and manage the health problems of his patients, especially as they present so often in family practice in an undifferentiated form, the family doctor needs to have an understanding and knowledge of the patient, the family and the community. He must be able to recognise the normal, and detect early deviations from the normal, define the problem, through appropriate and judicious investigations if necessary, and employ efficient clinical problem solving techniques, continuing care and/or referral to see the problems through to their end.

Prevention of illness and Health Education

The family physician carries the responsibilities of preventive care for his patients. Besides immunisations, health screening and periodic health examinations, he must be in a position to identify risks to health and detect physical or psychological disease early, take necessary action and thus prevent complications. He can employ his counselling and health education skills to teach the patient, his family and the community about their responsibility for their own health, about self-care and care of family members, and about strategies for lifestyle modification and health promotion and maintenance. He can utilise every consultation, even for minor problems, as an opportunity for health education and preventive care. Finally, he has an important role to play in the rehabilitation of those disabled through illness or injury to as near normal function as possible, and their restoration to work, home and society.

Problem management and continuing care

To be able to help with the health problems of all age groups, from the neonate to the elderly, he must be skilled in the management of all common physical and psychological illnesses, accidents and emergencies, in symptomatic treatment, in antenatal and postnatal care, in obstetrics and family planning, in therapeutic techniques and procedures appropriate to family medicine, and in counselling. He must be able to manage special groups of patients, viz. the patient with chronic problems, the terminally ill, the bedridden patient, patients with problems of living and members of at-risk families.

RESOURCE MANAGEMENT SKILLS

The family physician's capacity to cooperate with other colleagues, medical and non-medical, and to utilise and coordinate the contributions of members of related professions and specialists and others who have special skills to secure the best intersests of his patients, and the ability to accept and share both responsibility and credit will help inspire the confidence of the patient, family and community.

As the doctor of first contact he can also, within certain limits, help to control the use of extensive or unnecessary investigations or procedures, prescriptions for treatment, referral to specialists and admissions to hospitals. The ability to manage these resources for the maximum benefit of his patients will allow the family doctor to save money and time for his patients, and serve his professional responsibility to the community, of which he himself is a part.

PRACTICE MANAGEMENT SKILLS

Knowledge and appropriate use of the skills of practice management will make it easier on the doctor himself, besides his patients and his personnel. These skills include those of general administration of the practice, personnel management, patients' appointments, financial accounts, equipment and drugs maintenance and inventory, ethical and legal aspects of medical practice, proper medical record keeping, and effective time management.

Medical records must be precise, and structured such as will make continuing care a reality. They must also lend to assessment and audit of the clinical care provided and to any research projects. The doctor himself must be willing and able to critically evaluate his own work.

To be effective, the family physician must be able to use the short segments of time spent with each of his patients in consultation, as is the norm in family practice, to accumulate information, to complete his examinations and investigations, and to provide education.

Last but not least, the practitioner must recognise his own need for continuing education, leisure and time with his own family, and be able to allocate appropriate time and energy for each of these, so as to be the complete family physician.

Dr Moti Vaswani

RHEUMATOLOGY AND THE FAMILY PHYSICIAN

Bone aches, joint pains and body pains are common complaints that are presented to primary care doctors. Many will respond to symptomatic treatment and they are usually self-limiting. Notwithstanding this, it is important that we have a framework to approach such problems.

There is also a need to have a high index of suspicion. Two questions need to be addressed. Firstly, are we dealing with something more serious than the usual self-limiting conditions? Secondary deposits can present as bony pain. Multiple myeloma can be misdiagnosed for months as chronic backstrain. Secondly, is the patient likely to develop side effects from our therapy? Prolonged use of NSAIDs may put the patient at risk of peptic ulceration. Steroids may give dramatic results but have multiple side effects too. What is even worse is that indiscriminate intra-articular steroid injections in osteoarthritis will result in destruction and contribute to further problems. Allupurinol should be given with care or not at all to patients with gout and renal impairment.

There is a also a need to monitor the progress of the patient and to consider referral if the patient is not improving with initial management. An example of this situation is the patient with rheumatoid arthritis. Such a patient may need second line disease modifying drugs (DMARDs) to put the condition under control.

In this issue, the diagnostic approach to arthritic conditions is dealt with. When a patient presents with a joint pain, four questions need to be asked[1]. Is the problem articular or non-articular? Is it inflammatory or non-inflammatory? Is the involvement polyarticular or pauci-articular? Are there any extra-articular manifestations? Once it is decided that the condition is articular, then the next step is to decide if there is there is an inflammatory process. The issues are different. Osteoarthritis is non-inflammatory generally. The progression of disease cannot be halted by available drugs, but they can offer symptomatic relief. As osteoarthritis is non-inflammatory, paracetamol and analgesic doses of aspirin are as effective as more expensive alternatives. NSAIDs provide no more relief than paracetamol. Low analgesic doses of NSAIDs are no better than high, anti-inflammatory doses. Intra-articular steroids are generally not justified[2].

If the condition is an inflammatory one, one has to differentiate between septic arthritis, gout or pseudogout and the inflammatory polyarthritides. Diagnosis of septic arthritis and gouty arthritis is not difficult if a careful history and clinical examination are done. For the inflammatory polyarthritides, the diagnosis may be suspected by morning stiffness, joint swelling and warmth. Extra-articular manifestations like subcutaneous nodules, eye involvement and skin involvement are useful diagnostic cues. Often, in the early stages, there may be no physical signs and treatment initially may need to be based on history only.

There have been recent advances in the management of inflammatory polyarthritides with disease modifying drugs (DMARDs). These are slow acting, disease suppressing drugs. Drugs in this group are gold (oral or muscular), penicillamine, chloroquine, sulphasalazine, methrotrexate and azathioprine. The indications for such drugs are signs of active disease as reflected by ESR and progressive erosive changes on X-rays within a six month period[3]. These drugs should be given by the specialist however and the patient closely monitored.

The main role of NSAIDs is to provide symptomatic relief of pain and stiffness in patients with inflammatory joint disorders. They do not prevent erosions. In choosing NSAIDs, it is important to note that a new drug is not necessarily better than an established one. Special precautions need to be taken for those prone to steroid-induced gastropathy namely, those who have an established history of peptic ulcer disease and those who are receiving both NSAIDs and steroids. There is no need however, for routine antacid therapy in patients given NSAIDs.

Low back strain is common and a frequent cause for reporting sick. It is important that a thorough assessment is done to exclude serous causes and a plan of management instituted. Those who continue to have backache after the acute episode need further assessment to ascertain if the problem is organic, functional or both.

Osteoporosis is a topical subject. Primary prevention remains the most cost-effective approach. Adequate intake of calcium and exercise which is weight-bearing and skeletal-stressing are necessary. Walking, jogging, aerobic dance or cross-country runs are useful. Swimming is not helpful. Exercise to the point of producing amenorrhoea in menstruating women is associated with accelerated bone loss and should be discouraged. Some studies in males have also shown that high levels of exercises may lead to bone loss[4].

References

1. Alderdice C. Approach to the patient with polyarthritis. Can Fam Physician, 1990; 36:549-551, 553-554.

2. Robb-Nicholson L C and Liang M H. Osteoarthritis. in: Manual of clinical problems in adult ambulatory care. Edited by Laurie Dorbrand, Axalla J Hoole, and C G Pickard. Toronto: Little Brown 1992:286.

3. Goroll A H, May L A and Mulley. Management of rheumatoid arthritis . in: Primary Care Medicine. Third ed. Philadelphia: Lippincott, 1995:790-794.

4. Zikoski M W Osteoporosis. in: Ambulatory Medicine. Philadelphia: Prentice Hall 1994:456.

Dr Deidre Murugasu

DIAGNOSTIC APPROACH TO ARTHRITIC CONDITIONS

** R Ray MBBS, DPH (S'pore), FAMS.*
*** L G Goh MBBS, MMed (Int Med), FCGP, MRCGP (UK)*

HISTORY

When a patient presents with joint pain, the most important decision is to distinguish between an inflammatory and non-inflammatory process. Four questions help to determine the problem.

- Is the problem articular or non-articular?

  If it is outside the joint, decide if it is:

  - localised — consider bursitis, tendinitis
  - diffuse — consider fibrositis, polymyalgia rheumatica.

- Is it inflammatory or non-inflammatory?

  Morning stiffness and soft tissue swelling support an inflammatory process. See Table 1 for distinguishing features.

* *Dy Medical Director*
Family Health Services
Primary Health Division
Ministry of Health
Singapore

** *Senior Lecturer*
Dept of Community,
Occupational and Family Medicine
National University of Singapore

Table1: Distinguishing features in inflammatory and non-inflammatory joint disorders.

Symptoms & Signs	Inflammatory	Non-inflammatory
Morning stiffness	> 1 hour	< 1 hour
Fatigue	Marked	Occasional
With activity	Better	Worse
With rest	Worse	Better
Soft tissue swelling	Yes	Uncommon
Bony swelling	Uncommon	Yes

Source: Catherine Alderice. Can Fam Physician 1990; 36:553.

- Is the involvement polyarticular or pauci-articular?

  By definition, polyarticular involves five or more joints. Pauci-articular therefore involves four or fewer joints. Usually included in the latter group is mono-arthropathy.

  If the involvement is mono-articular, consider trauma, septic arthritis, gout and monoarticular stage of a polyarthritis. Of these, excluding septic arthritis is most important.

  If the involvement is polyarticular, the distribution of joints helps to delineate the underlying disorder:

  - inflammatory arthritides e.g., rheumatoid arthritis typically affect the feet, metacarpophalangeal joints, proximal interphalangeal joints and wrists.

- rheumatic arthritis is characterised by flitting joints rather than incremental involvement.

- non-inflammatory arthritides e.g., osteoarthritis, when they affect the hands, usually do so at the proximal interphalangeal, distal interphalangeal, and carpometacarpal joints. Metacarpophalangeal involvement is most uncommon in osteoarthritis; similarly, involvement of wrists, elbows, and ankles is also uncommon.

Are there any extra-articular manifestations? This final question often clinches the diagnosis, particularly if the possibility has been narrowed to an inflammatory arthropathy. Table 2 shows the extra-articular manifestations in inflammatory arthropathies.

Table 2: Extra-articular manifestations in inflammatory arthropathies

Inflammatory arthropathy	Extra-articular manifestations
Polyarthropathy	
– Rheumatoid arthritis	Extra-articular manifestations tend to occur later in the course of disease. Subcutaneous nodules, sicca symptoms (dry eyes and dry mouth); hand deformities — volar subluxation, swan neck, boutonniere deformity, ulnar deformity of the metacarpophalangeal joints — are common.
– Systemic lupus erythematosus	Extra-articular manifestations are usually prominent, often preceding joint complaints: alopecia, mouth ulcers, Raynaud's phenomenon, butterfly rash, photosensitivity and serositis.
– Psoriatic arthritis	In 15% of patients with psoriatic arthritis, the arthritis appears first and the typical skin rash develops months to years later. Typically the skin lesions and nail changes clinch the diagnosis.
– Chronic tophaceous gout	Tophaceous deposits may be found under the skin.
Oligoarthropathy	
– Ankylosing spondylitis	Irits, aortic incompetence.
– Reiter's syndrome	Conjunctivitis, keratoderma blenorraghica, balanitis in males.
– Psoriatic arthritis	Skin rash, nail changes reveal the diagnosis.
– Inflammatory bowel arthropathy	Ulcerative colitis and regional ileitis.
– Early rheumatoid arthritis	Subcutaneous nodules.

PHYSICAL EXAMINATION

Examination may be normal or there may be redness and swelling of affected joints, deformities and extra-articular manifestations.

Pain grading is helpful:

Grade 1 : joint pain but no tenderness on palpation

Grade 2 : definite pain on palpation with wincing

Grade 3 : pain so severe that on palpation the patient winces and withdraws.

X-RAYS AND LABORATORY INVESTIGATIONS

Laboratory data usually are not sufficiently specific for a particular diagnosis to be made. With the exception of a positive culture from the joint aspirate in septic arthritis, or monosodium crystals in acute gout there is no pathognomonic test for the rheumatic diseases.

In inflammatory polyarthropathy, initial investigations need only to be the following:

- complete blood count
- Erythrocyte Sedimentation Rate
- routine urinalysis
- Rheumatoid Factor
- Anti-Nuclear Antibodies

Symptoms that suggest a more diffuse process, such as fibrositis or polymyalgia rheumatica, should have more extensive investigations because some endocrine diseases, and even multiple myeloma or metastatic disease, can mimic them. Investigations should include:

- blood sugar assessment
- thyroid function tests
- calcium, phosphorus and alkaline phosphate levels
- serum protein electrophoresis.

Erythrocyte Sedimentation Rate (ESR)

The Erythrocyte Sedimentation Rate is a very useful test of inflammatory activity, particularly in patients with rheumatoid arthritis or polymyalgia rheumatica. In RA it is raised and very high in the acute stage.

Rheumatoid Factor

Rheumatoid Factor is an important test in confirming the diagnosis, but only if the positive results correspond to the patient's symptoms and current knowledge of rheumatoid arthritis. Early in the disease, it may be negative in rheumatoid arthritis but will normally turn positive within one year. Rheumatoid Factor is used mainly to confirm a diagnosis. It should never be used to monitor disease activity.

Anti-Nuclear Antibodies

Anti-nuclear antibodies (ANA) should be approached in the same way as a positive test for rheumatoid factor. Only if the patient's symptoms strongly suggest SLE should a positive test for ANA be taken as confirmation of the diagnosis. Like rheumatoid factor, ANA tests are not useful to monitor disease activity.

Table 3: Laboratory features of arthropathies

Arthropathy	Laboratory test & X-ray	Features
Rheumatoid arthritis	ESR	– Raised; very high in acute stage.
	RA factor	– If negative, may not exclude RA if other clinical features are suggestive.
	X-rays	– In later stages, periarticular osteoporosis and periosteal reaction occur.
Ankylosing spondylitis	X-rays	– Typical diagnostic features: blurring of margins of sacro-iliac joints, erosions and squaring of lumbar vertebrae.
Acute gouty arthritis	Uric acid	– Raised in acute stage: above 7 mg/100ml (363 micromoles/L) in males; above 6 mg/100ml (311 micromoles/L) in females
	S creatinine, Blood Urea	– Raised if kidneys are affected.
	X-rays	– Punched out juxta-articular erosions; degenerative changes at later stages of disease.
Osteoarthritis	ESR	– Normal.
	X-rays	– Narrowed joint space, irregular joint space; sclerosis of subchondral bone, subchondral cyst; osteophytes.

