THE SINGAPORE FAMILY PHYSICIAN

College of Family Physicians Singapore

Vol. 26 No. 1 / January – March 2000

MITA(P) No 385/03/99

The College Mirror

MITA(P) No 388/03/99
Issues: No 1 / Jan – Mar 2000

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Editorial

Updates on Asthma

Bronchial Asthma is a chronic condition that affects patients of all ages ranging from the pre-schoolers to the elderly. In Singapore the prevalence of bronchial asthma is about 2.0 to 2.4% in adults.[1] Twenty percent of school children are also affected by asthma. It is also the commonest occupational respiratory disease in Singapore. The asthma mortality between 1991-5 was 0.72 per 100,000 person years.[2] The total cost of asthma in Singapore was estimated at US $33.93 million per annum.[3] Inpatient hospitalisation accounts for the largest proportion of direct medical expenditure.

Asthma is a significant health care problem in Singapore. Family physicians are the key players in ensuring asthma is managed optimally at the primary care level. In this way asthma patients can maintain good quality of life, reduce days off work or school, reduce hospitalization, prevent further progression of the disease, and reduce unnecessary mortality.

Family physicians need to understand the pathophysiology of asthma and the recent advances in asthma therapeutics. Successful asthma management consist of 4 basic components of care, namely (1) accurate assessment of severity and monitoring of control, (2) management of trigger factors, (3) appropriate step-wise pharmacologic therapy, (4) patient education.[4]

Numerous clinical practice guidelines are published to assist physicians in making appropriate evidence-based clinical decisions in the care of asthma. These guidelines include the National Asthma Education Program Expert Panel Report 2, the British guidelines on asthma management as well as our local MOH clinical practice guidelines for the management of paediatric asthma.[5,6,7]

The focus of this issue of the Singapore Family Physician is on asthma. The articles will explore the various components of asthma care and deal with the management of specific groups of patients.

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Dr Tan Chee Beng
Honorary Editor

President's Column

Making a Statement for the Family Physician

The Family Physician is still the epitome of medical wisdom. He is still the Family Physician to his patients, to whom they can consult for all major and minor ailments in the family, and to take care of their families from "womb to tomb". And he will always be there to defend this role, no matter what may have been misrepresented by recent media articles, such as "Just where have all the patients gone?" (ST 15.4.00).

The College has rebutted that article with a reply that many Family Physicians/GPs constantly update themselves. This is no empty claim, judging from the many activities organised for and participated by family physicians. We have graduated from ad-hoc didactic lectures and talks to a more structured programme; a total of 13 CME events were jointly organised by the College with other organisations over the past calendar year, in addition to annual events such as the 7th Scientific Conference (Family Medicine: Facing Demographic Change) on 25-26 September 1999, the 4th Annual Surgical Update on 4-5 March 2000, followed by the hands-on Minor Surgical Procedure Course (which was 3 times oversubscribed) on 11-12 March 2000.

We have also informed the media that there is a GP training programme for medical undergraduates in their curriculum, a programme conducted by the Department of Community, Occupational and Family Medicine (COFM), at the National University of Singapore, a department set up in 1988 and working closely with the College and the Ministry of Health. We also have a postgraduate Family Medicine Teaching Programme, leading to the Master of Medicine (Family Medicine) degree. To date there are now some 120 specialised GPs in this discipline. In addition we have other short postgraduate courses such as the Graduate Diploma in Basic Ultrasonography.

The College is to launch another postgraduate diploma programme for the practising Family Physicians/GPs in July 2000 - the Graduate Diploma in Family Medicine (GDFM). This will be a two year vocational training certification to train primary care doctors to practice Family Medicine at an enhanced level. It will include distance learning, tutorials, practice skills course and elective short clinical course. More detailed information will be forthcoming in due course.

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Asthma Trends

Ong YY

Introduction

Asthma has become a significant worldwide health problem. It remains a costly clinical problem with a continuous need for new and innovative treatment. In asthma the areas of advancement have centred on

1) understanding of the pathophysiology

2) institution of treatment guidelines

3) the role of the newer drugs

4) research in the hope of preventing the evolving inflammatory allergic process.

The recent increase in asthma is the result of increased sensitisation to common inhaled allergens in the population.

Asthma symptoms in an individual may be due to a number of factors making avoidance difficult or impossible.

Airway Inflammation in Asthma

In the last 10 years the use of flexible fibreoptic bronchoscopy in numerous research studies to biopsy the airway wall has shown that asthma is a chronic inflammatory process.

This inflammatory process is brought on by cytokines involved in the inflammatory process. The mediators act on the bronchial smooth muscle, mucus glands, vascular endothelium, neural pathways to cause

1) contraction of the spiral smooth muscle in the bronchial wall

2) airway wall oedema

3) mucus plugging

4) eosinophil infiltration and remodelling of the asthmatic airway

- proliferation of epithelial cells
- hypertrophy of bronchial smooth muscle cells
- deposition of collagen below the basement membrane

The airway remodelling results in subepithelial fibrosis, bronchial muscle hypertrophy, mucus gland hyperplasia leading to enhanced 'resistant' airway dysfunction.

It is because of these irreversible changes that make the control of airway inflammation mandatory.

Prevalence

Asthma is increasing in prevalence all over the world affecting about 10% of children and 5% of adults. In many countries there is an increasing number of prescriptions for asthma drugs. Asthma prevalence is highest in industrialized countries but prevalence is rising in many developing countries.

In the UK, it is estimated that 4% of children between aged 0-3 years and 10% of children aged 4-17 years are on anti asthma medication.

In Japan the number of children who suffer from asthma is rapidly increasing from kindergarten to high school according to Ministry of Education statistics (1999). The number of asthmatics at middle schools reached 2% for the first time. The results showed that there is roughly one asthmatic child in every primary school classroom. Childhood asthma has become a major health concern.

The following have played an important role in asthma development

1) outdoor allergies eg house dust, moulds, grass/tree pollen

2) pollutants

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3) lack of physical activity

4) cockroaches

5) pet allergies eg animal hair

6) drugs (ßeta-blockers and non steroidal anti inflammatory drugs (NSAID)

7) respiratory tract viral infections

8) physical factors eg exercise

9) emotional/psychological reasons

A contributory factor to the rising prevalence in asthma is due in part to better diagnosis. Patient education, action plans and continuing medical education in asthma have all contributed to an increased awareness of how to manage the condition. Recent years have seen the development of clinics specially dedicated to asthma care locally. Personalized education material have been shown to improve asthma care.

Cigarette smoke may contribute to the increased prevalence of asthma as atopic conditions are seen in children of smokers. Asthma is more prevalent in young children under 25 years of age in households with at least one smoker.

The influx of the inflammatory cells is attributed to a network of interacting cytokines released in response to repeated allergen inhalation.

Drugs in Asthma

Cromolyns

Inhalation therapy with sodium cromoglycate is recommended as the first line prophylactic treatment for moderate asthma in children.

Inhaled Corticosteroid

Given that the inflammation associated with asthma plays an important role, it is recommended that anti inflammatory or 'preventor' medication becomes important. It seems logical to introduce inhaled steroids early. The aim of this strategy is to suppress inflammation which will reduce the need for symptomatic medication with bronchodilators. Inhaled glucocorticoids are considered the first-line treatment for patients with moderate to severe persistant asthma and remains the best initial therapy.

Theophylline

Oral theophylline is effective in the treatment of asthma even when serum theophylline concentrations are low. Although theophylline primarily is considered to be a bronchodilating agent it also increases mucociliary clearance and has anti inflammatory properties. Theophylline reduces microvascular permeability, inhibits the late phase bronchial response and may inhibit lymphocyte and eosinophilic infiltration of the airway. Theophylline, however, is associated with more adverse events.

The Leukotrienes

Leukotrienes are the first new class of drugs for asthma in 20 years. They are so called because they were initially identified in leukocytes and from the arrangement of 3 of their 4 double bonds in tandem (tri-ene) in the fatty acid structure. Leukotrienes are biologically active fatty acids derived from the oxidative metabolism of arachidonic acid, an integral component of the cell membrane.

In humans, the leukotrienes are produced by mast cells, basophilis, eosinophils, monocytes and macrophages. The leukotrienes influence the activity of target cells, eg, bronchal smooth muscle or vascular endothelium. The leukotrienes modifier drugs (LM) are the first specific mediator inhibitor/antagonists. The LM drugs inhibit inflammatory cell egress into the airway and these studies suggest that leukotrienes modifiers have anti inflammatory activity. The LM drugs have mild bronchodilator function apart from their anti inflammatory activity. They augment airway oedema, mucus hypersecretion and block the increase in leukotrienes in patients with spontaneous exacerbations of asthma and after antigen challenge.

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Their main value seems to be in combination with inhaled corticosteroids when the action is more effective than either drug alone.

Adding LM drugs to the treatment regimen is better than increasing the dose of inhaled steroids. Patients on medium to high doses of inhaled beclomethasone (1500-3000 mcg/day) are able to halve their steroid dose. Topical corticosteroids modulate the expression of a huge range of inflammatory molecules. In vivo steroids may not suppress leukotriene synthesis effectively.

The LM drugs may be particularly effective in patients with aspirin intolerant asthma and the only medical therapy effective in attenuating the aspirin induced asthma.

The other situation is in exercise induced asthma where the LM drugs are effective.

In mild intermittant asthma very few studies with LM drugs are available. Most of the studies have been on moderate persistant asthma where they have demonstrated improvement.

In one study, montelukast showed significant improvement in FEV, and daytime asthma symptoms. It had a faster and larger initial response than beclomethasone. However, 7 to 10 days after therapy was initiated the effect of beclomethasone surpassed that of montelukast.

While some regard the LM drugs as an alternative to inhaled corticosteroids, the latter appear to remain the best initial therapy based on currently available data.

Long acting ß2 agonists

There are 2 long acting ß2 agonists, formoterol and salmeterol which have been developed. They cause bronchodilation for at least 12 hours. They have been shown to reduce diurnal airway calibre fluctuations and asthma symptoms and they have become important agents in the therapy of patients with chronic asthma who are not fully controlled with inhaled anti inflammatory therapy.

Research

This is now being centred on early pharmacological treatment of children at high risk of developing asthma in the hope that this could possibly stop the evolving inflammatory allergic process.

There is a close association between early expression of atopic dermatitis and asthma in atopic children. The ETAC study (Early treatment of the atopic child) hopes to find whether anti-inflammatory treatment immediately instituted at the first signs of atopic dermatitis could stop the evolution from atopic dermatitis to asthma such that asthmatic symptoms will never be clinically expressed.

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Dealing with Asthma Triggers

Anne Hsu AL

Asthma is a chronic airway disease characterised by reversible bronchial obstruction, inflammation and hyperresponsiveness. Non-pharmacological measures such as trigger control and education have established their pivotal role in the management of asthma. There are studies suggesting that early exposure to triggers such as allergens, tobacco smoke, early hospitalisation for viral respiratory tract infections contribute to the primary development of asthma. In existing asthma, trigger eradication and avoidance measures lead to improvement in bronchial hyperresponsiveness, severity of symptoms, and requirement for asthma medication. Current asthma management guidelines require health care providers to assess patients for trigger factors. Although the majority of the trigger factors are environmental, some are endogenous. The trigger factors categorised into the following will be discussed:

1) Inhaled irritants and allergens
2) Respiratory infections
3) Rhinosinusitis
4) Gastroesophageal reflux
5) Exercise
6) Drugs and food
7) Endocrine factors

Inhaled Irritants and Allergens

The assessment of inhaled allergens or irritants should take into account the amount of exposure, the patient's sensitivity of allergens or irritants encountered, and the clinical significance of the exposure and sensitivity in the context of the patient's medical history. Inhaled irritants include environmental fumes (e.g. car exhaust), dust, chemicals (e.g. cleaning fluids and powders), smoke (including tobacco smoke), strong odors and aerosol spray, cold air and air pollutants. Recommended measures are irritant elimination and avoidance such as not allowing others to smoke inside the house, the use of unscented and nonaerosol products, and not exercising outdoors when pollution levels are high. Inhaled allergens include house dust mites, animal danders (usually dogs and cats), feathers, cockroaches, pollens, trees and fungal spores (such as mold and aspergillus). Allergen specific therapy will be discussed later.

Both inhaled irritants and allergens at the worksite can result in occupational asthma. Early recognition is important because once the airway is sensitised by these triggers, bronchoconstriction will be induced by minimal exposure, which may not be completely reversible with time (see Table 1). Referral to the occupational lung specialist is necessary for diagnosis (specific bronchoprovovation test may be necessary), and evaluation of workplace, relocation and workman compensation issues may need to be considered.

As for non-occupational related inhaled triggers, the predominant triggers vary with locale, lifestyle, season and climate. In Singapore, pollen and cold induced asthma are less common. The westernised indoor lifestyle trends and high humidity here make house dust mites as one of the commonest trigger factors of asthma. This is significant because we spend 80-90% indoor and about 25-33% of our time in the bedroom.

House dust mites are ubiquitous, invisible arachnids. Sensitivity to house dust mites can be evaluated with a skin prick test or measurement of serum Ig E level to house dust mites (RAST test). The advantage of the skin prick test is that a panel of selected antigens including house dust mites is done and it does not involve blood sampling. Sensitivity to the aeroallergen is rare if the test is negative whereas a positive test may not have relevance. Hence allergy testing should be done by clinicians who will interpret the results in the context of the medical history and recommend appropriate therapy. Allergen specific therapy includes allergen avoidance and immunotherapy.

House dust mites and pet dander eradication and avoidance are the most widely studied allergens. The essential measures for house dust mite avoidance are to remove carpets and stuffed toys,

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Occupation or Occupational Field	Agent
laboratory animal workers, veterinarians	dander and urine proteins
food processing	shellfish, egg proteins, pancreatic enzymes, papain, amylase
dairy farmers	storage mites
poultry farmers	poultry mites, droppings and feathers
granary workers	storage mites, aspergillus, indoor ragweed, and grass pollen
research workers	locusts
fish food manufacturing	midges
detergent manufacturing	*Bacillus subtilis* enzymes
silk workers	silk-worm moths and larvas
	Plant Proteins:
bakers	flour
food processing	coffee bean dust, meat tenderizer (papain), tea
farmers	soy bean dust
shipping workers	grain dust (molds, insects, grain)
laxative manufacturing	ispaghula
sawmill workers, carpenters	wood dust (western red cedar, oak, mahogany, zebrawood, redwood, Lebanon cedar, African maple, eastern white cedar)
electric soldering	colophony (pine resin)
cotton textile workers	cotton dust
nurses	psyllium
	Inorganic Chemicals:
refining	platinum salts
plating	nickel salts
diamond polishing	cobalt salts
stainless steel welding	chromium salts
manufacturing	aluminum fluoride
beauty shop	persulfate
refinery workers	vanadium
welding	stainless steel fumes
	Organic Chemicals:
manufacturing	antibiotics, piperazine, methyl dopa, salbutanol, cimetidine
hospital workers	disinfectants (sulfathiazole, chloramine, formaldehyde, psyllium, glutaraldehyde)
anesthesiology	enflurane
poultry workers	aprolium
fur dyeing	paraphenylene diamine
rubber processing	formaldehyde, ethylene diamine, phtyhalic anhydride
plastics industry	tolulene diisocyanate, hedamethyl diisocyanate, dephenylmethyl isocyanate, phthalic anhydride, triethylene tetramines, trimellitic anhydride, hexamethy tetramine
automobile painting	dimethyl ethanolamine toluene diisocyanate
foundry worker	furfuryl alcohol resin

Table 1: Agents Causing Asthma in Selected Occupations

wash bedsheets and pillowcases weekly in hot water (60° C), change old mattresses and pillows and avoid sleeping or lying on upholstered furniture. Optional or desirable measures are using miteproof mattresses or covers, pillowcases and blankets; and use HEPA (High Efficiency Particulate Air) filtration. The latter removes airborne mites but leaves undisturbed the major reservoir antigen in carpets, bedding, and upholstery. Acaricides and tannic acid have been used to kill mites and denature the proteineacous allergens but the limitations are the concern of chemical effects (although studies have reported on the safety profile of the agents marketed) on patients and cost related repeated and professional application. The other widely studied home allergen is pet dander. Cat allergen is more likely to cause sensitisation than that of dogs. The protein secreted by the cat's salivary, sebaceous, and lacrimal glands is the major allergen. The protein is very stable and retains its antigenic potency for at least a month. Therefore, even after permanent removal of the cat, it may take many months before the concentration of allergens in domestic dust falls. Advising patients to find a new home for their household pets may be one of the most unwelcome and unrewarding tasks. If, despite pointed education, the patient still finds it unacceptable to find a new home for the pet, measures that may reduce the allergen exposure load include regularly bathing the pet, removal of carpets, vacuuming, air filtration and the reduction of contact with the pets. Recommendations to deal with other home allergens are 1) control dust as much as possible by removing blinds and carpets, cleaning curtains and floors often, and dusting all surfaces frequently. House cleaning should be done by someone other than the patient, if unavoidable, the patient should use vacuum cleaner and mask with HEPA filters, 2) reduce cockroach infestation with control of food and water source and use of poison baits and boric acid, and 3) reduce mold by cleaning moldy surfaces and keeping the house well-ventilated and free of dampness.

Immunotherapy involves the injection of small amounts of specific allergen to desensitise a sensitised patient against allergic reaction. It is tedious and costly (monthly injection for 3-5 years) with poor compliance and can produce life threatening reaction in asthmatic patients. Hence, it is considered only when 1) there is clear evidence of a relationship between symptoms and exposure to an unavoidable allergen to which the patient is sensitive, 2) symptoms occur all or most of the year, and 3) there is difficulty controlling the symptoms with pharmacological measures.

Respiratory Infections

This is the commonest cause of asthma exacerbations requiring hospitalisation. Common respiratory infections associated with asthma exacerbations are sinus infections, colds, pharyngitis, bronchitis and pneumonia. Preventive measures include frequent hand washing, avoid crowded enclosed places and pneumococcal vaccination. Prompt specific therapy for respiratory tract infection and increasing drug regime for asthma control (e.g. doubling inhaled steroids for 2 to 3 weeks) may be necessary.

Rhinosinusitis, Nasal Polyps

Nasal and sinus disease can aggravate asthma through the following mechanisms 1) nasopulmonary reflex, 2) postnasal drip induced cough and irritability of the larynx and 3) aspiration of upper airway secretions into the lower airway. Treatment of upper airway inflammation and congestion results in better control of asthma. This includes restoration of nasal patency with decongestants, corticosteroids, cromolyn; control of nasal secretions with antihistamines, corticosteroids, nasal or antral toilet; and treatment of bacterial infection with antibiotics. Nasal polyps are best treated with corticosteroids and those with chronic disease despite corticosteroids may benefit from surgery. The usage of leukotriene modifiers for asthma control has also been associated with better control of allergic rhinitis. Allergen specific therapy is necessary for allergic patients.

Gastroesophageal Reflux (GER)

The prevalence of GER among asthmatics reported in the literature is estimated to be between 30-90 percent. The relationship between asthma and GER remains controversial based on the following observations 1) therapy of

GER may or may not improve asthma, 2) bronchoprovocation can induce GER and 3) respiratory symptoms occurs independently of reflux. Nevertheless, there is evidence of GER contributing to coughing and aggravation asthma especially at night. Studies show esophageal acid causes bronchoconstriction by a vagal mediated reflex and there is further augmentation of this response if microaspiration is present. Diagnosis is often made on history of belching, heartburn and nocturnal exacerbations. However, it should be noted that GER can be silent and 24 hours esophageal testing may be necessary. Medical therapy includes 1) physiologic and dietary measures such as weight loss if obese, avoiding food 4 hrs before bedtime, elevating the head of the bed by 6 to 8 inches, eating smaller, frequent meals, 2) reduction of acid reflux with prokinetic agents, H2 antagonist or proton pump inhibitors and 3) maintenance of esophageal sphincter pressure such as avoiding fatty meals, species, ethanol, caffeine, peppermint and methylxanthines. Surgical intervention is reserved for highly selective groups of patients when the above measures fail.

Exercise

Exercise-induced asthma (EIA) is a result of bronchospasm in the presence of underlying bronchohyperreactivity. This condition should be anticipated in all asthmatic patients. For some people with asthma, exercise is the only trigger. The mechanism is believed to be due to heat and water loss from hyperventilation during exercise. Bronchospasm generally reaches its peak about 5 to 10 minutes after cessation of activity and usually resolves in another 20 to 30 minutes. For some patients who engaged in continuous, repetitive exercise periods, EIA diminishes or is completely abated during a refractory period that usually lasts 2 hours after an exercise challenge. During this period, EIA is significantly reduced from its initial levels. Hence, adequate warming up to maximum exertion level may be helpful. When there is doubt, an exercise challenge can establish a diagnosis of EIA. In an exercise challenge, the patient exercises 8 minutes at 80 of the patient's maximum predicted heart rate. A drop in PEFR (peak expiratory flow rate) or FEV1 (forced expiratory volume at 1s) of greater that 15 percent is taken as positive.

The goal in the management of EIA is to enable patients to participate in any activity they choose without experiencing asthma symptoms, noting that many Olympic athletes have asthma. Patients should be advised on proper premedication and warm up to his maximum exertion level. The most common premedication is inhaled short-acting beta2-agonist 10-15 minutes before exercise. Other drugs found to be useful for EIA are inhaled long-acting beta2-agonist, cromolyn sodium, oral leukotriene modifiers and theophylline (takes one hour to reach peak levels and less effective than inhaled beta2-agonist). Persons with EIA should inform their teachers and trainers of the condition. This condition should not limit either participation or success in activities but may require the use of inhaled medication before activity and after, if needed.

Drugs and Food

Common drug sensitivity in asthmatic patients are nonselective beta-blockers (including eye drops), aspirin and other NSAIDs. Up to 20 percent of adults with asthma will experience severe and even fatal exacerbations of asthma after taking aspirin or other NSAIDs. The prevalence increases with increasing severity of asthma and also those with nasal polyps. Food allergies are not common. If a patient suspects a food allergy, he/she should be instructed to keep a foods diary to detect a possible correlation between certain foods and the occurrence of symptoms. Patients should read food labels if they are allergic to preservatives such as sulfites (potatoes, shrimp, dried fruit, beer, and wine). If suspected food allergic symptoms are pronounced, a referral to an allergist may be necessary because identification and control of food allergy can be difficult.

Endocrine Factors

Emotional expressions such as anger, laughing and stress have been associated with exacerbation of asthma. Avoiding stressful situations and learning relaxation techniques may be helpful. Premenstrual asthma (PMA) is a recognised

clinical entity. As many as 30-40% of female asthmatics experienced worsening of symptoms a week (peak at 2 to 3 days) prior to menstruation. Although case reports showed a change in physiologic tests (i.e. spirometry and bronchoprovocation test) in this group of patients, controlled studies did not demonstrate a significant difference in these tests. PMA is probably progesterone related and hence studies on hormonal modification appear to be a promising line of investigation. Meanwhile, management options that can be considered are adding long acting inhaled bronchodilator and/or increasing inhaled steroids prior to and during the period of asthma exacerbation.

Conclusion

Good communication with patients and patient education on asthma management, including dealing with trigger factors will improve treatment compliance and outcome. However, counselling should be cost-effective, as there is a non-linear link between the acquisition of knowledge with behavior. Asthmatics have different sensitivities to the above discussed trigger factors and some of these factors may be non-existent in many asthmatics. Therefore, asthma education should be special measures for certain risk groups. Patients with asthma at any level of severity should avoid 1) exposure to environmental tobacco smoke, 2) use of beat-blockers, 3) foods to which they are sensitive to and 4) exposure to allergens to which they are sensitive to. For patients with persistent asthma, the physician should investigate for allergens or irritant exposures and if necessary assess the significance of positive allergy tests in the context of the patient's medical history. Once the triggers of the patient's asthma have been identified, a plan should be devised to enable the patient to reduce the interference. There are 3 options in dealing with asthma triggers 1) avoid the trigger entirely (e.g. do not own pets if allergic to them), 2) limit exposure to the trigger if it cannot be completely avoided (e.g. leave the room if someone starts smoking) and 3) take an extra dose of bronchodilator before the trigger exposure if the first 2 options are not feasible (e.g. exercise induced asthma). Patients with persistent asthma should be treated for rhinitis, sinusitis and gastroesophageal reflux if present. Adult patients with severe persistent asthma, nasal polyps, or a history of sensitivity to aspirin or NSAIDs should be counseled regarding the risk of severe and even fatal exacerbations from using these drugs.

Further Reading

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Successful Asthma Management: Current Guidelines

Tan KL

Introduction

Asthma is a chronic inflammatory disease of the airways. The goals of treatment in asthma are to prevent chronic and troublesome symptoms and recurrent exacerbations of asthma, preserve optimal lung function and maintain normal activity levels. This approach to treatment would minimise the need for emergency department visits or hospitalisations and enhance the quality of life. The clinician should aim to provide optimal pharmacotherapy with minimal or no adverse effects.

Current guidelines

Some recommendations for the management of asthma that aims to bridge the gap between current evidence-based knowledge and practice and to assist clinicians in making appropriate decisions about asthma care are contained in the following guidelines:

1. Expert Panel Report 2: Guidelines for the diagnosis and management of asthma, National Institutes of Health, National Heart, Lung and Blood Institute, July 1997.[1] This publication is available on the internet at http://www.nhlbi.nih.gov/nhlbi/nhlbi.htm.
2. Global initiative for asthma (GINA). Asthma management and prevention, 1998.[2] This publication is also available online at http://ginasthma.com/.
3. The British guidelines on asthma management: 1995 Review and position statement.[3]

Foundation of effective asthma management

The four basic components of effective asthma management are:

1. Assessment of the severity of asthma and monitoring of the response to therapy using objective measures of lung function.
2. Comprehensive pharmacologic therapy for management of acute asthma exacerbations and chronic asthma.
3. Environmental control measures aimed at avoidance or elimination of factors that precipitate asthma symptoms or exacerbations.
4. Patient education fostering an active partnership among the patient, his relatives and the healthcare provider.

Assessment of severity and monitoring of control

To establish a diagnosis of asthma, the clinician should determine that episodic symptoms of airflow obstruction are present, airflow obstruction is at least partially reversible and alternative diagnoses that may mimic asthma are excluded. A careful medical history, physical examination and spirometry to demonstrate reversibility is essential to establish a correct diagnosis of asthma.[1] Spirometry is useful in both diagnosis and monitoring. Measurement of spirometry before and after inhalation of a short-acting bronchodilator helps to determine whether there is airflow obstruction and its severity and whether there is reversibility. A reduction in the ratio of forced expiratory volume in one second (FEV_1) to forced vital capacity (VC) from the predicted range is diagnostic of airflow obstruction.[4] The severity of airflow obstruction is classified on the basis of FEV_1, expressed as percent of predicted values. Significant reversibility is indicated by an increase in FEV_1 of at least 200mL and 12% after bronchodilator administration. A 20% or greater peak flow variability (eg. difference between morning and afternoon measurements of peak expiratory flow) indicates reversible airflow limitation consistent with the diagnosis of asthma. On the basis of symptoms and lung function, asthma severity may be classified into mild intermittent, mild persistent, moderate persistent and severe persistent.[1]

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For diagnostic purposes, spirometry is generally recommended over measurements of peak expiratory flow because of the wide variability in the peak expiratory flow reference values. Peak flow meters are useful monitoring tools and are recommended for patients with moderate-to-severe asthma. In these patients, the peak expiratory flow forms the basis for the asthma action plan.