Synovial fluid analysis

In oligoarthopathy and monoarthropathy, synovial fluid analysis is helpful. It is almost diagnostic in septic arthritis and in gouty or pseudogout arthritis. By contrast, such analysis does not help to differentiate the aetiology of polyarthropathy.

Reference:

Alderdice C. Approach to the patient with polyarthritis. Can Fam Physician, 1990; 36:549-551, 553-554.

OSTEOARTHRITIS

S C Ng, MBBS (S), MMed (Int Med), FAMS

SUMMARY

Osteoarthritis is a very common and age related form of arthritis. It is characterised by cartilage degeneration, bony overgrowth and some synovial hypertrophy giving rise to the typical clinical picture. Management should be mainly conservative. The patient should be educated about the condition and given physiotherapy and occupational therapy. Before starting drugs (e.g. non-steroidal anti-inflammatory drugs, analgesics), we have to consider other factors like risk of adverse reactions, efficacy, cost, etc. In some cases, the patient has to be sent for surgery. The indications for surgery are (1) when conservative measures have failed to reduce pain and disability, and (2) when the mechanical derangement contributes to further deterioration of the OA. Drugs specific for osteoarthritis are being developed but there is insufficient evidence to advocate their general use at this time.

INTRODUCTION

Osteoarthritis (OA) is the most common type of arthritis that we see in clinical practice. More than 80% of people aged above 75 years have OA. So as our population grows older, this condition will be even more common.

OA is often called degenerative arthritis as it is characterised by degeneration of the cartilage. Following this degeneration, there is bony outgrowth and in some cases inflammation of the synovium.

CLASSIFICATION

1. Idiopathic

(a) *Localised* — Heberden's nodes without other joint involvement.

Consultant Physician
Department of Medicine
Alexandra Hospital
Singapore 0314

(b) *Generalised* — involvement of three or more joints (usually small joints: predilection for postmenopausal women and episodic joint inflammation.

(c) *Erosive* — small joints of fingers are affected: with erosions, osteophytes and synovitis.

2. Secondary

(a) *Trauma* — acute, chronic

(b) *Congenital abnormality* — slipped femoral epiphysis

(c) *Metabolic* — gout

(d) *Endocrine* — acromegaly

(e) *Other bone disease* — osteonecrosis

(f) *Diseases of obscure aetiology* — Kaschin-Beck disease

RISK FACTORS FOR OA

These are divided into two categories: increased susceptibility and mechanical causes.

Increased Susceptibility

1. *Hereditary* — There is data to suggest a polymorphic genetic error in the gene for Type II collagen in those with generalised OA.

2. *Obesity* — The obese have a higher incidence of OA.

3. *Ligamentous laxity* — Patients with lax ligaments and weak muscles have more problems with OA when they grow old.

Mechanical Factors

1. *Trauma* — Men with monoarticular knee OA will often give a history of previous trauma to the joint.

2. *Abnormal joint shape* — e.g. patients with slipped femoral epiphysis have early OA of the hips.

3. *Sporting injury* — Recurrent minor injuries sustained during sports will lead to early OA.

CLINICAL FEATURES

Symptoms

1. *Pain* in the affected joint is the most common presenting symptom. The pain is usually insidious and aggravated by use of the joint. It is not possible to differentiate the quality of pain in OA from that due to other types of arthritis. There is also a poor correlation between pain in the joint and the radiologic appearance. This is particularly so for OA of the hands and less so for OA of the hips.

2. *Disability* is a problem. Patients with OA hands may have problems performing tasks with their hands; while patients with OA knees may complain of difficulty climbing stairs or unsteady gait. Some patients may notice loss of range of motion.

3. *Stiffness* in the morning is another symptom. However, unlike inflammatory arthritis (e.g. Rheumatoid Arthritis), the duration is usually less than 30 minutes.

Signs

1. *Tenderness* at the joint line and at the periarticular tissues is a good sign of disease in the joint.

2. *Swelling* of the joint can be detected. This is a combination of bony and synovial swelling. Popliteal cyst may be present.

3. *Crepitus* on movement of the joint is often felt.

4. *Deformity and malalignment* (e.g. varus deformity of the knees due to asymmetric involvement of the knee joint) can be obvious on inspection of the joint while weight bearing.

When examining patients with arthritis, always consider and look out for other causes of arthritis besides OA. These include Rheumatoid Arthritis (RA), Psoriatic Arthritis and Crystal Arthritis like gout and pseudogout. Patients with predominant proximal interphalangeal joint arthritis may well have RA or pseudogout. Patients with predominant distal interphalangeal joint arthritis may well have psoriatic arthritis and the doctor has to search for papulosquamous plaques in hidden areas like hair line, natal cleft and umbilicus.

On the other hand, when examining patients known to have other forms of arthritis, it is useful to look for the development of secondary OA.

Having arrived at a diagnosis of OA, it is often useful to look out for the rare patient with other underlying metabolic and neurologic diseases. On close examination the patient may be found to have acromegalic facies or hypothyroidism. If so, it is worthwhile checking the hormone levels. Patients may have features of Wilson's Disease or even Ochonosis and appropriate tests may then be done to diagnose these more exotic conditions. Neurologic conditions like Diabetic neuropathy and Tabes Dorsalis may cause a Charcot's joint which is severe OA secondary to loss of sensation of the joints.

INVESTIGATIONS

The following **radiologic features** may be seen in OA. There may be decreased joint space due to loss of cartilage. This decrease is often asymmetrical unlike the loss of joint space in RA. There may be subchondral bone cysts due to increased activity of the subchondral bone. The osteophytes seen on X-rays are due to bony overgrowth and marginal lipping. In severe cases, there will be abnormal alignment and even collapse / fracture of the bone.

There are no specific **blood tests** to diagnose OA. Tests are often ordered to look for other causes of the arthritis. These include the Erythrocyte Sedimentation Rate and C-Reactive Protein which are both markers of inflammation. The Rheumatoid Factor (RF) and anti-nuclear antibody (ANA) are often ordered. When interpreting these tests, always look at the entire clinical picture instead of making a diagnosis based on these serologic tests. There are many other causes of a positive RF and ANA and increasing age alone can cause them to become positive. When in doubt, particularly in those with inflammatory OA, follow-up of the patient and time will tell.

MANAGEMENT

1. **Confirm diagnosis,** consider differential diagnosis e.g. RA, which may require a different approach to treatment.

2. Decide what the **main problem** is — pain, disability, side effects of drugs, etc.

3. **Educate and reassure** the patient. Some are actually worried that the arthritis may lead to a stroke! Discuss with them the natural history of the arthritis. In general, OA tends to deteriorate very slowly and there are periods of exacerbation and remission.

4. **Assess the medical condition** of the patient to decide on the optimal treatment. Before sending patients for physiotherapy, ascertain that they are fit for the exercises which are prescribed for the OA. Before starting on Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), look out for medical conditions which put the patient at higher risk for NSAID complications — renal disease, hypertension, diabetes and small vessel disease (these increase the risk of NSAID nephropathy). Look out also for those who have underlying peptic ulcer disease as NSAIDs will aggravate it.

5. Start **physiotherapy** — do range of motion exercises, muscle stretching and strengthening exercises. Modalities like hydrotherapy and the various heat and cold therapies are useful for pain relief. Most important of all are the joint protection exercises and use of devices to protect the joints.

6. **Pure analgesics** like acetaminophen are good drugs to start with. They can be prescribed as a regular medication to suppress chronic pain or only when necessary. They are generally safer than Non-steroidal Anti-Inflammatory Drugs. Some are marketed as a combination drug with codeine and there is always a potential for drug dependence. Topical analgesics are available. The recently introduced topical capsicum which acts to release substance-p from the nerve endings has been shown to be quite useful in OA.

7. **Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)** are often used as first line drugs for OA and all types of joint pains. They are most effective for those with a lot of inflammatory changes and morning stiffness.

   There have been claims that some NSAIDs are "chondroprotective" while others accelerate the degenerative process of OA. Much of these claims are based on laboratory experiments and not human studies. Hence they should not dictate clinical decisions at this time.

   As NSAIDs have a long list of potential side effects, they should be prescribed with great care in the elderly with OA. It is safer to prescribe a lower dose and a brief course of NSAIDs instead of the daily high doses needed in RA. Topical NSAIDs are very useful. They are safer because of the much lower doses and there is direct penetration of the drug into the joint. Patients also seem to like the act of applying medication directly onto the painful joint.

8. **Specific Drugs for OA** have been marketed. Drugs like hyaluronan and glycoaminoglycan polysulphate are advertised as chondroprotective. In vitro they do inhibit protease and in animal models they have been shown to have some beneficial effect on the cartilage. However there are no good controlled human studies. Locally, courses of intra-articular hyaluronan have been tried but the results are inconsistent. More work needs to be done before this category of drugs can be recommended for general use.

9. **Surgery** can be offered to some of the patients. It is best to seek the opinion of an orthopaedic surgeon in those patients who may need surgery. In general, those with severe symptoms but not much radiologic signs may benefit from arthroscopic irrigation of the joint. Patients with more severe disease but not much cartilage loss may benefit from joint debridement. Osteotomy is useful to correct significant malalignment. Patients with severely damaged joints may need to undergo joint arthroplasty.

CONCLUSION

Much research is underway to further our understanding of cartilage, bone and synovium in OA. There has also been a lot of work done to unravel the genetics of the different types of OA. New drugs are being tested but no miracle drug has yet been found. At this time, conservative treatment and using time tested drugs carefully is still the best way to treat OA.

Bibliography

Brandt, Mankin: Pathogenesis of OA; Clinical features of OA; Management of OA in W N Kelly et al (eds). Textbook of Rheumatology, Saunders 1993, pp 1355-1399.

Jamieson, Barry: Osteoarthritis. in: Paget et al (eds). Manual of Rheumatology and Outpatient Orthopaedic Disorders, Little Brown and Company 1993, pp 323-334.

Dequeker, van de Putte, Dieppe, Cooper, Campion, Watt, Kuettner, Bullough: Osteoarthritis. in: Klippel and Dieppe (eds). Rheumatology, Mosby 1994, pp 7, 1.1-8.8.

OSTEOPOROSIS : AN OVERVIEW

K Q Yeo, MBBS, MMed (Surg), FRCS, MCH (Orth) (L'pool), FAMS

INTRODUCTION

Osteoporosis has been termed the "silent epidemic" in the west because of its prevalence, the socio-economic price it exacts and the lack of attention paid to it by most doctors. There has been a swing of the pendulum the other way over the last ten years and there are now advocates for early detection and prevention.

In the context of Singapore, the limited statistics that we have are not compelling enough to support such an approach and treatment must be tempered by common sense and a consideration of cost effectiveness. (Table 1)

NATURE OF OSTEOPOROSIS

Osteoporosis occurs when there is a reduction in bone mass to the extent that the skeleton is unable to withstand the normal stresses of daily living resulting in either deformities or fractures. It is important to note that there are no biochemical changes associated with osteoporosis. It is the measurement of bone mass that allows us to detect the decreased bone mass.

The cost to the community has been clearly shown in the west. (Table 2)

Table 1: Incidence of hip fractures in Singapore (1980)[1]

	Total population (million)	Population aged over 60 years (million)	No. fractures	Incidence in total population (%)	Incidence in population aged over 60 years (%)	Incidence relative to Singapore Total > 60 years	
Singapore	2.41	0.1735	258	0.011	0.15	–	–
UK	55.90	11.1000	42,000	0.075	0.38	7.0	2.5
Italy	56.90	10.0000	43,000	0.076	0.43	7.1	2.9
USA	223.20	33.000	200,000	0.090	0.61	8.4	4.1

Table 2: Economic cost of osteoporotic hip fractures[2]

	Total population (millions)	Number of fractures (per year)	Annual incidence (per 1000)	Estimate cost (per year)
USA 1980	223.2	200 000	0.89	> 2.8 million US$
Italy 1980	56.9	40 000	0.70	> 200 billion lire
UK 1980	55.9	42 000	0.75	> 200 million pounds

Consultant Orthopaedic Surgeon
3 Mount Elizabeth #08-10
Mount Elizabeth Medical Centre
Singapore 0922

NORMAL PHYSIOLOGY

Bone is a metabolically active structure where the process of destruction and repair is continuously taking place. The balanced state can be upset either by a increase in destruction or a reduction in repair.

The process is controlled by genetic and environmental factors. (Fig 1 and Fig 2)

This results in two types of osteoporosis in women with differing characteristics (Table 3).