Pharmacologic therapy

Underdiagnosis and inappropriate therapy are major contributors to asthma mortality and morbidity. Short-acting inhaled beta2-agonists taken as needed is the therapy of choice for relief of acute asthma symptoms (acute reliever). Short-acting inhaled beta2-agonists are also used in the prevention of exercise-induced bronchospasm. Short-acting inhaled beta2-agonists taken as needed to treat acute symptoms are usually sufficient therapy for mild, intermittent asthma. Persistent asthma, either mild, moderate, or severe, is controlled with daily anti-inflammatory therapy (chronic preventer). Inhaled corticosteroids are the most potent inhaled anti-inflammatory agent currently available. Early intervention with inhaled corticosteroids can improve asthma control and normalize lung function and may prevent irreversible airway injury.[1]

A stepwise approach to pharmacologic therapy is recommended for long term management of asthma. Pharmacologic therapy is usually iniatiated at a higher level at the onset to establish prompt control and then stepping down once control is established. The reduction in inhaled steroids should be gradual as patients may deteriorate as the steroids are being withdrawn. The British guidelines recommend that reductions should take place every 1-3 months by decreasing the dose of inhaled steroid by approximately 25-50% at each step.[3] Reduction in dose should be monitored by symptoms, spirometry or peak flows, frequency and severity of exacerbations and $ß_2$ agonist (acute reliever) use. Increasing use of short-acting beta2-agonists for acute asthma symptoms indicates inadequate control of asthma and the need for increasing anti-inflammatory therapy. Regularly scheduled, daily use of short-acting beta2-agonist is not recommended. Increasing use of beta2-agonist has been associated with increased risk for death or near death in asthmatic patients.[5] Nocturnal symptoms are controlled with long-acting inhaled beta2-agonist or sustained-release theophylline.

Current guidelines consider that further studies and clinical experience are needed for leukotriene modifiers before any positioning recommendations can be made.[1,3]

Management of acute asthma exacerbations includes inhaled beta2-agonist to provide prompt relief of airflow obstruction, increasing the dose of inhaled corticosteroids and initiating a short course of systemic corticosteroids for moderate-to-severe exacerbations to suppress and reverse airway inflammation. Patients with moderate-to-severe persistent asthma and patients with a history of severe exacerbation should be provided with a written action plan to guide patient self-management during acute asthma excerbations.[1]

Review of patient technique in using medications and drug delivery devices is mandatory. Response to therapy is monitored with serial measurements of lung function (spirometry or peak expiratory flow).

Control of trigger factors

In asthmatic individuals, exposure to irritants or allergens to which they are sensitive has been shown to increase asthma symptoms and precipitate asthma exacerbations. Asthmatic patients should be advised to avoid or control allergens, irritants or other factors that worsen their asthma. The Expert Panel Report 2 recommends that adult patients with severe persistent asthma, nasal polyps, or a history of sensitivity to aspirin or nonsteroidal anti-inflammatory drugs be counseled regarding the risk of severe and fatal exacerbations from using these drugs.

Rhinitis, sinusitis and gastroesophageal reflux should be treated when present. Annual influenza vaccinations are recommended for patients with persistent asthma.

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Asthma education

Patient education is an essential component of successful asthma management. Health care providers should teach patients the basic facts about asthma, roles of medications, inhaler/spacer technique, self-monitoring and asthma action plan. Asthma written self management plans encourages active patient participation in their management, reduces morbidity and health costs.[6] When asthma proves difficult to control on seemingly effective therapy, it is important to find out about any family, psychological or social problems which may be interfering with effective management.

Conclusion

To achieve better asthma control and outcomes, the clinician should integrate a holistic approach to good clinical practice and foster partnership in asthma care with the patient.

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Drug Therapy in Chronic Asthma

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Introduction

In today's practice, patients often seek medical attention only when unwell. Asthmatic patients are no exception. The occasional sensation of dyspnea, chest tightness and wheeze is so effectively relieved by beta agonist administration that there is a temptation to administer beta agonists on a regular basis to treat chronic asthma. With emerging recognition that chronic airway inflammation plays a pivotal role in the many symptoms of asthma, treatment strategies have been redirected from targeting the airway myocyte to antiinflammation therapy.

Controlling chronic inflammation in asthma: the crux of the matter

Our understanding of asthma has gone through evolutionary phases. Initially thought to be a psychosomatic disease, asthma is currently recognised as a chronic airway inflammatory disease even in disease quiescence. Indeed, even patients with mild asthma have been shown to demonstrate airway inflammation, characterized by infiltration of the mucosa and epithelium with activated T cells, mast cells and eosinophils[1]. In turn, airway inflammation leads to bronchial hyperresponsiveness, and contributes to further airflow limitation by mucus plug formation and airway edema. Untreated, chronic airway inflammation may progress to an irreversible stage of bronchial fibrosis with decline in lung function. While beta agonists provide quick symptom relief in an acute asthmatic attack, they do not reduce exacerbations, reduce nocturnal symptoms, and preserve lung function. Treatment philosophies have shifted toward anti-inflammatory therapy to address the problem at the root.

The patient's and doctor's goals in asthma management

The goals of asthma treatment include[2]:

- Minimal chronic symptoms, including nocturnal
- Minimal exacerbations
- No emergency visit
- Minimal need for rescue therapy
- No limitation to exercise
- Minimal adverse effects from medication

While asthma is not a curative condition, a whole armaturum of drugs are available to improve patients' quality of life and meet treatment goals. It should be realized, however, that medications do not drive the disease into remission, and the positive effects of medications are limited to the duration of treatment.

With the above goals in mind, it is necessary to identify the subgroup of asthmatics that require long-term preventive therapy. Currently, patients who experience one or more attacks per week, patients with more than 2 nocturnal attacks per month, with significant variability on peak flow monitoring should be considered for preventive therapy.

The most common anti-inflammatory agents used are inhaled corticosteroids, methylxanthines, antileukotrienes and cromones. It is realistic to explain to the patient that, unlike ß agonists, the onset of action of this class of drugs is slower, but the eventual effects would be seen in symptom reduction and prevention of irreversible airway changes.

Corticosteroids

To date, inhaled steroids are the most powerful tool in the long-term management of asthma with a broad spectrum of anti-inflammatory effects. Inhaled corticosteroids improve overall asthma management, reduce need for bronchodilator therapy, reduce hospitalisations, reduce airway hyperresponsiveness and improve pulmonary function. There is also evidence that early intervention with inhaled steroids may prevent the long-term decline in lung function arising from bronchial fibrosis.[3]

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Several caveats are relevant to inhaled steroids:

1. The onset of clinical improvement with inhaled steriods occurs after at least one week of regular use, and may take as long as 3 months to plateau[3]. Revisions in inhaled steroid dose may not be accurate if made prior to 3 months.

2. There is considerable inter-individual variation between patients in terms of steroid response. In general, however, steep increments in anti-asthmatic efficacy are seen in the initial 800µg/day of beclomethasone, budesonide and triamcinolone acetonide. Beyond 800µg/day, further increments in efficacy are less discernable. The converse is true with regards to steroid systemic effects. With doses in excess of 800µg per day, systemic side effects become more common[4]. Should asthma control remain poor despite inhaled steroids in the medium dose range, a rational approach would be to add an alternative class of treatment (eg antileukotrienes, low dose theophyllines and long acting ß agonists) rather than double the steroid dose.

3. Overtreatment with unneccessarily high doses of inhaled steroids results in higher local and systemic adverse effects. In order to prevent overtreatment, the inhaled steroid requirement should be reviewed on a regular basis. When the patient's asthma is well controlled, a reduction in inhaled steroids should be attempted. As a guideline, reductions should be done in one to three month intervals, with careful titration against symptom frequency and severity and ß agonist use. Local effects include candiasis and dysphonia. While systemic effects with inhaled steroids are much less likely compared with oral steroids, high doses of inhaled steroids have been associated with posterior subcapsular cataracts, ocular hypertension, skin bruising, osteoporosis and adrenal suppression. Measures to reduce systemic effects include mouth washing after inhaler dosing and incooperating the use if a large volume spacer. In children receiving > 400µg of budesonide or beclomethasone daily, regular growth monitoring becomes necessary.

4. The anti-inflammatory effects of inhaled steroids are maintained while on therapy. Following discontinuation of therapy, symptoms and airway hyperresponsiveness return to pretreatment levels.

Antileukotrienes

The cysteinyl leukotrienes are metabolites of arachidonic acid comprising leukotrienes C_4, D_4, E_4. The leukotrienes are potent inflammatory mediators that produce smooth muscle constriction, eosinophil chemotaxis, and increase microvascular permeability and edema formation. The cysteinyl leukotrienes are several orders more potent than acetlycholine and histamine in initiating bronchoconstriction. Antileukotrienes are unique in possessing dual antiinflammatory properties and bronchodilatory properties. They have shown particular efficacy against allergen induced, exercise induced and asprin induced asthma. In addition, they also reduce exacerbations and may treat co-existing allergic rhinitis. The practical advantage of antileukotrienes lies in easy dosing (oral formulation) and early onset of action (within the first 24 hours). Considered as add on therapy (rather than monotherapy) to inhaled corticosteroids in mild to moderate asthmatics, antileukotrienes may be considered in the following circumstances:

- When corticosteroids use is not feasible due to poor inhalation technique, or patient refusal to take inhaled steroids.

- As preventive therapy to aspirin, allergen, exercise induced asthma.

- As an alternative to further steroid increases in moderate to severe asthma

The overall response rate with antileukotrienes is generally less than that of inhaled steroids, at approximately 50%. Due to rapidity of action, response should be seen within 14 days, and non-responders should have their antileukotrienes discontinued[5].

Theophyllines

Previously considered a bronchodilator, low doses of theophylline have documented anti-inflammatory activities and have been recommended as a second line controller in addition to inhaled steroids. Although a less popular third line agent after long acting ß agonists and antileukotrienes, the advantage lies in its availability, oral formulation and cost. The disadvantages of theophylline include the narrow therapeutic window, and notorious potential for drug interaction.

Cromones

Used less commonly than the above drugs for chronic asthma, the mast cell stabilisers are well tolerated and useful in the context of atopic asthma, and exercise asthma. However, limitations to its use are cost (compared with low dose inhaled steroids) and dosing schedule (three to four times daily) which may affect compliance.

Poor response to treatment

Should a satisfactory response not be seen with treatment, the following factors should be considered rather than escalating pharmacological treatment:

1. Poor compliance from inadequate patient education, and poor inhaler technique
2. Presence of persistent triggers: allergens, gastroesophageal reflux, and sinusitis
3. Wrong diagnosis, for example, chronic obstructive pulmonary disease, heart failure, vocal cord dysfunction and endobronchial lesions simulating airway wheeze

Final point: Treat the patient, not just the asthma.

The advancing state of modern medicine often leaves the patient behind. There is a phenomenal choice of drugs and delivery devices available to treat chronic asthma. This has led to confusion in prescribing policies. However, focusing only on the pharmacologic aspect of asthma is akin to missing the forest for the trees. Pharmacoeconomic and physical constraints are a major limitation in treating asthma. For instance, patients that are less well to do may find it difficult to afford a monthly treatment prescription in the range of $100-200, patients with rheumatoid arthritis may not be able to actuate the metered dose inhalers, and some elderly patients find it extremely difficult to co-ordinate the delivery devices. Flexibility with regimes is necessary to accommodate different patients. For example, for the same drug, lung delivery can be increased by the appropriate use of spacers. Co-ordination difficulties can be overcome by dry powder inhalers, use of spacers or oral medications. Use of older drugs, for example, theophylline, should be considered if financial constraints are significant.

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Practical Aspects of Asthma Management in Children

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Summary

Bronchial Asthma is a common childhood problem affecting one of five school children. The social and economic impact is tremendous for the family. As practicing doctors we can help by giving the best care possible to the child. First, a correct diagnosis has to be made, then an accurate assessment of the severity of the disease and finally, plan for the best therapeutic options. The job has just begun. Long term follow up and monitoring is crucial to ensure good compliance. There are also the inter-current problems to be treated. The doctor cannot achieve all these alone. Partnership between the parents, caregivers and the doctors is the key to the success of therapy and the best outcome.

Introduction

Bronchial asthma affects about 20 % of the school going children in Singapore. The majority are mild and are best managed at the primary health level for cost effectiveness and convenience of the caregiver/parents. Childhood asthma is probably second only to acute respiratory tract infections, the commonest childhood illness. A rational and pragmatic approach is important in treating this group of patients. Below are some practical aspects of managing childhood asthma.

Making the right diagnosis

This is the first important step. Without an accurate diagnosis, no rational treatment can be given. No one has a problem making a diagnosis of asthma in a child who presents with recurrent cough with wheezing, which is precipitated by respiratory infections and aggravated by exertion. However, things are often not as straightforward. A small proportion present with chronic cough alone.[1] Often, this is a nocturnal cough and the child is very well the next morning at the clinic! Some children present with the problem of recurrent "chestiness", or gave a history of recurrent episodes of bronchitis or chest infections.

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For patients with atypical presentations, a detailed history is necessary to exclude conditions which mimic asthma. Patients with chronic sinusitis will also complain of recurrent cough and chestiness. Difficulty in breathing due to a blocked nose from chronic rhinitis may be attributed to asthma. Young infants with wheezing and cough but little evidence of a respiratory infection and who have a history of frequent regurgitation, are candidates at risk of chronic aspiration from gastroesophageal reflux.[2]

It is also important to ask about the onset of the problem. Congenital malformations must be excluded in children with a history of wheezing since birth, whereas a child with an acute onset of wheezing after eating "peanut porridge" may have aspirated a foreign body.

In patients whom asthma is suspected, careful questioning often reveals a personal or family history of asthma or asthma related allergies such as eczema, allergic conjunctivitis or allergic rhinitis.