Fig 1: Normal bone metabolism[3]

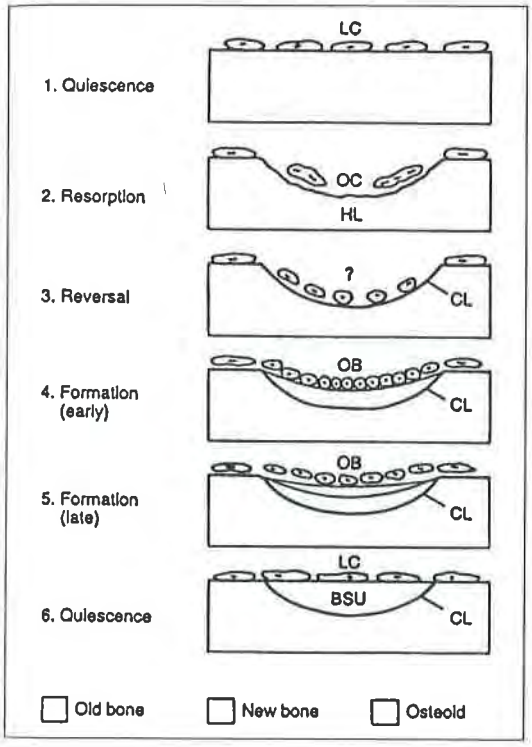


Fig 2: Irreversible bone loss[3]

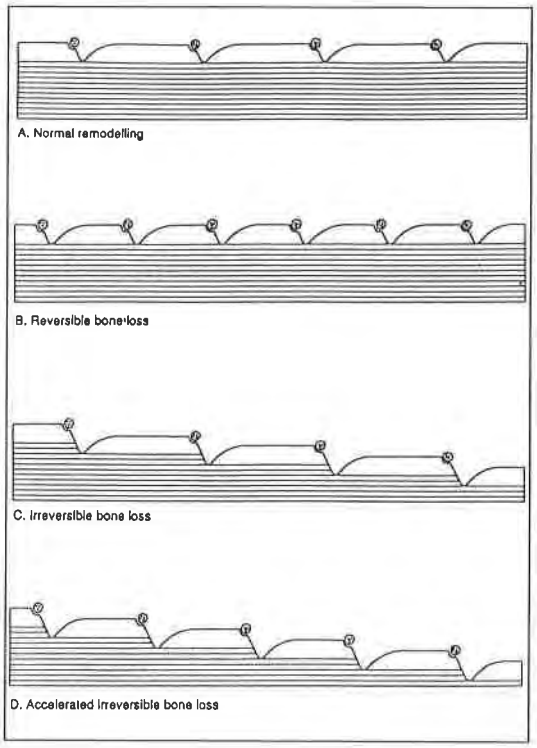
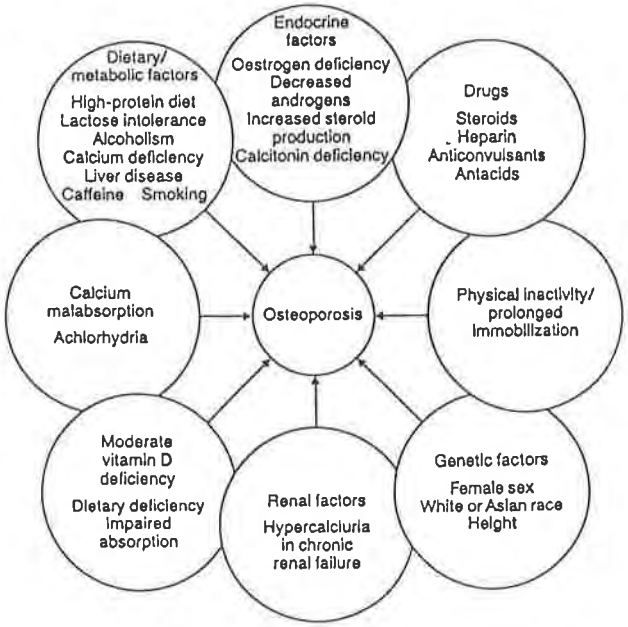


Table 3: Types of osteoporosis[3]

Parameters	Type of osteoporosis	
	Type I	**Type II**
Age	50-65 years	over 75 years
Sex ratio	6W / 1M	2W / 1M
Bone loss: type	trabecular	trabecular and cortical
rate	accelerated	not accelerated
Main type of fractures	spinal	femur, other long bones
Main aetiological factor	oestrogen loss	bone aging, hyperparathyroidism II
Parathyroid hormone level	reduced	increased
Calcium absorption	reduced	reduced
Synthesis of 1-25 $(OH)_2D_3$	secondary reduction	primary reduction

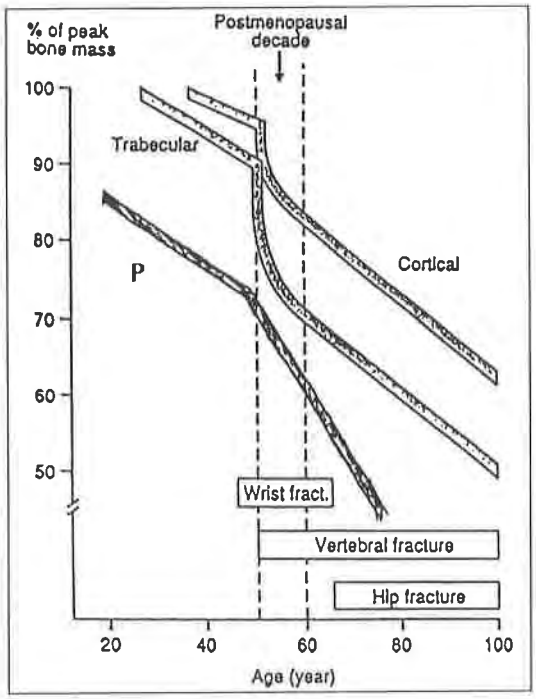
This may result from multiple causes which may be genetic or environmentally based (Fig 3).

Fig 3. Causes of osteoporosis[3]



The female pattern shows four phases and the male two phases (Fig 4).

Fig 4: Normal bone mass changes with age



P: Pathological Pattern. Lower peak bone mass and more rapid loss.

Female Pattern:

Phase I	: Build Up	: 0 - 30 years
Phase II	: Peak Bone Mass	: 30 years
Phase III	: Gradual Loss	: 30 years - Menopause
Phase IV	: Rapid Loss	: Post-menopausal

Male Pattern:

Phase I	: Build Up	: 0 - 30 years
Phase II	: Gradual Loss	: Throughout Adult Life

PATHOLOGICAL PATTERNS

These occur when there is a accelerated loss of bone.

The vexing question remains as to why some women lose bone mass faster than others, the so-called rapid bone losers as compared to the slow bone losers. Some advances have been made to enable us to identify the former group but the exact cause remains elusive.

CLINICAL PRESENTATION

The mode of presentation may be classified as:

1. **Asymptomatic**
   These are cases where a bone matrix study is done as part of the peri-menopausal or post-menopausal workup. This group may include those presenting with mild non-specific backache where osteoporosis is one of the possible causes.

2. **Symptomatic**
   A. ***Acute*** : These are cases presenting as fractures of the vertebrae, hip or wrist. In the very rare case there may be neurological deficits associated with the vertebral fracture. This may take the form of radiculopathy where the patient complains of a feeling of tightness at the level of the affected dermatome.

   B. ***Chronic*** : The patient may present with either backache or a feeling of inability to support the spine when sitting or standing. This worsens towards the end of day. It is not common for our local women to complain of the so-called "dowager's hump" as most patients accept the gradual spinal curvature as a natural process of ageing.

INVESTIGATIONS

It must be emphasized that there are no biochemical tests to confirm osteoporosis. Biochemical tests are done to exclude malignancies and osteomalacia. A suggested Osteoporotic Biochemical Profile for this purpose would be:

Blood : Haemoglobin level
Total White Count and a Differential Count
Peripheral Blood Film
Erythrocytic Sedimentation Rate
Serum Calcium and Phosphate levels
Serum Alkaline Phosphatase level
Serum Protein level and Albumin / Globulin ratio
(Protein Electrophoresis should be done only if there is a suspicion arising from a change of the Serum Protein level or a change of the Albumin / Globulin ratio).

Urine : Bence-Jones Proteins; this is not a very useful test but done for medicolegal reasons.

Special tests : These are done to detect rapid bone losers and will be discussed later.

Radiological studies (Sites of pain) : In the spine it is usually at the lower thoracic or thoracolumbar levels.

In the hip, it is important to take an x-ray if the patient complains of pain even if the injury is minor. An early or impacted fracture of the neck of femur would allow the patient to continue walking but if left alone will change into a badly displaced fracture leading to more major surgery and medicolegal problems.

Diagnostic Tests: These are tests designed to measure the bone mass. A variety of methods have been used:

Single photon absorptiometry
Dual photon absorptiometry
Dual energy x-ray absorptiometry (DEXA)
Neutron activation analysis
Ultrasound

All of these, excepting ultrasound, involve the emission of various isotopic or x-ray sources through the bone and measuring the transmitted radiation using various detectors.

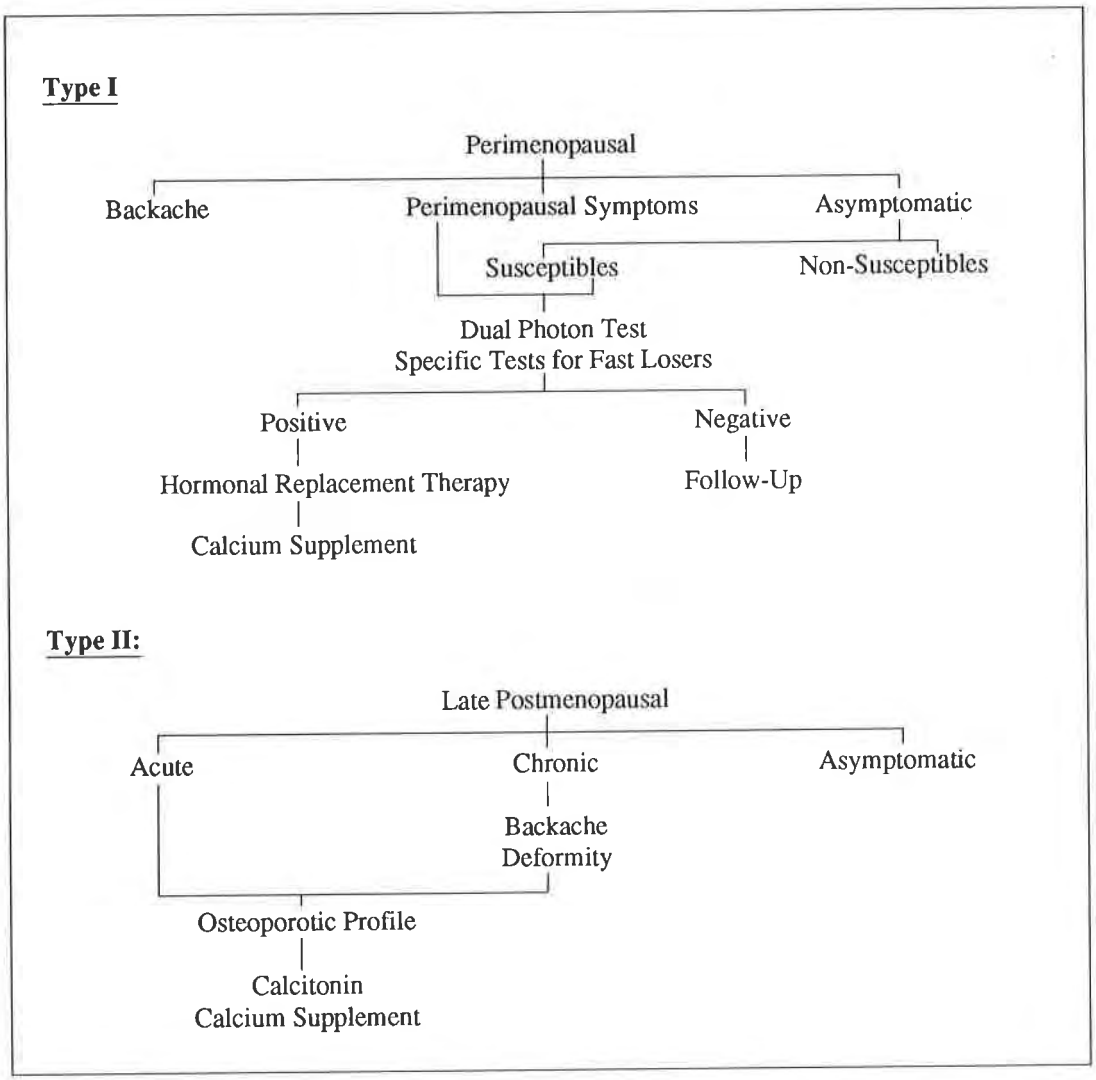
The attenuation of ultrasound signals during their passage through bone can also be used to determine bone mass. This is a promising and innocuous method.

MANAGEMENT

There is little controversy about the general management: Symptomatic treatment for the pain; rest during the acute phase; the use of analgesics and muscle relaxants; physical therapy and supportive corsets. The patient should be encouraged to move as soon as the pain is under control.

Controversies still exist with regards to:
1. Medications and their mode of action.
2. Preventive therapy.
3. The role of calcium and whether there is a place for prophylactic calcium therapy.

Below are the suggested algorithms for Type I and Type II osteoporosis treatment.



CONCEPT OF RAPID AND SLOW BONE LOSERS

It is easy to understand why a woman who starts off with a lower peak bone mass should be more susceptible to osteoporosis. It is however difficult to explain why those who start off with the same peak bone mass should have differing susceptibility to osteoporosis. Chestnut and his co-workers in 1987 developed a procedure to differentiate the two groups with some degree of accuracy. This is based on the movement of the body fat mass, urinary calcium and hydroxyproline, and the serum alkaline phosphatase. Using a calculation, they were able to predict with accuracy in three quarters of cases tested 3-monthly over two years as to who would be rapid bone losers. They have incorporated this into a little calculator for ease of determination. This has yet to gain wide acceptance but certainly would be useful if the concept is correct. However, it still leaves us without an answer as to why there should be two differing groups.

MEDICATION AND PROPHYLAXIS

In a concensual statement on osteoporosis published in 1987 in the British Medical Journal[4], a review of current approach to the use of medication and prophylaxis for osteoporosis was presented.

1. *OESTROGEN / PROGESTERONE THERAPY*

This is the only well established prophylactic measure for the reduction of osteoporotic fractures and is generally accepted for symptomatic cases in the perimenopausal patient. The minimal effective dose is the equivalent of 0.625 mg of conjugated oestrogen, 2 mg of 17 b-oestradiol, or 25 mg of ethinyloestradiol.

The disadvantages are well known:

a. Liver: there is an increase of hepatic protein synthesis, the effect of which is uncertain, and the possibility of the development of malignancy has to be borne in mind.

b. Uterus: there is an increase in endometrial hyperplasia leading to an increase in development of endometrial carcinoma in monotherapy. This is dose and duration dependent.

c. Breast: whether it leads to an increase in breast cancer is controversial, the majority opinion tending towards there being no increased risk.

2. *PROGESTERONE*

The use of progesterone with oestrogen controls vaginal bleeding and reduces the risk of uterine hyperplasia and the risk of uterine carcinoma.

However, there is a qualitative element here which is often left out of the argument for the use of hormonal replacement therapy. Most physicians would agree that there is a reduction in the risk of osteoporotic fractures and hence death from the ensuing complications. However, the one who should have died from the fracture would now be alive whereas the one who should not have had uterine carcinoma if she were not on hormonal therapy would now have died from uterine carcinoma.