It is always prudent to carry out a careful examination of all patients, especially those with chronic or recurrent symptoms. Whilst the presence of rhonchi on auscultation may suggest asthma, care must be taken to exclude other diagnoses. In a young infant less than 6 months of age, the commonest cause of wheezing is viral bronchiolitis. Sometimes it can be difficult to differentiate rhonchi from noisy breathing through an obstructed nose. It may help to ask the child who has a blocked nose to breathe through the mouth and when the noise disappears, it is the noise due to air moving through the obstructed nasal passage and not "rhonchi" which is due to obstruction in lower airways. In young infants, it is important to time the respiratory phase; inspiratory stridor caused by a floppy upper airway may be mistaken to be that of an audible wheeze if one is not careful.

The presence of other findings may point to other possible diagnoses. Children with congenital heart disease, particularly those with a large left to right shunt presenting with cardiac failure may closely mimic the signs and symptoms of asthma. In these children, a cardiac murmur will be present on auscultation of the heart. An infant with persistent wheeze may point to a fixed airway obstruction such as tracheal stenosis.

Often, the diagnosis of asthma can be confidently made after a detailed history and careful examination. We seldom need to carry out special investigations. However, if the symptoms are atypical, some investigations may be necessary. In children with chronic or severe symptoms, objective assessment may be helpful in planning the treatment strategy.

The commonest investigation is a CXR. This aids in the exclusion of a chronic infection such as PTB which could mimic asthma, particularly in young infants. Rarely, mediastinal masses may be picked up on a CXR. A short period of monitoring the Peak Expiratory Flow Rate (PEFR) with a portable Peak Flow Meter at home, may help to document the presence of reversible airway obstruction. This is a hallmark in the diagnosis of asthma. PEFR and spirometric measurements are useful tools in the assessment of the severity of the underlying asthma. Children with long term asthma should have at least one spirometric measurement as an objective assessment. It is well known that the severity of asthma is not only underestimated by patients but also by parents and doctors. For patients who have long term symptoms but are otherwise asymptomatic in between acute excerbations, at least one spirometric measurment is desired.[3] This should be carried out when the child is well. For clinics who do not have the facilities to carry out spirometric measurements, the child may be referred to a Respiratory Function Laboratory for the tests.

Choosing the optimal therapeutic option

Having made the diagnosis and after a careful assessment, it is important to select a suitable treatment option for the patient based on the severity of the disease. There are consensus statements on the management of paediatric asthma in Singapore which family physicians can use as guidelines.[3]

Doctors should keep in mind that parents expect good results in a short time with no adverse effects and minimal cost, though not necessarily in that order. It is, therefore, prudent to have a frank discussion with the parents on the options available and what are the realistic and achievable goals. The fact that treatment may be long term and changes to the treatment regime may be necessary must be clearly explained to them. It is a good idea to let parents know when they are likely to notice improvement in the child's symptoms. When kept well informed, parents are more likely to be cooperative and will have more confidence in the doctor. This will prevent them from doctor hopping when they do not see a response in one to two days. Make it clear that alternative strategies will be in place if the child does not respond well to the treatment. A referral for second opinion will be considered when necessary. One common complaint from parents is that family physicians are only "interested in treating symptoms rather than preventing symptoms". Parents are pleased with doctors who care for the well being of the child, and letting them know that you will not hesitate to send the child to another colleague whom you feel may help in the management, is reassuring and will boost their confidence.

The choice of treatment is largely dependent on the duration and severity of the symptoms, the age of the child and the financial situation of the family, especially when we are dealing with long term treatment. It is best to choose a treatment with a predictable efficacy and good outcome in the majority of patients.

Inhaler therapy is well documented to be both safe and efficacious. Even young infants can be put on inhaler therapy with the use of an appropriate spacer device. The efficacy of MDI with spacer is as good as that of nebuliser and has less effect on heart rate in children.[4]

For a quick response, particularly in children who have been symptomatic for long time, it is practical to start with maximal therapy for that

severity and to down regulate as soon as symptom control has been achieved. Children on inhaled steroid therapy usually show improvement within two to four weeks. Thereafter, the dose of steroids can be reduced or changed to sodium cromoglycate where appropriate.

For parents who have steroid phobia, the alternative anti asthma medications include nedocromil sodium (Tilade) and montelukast (Singulair). Currently, both are recommended for use in older children in our community.

Follow up and monitoring

Whatever treatment is offered, the regime must be kept as simple as possible, preferably not exceeding twice a day, to ensure compliance. It is equally important to make sure that the family can afford the expenses, otherwise alternative cheaper regimes should be given. To improve compliance, scheduled visits are useful for reassessment, re-enforcement and re-education. Objective measures like PEFR and spirometry may be good indicators of the progress made. Caregivers can be taught home management of milder exacerbations and be kept informed of the progress and future plans of stepping down therapy during these scheduled visits.

Most children will require 6 months to one year of anti inflammatory therapy. Treatment can be reduced slowly once the child has been well for 3 months. During the stepping down period it is necessary to inform parents of the possible relapse of symptoms and advise them on the measures to take if that happens.

Children who have relapse of symptoms after an appropriate course of anti inflammatory treatment will require a full assessment, for these children who may have persistent asthma or there may be other concurrent medical problems like allergic rhinosinusitis.

Conclusion

Bronchial asthma is a common childhood disorder which requires much attention from the attending doctor. After making a diagnosis, the best therapeutic option has to be selected and continuous care and monitoring is essential to the success of the therapy. The best outcome is only possible with a good partnership between the parents/caregiver and the family doctor. Only a small number of patients with chronic or problematic symptoms need to be referred to a specialist for management. In this group of patients, continued support from the family physician is important for the total care of the patient.

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Inhalational Therapy in the Management of Childhood Asthma

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Introduction

The prevalence of bronchial asthma in school children has risen from 5.5% in 1969, to 14.7% in 1987, and 20% in 1994. Clinical practice guidelines have been developed to help physicians in selecting the appropriate drugs in the management of childhood asthma[1]. However, the choice of the method of delivery is as important as the choice of drugs. Optimal delivery of the drug is vital to the success of the therapy.

Drugs may be administered to the asthmatic patient by three main routes: orally, by inhalation and by injection. Injections are mainly reserved for moderate to severe asthmatic exacerbations which are managed in hospitals and will not be discussed here.

Oral vs inhaled therapy

Inhalational therapy is preferable to oral treatment in the management of childhood asthma due to the advantages of a low, targeted dose, reduced side effects and faster onset of action.

1. Beta$_2$ agonists

There are considerable disadvantages in using the oral route. The dose of beta$_2$ agonist given is usually 10 to 20 times higher than inhaled medications, thus producing unwanted side effects of tremors and tachycardia. The other problem is the onset of action. Oral salbutamol takes at least half to one hour to act, while inhaled salbutamol is effective within minutes. Hence, children who require bronchodilator intermittently are advised to use an inhaler.

2. Steroids

Oral steroids are useful in acute severe asthma but its use should be restricted to patients who do not respond to inhaled salbutamol and inhaled steroids during an acute exacerbation. Any child who receives more than 4 courses per year of oral steroids is at high risk of developing complications such as osteoporosis, stunting of growth, suppression of the hypothalamic-pituitary-adrenal axis etc. Patients who need frequent courses of steriods should have their maintenance medications reviewed with the intention of stepping up the preventive therapy.

Inhalational therapy

Four different inhalational systems constitute the cornerstone of inhalational therapy in childhood asthma:

1. Metered dose inhalers (MDI)
2. MDI with a spacer / aerochamber
3. Dry powder inhalers (DPI)
4. Nebulizers

Each differs with regards to the methods of generating particles of desirable size, optimal inhalational technique and ease of use. It is thus important for physicians to understand the correct inhalational technique and the optimal age limit for the various devices to ensure maximal delivery of drugs to the patients. Many investigators have reported high frequencies of incorrect use as a direct cause of treatment failure in asthmatic children.

General principle of aerosol deposition

The larger the particle size, the greater the deposition in the oropharynx and the large airway in humans. In general, the desirable intrapulmonary deposition of an aerosol particle in asthmatics is greater with particles in the range of 2 to 5 μm in diameter. Aerosol deposition is also affected by the breathing pattern, the inspiratory flow rate (especially in DPI) and the electrostatic charge (in spacers).

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A) MDI

In MDIs, the active drug is dispersed throughout a chlorofluorocarbon propellant in the canister. The tendency for the fine particles to agglomerate is prevented by a surfactant. The patient activates a dose from the aerosol by pressing down the canister, thus releasing a metered dose of the drug, propellant and surfactant into the air. The canister should be shaken before activation to ensure proper mixing of the content. Aerosol should not be stored in a refrigerator because this will decrease the vapour pressure of the propellants and affect the delivery of the drugs.

Inhalation technique : (Fig 1)

Metered dose inhalers (MDI)

1. shake the canister
2. position upright and direct into the month
3. discharge the canister at the beginning of inspiration
4. inspire slowly and hold breath for 10 seconds
5. wait 1 minute to inhale the next dose

Problem with the use of MDI:

Correct administration of MDI without spacer device is difficult in children due to the following reasons:

- Difficulty in the co-ordination of actuation and breathing
- "Cold Freon effect" ie the patient stops inhalation when the cold aerosol particles reach the soft palate
- Actuation of aerosol into the mouth followed by inhalation through the nose

Breath actuated devices

Breath actuated MDI (eg autohaler, accuhaler) removes the problem of coordination. It may be used in patients older than 6 years of age with proper counselling.

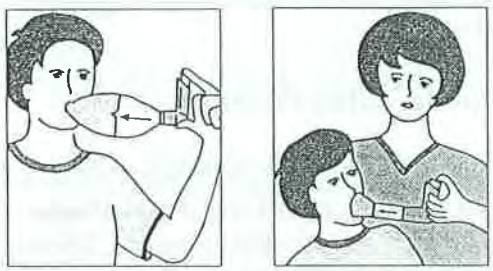
B) MDI with Spacer

Types of spacers:

- plastic spacer - small volume eg aerochamber
  - large volume eg volumatic spacer, nebuhaler
- Non-electrostatic spacer – metallic spacer

Using a spacer with the MDI slows down the velocity of the aerosol particles and reduces their size. It also allows evaporation of the propellants before inhalation, thus reducing the side effects such as heart palpitation and bronchoconstriction. The spacer has a one-way valve which opens during inspiration and closes during expiration.

Inhalation technique: (Fig 2)



Metered dose inhalers with spacer devices

1. shake the MDI canister
2. insert into one end of spacer
3. fire the canister into the spacer
4. take 5 normal breaths through the mouth piece
5. with aerochamber, the face mask covers the nose and the mouth and the child takes 5 normal breath through the mask
6. remove the spacer from the mouth and face
7. wait for 1 minute before the child repeats the above procedure

Advantages of spacer with MDI

- Eliminates the problem of non-coordination
- Increases lung deposition from about 10% with MDI to about 20% with spacer and MDI
- Decreases oropharyngeal deposition. This is especially important in the case of inhaled steriods
- Decreases 'cold freon' effect of aerosol
- Useful in an acute exacerbation when the patient has a decreased inspiratory flow rate

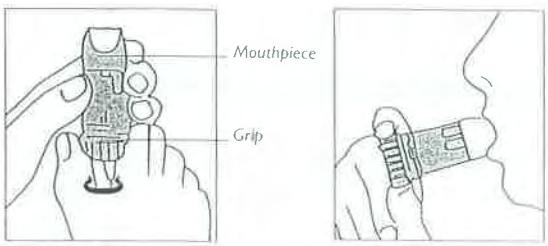
Problems with MDI and Spacer

- A spacer is bulky and is difficult to carry around. It is best used for giving prophylactic treatment at home in the morning and evening
- Electrostatic charges on the wall of the spacer attracts the drug particles thus reducing the delivery of the drug. This effect is overcome after repeated use when the lining of the wall is saturated. Priming a new spacer with 20 actuations before use also achieves the same effect. A metallic spacer (not yet available) eliminates this problem.
- Large volume spacers deliver more drugs than small volume spacers. A spacer device with a face mask is recommended for children below 3 years. Older children who are able to breathe through a mouth piece will be encouraged to use a volumatic spacer or a nebuhaler.

Dry Powder Inhalers (DPI)

In dry powder inhalers eg, turbuhaler, the drug is provided in finely milled powder in blisters or capsules which are individually packed. It is often mixed with a carrier substance, eg lactose, in large aggregates (>60 μm). The turbulent airstream created during inspiration breaks up the large aggregates into small particles to be delivered to the lower airways. Therefore, a minimum inspiratory flow of 30 l/min is required to effectively deliver the drug, hence, DPI is usually recommended for children older than 6 years of age.

Inhalation technique (Fig 3)



Turbuhaler

1. Unscrew and remove the white cap
2. Twist the grip fully to the right, then twist back to the left until it clicks. This will load the inhaler
3. Breathe out gently. Then place the mouthpiece between the lips and inspire deeply, and hold breath for 10 seconds. Repeat if a second dose is needed.
4. Replace the cap

Advantages of the turbuhaler

- Studies done on school children suggest that intrabronchial deposition is twice as high with turbuhaler than after treatment with a metered dose inhaler with a nebuhaler attached[2]
- Compact and portable
- No propellants needed
- Easier to use than MDI alone

Problems with DPI

- Insufficient inspiratory flow (minimal 30 l/min) to activate the drug, especially in acute exacerbation of asthma. This can be overcome by proper training.
- Children may exhale through the inhaler, thus blowing the drug out of the inhaler. Proper training will eliminate this problem
- Moisture sensitive, hence, DPI should always be re-capped after use

Nebulizers

In a jet nebulizer, air or oxygen from either a wall source, compressor or cylinder, flows through a narrow venturi where negative pressure is created. Liquid is drawn up in a feeding tube and is fragmented into droplets. Smaller droplets are carried away in the inhaler airsteam. The remaining large droplets impact on the baffles, return to the reservoir and are nebulized. There will always be a residual volume of 0.5 to 1 ml of solution left behind. Decreasing the volume fill below 4 ml will increase the concentration of the drugs left behind in the reservoir. However, increasing the initial volume fill prolongs the time of nebulization to more than a few minutes, which is not tolerable to most young children. The flow rate of the driving gas is also an important factor in determining the aerosol deposition. Flow rates of 6 l/min maximises the nebulizer function.