If used, hormonal replacement therapy should start within five years of the menopause and should be kept on for ten years.

3. *CALCIUM SUPPLEMENTS*

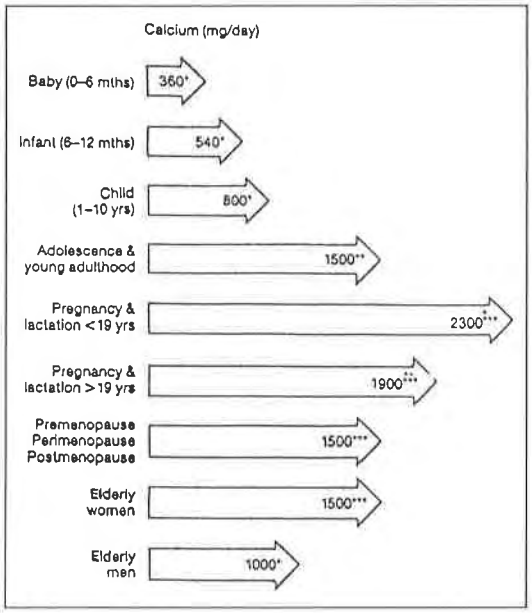
The recommendated daily intake of elemental calcium is 800 to 1000 mg. We have no data on local intake, but Hongkong studies show a fairly large proportion of Chinese women having a daily intake below the recommended amount.

Calcium supplement is generally accepted to be useful in building up peak calcium mass, especially where there is a suspicion of dietary inadequacy. Its usefulness in the post-peak period is debatable. There is no convincing proof that excessive intake will lead to renal stones. However, it is wise not to prescribe prophylactic calcium supplements for those with a history of renal stones.

The type of calcium has very little effect on efficacy except from the point of view of compliance. Most older patients find it easier to use soluble calcium preparations; in addition there is a slight risk in the use of "earth" or "natural" calcium, as calcium is often found in conjunction with heavy metals such as lead.

The recommended dosage for the symptomatic patient is 1500 mg a day.

Fig 5: Suggested calcium requirements[3]



4. *EXERCISE*

Most physicians would agree on exercise in moderation (gravitational and not anti-gravitational exercises such as swimming). Excessive exercise however can lead to oligomenorrhea or amenorrhea and loss of bone.

5. *FLUORIDE*

Fluoride increases trabecular bone mass and not cortical bone mass. It may in fact decrease cortical bone mass leading to fracture susceptibility in the long bones. It is useful in osteoporotic vertebral fractures. Its efficacy in the prevention of osteoporosis is debatable. Side-effects include gastro-intestinal symptoms, bone pain and arthralgia. The recommended dose of elemental fluoride is 20 mg a day taken with calcium supplement. The duration of therapy is not less than five years.

6. *VITAMIN D*

There is no proof that Vit D is of use in the prevention or treatment of osteoporosis.

7. *ANABOLIC HORMONES*

Anabolic hormones increase bone mass. However the side-effects do not allow for general use. The side-effects include virilization, increased plasma lipoprotein, susceptibility to coronary heart disease and liver dysfunction leading to tumours. They are useful probably only for the subset of glucocorticoid mediated osteoporosis.

8. *CALCITONIN*

Calcitonin is generally accepted to possess no serious side-effects. It acts as a inhibitor of bone resorption. It is useful as a analgesic in the initial two weeks of treatment. It acts by reducing osteoblastic activity. Dosages and duration of use are still not standardized.

9. *DIPHOSPHONATE*

Like calcitonin, it acts as a inhibitor of bone resorption. It has no analgesic property. Regimes of therapy are not well established.

10. *SPECIAL REGIMES: CYCLICAL AND COHERENCE REGIMES*

Cyclical regimes generally consist of the intermittent use of inhibitors of bone resorption such as calcitonin or diphosphonates. The intermittent use of bone formation stimulators such as parathormone or fluoride is not well explored.

Coherence regimes are based on the sequential application of an activator and inhibitor of bone formation leading to a nett increase in the rate of bone remodelling. A typical regime is that recommended by Dr Harold Frost. This is known by the acronym of ADFR: Activate, Depress, Free and Repeat. These regimes have yet to gain general acceptance.

CONCLUSION

While there has been an increasing interest in osteoporosis over the last fifteen years, we have yet to have a full understanding to the causes. Therapy for the most part is still empirical. With deeper understanding of bone metabolism at the cellular level, we will hopefully be able to be a bit more scientific in our approach in the future.

References

1. Table 1: Osteoporosis and the incidence of hip fractures in Singapore. S T Lee: 3rd Asian Symposium on Osteoporosis. Nov 8, 1992, Pg 6-10.
2. Table 2: Epidemiology and financial aspects of osteoporosis. C Gennari: Osteoporosis. Eds: D V Cohn & T J Martin, Pg 1-2.
3. Table 2: Fig 1 - 4: Courtesy of Sandoz
4. Consensual statement on osteoporosis. British Medical Journal Vol 295, 10 Oct 1987, Pg 914-915.

LOW BACK PAIN

S K Tan, MBBS , FRCS (G) ,FAMS ,PPA

Low back pain in a extremely common symptom encountered in clinical practice. It accounts for a very significant proportion of man-hours lost due to sickness and hence is a major problem to business and industries. While it is possible to reduce this problem, the solutions to some of the causes are not easy as they involve not only medical issues, but socio-economic, psychological and psychiatric ones as well. The doctor treating such patients therefore must have a clear overview of the problem so that he or she can then come to an unequivocal working diagnosis as far as possible. This will then allow him to formulate a treatment plan tailored to the individual patient.

CAUSES OF LOW BACK PAIN

The first thing one must realise is that this is a symptom and not a diagnosis — a basic enough fact but often forgotten. A second common mistake is to assume that this is spinal in nature. In fact, back pain arising from the spine and its supporting structures are not necessarily the only major cause. The causes of low back pain are extremely numerous and vary tremendously in different groups of individuals. Broadly, causes of low back pain may be classified as:

Chairman
Division of Surgery
Sr. Orthopaedic Surgeon and
Head, Dept of Orthopaedic Surgery 'O'
Singapore General Hospital
and
Clinical Associate Professor
National University of Singapore

Organic causes

Vascular – these are due to vascular problems or malformations e.g. haemangiomas around the spine or an aneurysm of the aorta with pressure on the vertebral column.

Viscerogenic – these are due to disease of abdominal viscera e.g. peptic ulcers, retroverted uterus, renal calculi, chronic pancreatitis.

Neurogenic – conditions affecting the neural contents of the spinal e.g. meningism, meningiomas etc.

Spondylogenic – this group can be further subdivided into:-

(i) *osseous* origin e.g. osteoporsis, spondylosis, vertebral body trachea, etc.

(ii) *soft tissue* causes e.g. chronic or acute sprains of the paravertebral muscles.

Discogenic – these are disc diseases e.g. disc prolapse or disc degeneration. Disc prolapse is more common in younger patients while disc degeneration occurs more in the elderly.

Miscellaneous – e.g. systemic diseases like rheumatoid arthritis or SLE.

Non-organic causes

Psychogenic – these patients do not have any underlying disease but the pain is a manifestation of an underlying emotional or psychological reaction to stress.

Postural and mechanical – patients who have poor posture, are over-weight, or work in ergonimacally incorrect environment will commonly suffer from back pain.

DIAGNOSIS

It is essential, as mentioned above, to have a correct working diagnosis to enable a successful treatment outcome. The diagnosis, like all fields of clinical medicine, is dependent upon:

History

A good, concise but adequate history is the most important aid in coming to an accurate diagnosis. In fact, it is sometimes said that 60% of the diagnosis is made from the history alone. In addition to the usual things asked in the history, the following should be noted if the pain originates from the spine:

- duration, regularity and nature of the pain
- aggravating and relieving factors
- associated symptoms of parasthesia and numbness
- nature of work and work environment

While taking the history, the patient's emotional and psychological make-up is being assessed continuously to determine its influence on the problem.

Physical Examination

More mistakes are often made from want of a proper and systematic examination. Examination of the spine (if the history points to a pathology there) should be conducted along the following lines:

i) Mechanical status of the spine:
   - mobility – flexion, extension, lateral flexion and rotation
   - stability – pain associated with normal movements of the spine

ii) Neurological component:
   - Motor – power of muscle groups of the lower limbs
   - sensory – check integrity of the dermatomes
   - autonomic – bladder, bowel (anal tone) functions and reflexes

The history and physical findings should provide enough data to come to a working provisional diagnosis to either commence treatment or proceed with some basic investigations:

Investigations

i) Haematological:
full blood count, ESR

ii) Biochemical:
uric acid, SAP, urea, electrolytes, etc.

iii) Immunological:
RA factor, C-reactive protein, ANF, PSA

iv) Imaging modalities:
plain radiographs of the spine
C. T. scan (with or without contrast)
MRI (magnetic resonance imaging)

TREATMENT

Once a cause for the pain is established, treatment is fairly straight forward, i.e. treat the cause e.g:

Prolapsed disc – Conservative: rest, short wave diathermy and traction, analgesics and abstinence from heavy strenuous physical activities. Surgery is only indicated in a small proportion of cases. Indications for surgery are:

Absolute : central disc prolapse with bladder or bowel incontinence

Relative : failed conservative treatment with intolerable pain (sciatica).

Be very cautious about offering surgery for prolonged backache without sciatica as the results of such surgery are unpredicable.

A change of lifestyle, work habits or even change of occupation may be necessary to tackle the back pain of postural or mechanical origin. Counselling and psychiatric support may be necessary. Often the practitioner may be faced with a problem which is either fairly personal in nature. Such problems usually do not surface early and only through repeated reviews can one elicit the nature of the underlying condition. Thus treating the family and the environment may be the solution to the patient's symptoms.

USE OF DISEASE MODIFYING ANTI-RHEUMATIC DRUGS

S C Ng, MBBS (S), M Med (Int Med), FAMS

SUMMARY

Disease Modifying Anti-Rheumatic Drugs (DMARDs) are drugs used to modify the long term outcome of Rheumatoid Arthritis. Currently there are very few DMARDs available for use, and all of them are toxic. However with careful monitoring these drugs can be used successfully to improve the quality of life of the patients and reduce the deformities and disability. DMARDs can be used singly, or in combinations when the disease is more aggressive. There has been a recent trend to use DMARDs earlier in the course of the disease because it is now realised that aggressive RA can cause erosions within two years of onset and if left untreated for too long the RA becomes more difficult to treat. Though this new approach sounds logical there is still no firm evidence that it is really more effective and just as safe as the traditional and more conservative approach. More studies are needed to prove it.

INTRODUCTION

Disease Modifying Anti-Rheumatic Drugs (DMARDs) as the name suggests are drugs that modify the outcome of the rheumatic disease. This is in contrast to Symptom Modifying Anti-Rheumatic Drugs or SMARDs (e.g. non-steroidal anti-inflammatory drugs) which act quickly to modify the symptoms experienced by the patient but may not improve the long term outcome of the disease. We use SMARDS for all rheumatic diseases but DMARDs are needed only for those conditions which deteriorate in the long term if treated only with a SMARD.

Although the term DMARD was mainly used to describe drugs used in Rheumatoid Arthritis, the real DMARDs in rheumatic diseases are antibiotics for septic arthritis (because they kill the bacteria causing the infection) and urate lowering drugs for gout. This article focuses only on the DMARDs used in RA.

Consultant Physician
Department of Medicine
Alexandra Hospital
Singapore 0314

Over the years several other names have been used to describe this group of drugs. The term Slow Acting Anti-Rheumatic Drugs (SAARDs) was coined to emphasise the fact that several weeks or even months may pass before any clinical benefit can be seen. Another name used is Remission Inducing Drug (RID). This name is as optimistic as the term SMARD. The most recent name given is Disease Controlling Anti-Rheumatic Drugs (DCART). To be classified as a DCART the drug, when used for at least one year, must be shown to have the following actions:

1. improve and sustain physical function
2. decrease inflammatory synovitis
3. prevent or significantly decrease progression of structural joint damage (erosions).

TYPES OF DMARDs

The currently used SMARDs are gold (injectable and oral), D-penicillamine, sulphasalazine, methotrexate, azathioprine, anti-malarials (e.g.

hydroxychloroquine), cyclophosphamide and cyclosporin A. Review of studies done on these drugs used in a conventional way has shown improved quality of life of the patients but many of these patients do end up with some deformity and disability.

Gold

Gold was first used as medicine more than 4000 years ago by the Egyptians. It was in 1935 that Forrestier found that gold was effective in the treatment of RA. Since then trials conducted by the Research Subcommittee of the Empire Rheumatism Council and the Co-operating Clinic for the Systematic Study of Rheumatic Diseases of the American Rheumatism Association have proven the benefit of gold in RA. Gold slightly retards erosions in RA. Before starting gold therapy, we have to ensure that the kidney, liver and blood counts of the patient are normal. After a test dose of 10 mg of injection myocrysine or solganol, the patient can be given weekly doses of 50 mg of the drug with monitoring of the urine and blood. The risk of side effects increases with the cumulative dose of gold. The side effects include:

Adverse effects of injection gold:

mucocutaneous lesions (15-30%), nephrotic syndrome (3-7%), thrombocytopenia (3%), aplastic anaemia, enterocolitis, transaminitis and cholestasis, hypersensitivity pneumonitis, peripheral and cranial neuropathy, post-injection vasomotor syndrome and post-injection non-vasomotor syndrome.

Adverse effects of oral gold:

gastrointestinal upset (40%), rashes (30%), stomatitis and conjunctivitis (10%), proteinuria, thrombocytopenia and leucopenia.

Patients given gold must be carefully monitored before their injections. They should be asked about oral ulcers and skin rashes or itchiness and their urine and blood counts must be checked according to the schedule given. The doctor should also be on the lookout for the other side effects above.

D-penicillamine

This drug was first found to be useful for RA in 1970. As the initial experience with the drug found it toxic because of the high doses used, the advice on use of this drug was to "go low, go slow". Various trials were carried out and showed that it was effective particularly at the higher doses. Some authors suggest that D-penicillamine is useful for extra-articular RA including rheumatoid vasculitis and nodules. Toxicity is a big problem.

Adverse effects of D-penicillamine:

dysgeusia (12.8%), rashes (11.8%), proteinuria (9%), gastrointestinal distress (7.3%), thrombocytopenia (7.1%), nausea (4.5%), neutropenia, oral ulcers, autoimmune phenomenon (drug induced SLE, polymyositis, Goodpasture's syndrome, myasthenia gravis, pemphigus) and teratogenesis.

Patients taking this drug must be followed-up and have their blood counts and urinalysis monitored regularly according to the protocol. Toxicity is often dose related and locally the usual dose used is 250 mg per day even though the textbooks advise doses of up to 750 mg per day.

Anti-malarials

Anti-malarials were found to be effective for SLE and RA after the chance use of the drug for DLE in 1951. These drugs have a very slow onset (full effects may take months) and are considered to be fairly mild but safe. In view of their relatively good safety profile, anti-malarials are often used in combination with other SMARDs. Though declared as fairly safe (no life threatening toxicity), there are many potential adverse effects.

Adverse effects of anti-malarials:

blurred vision with difficulty in accommodation, retinopathy and corneal deposits, myopathy, cardiomyopathy (in the event of an overdose), gastrointestinal upset, marrow aplasia, ototoxicity and skin pigmentation.

Patients who are prescribed these drugs on a long term basis must be followed up by the ophthalmologist every six months to look for silent retinopathy. It is generally felt that retinal toxicity is related to daily dose rather than the total cumulative dose. The full blood count should also be checked periodically. Hydroxychloroquine is generally less toxic than chloroquine.

Sulphasalazine

This drug was first synthesised for the treatment of RA in 1930. However forty years elapsed before the true value of sulphasalazine as an anti-rheumatic drug was appreciated. It is made up of sulphapyridine and 5-aminosalicylic acid and the active component is the antibiotic portion which is the sulphapyridine. Trials have shown that it is as effective as gold in RA. Sulphasalazine is more effective than hydroxychloroquine in slowing down the progression of bony erosions. Like the anti-malarials, this is a relatively safe drug but has known side effects that we have to watch out for.

Adverse effects of sulphasalazine:

nausea, vomiting, abdominal pain, headache, fever, light headedness, dizziness are all more common; rashes, anaemia, leucopenia and thrombocytopenia, hypersensitivity (fever, rash, lung infiltrates and hepatitis) and oligospermia.

Patients are generally started on 500 mg (1 tablet) per day and if they tolerate the drug, the dose is increased to 2 or 3 gm a day in divided doses depending on the response. One problem with this drug is that the patient has to take up to six tablets a day (unlike D-penicillamine or hydroxychloroquine which is a one-tablet-a-day dosage). Patients given this drug should have their full blood count monitored regularly.

Methotrexate

Methotrexate (MTX) has been used for RA since 1972 when it was shown that low dose MTX was useful for RA. Since then many studies have shown that it is an effective SMARD. Currently it is one of the most popular anti-rheumatic drugs in the USA. Compared to the other SMARDs, it has a relatively rapid onset of action. It is given as a weekly dose starting from 5 mg / week to 20 mg / week.

Adverse reactions of methotrexate:

gastrointestinal upset in 10% (nausea, loss of appetite, diarrhoea and stomatitis), alopecia, ultra violet light sensitivity, vasculitis, exacerbation of rheumatoid nodules, megaloblastic anaemia, leucopenia, thrombocytopenia, pulmonary hypersensitivity, teratogenicity, oligospermia, central nervous system problems (like dizziness, headache, mood alteration and memory impairment).

The most well known of all the toxicities is hepatotoxicity. It is now known that the risk for significant hepatotoxicity is 1:1000 at five years. The possible risk factors for hepatotoxicity are increased dose, increased age and associated alcohol consumption. There is no need for a pretreatment liver biopsy unless there is a past history of liver disease or alcohol intake, but a biopsy of the liver should be done after a total dose of 1.5 mg or about two to three years of therapy. A large study has shown that after 1.5 gm of methotrexate, the liver biopsy showed mild fibrosis in 8%, moderate fibrosis at 0.5%, and cirrhosis in 0.1% of patients. The morbidity and mortality of liver biopsy done either as an inpatient or outpatient procedure is 0.03% each. Patients who are on MTX have to be monitored regularly and have their blood count and liver and renal function tests done periodically. A baseline CXR is recommended but regular follow-up CXR is not necessary.

Other Drugs

The other drugs used for RA include **azathioprine** which is effective also as a steroid sparing agent. There is an increased risk of malignancy in patients on azathioprine, particularly Non-Hodgkin Lymphoma. **Cyclophosphamide**, though a very effective SMARD, should not be used routinely for RA. This is because of the serious adverse side effects known. These include carcinogenicity, infertility, marrow suppression and bladder toxicities. Its only role is in the treatment of severe RA with vasculitis. **Cyclosporin** is an effective drug for RA but is difficult to use because the

lower doses do not work and the higher doses are toxic particularly to the kidneys. The concomitant use of NSAIDs further compounds the nephrotoxicity. The best dose is probably about 5 mg / kg / day in divided doses. The blood pressure and renal function have to be monitored carefully. Other side effects like hepatotoxicity, nausea, vomiting, tremors, paraesthesia, gum hyperplasia and hirsutism are also seen.

Steroids

Steroids are the most potent and fastest acting anti-inflammatory drugs. Steroids are best classifies as Symptom Modifying Anti-Rheumatic Drugs but there is some suggestion that they may have some disease modifying activity. The major problem with steroids is the high incidence of adverse effects and hence they should be used together with another SMARD.

Adverse effects of steroids:

cushingoid facies with the moon face, buffalo hump, striae, osteoporosis, ischaemic necrosis, steroid myopathy, sub-capsular cataract, acute anterior uveitis, glaucoma, impaired glucose tolerance, impaired lipid metabolism, sodium retention, potassium loss, increased risk of infections, increased risk of NSAID gastropathy, intestinal perforation, pancreatitis, steroid psychosis, seizures, cortical atrophy and benign intracranial hypertension.

Hence before starting the patient on steroids, the doctor should tell the patient about the many potential side effects of the drug. Then explain that it is a very useful drug because it is very powerful drug and acts very quickly to relieve the symptoms of inflammation. When the patient appreciates the danger of prolonged steroid ingestion, he or she will be very cooperative when it is time to discontinue it. Sometimes the patient will even ask the doctor if the steroids can be stopped. We must always start steroids together with a DMARD. In this way the patient immediately feels better due to the steroids and after a few months when the DMARD starts to take effect, the steroids can be tapered off. Steroids can also be used for intra-articular injections or as bolus doses in cases with severe systemic flare despite SMARDs.

CHOICE OF DMARDs

In 1993, Paulus reviewed the literature and found that if we were to look at individual drug trials, almost every drug would have a study to show that it is superior to another. D-penicillamine was the better drug in 2 out of 6 trials. Injectable gold was the better drug in 3 out of 8 trials. Auranofin was the better drug in 1 out of 4 trials. Methotrexate was the better drug in 1 out of 2 trials. Sulphasalazine was the better drug in 1 out of 3 trials. Anti-malarials were the better drug in 1 out 5 trials.

Another way to chose a DMARD was suggested in a meta analysis of second line drugs in 79 trials. When we compare improvement in composite index, the most effective drug was methotrexate followed by sulphasalazine and D-penicillamine. When we compare improvement in joint counts, methotrexate and sulphasalazine were again the top two drugs. When comparing toxicity, the most toxic drug was injection gold followed by D-penicillamine. Anti-malarials were the least toxic drug. The drugs with the fewest dropouts due to toxicity were anti-malarials and methotrexate. However many other factors have to be considered. These include previous drug allergies, renal function, liver function, cost of drugs, convenience of daily doses versus weekly injections and patient preference.

WAYS TO USE DMARDs

In the past few years, concepts regarding the treatment of RA have changed dramatically. Previously, doctors have been very conservative and tended to use only NSAIDs for very long periods until bony erosions and deformities were obvious. Then they may have started a SMARD. This conservative approach did not adequately treat those who had aggressive disease. The present proposal is to identify the group of patients with aggressive disease and adopt a more aggressive approach in their treatment. Indeed, now that we know erosions can occur as early as two years from the onset of the disease, starting DMARDs late may well be too late.

With the above concept in place, there are now three ways to use DMARDs:

The Traditional Approach

The traditional approach is to use on NSAID and add one DMARD to treat the RA if the disease is not adequately controlled. The decision to start the DMARD is made much earlier than in the past, and any one of the DMARDs listed above can be used. This approach is best suited for patients with mild disease. Some patients may never need a DMARD and all symptoms and signs of inflammation go away with NSAIDs alone.

Combination of Two DMARDs

This is a more aggressive approach and the hope is that two drugs may have additive efficacy without any increase in toxicity. The results of open trials were encouraging but controlled trials were less so. Various combinations have been tried and most of them include an anti-malarial because of the relative safety of this group of drugs.

Multiple DMARD Step Down Bridge Approach

This is the most aggressive strategy of all. The RA is treated like "cancer" with multiple DMARDs initially to "achieve remission" and then the more toxic drugs are discontinued, leaving the less toxic drugs to "maintain remission." An example of such a combination is steroids, methotrexate, azathioprine, gold and hydroxychloroquine. Once the RA is controlled the more toxic drugs are discontinued until only hydroxychloroquine is left. As the potential for toxicity is great, only patients identified to have aggressive disease should be treated in this fashion. Patients who fall into this category have the following characteristics:

> multiple actively inflamed joints especially the metacarpal-phalangeal joints, high titre of rheumatoid factor initially, high ESR initially, extra articular disease, presence of rheumatoid nodules, thrombocytosis, increased circulating immune complex and presence of HLA DR4, Dw4 and Dw14.

Though logical, this approach is untested and should not be recommended for routine use at this time.

CONCLUSION

DMARDs are an important part of the treatment of RA because they modify the outcome of the disease. They are all toxic and should be used with care. Currently we have only a few to choose from and none is perfect. As we begin to understand the pathogenesis of RA we hope to find new and better DMARDs. In the meantime we have to find better ways to use the available ones.

ORIGINAL ARTICLE

ANXIETY-DEPRESSIVE NEUROSIS IN CHINESE PATIENTS

W F Tsoi, MD, DPM, FRCPG, FRANZCP, FRCPsych

SUMMARY

This paper studies the symptomatology of 120 consecutive Chinese patients diagnosed as anxiety-depressive neurosis. The mean duration of their illness was about 6 years. The ten most frequent individual symptoms were (1) feeling tired, (2) anxious, (3) giddy, (4) headache, (5) depressed, (6) insomnia, (7) tense, (8) palpitation, (9) irritability and (10) chest discomfort. There was considerable overlap of symptoms between the anxiety and depressive neuroses. There was no differences between the males and females. The older patients had more symptoms and a longer duration of illness. Previous studies also showed an overlap of symptoms between anxiety and depressive neurosis. They could be classified as a single mixed anxiety-depressive disorder. The pharmacological and psychological treatment of anxiety neurosis and depresive neurosis is similar.

Key words:
anxiety, Chinese, depression, neurosis, symptomatology.

INTRODUCTION

The aim of this paper is to study the symptomatology of anxiety-depressive neurosis in Chinese patients. In primary medical practice, neurotic disorders (new term for neuroses) can be divided clinically into two broad groups according to their symptomatology and prevalence.

1. A group of anxiety-depressive neurosis which covers the categories anxiety neurosis, depressive neurosis, neurasthenia and mixed anxiety-depressive neurosis. These disorders are common but their symptoms are ill-defined, mixed and multiple, and do not easily differentiate these four conditions from each other.

2. A group of less common neurotic disorders which covers the categories panic disorder, phobia, obsessive compulsive disorder, conversion hysteria and dissociative hysteria. Their clinical pictures are fairly well defined.

The symptoms of anxiety-depressive neurosis are not only ill-defined, but are to some extent influenced by the patients' personal and cultural experience e.g. it was found that Chinese patients tend to somatize their anxiety and depressive symptoms [1,2,3].

The aim of this study is to present the symptomatology of anxiety-depressive neurosis in a group of Singapore Chinese patients and to find out whether these symptoms represent one or more than one disorder.

Consultant Psychiatrist
Gleneagles Medical Centre #10-08
6 Napier Road
Singapore 1025

Work carried out at,
Department of Psychological Medicine
National University of Singapore

MATERIALS AND METHODS

The subjects were 120 consecutive patients attending the psychiatric out-patients clinics of the University Department of Psychological Medicine in the Singapore General Hospital over a 4 year period, presenting with anxiety, depressive and somatic symptoms for which there was no detectable organic pathology. A psychiatric examination was carried out by the author and only those patients diagnosed as "Anxiety Neurosis" and "Depressive Neurosis" according to The World Health Organisation International Classification of Diseases 9 edition (ICD-9)[4] criteria were included. The patients were given a check-list of 6 groups of symptoms: pain, discomforts, autonomic symptoms, anxiety symptoms, depressive symptoms and insomnia (appendix 1). The statistics used for comparing the symptoms of anxiety and depressive neuroses is Chi-square.

RESULTS

There were about equal numbers of male (58) and female (62) patients. The age ranged from 18 to 76 with the mean at 35.3 years. Most of them (63%) belonged to the age group 20-39 years. Only 2% were 60 years and above (Table 1). There were no significant differences between the males and females in their educational background. Half (50%) were English educated and about three quarters (73%) had received at least secondary education.

The presenting class of initial complaints in order of frequency were discomfort, pain, insomnia, anxiety and depression. The mean duration of their illness was about 6 years and the mean duration of their present symptoms was about 10 months. There were no differences between the male and females (Table 2).