An ultrasonic nebulizer uses a piezo electric crystal vibrating at a high frequency to generate a formation of liquid in the nebulizer. It works quietly, but cannot be used for suspension, eg, budesonide suspension. Newer nebulisers such as sidestream nebulisers are more effective and allow increased drug delivery.

Problems with nebulizers

- Children should inhale through a well-fitted mask. Any inhalation through a mask that is 2 to 3 cm away from the face, reduces the drug delivery by about 50%[3].
- It is bulky, inconvenient and not portable.

With adequate training, most children can be trained to use inhalers. Recent studies have shown that even in acute exacerbations, MDI with spacer produce outcomes that are equivalent to nebulizers.[4] Nebulizers should be limited to a minority of children who absolutely refuse to use an MDI with or without spacer / mask.

Conclusion

The choice of an inhaler device in children should be based on an understanding of the advantages and problems of each device. The decision to select a particular method should be individualised and based on the child's age, his ability to use it, his preference and the cost of the medicines. The appropriate choice of inhaler is very important to ensure the patient's compliance with therapy to maximise the efficacy of treatment.

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Choice of inhaler device for children

According to the British Thoracic Society Guideline in 1997

	1 – 2 years	3 – 5 years
MDI + spacer and face mask (or aerochamber)	1st	2nd
MDI + spacer	2nd	1st
Nebuliser	3rd	3rd

Dry powder inhalers and the breath actuated MDI will be recommended for children older than 6 years old.

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Your Job and Asthma

Lee HS

Summary

A significant proportion of adult asthma may be caused or aggravated by occupational factors. Occupational asthma is the most common occupational respiratory disease in Singapore. The definition, common causative agents and the diagnosis and management of a patient with occupational asthma are discussed. It is important in the total management of the asthma patient that causal or aggravating factors be identified and advice be given to avoid exposure to such factors. Cases of suspected occupational asthma should be investigated to confirm the diagnosis and removed from further exposure to the causal or aggravating factors, where feasible. Early diagnosis and removal from further exposure could result in a better prognosis for the patient.

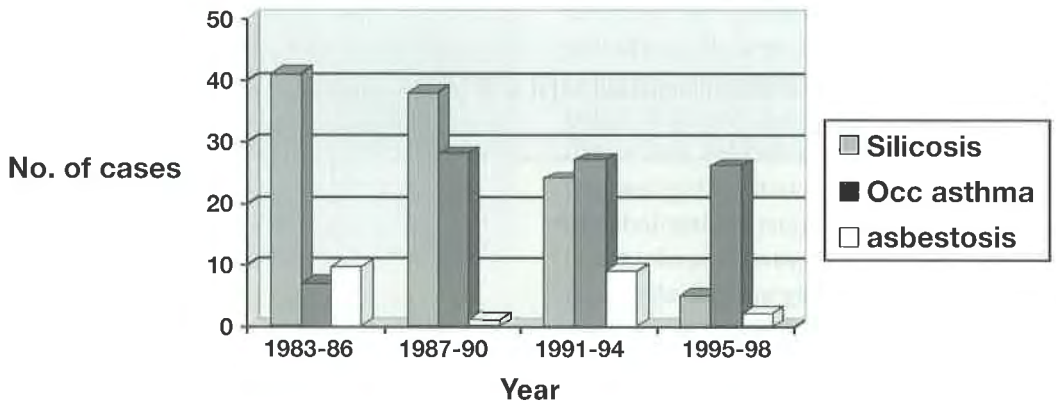
Introduction

Asthma occurs commonly in the general population and among asthmatic adults, a proportion of the cases might be caused by or aggravated, to varying degrees, by occupational exposures. It has been estimated that up to 15% of adult asthma may be caused by the work environment[1]. In a study conducted in Singapore, higher associated risk of asthma was found among service workers and manufacturing workers (in particular cleaners, textile workers, electronic production operators, printers and construction or renovation workers) while a reduced risk was found for professional and administrative workers[2].

Asthma may contribute significantly to medical expenses and sickness absence. Depending on the severity of the asthma and the nature of the job, it may affect his or her job performance or his fitness to work in certain types of jobs. As a physician, we have an important role in the management of a worker with asthma. This includes the identification of cases of work-related asthma, medical treatment, advice on avoidance of trigger factors and recommendations on job transfer and fitness to work, where indicated.

In the industrialised countries, eg the United Kingdom[3], occupational asthma is the most common occupational respiratory disorder while in developing countries such as China, silicosis is the leading occupational lung disease[4]. Occupational asthma is the most common occupational respiratory disease in Singapore[5]. It was made a notifiable industrial disease under the Factories Act in 1985. It is also a compensable disease under the Workmen's Compensation Act.

Occupational Lung Diseases in Singapore, 1983-1998



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Definition of occupational asthma

Previously, occupational asthma was defined rather narrowly as "asthma induced by sensitization to an agent inhaled at work". More recently it was defined as "a disease characterised by variable airflow limitation and/or airway hyperresponsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace"[6]. This latter definition includes cases of asthma caused by mechanisms other than allergy, eg inflammation from irritants or those caused by unknown mechanisms as in isocyanate asthma. The presence of pre-existent asthma should not preclude the diagnosis of occupational asthma, if it can be shown that the patient developed asthmatic attacks as a result of exposure to the working environment. In a very broad sense, occupational asthma is asthma caused or aggravated by the work environment. However, the work factors (which should be specific) should cause significant aggravation compared to non-work factors.

Why is it important to investigate and confirm the diagnosis of occupational asthma?

1. **Identify the specific cause**
   - advise worker to permanently avoid further exposure
   - reduce morbidity and medical leave
   - prevent the risk of a severe or fatal asthmatic attack in the workplace
   - reduce the risk of permanent airway damage

2. **Provide a clear basis for action**
   - recommending job transfer
   - future job placement

3. **Medico-legal reasons**
   - notifiable industrial disease
   - compensable occupational disease

What are the occupation at risk and common causative agents?

Common causes of occupational asthma in general and in Singapore are given in tables 1 and 2.

Table 1 Some Common Causes of Occupational Asthma

Agents	Occupations
Isocyanates eg toluene diisocyanate (TDI)	Polyurethane foam workers Spray painters and varnishes Insulation workers
Acid anhydrides	Chemical workers making or using eg phthallic anhydride epoxy, alkyd or polyester resins Spray painters
Aliphatic amines eg ethylene diamine	Spray painters
Pharmaceuticals eg antibiotics, glutaraldehyde	Pharmaceutical technicians Health care workers Veterinary workers Animal feed workers
Soldering flux eg colophony	Soldering operators
Coolant oil mist Welding fumes	Machinists Welders
Wood dust	Carpenters
Metals and their salts Eg nickel, cobalt, chromium	Electroplaters Welders
Foodstuffs eg soybean, flour	Food processing workers
Enzymes eg B subtilis	Bakers Detergent workers
Animal products eg dander, excreta, urine	Laboratory technicians Zoo keepers

Table 2: Occupational Asthma by Causative Agent in Singapore, 1983-1998

Causative Agent	No of Cases (%)
Isocyanates	27 (30.7)
Solder flux	12 (13.6)
Welding fumes	9 (10.2)
Wood dust	4 (4.5)
Pharmaceutical Drugs	4 (4.5)
Grain dust	3 (3.4)
Amines	3 (3.4)
Acid anhydrides	2 (2.3)
Coolant mist	2 (2.3)
Others	22 (25.0)
Total	**88 (100)**

Workers at risk include spray painters, welders, solderers, chemical process workers, insulation workers, bakers, carpenters, machinists, pharmaceutical workers and workers involved in the manufacture of polyurethane foam products, eg cushions and foam mattresses. The most common causative agent is a group of chemicals called isocyanates, eg. toluene diisocyanate (TDI). They are used as hardeners or curing agents in polyurethane paints, varnishes, glues and in the manufacture of polyurethance foam products. The common causative agents may vary from country depending on the type of industries.

Clinical Presentation and Diagnosis

The approach to diagnosis is made simpler by asking these questions. Firstly, is there asthma? The history may reveal symptoms suggestive of asthma such as cough, breathlessness, chest tightness and wheezing which varies in severity from time to time. The asthma reaction may be immediate, delayed or there may be a dual reaction. There may be accompanying rhinitis. Sometimes wheezing may not be present and the only symptom may be a persistent cough especially in the early stages. Other evidence include clinical documentation of an asthmatic attack or a positive methacholine challenge test.

Secondly, is there a work-related pattern of symptoms? Ask for improvement of symptoms when away from work and recurrence of symptoms when back at work. While the history is a very important screening tool, by itself it may not be a satisfactory means of diagnosing occupational asthma. Objective evidence is often necessary to document that the asthma is work-related.

Serial Peak Expiratory Flow Rate (PEFR) Monitoring

Serial PEFR monitoring is now an established tool in the investigation of occupational asthma.

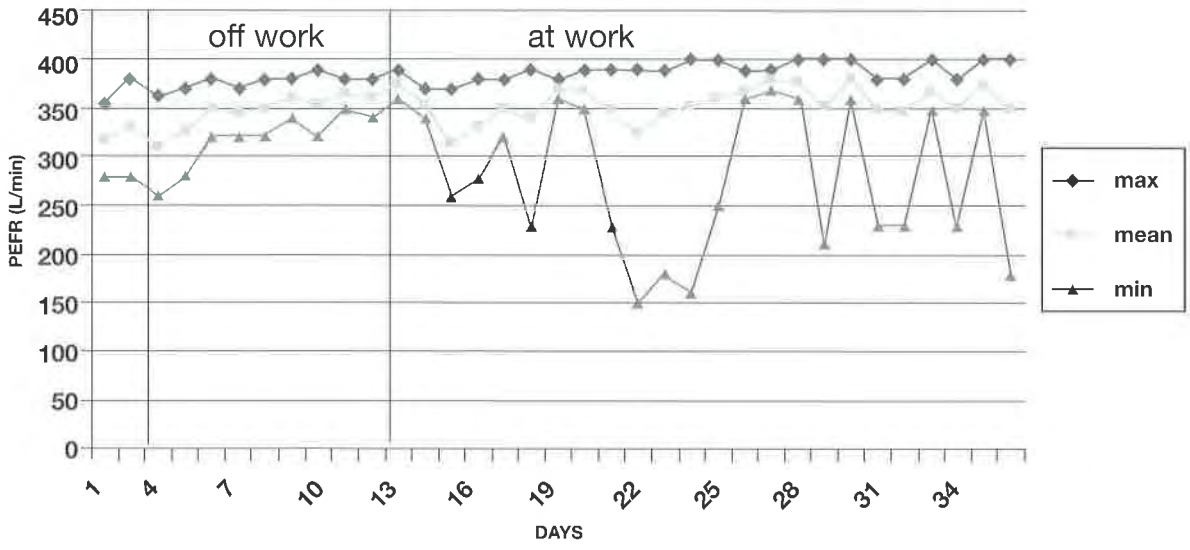

Figure 1. Serial PEFR of a patient with occupational asthma to cyanoacrylate glue

Patients are instructed on the proper use of a portable PEF meter and told to record their PEFR every two to three hours during the waking hours for about three weeks. This should include periods at work and away from work. The period away from work should be about 7 to 10 consecutive days. Each time the best of three readings is taken. The daily maximum, minimum and mean readings are plotted on a graph.

A diurnal variation of 20% or more is suggestive of asthma. Diurnal variation is calculated as the difference between the maximum and minimum PEFR expressed as a percentage of the maximum for each day. Look for evidence of improvement when at home and for deterioration when at work. Deterioration is shown by the falling mean PEFR and a widening diurnal variation. An example of the serial PEFR of a worker confirmed to have occupational asthma to cyanoacrylate glue is shown, (see figure 1). The serial PEFR can confirm the presence of work-related airways obstruction but does not tell us the specific cause of the asthma.

Thirdly, what is the specific cause of the asthma? To know the specific cause, we must know what the worker is exposed to in his work environment. A workplace visit together with a literature search will tell us what possible known asthma causing substances he may be exposed to. If he is exposed to only one known asthma causing agent, and has a serial PEFR showing a work-related pattern, one may presume that his asthma is caused by that known agent.

However, the patient may be exposed to several known causes of asthma in his workplace. He may be exposed to chemicals not previously reported to cause asthma. Or he may have already left his workplace and it is not possible to do a serial PEFR at his previous workplace. In such situations, a specific bronchial provocation test to the suspected agent is indicated.

Specific Bronchial Provocation Test (BPT)

This is done on an in-patient basis so that any asthmatic reactions, especially delayed reactions can be properly documented and treated. Ideally, the patient should have his asthma stabilised first and not be on any medications. On the first day, he monitors his PEFR three hourly for 24 hours. On the second day, he is exposed to a control substance, eg lactose dust or some other inert substance, and his PEFR monitored. If there is no reaction, he is exposed to the suspected agent on the third day and his PEFR monitored. A fall in the PEFR by 20% or more from the baseline in the presence of a negative control challenge can be considered a positive BPT. The exposure levels should not exceed workplace levels and the permissible exposure limits of the substance. The actual levels and duration of exposure would depend on the agent and the patient. In general, these should be kept as low and as short as possible.

Management

Once the case is confirmed, the patient should be permanently transferred from exposure to the specific causative agent. He should be told what is the cause of his asthma and what to avoid in future - this may include other jobs which may expose him to a similar substance. His asthma should be medically treated according to existing protocols and he should be informed that his symptoms may persist for a while even after his transfer. About 50% of cases continue to have persistence of symptoms even after ceasing exposure and one of the factors said to contribute to this is a prolonged period of continued exposure after the development of the asthma[7]. It is, therefore, important to diagnose cases early and to transfer them from further exposure.