Table 1: Basic Characteristics of Patients

	Male N=58		Female N=62		Total N=120	
AGE						
Mean Age	34.5 yr		36.0 yr		35.3 yr	
Std Dev	11.7 yr		11.3 yr		11.5 yr	
EDUCATION						
English stream	26	45%	34	55%	60	50%
Chinese stream	32	55%	28	45%	60	50%
Primary	11	20%	18	30%	29	25%
Secondary	31	55%	25	41%	56	48%
Above Secondary	15	27%	18	29%	33	28%

Table 2: Main Complaints

	Male		Female		Total	
SYMPTOM TYPES						
discomfort	20	35%	23	37%	3	36%
pain	9	16%	12	19%	21	18%
insomnia	8	14%	12	19%	20	17%
anxiety	8	14%	8	13%	16	13%
depression	8	14%	5	8%	13	11%
MEAN DURATION						
Illness	79.1 mth		66.2 mth		72.5 mth	
Present Symptoms	10.5 mth		9.6 mth		10.0 mth	

The 15 most frequent individual symptoms (Table 3) were feeling weak or tired, anxious or worries, giddy or dizzy, headache, feel sad or depressed, cannot fall asleep at night, feel tense or cannot relax, palpitation, irritability, and chest discomfort. There were no significant differences in the frequency of most of the symptoms between the males and females, between their educational stream (English or Chinese educated), and their education levels (primary, secondary and post-secondary). The most frequent anatomical sites for somatic symptoms were the head, chest and abdomen.

When the patients were divided into two groups according to their ages: younger patients (age 10-39 years) and older patients (age 40-70 years), the older patients had a longer duration of illness (14.3 months) than the younger patients (8.2 months). The older patients also had more symptoms.

Table 3: Most Frequent Symptoms (Affecting more than 50% of Patients)

Symptoms	Male	Female	Total
1. feeling weak or tired	85%	77%	81%
2. anxious or worries	81%	71%	76%
3. giddy or dizzy	67%	76%	72%
4. headache	62%	76%	69%
5. feel sad or depressed	69%	65%	67%
6. cannot fall asleep at night ...	60%	69%	65%
7. feel tense or cannot relax... ...	66%	60%	63%
8. heart beating fast...	57%	63%	60%
9. feel angry easily or irritable ...	64%	55%	59%
10. chest discomfort	52%	63%	58%
11. feel frightened for no reason ...	59%	55%	57%
12. have poor concentration... ...	64%	47%	55%
13. difficulty breathing...	52%	57%	54%
14. have poor memory...	53%	50%	52%
15. chest or heart pain...	48%	53%	51%

Those patients diagnosed as anxiety neurosis did not differ significantly from those diagnosed an depressive neurosis in their age, duration of illness and the duration of current episodes (Table 4). The anxiety neurosis patients had more complaints of chest discomfort, difficulty breathing, excessive sweating and feeling tense than the depressive neurosis patients.

Table 4: Breakdown of Symptoms by Diagnosis

Symptoms	Anxiety N=83	Depression N=37	Total N=120	Signi-ficance
Pain Symptoms				
headache	65%	78%	69%	NS
chest or heart pain	54%	43%	51%	NS
back or kidney pain	35%	49%	39%	NS
joint pain	37%	41%	38%	NS
abdominal pain	36%	41%	38%	NS
neck pain	31%	49%	37%	NS
bone pain	35%	22%	31%	NS
muscle pain	31%	30%	31%	NS
Somatic symptoms				
weak or tired	82%	78%	81%	NS
giddy or dizzy	70%	76%	72%	NS
palpitation	66%	46%	60%	NS
chest discomfort	65%	41%	58%	P<0.05*
difficulty breathing	61%	38%	54%	P<0.01*
stomach discomfort	45%	51%	47%	NS
limb numbness	47%	35%	43%	NS
vomiting feeling	37%	41%	38%	NS
fainting feeling	33%	30%	32%	NS
Autonomic Symptoms				
hands trembling	48%	38%	45%	NS
sweat a lot	48%	27%	42%	P<0.05*
feeling cold	30%	27%	29%	NS
constipation	22%	30%	24%	NS
frequency micturation	22%	16%	20%	NS
Anxiety Symptoms				
anxious or worries	80%	68%	76%	NS
tense (cannot relax)	69%	49%	63%	P<0.05*
angry, irritable	58%	62%	59%	NS
fear for no reason	61%	46%	57%	NS
restless	43%	43%	43%	NS
phobia	27%	30%	28%	NS
Depressive Symptoms				
depressed	64%	73%	67%	NS
poor concentration	55%	54%	55%	NS
poor memory	49%	57%	52%	NS
feel useless	36%	49%	40%	NS
guilt feelings	25%	32%	28%	NS
suicide idea	24%	30%	26%	NS
Sleep Disturbance				
insomnia	68%	60%	65%	NS
frequent waking	35%	24%	32%	NS
wake up very early	23%	30%	25%	NS
dreams or nightmares	25%	24%	25%	NS

* Statistics used: chi-square.

DISCUSSION

The term neurosis was first introduced in the 18th century by Cullen in his system of classification of diseases as "disorders of the nervous system" which is equivalent to the present day neurological disorders. In the late 19th century, Freud separated those neuroses without an organic basis and called them psychoneurosis, in the belief that the origin of psychoneurosis could be traced to unconscious conflicts in the mind between basic needs (sex and aggression) and their fulfillment. The term neurosis is unsatisfactory. There are many definitions but none are universally accepted.

In psychiatry, neurosis is a descriptive term for a group of minor psychiatric illness which are classified according to their main symptoms: e.g. anxiety neurosis, depressive neurosis, phobic neurosis, obsessive-compulsive neurosis, hypochondriacal neurosis, hysterical neurosis.

The term "neurosis" is now being replaced with a less emotional term "disorder" e.g. anxiety disorder, phobic disorder, obsessive-compulsive disorder, conversion hysterical disorder, etc. Because of the lack of well-defined organic pathology and the subjective nature of the complaints, the subtypes are unstable and are not aetiologically or genetically distinct disease entities.

The two most common neuroses are anxiety neurosis and depressive neurosis. Anxiety and depressive neuroses were traditionally classified under the heading of "neurotic disorders". The World Health Organisation International Classification of Disease 10th edition (ICD-10)[5], classifies anxiety neurosis and depressive neurosis under mood disorders. It also recognizes a mixed anxiety and depressive disorder.

Anxiety neurosis used to be divided into acute, subacute and chronic subtypes. Acute anxiety disorder is being renamed as panic disorder. Subacute anxiety neurosis is being replaced by the term generalized anxiety disorder. What was known as chronic anxiety neurosis would now be covered by a number of conditions such as somatization disorder, hypochondriasis, or neurasthenia depending on their main clinical symptoms.

Depression was divided into psychotic, endogenous, reactive, neurotic and masked depressions. While there are clear cut differences between endogenous depression and acute anxiety neurosis, it is not so easy to separate the other subtypes e.g. neurotic and masked depression from chronic anxiety neurosis, somatization neurosis, hypochondriasis and neurasthenia.

The overlap of symptoms between anxiety neurosis and depressive neurosis has been well documented. As early as 1934 Lewis[6] noted that the relation of anxiety to depression is intimate. According to Cleghorn (1970)[7] the terms "anxiety neurosis" and "depressive neurosis" should be replaced by "anxiety-depressive syndrome". Symptoms of anxiety seldom occur without symptoms of depression. Hamilton (1981)[8] found that depressed mood occurs in 71% and 95% in anxiety disorders and anxiety symptoms occur in 86% and 96% of males and females respectively with depressive illness. Curran and Partridge (1970)[9] grouped anxiety neurosis under affective disorders. Goldberg (1976)[10], who used the General Health Questionnaire (GHQ) to assess symptomatology in anxiety and depression, was unable to find any principal component analysis that separated anxiety and depressive symptoms into two distinct dimensions. Anxiety and depressive disorders include somatic symptoms, and somatoform disorders appear to be related to the affective and anxiety disorders[11,12,13]. Somatization is common in Asian patients[1,2,3]. Tyrer et al (1987)[14] found that symptoms of anxiety, panic, depression, hypochondriasis and phobias were recorded in 78 psychiatric patients with depressive, anxiety and phobic neuroses. The results showed great temporal variability of anxiety and depressive symptoms. These symptoms were also poorly related to diagnosis. Their findings suggest that classifications of neurotic disorder based on presenting anxiety and depressive symptoms are unsatisfactory and that many patients could be classified as a single mixed disorder. To support this, the WHO-ICD10[5] has created a new entity known as mixed anxiety and depressive disorder.

To further support the hypothesis, the pharmacological treatment for acute anxiety neurosis (panic disorder) is similar to that for endogenous depression. The severity of symptoms

of anxiety and depression does not predict response to amitriptyline and diazepam. The response to drug treatment does not predict a distinction between anxiety and depression[15]. Both conditions – acute anxiety neurosis (panic disorder) and endogenous depression (major depression) – are currently treated with antidepressants. The treatment of choice for chronic anxiety neurosis and neurotic depression is psychotherapy.

While endogenous depression and panic disorder are relatively uncommon in general medical practice, anxiety-depression (chronic anxiety neurosis and neurotic depression) is the most prevalent psychiatric disorder in the community.

CONCLUSION

The results of this study on 120 Chinese patients show that it is not easy to differentiate anxiety neurosis from depressive neurosis. The patients have similar clinical manifestations and they present mainly with somatic complaints. It is therefore important to consider anxiety-depression as a differential diagnosis in any patients who present with multiple physical complaints and to consider antidepressants if they do not respond to antianxiety drugs.

References

1. Kleinman A M. Depression, somatisation and the "New Cross-Culture Psychiatry". Soc Sci Med 1977; 11:3-10.
2. Kung N Y T, Lau B W K, Chung J T C: Masked depression in general practice in Hong Kong. Singapore Med J 1982; 23:220-5.
3. Tseng W S: The nature of somatic complaints among psychiatric patients: the Chinese case. Comprehens Psychiat 1975; 16:237-45.
4. World Health Organisation International Classification of Diseases 9th edition (ICD-19). WHO 1978.
5. World Health Organisation International Classification of Diseases 10th edition (ICD-10). WHO 1990.
6. Lewis A J: Melancholia – a clinic survey of depressive states. J Ment Sc 1934; 80:277-278.
7. Cleghorn J: The anxiety-depression syndrome. Psychosom 1970; 11:438-441.
8. Hamilton M: The assessment of anxiety states by rating. Br J Med Psychol 1981; 32:50-55.
9. Curran and Partridge: Psychological Medicine. Oxford University Press, 1970.
10. Goldgerg DP, Rickels K, Downing R, Hesbacher P: A comparison of two psychiatric screening tests. Br J Psychiatry 1976; 129:61-67.
11. Fabrega H, Mezzich J, Jacob R, Ulrich R. Somatoform disorder in a psychiatric setting. J Nerv Ment Dis 1988; 431-439.
12. Liskow B, Othmer E, Jenick E C et al: Is Briquet's syndrome a heterogenous disorder? Am J Med 1986; 143:626-629.
13. Lloyd G G: Psychiatric syndromes with a somatic presentation. J Psychosom Res 1986; 30:113-120.
14. Tyrer P, Alexander J, Remington M, Riley P: Relationship between neurotic symptoms and neurotic diagnosis: a longitudinal study. J Affect Disord 1987; 13:13-21.
15. Johnstone E C, Owen D G C, Firth C D, et al: Neurotic illness and its response to anxiolytic and antidepressant treatment. Psychold Med 1980; 10:321-328.

APPENDIX

NAME _____ REF NO _____

Please circle "yes", those symptoms which bothered you during the PAST ONE MONTH.

1.	headache	yes
2.	neck pain	yes
3.	chest or heart pain	yes
4.	stomach or abdominal pain ...	yes
5.	backache or kidney pain	yes
6.	joint pain or rheumatism... ...	yes
7.	bone pain	yes
8.	muscle pain	yes
9.	other aches or pain	yes
11.	giddy or dizzzy	yes
12.	fainting or fainting feeling ...	yes
13.	chest discomfort	yes
14.	heart beating fast...	yes
15.	difficulty breathing	yes
16	stomach full or discomfort ...	yes
17.	vomiting or vomiting feeling ...	yes
18	numbness of hands or legs ...	yes
19	feeling weak or tired	yes
21.	sweat a lot or cold sweat... ...	yes
22.	shaking or trembling of hands ...	yes
23.	need to pass urine frequently ...	yes
24.	pass motion many times	yes
25.	difficulty with constipation ...	yes
26.	feeling cold	yes
31.	anxious or worries	yes
32.	feel frightened for no reason ...	yes
33.	afraid of harmless things... ...	yes
34.	feel tense or cannot relax ...	yes
35.	feel restless – cannot sit still ...	yes
36.	feel angry easily or iritable ...	yes
41.	feel sad or depressed	yes
42.	have feelings of guilt	yes
43.	feel useless (not a useful person)...	yes
44.	have suicide ideas or wish to die...	yes
45.	have poor memory	yes
46.	have poor concentration ...	yes
51.	cannot fall asleep at night ...	yes
52.	wake up easily at night	yes
53.	wake up very early e.g. 4 a.m. ...	yes
54.	have bad dreams or nightmares ...	yes
55.	cannot wake up in the morning ...	yes
56	feel sleepy during the day ...	yes

ORIGINAL ARTICLE

TREATMENT OPTIONS FOR GALLBLADDER STONES

M Chellappa, MBBS, MS, FRCS, FACG, FACS, FAMS

INTRODUCTION

Gallstones are common. The high prevalence of gallstones has become more apparent after the introduction of ultrasonography because even a routine medical checkup in which an ultrasonography is included picks up a patient with gallstone disease. The incidence is higher in women and it also occurs in an earlier age in them. Quite a significant percentage of the gallstones are asymptomatic and the dictum that asymptomatic gallstones should be left alone still holds good. This has been reemphasised by a study done in 1983 which showed that 13 percent of the patients with gallstones developed biliary pain, and complications developed in only 2 percent. Although this study was done in the era where laparoscopic cholecystectomy was not prevalent, the reduced morbidity and more patient compliance for laparoscopic cholecystectomy has not changed the general rule that only symptomatic gallstones should be treated.

There are a few exceptions to this rule, such as:

(1) patients with large single stones of significant size who may run a high risk of developing gall bladder cancer, at least statistically, and

(2) patients suffering a combination of diabetes and gallstones.

In the latter, the higher chance of them developing morbid complications after an acute cholecystitis would clearly warrant an elective surgery even in the absence of symptoms. Patients suffering from symptomatic gallstones have got three options:

(a) dissolution therapy,
(b) extracorporeal shock wave lithotripsy, (ESWL), and
(c) cholecystectomy

Other procedures like contact dissolution therapy and cholecystolithotomy (where through intervention just the stones were removed) have had limited success and have been abandoned as routine procedures in clinical practice.