Some common reasons for persistence of asthma after transfer of worker with occupational asthma from exposure to causative agent

1. Still exposed indirectly to the causative agent
2. Exposure to other irritants
3. Inadequate medication
4. Poor compliance to medication
5. Insufficient time for recovery

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Workmen's Compensation

If he is a workman by definition, he may be eligible for Workmen's Compensation. Under the Workmen's Compensation Act, a workman with occupational asthma may be entitled to temporary disability benefits (ie medical leave and treatment costs) as well as permanent disability benefits. For the assessment of permanent disability, the patient should be removed from further exposure to the causative agent for at least a year and his asthma optimally treated. The disability would depend on the lung function. However, patients who require daily maintenance medication to control their symptoms may be considered mildly impaired even though their lung function is normal and be awarded between 5-20% disability depending on the medication required[8].

Prevention

Substitution of chemicals known to cause asthma should be considered, if this is feasible. Otherwise workplace exposures to asthma causing agents should be kept as low as possible to minimise the risk of occupational asthma through engineering controls such as enclosures, local exhaust ventilation systems, respiratory protection and other administrative procedures. Workers should be educated on the symptoms of asthma and encouraged to seek early medical attention.

Fitness to Work

A worker with asthma may have to be assessed for this fitness to work in a particular job. It would be relevant for the doctor to know both the patient's work capacity as well as the job demands. This subjective effort tolerance of the patient and the lung function can be used to estimate his physical work capacity. Other medical conditions and his cardiac status are also relevant. The attitude of the patient and how he perceives his disease and his work are important factors too.

The physical demands of the job and its component tasks need to be assessed in relation to the patient's capacity to meet them. How much physical effort is required? What is the pace of work? Does he need to use respirators? Breathing through a high resistance filter may be demanding for a severely impaired worker. Medical examination for fitness to use respirators is one of the components of a respiratory protection programme[9].

The environment should also be assessed. Would he be exposed to dusts, fumes or irritant gases? Would these aggravate his asthma? Would he be exposed to substances that he may be allergic to?

Conclusion

In any worker suffering from asthma, the physician should consider the possibility of occupational factors in the causation or aggravation of the condition. The total management of the patient includes identifying likely trigger factors and advising the worker to avoid exposure to these factors, whether occupational or otherwise. Early diagnosis and removal from further exposure could result in a better prognosis for the patient.

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Exercise and Asthma

Ong KC

Introduction

Bronchial asthma is a chronic obstructive airway disease characterized by airway inflammation and hyper-responsiveness. Multiple factors can trigger acute asthmatic attacks and exercise is one of the most frequent precipitants. Although exercise-induced asthma (EIA), defined by an attack of asthma induced by exercise, has been reported to affect 50 to 80% of patients with asthma, it is generally believed that, given sufficient exercise intensity, *all* patients with asthma will be affected by EIA[1]. Hence, the association between asthma and exercise is a very common occurrence indeed.

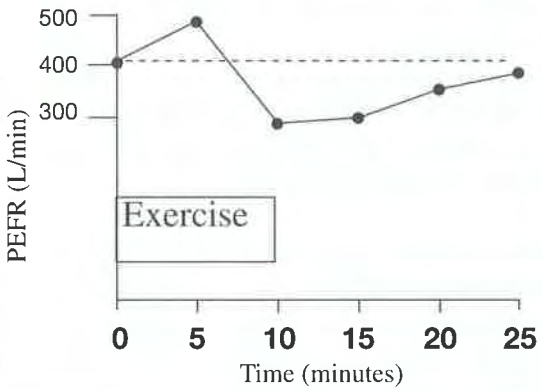
This article examines the relationship between exercise and asthma from two different perspectives. Firstly, the relationship between exercise and EIA will be reviewed. Secondly, the influence of asthma on the patient's exercise capacity as well as the role of exercise reconditioning in the holistic management of the asthmatic patient will be discussed.

How does exercise affect asthmatics?

EIA should be suspected in a patient with asthma who has symptoms of wheezing, coughing, shortness of breath and chest pain or discomfort during and shortly after exercise. The symptoms are most intense for 5 to 10 minutes and usually resolve within 15 to 30 minutes following exercise cessation.

These symptoms are correlated to pulmonary function measured serially before, during and after exercise as shown in Figure1. Bronchodilatation, probably as a result of the release of endogenous cathecholamines, is the initial response to exercise in both normal individuals and those with asthma. This response is transient, peaks at mid-exercise, and returns to baseline at the end of exercise. In patients with asthma, however, progressive bronchoconstriction ensues with maximal obstruction occurring 5 to 10 minutes after the cessation of exercise. Spontaneous remission

Figure 1. Serial monitoring of peak expiratory flow rates (PEFR) before, during and up to 25 minutes following the cessation of exercise in a patient with EIA.



usually follows, so that pulmonary function returns to baseline within 30 to 60 minutes. Three major theories have evolved over the past few decades with regard to the specific inciting stimulus for EIA: heat loss, water loss (hyper-osmolarity) and rewarming of the airway. Of these, the water loss hypothesis of EIA is best accepted[2]. The evaporative loss of water from the respiration mucosa during exercise causes a transient hyperosmolarity of the periciliary fluid. This, in turn, induces not only the release of mediators from mast cells but also compensatory dilation of the bronchial vasculature. Inflammatory mediators further enhance vasodilation and induce bronchial smooth muscle contraction, resulting in airway obstruction.

As the symptoms of EIA are very nonspecific, objective documentation of airflow obstruction following an exercise challenge and reversibility after exercise may be required to make the diagnosis of EIA. This can be done by exercise challenge (or bronchoprovocation) testing. In this setting, the patient performs exercise of an intensity standardized to increase the individual's heart rate to 80% of the predicted maximum for a duration of 6 to 10 minutes on a treadmill or

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bicycle ergometer. Serial measurements of peak flow or FEV1 are recorded before exercise and every 5 minutes thereafter, for a total duration of 30 minutes after the cessation of exercise. A positive response is denoted by a 10% (probable EIA) or greater (>15% = definite EIA) decrement in peak flow or FEV1, typically occurring 5 to 10 minutes after exercise is discontinued[3]. Recently, challenge tests for EIA that do not require exercise, eg hyperventilation of dry air and inhalation of mannitol[4] have proven useful and much more convenient to administer. In the field, the diagnosis of EIA can also be made with objective documentation of air-flow obstruction by the serial monitoring of peak flows before and after exercise and with the onset of symptoms, before and after bronchodilator.

There are three factors contributing to the severity of exercise-induced airway obstruction: minute ventilation (exercise intensity), temperature and humidity of the inspired air (climatic conditions), and baseline airway reactivity. There are differences in asthmogenicity between various sports related to differences in the inherent minute ventilation and climatic conditions associated with each particular sport. For example, physical activities requiring high levels of minute ventilation, eg basketball or rugby, produce a greater degree of airway obstruction than activities associated with a lower level of minute ventilation, say golf or gymnastics. Likewise, warming and humidifying the inspired air decreases the severity of obstruction, whereas cooling and drying the inspired air exacerbates obstruction. Therefore, activities associated with warm and humid conditions such as swimming and water polo are less asthmogenic than activities associated with cool and dry climatic conditions like ice hockey and skiing.

How does asthma affect exercise capacity?

If left untreated, asthma limits tolerance for even mild degrees of physical activity and exercise. Because of this exercise intolerance and for fear of inducing acute episodes of asthma, previous philosophy was to limit or even exclude exercise in patients with asthma. With a better understanding of the pathogenesis of the disease as well as the development of newer and improved medications, even individuals with moderate or severe asthma can participate in recreational or even competitive sports as long as the disease is optimally controlled. A frequently quoted illustration in support of this is the fact that 11% of the 1984 US Summer Olympic team had EIA but they went on to win 41 medals[5].

As baseline airway reactivity is one of the 3 major variables influencing the severity of exercise-induced airway obstruction, any therapeutic approach for reducing exercise-induced symptoms in a patient with asthma should start with optimizing the control of asthma by reducing airway inflammation and bronchial hyperreactivity. Many asthmatic patients are often undertreated and EIA may be a reflection of this. Established guidelines[6] which provide optimal step-wise care of patients with asthma and recommendations for managing or eliminating common trigger factors such as allergic rhinitis, sinusitis, allergen exposure which may nonspecifically increase bronchial responsiveness are currently available.

Next, specific strategies to prevent EIA in susceptible individuals should be considered. These involve both non-pharmacologic and pharmacologic modalities. Non-pharmacologic modalities include appropriate warm-up exercises prior to the onset of exercise and reducing the exposure to cool dry air during exercise. In approximately 40% to 50% of patients with asthma, a mild attack of asthma makes the patient less responsive (or refractory) to an identical exercise task performed within 1 to 2 hours. This is known as the refractory period. The mechanism of this response is at present unknown but patients who develop this response may be able to use this to their advantage by inducing the refractory state with a series of warm-up exercises approximately 45 to 60 minutes before the onset of exercise. As there are no clinical or spirometric parameters that can accurately identify patients who are capable of inducing a refractory period, empirical induction for all patients with EIA is recommended. The intensity of warm-up exercises necessary to induce a refractory period remains to be established but multiple sprints of 30-seconds duration, 2 minutes apart before prolonged exercise have been reported to reduce

the severity of EIA. Reasonable precautions to reduce exposure to cool dry air during exercise cannot be overemphasized but this is not as much a problem locally as in temperate countries.

Many different kinds of medications may be used in the prevention of EIA[2]. Generally, beta-adrenergic agonists are very effective, being able to reduce or completely attenuate EIA in 90% of patients. In addition, they may be used as a rescue agent to treat acute bronchopasm. Therefore, they are widely considered as first line drugs for EIA. In view of its potent bronchodilator effect and rapid onset of action, most patients require 2 to 4 puffs of salbutamol 15 minutes before the onset of exercise to prevent EIA. The beta-agonist inhaler should be used between the warm-up and subsequent exercise, either as a rescue agent (if needed) or as part of a preventive regimen. It is note-worthy that whereas inhaled and nebulized formulations of beta-agonists are approved, all oral forms of the drug are banned in international competitions. Newer long-acting beta-agonists (eg salmeterol and formoterol) are also efficacious in preventing EIA and have a much longer duration of action. The use of anti-leukotrienes for prevention of EIA is also promising.

In spite of maximal treatment for chronic asthma and preventive modalities for EIA, there are some difficult-to-manage patients who have such a high baseline level of airway reactivity that they become symptomatic with even minor degrees of physical activity. These patients should undergo further exercise testing (with their standard premedicating regimen) to determine if bronchospasm is really the cause of their symptoms and to exclude other causes of exercise limitation, eg poor physical fitness, muscular weakness, ischemic heart disease. Cardiopulmonary exercise testing (CPET) which is an integrative evaluation with gas exchange and ventilatory measurements added to the monitoring of the electrocardiogram and blood pressure during exercise is ideal for this purpose.

As a group, individuals with asthma are physically deconditioned (unfit). It is a misconception that exercise-induced bronchospasm is the limiting factor to exercise for patients with asthma. Psychological factors and physical deconditioning are more common contributory factors to exercise intolerance than exercise-induced bronchospasm[7]. Patients with inadequately treated airway obstruction may experience breathlessness at lower levels of activity than individuals without asthma. To avoid this sensation, these subjects exercise less, leading to decreased physical fitness. As a result, even lesser degrees of exercise induce breathlessness, creating a vicious cycle (known as the dyspnea spiral) ending in a sedentary and physically unconditioned person. Surely the logical thing to do for the majority of patients with asthma in order to evade the dyspnea spiral is more regular exercise rather than avoidance or abstinence. In addition to improved fitness, the potential benefits of exercise on asthma include possible attenuation in airflow obstruciton to exercise, decreased frequency and severity of acute exacerbations, decreased use of medications and absenteeism, and improved self-confidence.

To reap the benefits of exercise, asthmatic patients who have poor exercise performance resulting from either sub-optimal control of the disease or physical deconditioning should ideally participate in exercise programs and be supervised qualified physicians and physical therapists during exercise. Exercise prescription should be individualized, varying with the patient's disease severity, fitness level and preference of activities. The American College of Sports Medicine (ACSM) has established the goal of exercise programs to be performance of aerobic exercise for at least 20-30 minutes duration 3 to 5 days per week in order to attain physical reconditioning in healthy individuals[8]. The exercise intensity should be between 50% and 85% of the patient's maximum oxygen consumption (as measured by CPET). These guidelines for exercise prescription have frequently been applied to individuals with chronic respiratory disorders with successful results. In individual with asthma, the level of the initial exercise intensity should be above the lower limit necessary for a training effect (using the ACSM guidelines) but at a level that does not require a minute ventilation higher than the patient can sustain continuously for at least 20 minutes (as determined by the patient's level of dyspnea or ventilatory reserve during exercise). The appropriate level of entry, rate of build up and

expected achievable goals of any exercise program should be discussed with the patient to obtain his or her maximum cooperation.

Conclusion

EIA frequently affects patients with asthma. Appropriate treatment of resting airway obstruction and hyper-responsiveness in conjunction with preventive measures and premedications is critical in reducing this occurrence. As factors other than exercise-induced bronchospasm commonly limit exercise performance in patients with EIA and different causes of exercise limitation may not be distinguishable from symptoms alone, all patients with EIA and reduced exercise performance should receive careful evaluation and exercise prescription to maximally derive the benefits of regular exercise. With proper recognition, evaluation and treatment, the effects of EIA on the individual can be minimized, thereby not only allowing patients to engage in recreational exercises but also to excel in competitive sports at the highest level.

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A Mental Health Literacy Survey of Private General Practitioners and the Government Polyclinic Doctors in Singapore

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Summary

Objective: To study and compare the views of private general practitioners and government polyclinic doctors regarding the diagnosis and management of three major psychiatric disorders.

Method: The respondents were required to read a psychiatric case vignette and then complete a structured questionnaire. They were randomly allocated a case vignette of depression, schizophrenia or of mania, with 540 questionnaires distributed to the sample of private general practitioners, and 151 distributed to the doctors attached to the government polyclinics. The respective response rates were 35% and 51%. Judgements regarding the diagnosis and management of the psychiatric disorders were then compared between the two groups.

Results: The study showed that the diagnostic accuracy for both groups was higher for depression and schizophrenia than for mania, with the accuracy rate for the last disorder being of some concern. Both groups generally viewed psychiatrists and other doctors as likely to be helpful, while traditional healers and their practices were seldom rated as likely to be helpful. The respondents also acknowledged the importance and selective relevance of psychotropic medication, as well as a range of other broad non-drug strategies. However, sleeping pills and benzodiazepines were rated as helpful by a high percentage of respondents, which is a concern when dependence is a common problem.

Conclusions: As the main providers of primary care to the community, both groups were highly accurate in recognising the symptoms of depression and of schizophrenia. Results suggest that medical education updates could well focus on the diagnosis of mania (or bipolar disorder) and its distinction from schizophrenia, on conservative use of sleeping pills and benzodiazepines, and on some minor issues identified in the survey.

Key words: mental health surveys, depression, schizophrenia, mania.

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Introduction

Jorm et. al[1] defined "mental health literacy" as referring to the "knowledge and beliefs about mental disorders which aid their recognition, management or prevention". They developed a strategy for examining the extent to which two mental disorders (i.e. depression and schizophrenia) were correctly identified from separately presented case vignettes. The respondents were then assessed for their beliefs about a range of issues, in particular, the helpfulness of a range of interventions for each condition, and the likely outcome for those disorders with and without treatment.

In 1998, Woodbridge Hospital and the Institute of Mental Health conducted a mental health literacy study (Parker et. al, 1999[2]) of its professional staff (i.e. psychiatrists, nurses, psychologists, social workers and the occupational therapists), which identified primary practitioners as being very helpful for those with such disorders. In Singapore, primary medical care is provided by private general practitioners and government polyclinic doctors who may be the first point of access for those with psychiatric disorders. The present study contrasts the diagnostic judgement and management views of these two groups regarding three important psychiatric disorders: depression, mania and schizophrenia.

METHOD

Samples

We distributed 540 questionnaires (comprising 180 of each case vignette) to a randomly selected one-in-three sample of the private general practitioners (GPs). The list was obtained from a local pharmaceutical company. A total of 151 questionnaires were sent to all the 151 doctors (OPDs) practising in the government polyclinics. Questionnaires to both groups sought anonymous responses. A second questionnaire with the same case vignette was sent to the same GP after a few weeks as a reminder if the earlier set had not been returned. As for the OPD sample, the respective heads of the polyclinics were contacted by phone and by letter to remind their staff to return the questionnaires.

At the beginning of the questionnaire, all primary physicians were asked about their years practising as a doctor and whether they had had any prior psychiatric training. Their age and sex were also obtained.

All the primary physicians were randomly allocated a case of major depression, mania or schizophrenia. The case vignettes of depression and schizophrenia were identical to the Australian study by Jorm. The Mental Health Literacy Working Party at Woodbridge Hospital and the Institute of Mental Health developed the case vignette of mania for its professional staff survey, and subsequent use in this study.

The depression vignette described a "Mr A is 30 years old. He has been feeling unusually sad and miserable for the last few weeks. Even though he is tired all the time he has trouble sleeping nearly every night. Mr A doesn't feel like eating and had lost weight. He can't keep his mind on his work and puts off making any decisions. Even day to day tasks seem too much for him. This has come to the attention of Mr A's boss who is concerned about his lowered productivity."

The mania vignette described a "Mr A who is 27 and lives with his parents. He has been employed for most of the time since leaving school, but has recently left his job as a salesman. He has never taken any illicit drugs. His parents state that in the last three weeks he has been extremely active, requiring less sleep and not appearing tired, being talkative and disinhibited and on occasions- quite irritable. He claimed to have invented a machine for curing cancer and wished to go to the U.S. to sell it. When stopped by his parents he became violent, and they had called the police."

The schizophrenia vignette described a "Mr A is 24 and lives at home with his parents. He has had a few temporary jobs since finishing school but is now unemployed. Over the last six months he has stopped seeing his friends and has begun locking himself in his bedroom and refusing to eat with the family or to have a bath. His parents also hear him walking about his bedroom at night while they are in bed. Even though they know he is alone, they have heard him shouting and arguing as if someone else is there. When they try to encourage him to do more things, he whispers that he won't leave home because he is being spied upon by the neighbour. They realise he is not taking drugs because he never sees anyone or goes anywhere".

The respondents were asked to choose the most appropriate diagnosis from the following list of options after reading the case vignette: stress, depression, physical weakness, schizophrenia/ paranoid schizophrenia, mental weakness, mania, anxiety, being possessed, 'others' (and specify), 'nothing' and 'don't know'.

Respondents were then requested to tick one of the options that might "best help" Mr A. They then rated the extent to which a number of resources (16 options), medications (9 options) and activities/therapies (17 options) might be 'helpful', 'harmful', 'neither', 'depends' or 'don't know' for the patient portrayed in the case vignette.

Results

There was a 38% response rate to the 691 questionnaires distributed, with the response rates being 35% (187/540) for the GPs and 51% (77/ 151) for the OPDs.

The GPs were mainly in the age group of 30 to 39 years old and 40 to 49 years old (31% and 32% respectively of the GP respondents). 21% were aged 50 to 59 years old and a significant 15% were 60 years and above. In contrast, the OPDs were mainly in the age group of 30 to 39 and less than 30 years old (53% and 37% respectively of the OPD respondents). Of the GP respondents, 77% were males and 23% were females. The reverse was found in the OPD cohort (43% were males and 57% were females).

The GPs were distributed across a range of services (i.e. 65% in HDB residential, 17% in private residential and 14% in companies or factories). The mean years practising as a doctor were 21 years for the GPs as against 9 years for the OPDs, while 12% of the GPs and 46% of the OPDs had had some psychiatric training.

Diagnostic 'accuracy' was assessed by comparing the rates of selecting the correct vignette diagnosis from the eight nominated options. The depression vignette was correctly identified by 99% of the GPs and 100% of the OPDs, the mania vignette by 50% and 72%, and the schizophrenia vignette by 92% and 100% respectively. Incorrect diagnostic 'spillage' varied. For depression, 1% of the GPs chose the 'stress' diagnosis. For mania, incorrect diagnostic options chosen by the GPs were 'schizophrenia' (46%), 'depression' (4%), and by the OPDs, 'schizophrenia' (17%) and 6% each for 'anxiety' and 'other'. For schizophrenia, the incorrect diagnostic options chosen by GPs were 'physical weakness' (5%) and 2% each for 'depression' and 'mania'.

Respondents were next required to nominate which single option would allow Mr A to be best helped, with the options listed as: 'talk things over with friends or family', 'Mr A must first recognise he has a problem', 'go to a polyclinic', 'go to a family doctor', 'go to a traditional healer (e.g. Bomoh, Temple medium, etc)', 'see a psychologist', 'see a church minister', 'see a counsellor to have counselling', 'take medication' and 'see a psychiatrist'. Respondents were also allowed to answer 'other (and to specify)' or 'don't know'.

For depression, 34% of the GPs nominated 'Mr A must first recognise he has a problem' and 'go to a family doctor' each as their 'best help' option, while 35% and 24% of the OPDs nominated those respective categories accordingly as their 'best help' option; 25% of the GPs and 24% of the OPDs nominated 'see a psychiatrist' as their 'best help' option; while 1% of the GPs and 10% of the OPDs rated 'talk things over with friends or family' as the 'best help' option.

For mania, 81% of the GPs and 89% of the OPDs rated 'see a psychiatrist' as their 'best help' option. The GPs also nominated 'go to a family doctor' (13%) and 'Mr A must first recognise he has a problem' (6%) as 'best help' options. The residual 'best help' options for the OPDs were 'to talk things over with friends or family' and 'other' (6% each).

For schizophrenia, 83% of the GPs and 92% of the OPDs nominated 'see a psychiatrist' as their 'best help' option. The residual 'best help' options by the GPs were 7% for 'Mr A must first recognise he has a problem' and 11% for 'go to a family doctor'. The residual 'best help' options chosen by the OPDs were that 'Mr A must first recognise he has a problem' or 'go to a polyclinic' (4% each).

Next, respondents were required to rate the extent to which a range of resources, medication and activities/therapies might be helpful or harmful. Aggregated (i.e. all doctors) percentage 'helpfulness' rates allow comparisons of the listed options and the responses of all primary physician respondents to the three disorders to be made. For example, a psychiatrist was rated as likely to be helpful (96%, 99%, 99%) for depression, mania and schizophrenia respectively. Similarly, seeing a traditional healer for the three disorders was viewed as unlikely to be helpful (2%, 2% and 1% respectively).

The coding options also allowed each intervention to be scored as '3' if rated as 'helpful', '2' if rated as 'neither' or 'depends', and '1' if rated as 'harmful', while any 'not known' response option was scored as missing data. Thus, a mean rating in excess of 2.0 would argue for a treatment being

viewed as generally likely to be 'helpful' (where 3.0 would be the ceiling rating), while a mean rating of less than 2.0 would argue for a treatment being viewed as generally likely to be 'harmful' (with 1.0 representing the extreme expression of harm). Group differences were examined by use of the Mann-Whitney U-test.

Table 1. Ratings by all doctors of judged helpfulness (assessed as percentages) of differing resources and medications for those with depression, mania and schizophrenia, and comparisons of mean scores generated by GPs and OPDs.

Resources and medication	All doctors (% helpful)			Depression		Mania		Schizophrenia	
	Dep	Mania	Schiz	GP	OPD	GP	OPD	GP	OPD
RESOURCES									
An A&E doctor	91	86	82	2.92	2.90	2.84	2.89	2.82	2.83
A polyclinic doctor	71	75	81	2.72	2.66	2.73	2.83	2.77	2.88
A general practitioner	97	91	92	2.97	2.97	2.90	2.94	2.92	2.92
A pharmacist	10	4	6	1.86	1.96	1.71	2.11**	1.84	2.13**
A Chinese physician	8	2	1	1.49	1.74	1.29	1.33	1.49	1.46
A counselor	75	47	48	2.68	2.86	2.38	2.50	2.43	2.58
A social worker	51	28	43	2.40	2.66*	2.14	2.44*	2.35	2.58
A counseling service	72	36	35	2.65	2.83	2.20	2.53	2.29	2.42
A traditional healer	2	2	1	1.25	1.39	1.18	1.12	1.26	1.13
A psychiatrist	96	99	99	2.96	2.97	2.98	3.00	2.98	3.00
A psychologist	79	52	54	2.80	2.72	2.47	2.56	2.56	2.50
Close family members	67	55	57	2.65	2.69	2.43	2.78*	2.47	2.79*
Close friends	71	49	48	2.67	2.76	2.33	2.78**	2.39	2.63
A Chinese or Malay medicine shop dispenser	2	0	1	1.31	1.43	1.16	1.33	1.27	1.21
A religious leader	52	21	29	2.33	2.69**	1.96	2.18	2.09	2.25
A specialist doctor	8	0	0	1.53	1.90**	1.10	1.17	1.22	1.26
MEDICATION									
Vitamins & minerals	21	2	4	2.21	2.14	1.98	2.00	2.00	2.00
Tonics or herbal medicines	11	0	0	2.04	2.07	1.82	1.89	1.90	1.91
A purging medicine	0	0	0	1.14	1.31	1.16	1.22	1.32	1.25
Antidepressants	94	34	35	2.97	2.83*	2.16	1.78	2.20	2.17
Antibiotics	1	0	0	1.31	1.59**	1.31	1.50	1.48	1.58
Mood stabilizers	37	77	44	2.28	2.07	2.73	2.67	2.36	2.17
Sleeping pills	40	50	40	2.06	2.14	2.35	2.47	2.30	2.05
Anti-psychotic or major tranquilizers	20	90	92	1.82	1.74	2.88	2.89	2.86	3.00
A benzodiazepine	46	55	58	2.33	2.11	2.50	2.41	2.53	2.54

*p<0.05, **p<0.01, ***p<0.001 GP = Private General Practitioner, OPD = Government Polyclinic Doctor

Table 2. Ratings by all doctors of judged helpfulness (assessed as percentages) of differing activities and therapies for those with depression, mania and schizophrenia, and comparisons of mean scores generated by GPs and OPDs.

Activities and therapies	All doctors (% helpful)			Depression		Mania		Schizophrenia	
	Dep	Mania	Schiz	GP	OPD	GP	OPD	GP	OPD
ACTIVITIES									
Becoming physically more active	78	30	45	2.77	2.79	2.15	2.24	2.47	2.38
Reading about people with similar problems	80	46	51	2.77	2.86	2.34	2.56	2.50	2.35
Being kept at home	0	0	0	1.04	1.07	1.06	1.17	1.12	1.13
Getting out and about more	70	29	39	2.68	2.69	2.04	2.22	2.41	2.29
Attending courses on relaxation	83	46	40	2.86	2.68	2.48	2.28	2.37	2.33
Cutting out alcohol	62	68	51	2.59	2.59	2.68	2.50	2.44	2.63
Taking a holiday	76	34	17	2.71	2.86	2.25	2.35	2.12	2.17
Having a rest	60	34	18	2.47	2.76*	2.31	2.39	2.15	2.21
Occasional alcoholic drink to relax	14	5	1	1.79	1.86	1.43	1.53	1.68	1.46
A special diet	4	9	3	1.82	1.73	1.91	1.94	1.93	1.91
THERAPIES									
Treatment from a traditional healer	3	0	1	1.35	1.54	1.22	1.33	1.34	1.42
Psychotherapy	75	64	74	2.79	2.66	2.69	2.50	2.75	2.67
Hypnosis	32	27	23	2.22	2.05	2.16	2.13	2.22	2.14
Admitted to a psychiatric ward of a general hospital	31	71	86	1.99	2.07	2.71	2.44	2.83	2.92
Admitted to a psychiatric hospital	29	77	86	1.96	1.96	2.66	2.72	2.83	2.92
ECT	19	41	41	1.68	1.96	2.34	1.94	2.25	2.42
Acupuncture	5	2	3	1.85	1.90	1.95	1.80	1.94	1.90

*p<0.05, **p<0.01, ***p<0.001

GP = Private General Practitioner, OPD = Government Polyclinic Doctor

First, we consider ratings by the combined sub-groups of primary physicians. For the three vignettes, most respondents rated psychiatrists, private general practitioners, A&E and government polyclinic doctors as likely to be helpful. This was followed by other professionals (e.g. psychologists, counsellors) as well as family and friends. Pharmacists, traditional healers and specialist doctors were seldom rated as likely to be helpful.