ORAL DISSOLUTION THERAPY FOR GALLSTONES

Patients suitable for treatment with gallstone-dissolving drugs should have a functioning gall bladder. This can be easily established by performing oral cholecystography. The stones should also be radiolucent and less than 15 mm in size. Very small and lucent stones on oral cholecystography are commonly rich in cholesterol and are suitable for dissolution therapy. Dissolution therapy has no effect on calcified or pigment stones, nor on stones within a non-functioning gall bladder. Patients who develop cholesterol stones are thought to do so because they secrete bile which is saturated with cholesterol and this could be the result of either an increased secretion of cholesterol or reduced bile acids in bile. They are particularly given bile acids to increase the concentration in bile and they also inhibit cholesterol synthesis and thereby they reduce the saturation of cholesterol in bile. Chenodeoxycholic acid and ursodeoxycholic acid are two natural acclaimed bile acids that are used orally for

Consultant Surgeon
Mount Elizabeth Medical Centre #14-15
Singapore 0922

gallstone dissolution. They produce their effects by different mechanisms. Chenodeoxycholic acid reduces HMG-CoA reductase activity, reducing the synthesis and secretion of cholesterol, and it has no effect on cholesterol absorbtion. On the other hand, ursodeoxycholic acid reduces cholesterol secretion but it has very little effect on cholesterol synthesis. Both these drugs have been widely used in clinical practice and appear to be safe. Chenodeoxycholic acid sometimes may cause diarrhoea and it also causes minor lipid alterations and thus has a small theoretical risk of promoting atherosclerosis. It can also cause minor reversible changes in serum liver enzymes. On the other hand, ursodeoxycholic acid does not cause any adverse side effects and is the preferred single agent in treating gall stones.

Rowachol is a non-bile acid compound which has been reported to dissolve gall stones in clinical practice. It is an inexpensive preparation of six cyclic monoterpene oils, including menthol. It has been shown to depress HMG-CoA reductase and inhibit nucleation of crystals in bile. It has minimal side effects and also enhances the cholelitholytic effect of bile acids.

Treatment of gallstones in suitable patients by oral dissolution therapy is prolonged, stretching over a period of one to two years, and is successful in 60 percent of the patients who have been found to be suitable to undergo this treatment. It can only be used as the treatment of choice in patients with lucent, floating small stones which are symptomatic only occasionally. Only 20 percent of the patients fulfill such requirements.

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY

The extremely high degree of success of ESWL in the management of renal calculi fostered the enthusiasm to use the same technique for the management of gall stones. After fragmentation, the presence of a continued stream of urine ensured the washing down of fragmented renal calculi but on the other hand, the gall bladder is not blessed with such a steady stream of fluid. Also the contraction of the gall bladder is often slow and incomplete and leaves a residual volume of 50 percent or more in patients, especially in those with gallstones. Furthermore, the cystic duct is often a narrow channel of 1-3 mm in size and is difficult to navigate. Because of this, the use of ESWL has been very restricted and selective and associated with a fair degree of recurrence in spite of a successful fragmentation.

Selection criteria for patients to undergo ESWL would include a functioning gall bladder as shown by an oral cholecystogram and lucent gallstones of less than 30 mm in size or not more than 3 radiolucent stones making up a stone mass of 30 mm. After a successful fragmentation, these patients should undergo oral dissolution therapy for a period of 12-18 months. It has been generally found that only 15 percent of the gallstone patient population fit in with this criteria for inclusion under ESWL. There is a very low, 1 to 2 percent, incidence of acute pancreatitis in the post-ESWL period. On the other hand, there could be episodes of biliary colic in as high as 30 percent of the patients.

CHOLECYSTECTOMY

Carl Langenbusch performed the first cholecystectomy in 1982 for treating a patient with cholelithiasis and since then it has been the gold standard with which the other modalities of treatment are compared. The risk of complications is less and the mortality is less than 0.3 percent. Cholecystectomy not only eradicates the symptoms related to gallstones but at the same time prevents future complications. Patients after cholecystectomy are rarely disturbed by persistence of the symptoms, and the so-called post-cholecystectomy syndromes highlighted, in many cases, related to either a wrong diagnosis or residual stones. An increased risk of developing large bowel cancel after cholecystectomy has been reported by some investigators but this has not been confirmed by many others.

Although cholecystectomy has been the best form of treatment for cholelithiasis, the conventional surgery has been associated with a significant stay in the hospital and a prolonged period of recovery before return to normal work. Therefore to reduce cholecystectmy to its least invasive form has lead to the recent development of laparoscopic cholecystectomy. The advantages of laparoscopic

cholecystectomy are a good cosmetic result, rapid resolution of post-operative pain, reduction of duration of hospitalization and a quicker return to work after surgery.

The indications for laparoscopic cholecystectomy are similar to open cholecystectomy. After gaining experience, 95 percent of the patients with gallstone disease can be successfully dealt with by laparoscopic cholecystectomy. Patients with previous history of jaundice or a dilated common bile duct as shown in ultrasound would have to undergo a pre-operative ERCP to explore any common duct stones. Patients with common bile duct stones can also be managed by a pre-operative ERCP, subsequently followed by a laparoscopic cholecystectomy. Laparoscopic cholecystectomy is especially indicated in obesity although it is slightly technically more demanding. Post-operative morbidity of obese patients is much, much less after laparoscopic cholecystectomy than after open cholecystectomy. Patients with severe cardio-respiratory disease can still undergo a laparoscopic cholecystectomy but it may be wiser to use the gasless technique rather than a pneumoperitoneum.

The technique of laparoscopic cholecystectomy has been standardised. Under general anaesthesia, the patient's abdomen is insufflated with carbon dioxide and a 10/12 mm port is inserted into the umbilicus for introduction of the laparoscope. The laparoscope is later connected to a video camera and a TV monitoring system and the rest of the procedure is performed under TV monitoring control. Three more ports, one 10 mm and two 5 mm, are further used to facilitate the retraction of the gall bladder and performance of the procedure. The cystic duct and the artery are ligated with clips and subsequently the gall bladder is separated from the liver bed either by diathermy or by laser. Finally the gall bladder is removed through one of the ports. The patient generally stays in hospital for about 24 to 48 hours after the surgery and returns back to work after about 4 to 5 days. Although laparoscopic cholecystectomy has been widely accepted as the treatment of choice for symptomatic gall stones, there have been initial reports of some instances of bile duct injuries due to improper conduct of this procedure. With meticulous dissection and due care in visualising the anatomy, it is possible to perform this procedure with utmost safety. In a personal series of over 1600 cases, the incidence of bile duct injury was nil. In case of difficulty, operative cholangiogram can be performed.

CONCLUSION

The options available for patients with gall bladder stones have evolved to near perfection over the past years. Unlike peptic ulcer disease, medical treatment is not displacing surgery. On the other hand, surgery is becoming less invasive.

MANAGEMENT OF ARTHRITIC PROBLEMS

** R Ray MBBS, DPH (S'pore), FAMS*
*** L G Goh, MBBS, MMed (Int Med), FCGP, MRCGP (UK)*

GENERAL PRINCIPLES

From the standpoint of management, patients with arthritic conditions can be divided into three groups:

- **Inflammatory arthropathy and physical examination is positive**
  Patients whose history indicates inflammatory polyarthropathy and who have objective evidence of joint involvement are usually fairly easy to manage. If gouty arthritis or septic arthritis is present, the treatment is specific. In inflammatory polyarthritis, the initial management is symptomatic. Self-limiting conditions, particularly a viral illness (which can mimic rheumatoid arthritis), will resolve within six weeks.

  If symptoms persist beyond six weeks, one must establish the most likely diagnosis and then treat the symptoms as they occur; the need for second line drugs may need to be sought.

- **Inflammatory arthropathy but physical examination is normal**
  These patients probably have early arthritis like rheumatoid arthritis but may not have yet developed recognisable features. The patient may develop new symptoms over time or will have a complete resolution of their symptoms. Treatment at this time is with NSAIDs and they should be followed up more closely than the other two groups.

- **Non-inflammatory arthropathy**
  These are patients who have no inflammatory features and results of physical examination may be normal or abnormal. Advice on judicious exercise, and weight reduction of the overweight, is needed.

* *Dy Medical Director*
Family Health Services
Primary Health Division
Ministry of Health
Singapore

** *Senior Lecturer*
Dept of Community
Occupational and Family Medicine
National University of Singapore

DRUG TREATMENT

The drugs for treatment of arthritic conditions fall into five groups: simple analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), second-line antirheumatic drugs, corticosteroids and immuno-suppressives.

Simple analgesics

Simple analgesics are drugs of first choice to relieve pain caused by non-inflammatory arthropathies such as cervical or lumbar spondylosis and osteoarthritis. In rheumatoid arthritis they are unlikely to be helpful if there is active inflammatory disease with morning stiffness, but they may be helpful in pain relief caused by long-standing destructive disease where the inflammatory element has burnt out. They need to be prescribed only on-demand.

Paracetamol is the first choice simple analgesic for the relief of non-inflammatory arthritic pain.

Non-steroidal anti-inflammatory drugs (NSAIDs)

The main role of NSAIDs is to provide symptomatic relief of pain and stiffness in patients with inflammatory joint disorders. They do not prevent erosions.

Table 1: Classification of NSAIDs

Generic name (Brand name)	Class	Dose Per Talbet	Adult Dose
Indomethacin (Indocid)	Phenylacetic acid derivative	25 or 50 mg	25 mg TDS 50 mg ON
Diclofenac (Voltaren)	Phenylacetic acid derivative	25 mg	25-50 mg BD to TDS
Ketoprofen (Orudis)	Propionic acid derivative	50 mg	50 mg TDS
Naproxen (Naprosyn)	Propionic acid derivative	250 or 500 mg	250 mg - 500 mg BD
Mefenamic acid (Ponstan)	Fenamate	250 mg	250-500 mg TDS
Piroxicam (Feldene)	Oxicam drivative	10 or 20 mg	20 mg OM single dose

NSAIDs are indicated in rheumatoid arthritis, ankylosing spondylitis and other seronegative arthritides. They are also indicated, with the exception of salicylates (which in low doage inhibit renal excretion of urate), in acute gout or pseudogout. They may be helpful in relieving pain associated with osteoarthritis that has not responded to a simple analgesic.

Guidelines for use

- Be familiar with at least one from each class, particularly dosage and side-effects. Choose one of the established drugs first. A new drug is not necessarily better.
- In general the propionic acid derivatives are best tolerated and are the drugs of first choice in the treatment of rheumatoid arthritis.
- A diary of symptoms may indicate the best time to administer drugs, e.g. at night to relieve early morning stiffness.
- If a particular NSAID is going to help, the patient will usually notice some improvement within 48 hours, and certainly within two weeks.
- There are marked individual variations in response (as well as in side-effects) to a particular NSAID, so that if the first NSAID does not produce a response in maximum tolerated dosage, it is worth trying an alternative.
- Combinations are to be avoided, as they are no more effective than the equivalent dose of a single drug given alone, may interact with one another and may also increase the risk of side-effects.

Side effects of NSAIDs

- hypertension; indomethacin in particular may cause a sharp rise of blood pressure.
- gastroduodenal injury: dyspepsia, erosions and ulcerations
- fluid retention
- impaired liver function
- may precipitate bronchial asthma

Special precautions in the use of NSAIDs

- *Past allergic reactions.* Avoid NSAIDs in patients allergic to Aspirin. Be aware that allergy to one NSAID has increased risk of allergy to another NSAID, even of a different class.
- *Atopy and bronchial asthma.* Caution in prescribing NSAIDs. Observe patient for an hour in the clinic after the first dose.
- *Heart failure, renal impairment.* Fluid retention may result. Review patient more frequently; prescribe a lower dose.
- *Hypertension.* Until more data is made available, the prescription of any NSAID to a hypertensive patient should be followed by more frequent blood pressure measurements.

Indomethacin in particular may cause undesirable increases in blood pressure in some patients.

- *Elderly.* Use smaller doses.
- *Pregnancy.* Avoid throughout pregnancy.
- *Lactation.* Only a very small amount is secreted into breast milk.
- *Patients at risk of NSAID gastroduodenal injury*:
  - recent peptic ulceration (within 5 years)
  - steroids given in addition to NSAIDs.

  Co-administration of anti-ulcer therapy is needed for such patients.

NSAID-induced gastroduodenal injury

- *NSAID induced dyspepsia.* This is the most common form of injury. Manage by stopping or decreasing the drug dosage, taking the drug with food or concomitantly administering H_2 blockers in full split doses. NSAID induced dyspepsia, in the absence of anaemia, signs of GI bleeding, weight loss or other worrisome associated symptoms is not an indication for endoscopy.
- *NSAID induced erosions.* For patients who undergo endoscopy, the most common endoscopic abnormality is erosions. These are more often in the stomach than the duodenum. Erosions seldom cause clinically significant bleeding and are by definition insufficiently deep to cause perforation. They can be managed in a similar fashion to NSAID dyspepsia.
- *NSAID ulcer.* NSAID-induced ulcers are more common in the stomach than in the duodenum.
  - *NSAID -nduced duodenal ulcer* is relatively easy to heal and can be treated with standard split doses of H_2 blockers, full doses of sucralfate or omeprazole. In general, NSAID-induced duodenal ulcers can be healed while the patient continues to take the NSAID.
  - *NSAID-induced gastric ulcer* is more difficult to heal, particularly if it is are large (>5 mm) or if the patient continues to take the NSAID. If the NSAID is to be discontinued, the treatment is with H_2 antagonist or omeprazole. Sucralfate is not effective. If the NSAID is to be continued, the treatment is with omeprazole. Misoprostol will heal NSAID-induced gastric ulcers even while the patient continues to take the NSAID. However, a relatively low healing rate of 60% and the toxicity of the drug makes it unacceptable for this use.

Second-line anti-rheumatic drugs or disease modifying drugs (DMARDs)

These are slow-acting, disease suppressing drugs. Drugs in this group are gold (oral or intramuscular), penicillamine, chloroquine, sulphasalazine, methotrexate and azathioprine. All are given on a long-term basis and have a disease suppressing effect that is delayed for two to three months. As these drugs have no immediate anti-inflammatory effect, they are often given in combination with an NSAID. Specialist referral is appropriate for most patients who require this type of drugs.

Indications

- Rheumatoid arthritis or the peripheral arthritis of the seronegative arthritides in patients who have persistently active inflammatory disease not responding to NSAIDs within a six month period.
- Progressive erosive changes on x-rays.

Principles in the use of DMARDs

- Begin early, that is within a six month period, if active disease persists.
- Patients should be monitored regularly for adverse effects when on these drugs.

Corticosteroids

Steroids are not curative and do not prevent joint destruction. They are however useful in the following situations:

- intolerable pain with progressive disability — short term use only until DMARDs begin to take effect.
- elderly patients threatened by complete dependency.

- vasculitis

- local injections: tenosynovitis, tendinitis, bursitis or nerve entrapment or acutely inflamed joint. Intra-articular injections should not be given more than three times a year.

Do not start steroids without a specialist assessment. Although the initial response is usually good, the dose needs to be increased and it is difficult to take patients off steroids once started. Side-effects are then inevitable.

Immunosuppressive drugs

These include drugs such as azathioprine, cyclophosphamide and chlorambucil. They are drugs to be initiated by the specialist physician and are reserved for serious forms of arthritis or connective tissue disease that are unresponsive to other agents. Immunosuppressive drugs have potential serious side-effects and require careful monitoring for their effect on the bone marrow.

OTHER TREATMENT

If symptoms are poorly controlled, or if there is disability or deformity, consider:

- Splinting joints to rest them and also to prevent or correct deformities (refer to a physiotherapist).

- Referral for intra-articular injections of corticosteroids — most rheumatologists would inject one joint only 2-3 times a year to avoid the risk of joint damage.

- Referral via the rheumatologist to a surgeon or directly to a surgeon for correction of deformity, replacement or arthrodesis of joints to reduce pain.

MANAGEMENT OF COMMON ARTHRITIC CONDITIONS

OSTEOARTHRITIS

The progression of disease cannot be halted by available drugs, but these can offer symptomatic relief. Surgical intervention should be restricted to patients who do not respond to conservative management and are so disabled that they cannot get through their activities of daily living.

Analgesics and NSAIDs

- Paracetamol and analgesic doses of aspirin are as effective as more expensive alterantives.

- NSAIDs provide no more relief than paracetamol. Low, analgesic doses of NSAIDs are no better than high, anti-inflammatory doses.

- Narcotic analgesics (e.g. codeine) should be used sparingly if at all, and only for acute disabling pain interfering with essential activity.

- Intra-articular steroids are generally not justified.

Exercise

- Restoring the joint alignment through strengthening the supporting muscles can help to reduce pain e.g., quadriceps exercise for osteoarthritis of the knee.

- Aerobic exercise programmes with full or partial weight bearing can greatly enhance endurance, walking distance and a sense of well being.

- Excessive joint strain, such as results from stair climbing, should be reduced to the degree possible.

- Supervision and patient education are essential to a successful exercise programme.

Weight reduction

- Even modest degree of weight loss helps to relieve mechanical stress on hip and knee joints.

- An assisted, supervised programme is essential, because the obese person with osteoarthritis is likely to feel that the advice to lose weight and exercise is otherwise beyond his or her capacity.

Heat

- This may provide subjective comfort even though controlled trials show no benefit.

Surgery

- Surgical consultation is indicated in the patient who is failing with conservative management.

The consultation should be viewed with the patient as an opportunity to weigh treatment options, rather than automatic surgical intervention.

- Surgery should only be offered to those who are mentally and physically healthy enough to tolerate surgery and sufficiently motivated to carry out the exercise needed to ensure full rehabilitation.

GOUTY ARTHRITIS

Acute Gout

- Rest, elevation, aspiration of joint if required.
- If patient is on thiazide therapy for hypertension, substitute with another antihypertensive.
- Start treatment early: either with NSAIDs alone, or together with colchicine.
- Colchicine - give 1 mg initially and then 500 mcg 2 hourly until relief of pain is obtained or vomiting or diarrhoea occur; maximum 8 mg.

Chronic Gout

Indications to start with allopurinol

- Recurrent attacks of gout, preferably after third attack.
- Chronic tophaceous gout.
- Hyperuricemia, arthritis and renal disease.
- Serum uric acid > 9 mg/100 ml (540 micro moles per litre).

Dosage

- Start with allupurinol 100 mg/daily.
- The dose should be reduced in renal failure.

Patient to be warned

- of possibility of acute attack with allopurinol if NSAID is not taken in the first week of therapy with allopurinol.
- to avoid excessive purine intake especially with alcohol. (Give patient low purine diet sheet).
- to avoid dieting severely as this may precipitate an acute gouty attack.
- to avoid a high purine diet.

Follow Up

- check serum uric acid level after a month. Increase allopurinol to 200 - 300 mg to maintain normal urate concentration.
- once controlled, the patient needs to be seen only once every six months to check BP and renal function (serum creatinine).

RHEUMATOID ARTHRITIS

The clinical course of rheumatoid arthritis is extremely variable, so individualisation of treatment is essential.

Nonpharmacologic measures

Exercise

This helps to maintain range of motion and muscle strength. The goals are to strengthen supporting muscles and minimise the chances of post-inflammatory contracture.

- Range of movement muscle exercises of affected joints should be taught to the patient.
- If pain is too severe for active exercises, passive exercises can be carried out by the physiotherapist; joints with effusion should be exempted.

Rest and splinting

- This is indicated only during times of severe swelling and pain; in the patient with mild to moderate disease, complete bedrest can be harmful.
- Selectively resting individual joints by splinting can help relieve pain and prevent contracture of severely inflamed joints, especially those too swollen to exercise. Splinting the wrist joint is an example.

Dietary measures and supplements

- Based on studies of fish-oil supplements, diets high in fish and plant fatty acids and low in those from animal fat may achieve a modest degree of subjective improvement.

Pharmacologic therapy

- This includes aspirin, NSAIDs and disease modifying drugs. The indications, dosages and precautions have been described earlier.

- Traditional medical management of rheumatoid arthritis has been a pyramid approach starting with patient education, physical and occupational therapy, NSAIDs and then advancing to disease modifying drugs. Cartilage and bone may be irreparably damaged by the intense synovial inflammation with accompanying joint deformity and muscle wasting if the disease is managed this way.

- The current treatment is to monitor the inflammatory activity and response to NSAIDs; disease modifying drugs are introduced within six months if there is continued activity.

Monitoring

- Disease activity and response to therapy are best monitored by reproducible measures such as duration of morning stiffness, erythrocyte sedimentation rate, number of tender swollen joints, and grip strength (which can be measured with a blood pressure cuff). Time to walk 15 m is also a useful measure.

- At the moment, one estimates the likelihood of disease progression by the duration of symptoms. If there is no improvement over six months, DMARDs need to be considered.

Surgery

Arthroplasty is an important component of therapy in patients with destroyed joints. Hip and knee procedures are most successful; hand, wrist, elbow and ankle reconstructions are less certain in outcome.

ANKYLOSING SPONDYLITIS

The primary objectives in the management of ankylosing spondylitis are:

- Pain relief

- Control of inflammation

- Early institution of strengthening exercises

- Maintenance of good posture and function.

Ankylosing spondylitis is one disease in which rest is positively harmful. Exercise should be a daily routine and should include breathing exercises and spinal extension exercises. Patients should be advised on avoidance of spinal trauma and to stop smoking. NSAIDs are still the main drugs used. There is no known modifying agent for ankylosing spondylitis.

PSORIATIC ARTHROPATHY

The management of psoriatic arthropathy is similar to that for rheumatoid arthritis except for the avoidance of antimalarials and steroids. Antimalarials exacerbate skin lesions while steroid withdrawal may precipitate a flare up of the skin lesions.

References

1. Manual of clinical problems in adult ambulatory care Edited by Dorbrand L, Hoole A J, and Pickard C G. 1972, printed in USA, Chapter 10: Muscular Skeletal problems pg 283-339.

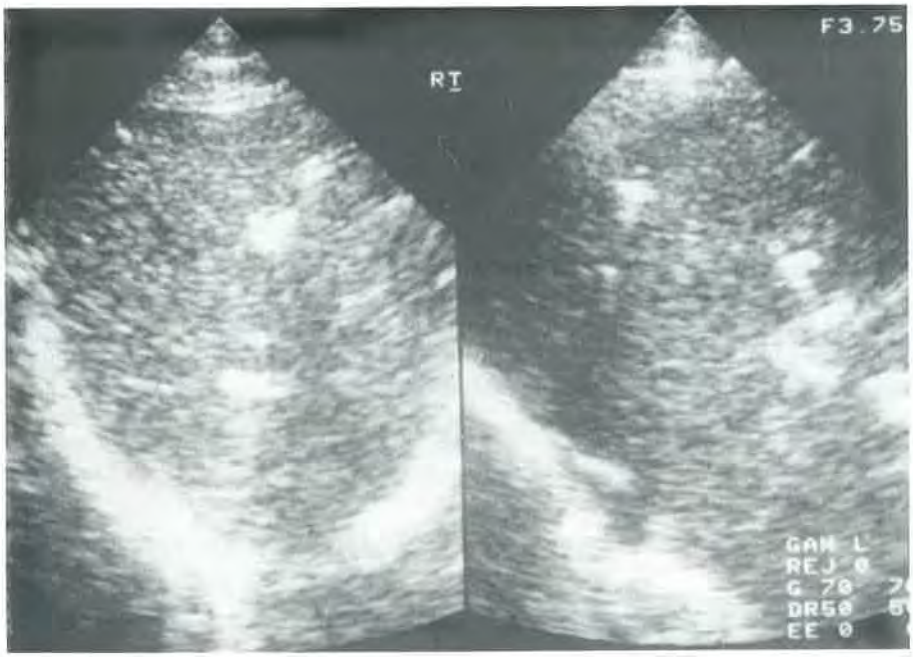
2. Soll A H, Weistein W M, Durara J, McCarthy D. Non-steroidal anti-inflammatory drugs and peptic ulcer disease. Ann Intern Med 1991; 114:307-19.

X-RAY QUIZ

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

History:

46 year old lady with fever and right hypochondrial pain.
An ultrasound of the liver was performed.
What is your diagnosis?



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

Answers on next page

ANSWERS TO X-RAY QUIZ

Answers:

Ultrasound image of the liver shows liver parenchyma echoes to be normal. However, there are brightly echogenic spots, which are rather linear and cast streaking artifacts alternating between posterior enhancement and shadowing. No cavities noted.

Diagnosis:

Aerobilia. Air is a poor transmitter of sound waves. It tends to scatter the focussed beam resulting in a mixed picture of enhancement or shadowing beyond.

Water or bile are good echotransmitters; the intrahepatic ducts will appear as parallel lines and do not cast shadows beyond it.

Sometimes gas under the diaphragm in cases of perforated viscus also be picked up by ultrasound.

Causes of Aerobilia:

I. Due to incompetence of the sphincter of Oddi:
   a) In the elderly (tend to have patulous sphincter).
   b) Following passage of gall stone.
   c) Post procedural – e.g. sphincterotomy at ERCP.

II. Spontaneous biliary fistula:
   a) Passage of a gall stone directly from an inflamed gall bladder into the bowel. Commonly into the duodenum, but may also be into colon.
   b) Duodenal ulcer perforating into common bile duct.
   c) Malignancy or traumatic fistulae.

WORLD HEALTH ORGANIZATION PUBLICATIONS

NEW BOOK ANNOUNCEMENTS

HOME-BASED MATERNAL RECORDS
Guidelines for Development, Adaptation and Evaluation
1994, viii + 85 pages
ISBN 92 4 1544464 3

This book provides a comprehensive guide to all aspects of the development, adaptation, and use of home-based maternal records as an exciting new tool for reducing maternal and perinatal morbidity and mortality. Home-based maternal records, which are retained by the woman and serve as her "passport" to appropriate health care, are simple cards designed to facilitate the easy recording and interpretation of comprehensive information on the health status of a woman before her first pregnancy, during the current pregnancy, delivery, postpartum and neonatal periods, and during two subsequent pregnancies. The cards can also be used to record information during the periods between pregnancies and on the woman's breast-feeding, family planning, and tetanus toxoid immunization status.

Though simple in concept and design, the cards have demonstrated their effectiveness as a tool for the early detection of risk factors, the promotion of timely referral, the monitoring of women's health for periods of up to 10 years, and the education of women about health, nutrition, and family planning. Home-based maternal records have also shown their potential to encourage more appropriate referrals and better utilization of health services, to promote self-diagnosis and self-care, to foster greater community involvement, and to facilitate the collection of health information.

The book draws on experiences and lessons learned during the extensive field testing of home-based records as part of a WHO collaborative evaluation involving 20 centres in 14 countries. While a WHO prototype record is presented as a model, emphasis is placed on the best ways to adapt this prototype to local conditions, test its effectiveness, pinpoint problems, and find solutions, even when resources are scarce and populations largely illiterate.

CONTRACEPTIVE METHOD MIX
Guidelines for Policy and Service Delivery
1994, viii + 143 pages
ISBN 92 4 154459 7

This book provides a comprehensive guide to the many factors that must be considered when planning to expand the range of contraceptive methods offered by family planning programmes. Noting that the reproductive health needs of women vary greatly, the book shows how the provision of a range of different methods can improve user satisfaction, enhance a programme's reputation, increase contraceptive prevalence, and thus contribute to the ultimate goal of reducing unwanted fertility.

The book's first three chapters explain how an appropriate mix of contraceptive methods contributes to both overall reproductive health and the increased prevalence of contraceptive use, and provide a detailed guide to the advantages and disadvantages of all currently available contraceptive methods. Each method is assessed in terms of recent data on effectiveness, safety, confirmed or suspected risks to health, contraindications, appropriateness to the specific needs of users, factors influencing user satisfaction, and demands on programme staff, time, and resources.

Subsequent chapters present information that can help programme managers understand the factors that influence a client's choice of method and then adjust their programmes to provide the best possible contraceptive method mix consistent with local needs and available resources. The importance of helping couples make informed choices is underscored in a chapter on information, education and communication, which includes extensive advice on counselling. Additional guidelines, checklists, model forms, and sample protocols are set out in a series of annexes.

Printed by Nam Sing Book Store