As for the utility of drugs, the doctors as a group rated antidepressants as likely to be helpful for depression only and the use of antipsychotic medication as likely to be helpful for mania and schizophrenia. 77% of the respondents rated mood stabilizers to be helpful in mania whilst 37% and 44% rated the drugs as likely to be helpful in depression and schizophrenia respectively. As a whole, between 40 to 58% of the doctors also viewed sleeping pills and benzodiazepines as likely to be helpful in the three disorders, and with rates seemingly uninfluenced by the diagnosis. Vitamins, minerals, tonics, herbal medicines and purging medicine were seldom rated as likely to be helpful.

In terms of activities and therapies, the primary physicians were unlikely to rate psychiatric admission or electroconvulsive therapy (ECT) as helpful for depression. Conversely, relaxation therapy, self-help books, being physically active, having a holiday, psychotherapy, and ceasing alcohol were regarded as helpful strategies for depression. Psychiatric admission was rated by most as helpful for mania and schizophrenia, and for these conditions, activities such as ceasing alcohol, psychotherapy, relaxation therapy and self-help books were also rated as likely to be helpful by some. For all three conditions, treatment from a traditional healer and treatment such as acupuncture and special diets were seldom rated as likely to be helpful.

Next, we compared mean helpfulness generated by each group. The OPDs (compared to the GPs) rated pharmacists and close family members as likely to be helpful (significant for mania and schizophrenia) and social workers as likely to be helpful (significant for depression and mania). Religious leaders and specialist doctors were also rated by OPDs (when compared to the GPs) as likely to be helpful for depression (statistically significant) and close friends as a helpful resource for mania (also statistically significant). For medications, the GPs were more likely to rate antidepressants as helpful for depression. Few differences were suggested for activities and therapies.

Discussion

The total response rate of 38% is low, but perhaps higher than might be expected for primary physicians in view of their busy work commitment. The higher response rate from the government polyclinics is likely to reflect the direct involvement of the Director of the Family Health Service who oversees the OPD doctors. The GPs were older and were less likely to have received prior psychiatric training. Currently, OPD doctors can choose to do a six-month elective posting in Psychiatry during their three years training-ship in pursuing their Master of Medicine (Family Medicine) degree and this is likely to have contributed to this difference in the younger OPDs.

Among the GPs, diagnostic accuracy was highest for depression (99%) and schizophrenia (92%) and low for mania (50%). This trend was also seen in the OPD cohort: depression (100%), schizophrenia (100%) and then mania (72%). Specifically, mania was misdiagnosed by 46% of the GPs and 17% of the OPDs as schizophrenia. These rates are of some importance as the long-term management strategies for schizophrenia and mania are quite different, while the outcome is also generally quite different.

In terms of resources and medications, the whole group of physicians rated the helpfulness of psychotropic medications in broad line with current psychiatric theories, although the relatively high rates of benzodiazepines and sleeping pills are of concern when risks of dependency on these drugs are substantial. As the respondents were essentially 'western trained' doctors, it was not surprising to notice the low preference for traditional healers and for Chinese or Malay medicine shop dispensers, and for alternate medicines. One could only conclude that

the pharmacist was not highly rated because of the culture in Singapore whereby almost all patients would have consulted a doctor before getting their medication from the pharmacy.

High helpfulness ratings for counsellors, family members and friends, relaxation therapies, psychotherapy, physical activities and self-help books indicated a pluralistic approach to factors that might assist those with such disorders.

When the GPs and OPDs were directly compared on these variables, few significant differences were identified, and it would be difficult to draw any conclusion about the GPs and OPDs being likely to differ in clearly detectable directions over the management of such disorders.

This survey provides important information about the capacity of the primary care doctors in Singapore to make an accurate diagnosis of psychiatric disorders. It also allowed us to gain insight into their judgement about the comparative utility of a range of treatments, resources, medication and activities. Continued medical education will play an important role in updating them on the advances in psychiatric practices, and data from such literacy studies serves to highlight their current knowledge base and topic areas for focussed consideration.

Acknowledgements

We thank all the private general practitioners and polyclinic doctors who participated in the survey. We also like to thank Janssen-Cilag for the providing the list of the private general practitioners.

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The Singapore Family Physician

Allopurinol Hypersensitivity Syndrome: A Case Report

Tay YK, Ling YK

Abstract

A favourable outcome was seen in a middle-aged Chinese male who experienced a major reaction from allopurinol. This was apparently prescribed because of mild hyperuricaemia and the presence of heel pain. The patient suffered from a generalised maculopapular rash and hepatitis requiring hospitalisation. He responded to moderately high doses of systemic steroids. Fatal cases of allopurinol-induced hypersensitivity syndrome have been reported. The purpose of this report is to emphasize the features of the allopurinol hypersensitivity syndrome and that allopurinol should only be prescribed for legitimate indications.

Case Report

A 35-year-old Chinese male had a history of hypertension on dietary control. He presented with mild right heel pain and mild hyperuricaemia (uric acid of 8.6 mg/dL) to a private practitioner who prescribed allopurinol 300 mg daily.

Two weeks later, he developed an insidious onset of a patchy red rash over the face, upper chest and back. Over the subsequent two weeks, the rash became progressively worse cumulating in facial swelling and a generalised rash with mild pruritus. Intermittent fever was also experienced. There were no oral or genital ulcers and no breathlessness. The initial impression was measles and he was treated symptomatically with antihistamines and non-steroidal anti-inflammatory drugs (NSAIDs). Allopurinol was stopped voluntarily by the patient with the onset of the rash.

He was referred to the National Skin Centre for further assessment. Physical examination revealed a generalised rash over the trunk and limbs (*Figure 1*).The rash was maculopapular in nature with some targetoid-like lesions. Purpuric stains were seen in areas of minimal trauma (suction leads of electrocardiography) and on the legs (*Figures 2 and 3*). The face was swollen and mildly scaly.

Figure 1.
Confluent rashes over the trunk giving an erythematous flush similar to measles

Figure 2.
Maculopapular rash with areas of purpura over the legs

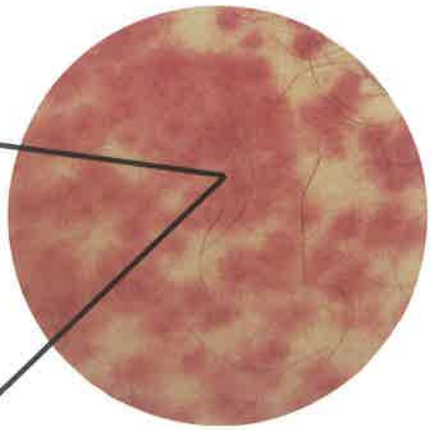

Figure 3.
Close-up of the targetoid-like rash

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He was mildly jaundiced (peak serum bilirubin of 99 µmol/L) and there was biochemical evidence of liver transaminitis (ALP: 229 U/L, AST 320 U/L, ALT 1180 U/L) even though the liver was barely palpable and non-tender. There was no serological evidence of viral hepatitis A, B and C. Ultrasound examination was consistent with fatty liver.

He was treated with 60 mg/day of prednisolone (about 1 mg/kg/day) and this was gradually tailed off over a period of 8 weeks. He made an uneventful recovery.

Discussion

Allopurinol hypersensitivity syndrome is an infrequent adverse reaction to allopurinol with significant morbidity and mortality, requiring prolonged hospitalisation in some cases. While a severe adverse reaction was found in one out of 260 hospitalised patients (0.4%) in one study,[4] the exact incidence of allopurinol hypersensitivity syndrome is unknown.

In contrast to other hypersenitivity reactions, the onset of allopurinol hypersensitivity is highly variable and may be delayed for several months. On average, signs appear one and a half months (47±109 days) after the initiation of allopurinol therapy.[8] Pruritus may forewarn the onset of the syndrome. The cutaneous signs include maculopapular rash and exfoliative dermatitis, and less commonly, Steven-Johnson syndrome and toxic epidermal necrolysis.[5] Often, fever and leukocytosis with eosinophilia[2,5] are present. Abnormal liver function tests are encountered in most cases, despite the absence of hepatomegaly. Complications such as respiratory distress, renal failure and disseminated intravascular coagulation may be fatal.

While there is no way to predict the likelihood of developing hypersensitivity reactions, patients with renal insufficiency or hypertension, especially those on thiazide diuretics, appears to be more at risk.[1] The mortality rate can be as high as 30% in the presence of elevated AST.[8]

While there is currently no satisfactory treatment for allopurinal hypersensitivity syndrome, prolonged use of moderately high dose of systemic steroids may assist in its control.[3] Systemic steroids need to be given for a fairly long period of time; too fast withdrawal of steroids will lead to a rebound with reappearance of symptoms and signs of the syndrome. Early recognition, withdrawal of allopurinol and appropriate supportive therapy are important.

Asymptomatic hyperuricaemia is not an indication for starting therapy to lower serum uric acid. [7] In fact, early treatment of asymptomatic hyperuricaemia to reduce the risk of renal damage, stones or gouty arthritis is unproven and unjustified. However, it is disturbing to realise tht asymptomatic hyperuricaemia is the most frequent indication for allopurinol treatment.[5,6]

Table1: Indications for use of allopurinol[7]

Visible tophi
Major uric acid overproduction as in secondary gout
Frequent gouty attacks unresponsive to prophylactic colchicine or NSAIDs
Recurrent uric acid renal calculi
Recurrent calcium oxalate renal calculi with hyperuricosuria

Table 2: Unjustified indications for use of allopurinol[7]

Asymptomatic hyperuricaemia
Acute attacks of gout
Non-tophaceous gout

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Allopurinol is an effective and valuable drug. However, due to its potential adverse effects, it should only be administered for accepted indications with the appropriate dosage adjustment according to the patient's renal function. This may be the only way of minimizing the incidence of allopurinol hypersensitivity syndrome.

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NSAIDs Induced Gastropathy and its Practical Management

Cheong WK

Introduction

The side effects of non-steroidal anti-inflammatory drugs (NSAIDS) are not restricted to the stomach and duodenum. NSAIDS can also adversely affect the kidney[1], liver[2], cardiovascular and haematological system in addition to the entire gastrointestinal (GI) tract[3,4,5,6] from the esophagus to the colon. Ulcers, haemorrhage, perforation and strictures of the small intestines and colon, various forms of colitis (eosinophilic, collagenous, pseudomembraneous) and small bowel enteropathy characterised by malabsorption and chronic diarrhoea have been reported.

NSAIDs Gastropathy[7]

This term encompasses a spectrum of acute and chronic lesions of the stomach and duodenum associated with NSAIDs. Within 90 minutes of aspirin ingestion, gastric intramucosal petechiae and hemorrhage are evident, to be followed by gastroduodenal erosions. These lesions are of uncertain clinical relevance as spontaneous resolution may occur, despite continuation of aspirin, a phenomenon referred to as adaptation. The mechanism of, and the factors promoting adaptation are poorly understood. Failure of adaptation leads to the development of gastroduodenal ulcers. NSAIDs can also exacerbate pre-existing peptic ulcer disease and retard ulcer healing despite effective acid suppressive medication.

Incidence and magnitude of problem

The ARMIS registry[8] prospectively captures data relating to outcomes, drug side effects and economic impact of rheumatoid arthritis and osteoarthritis from 17 participating US and Canadian based centres. The current databank is estimated to comprise more than 36 000 patients and more than 300 000 patient years of follow up. Information from this databank indicates the following:

- 15% of NSAIDs users develop dyspesia
- 40%-50% develop duodenal or gastric ulcers sometime in the clinical course
- 1-4% develop clinically significant complications per year from these ulcers
- 92.5% of admissions in rheumatoid arthritis patients are for NSAIDs related complications
- there is a 0.22% annual death rate for GI complications related to NSAIDs use

NSAIDs increases the risk of developing serious GI side effects by 3-5 times and this risk increases with increasing age. Patients older that 60 years have 13 times increased risk compared with 3 times for those younger than 60 years[9,10].

Are symptoms useful indicators of NSAIDS gastropathy?

Dyspeptic symptoms are commonly experienced while taking NSAIDs but are very poor predictors of actual gastroduodenal damage. 50% of symptomatic patients will endoscopically not demonstrate any lesions. Conversely, 40% of those endoscopically documented to have erosive gastritis are asymptomatic. In fact, 42-81% of NSAIDs users develop complications (bleeding, perforation) without prior warning symptoms[11].

Are there identifiable risk factors to the development of NSAIDs related GI complications?

Certain factors, particularly if cumulative, will identify a candidate as high risk. They include[12]:

- history of previous peptic ulcer disease with or without complication (risk increases 13 times)
- concomitant use of anticoagulants or steroids (risk increases 6-10 times)
- >60 years of age (risk increases 3-4 times)
- the dosage NSAIDS used. High dose increases risk by 3 times over low doses

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It is important to appreciate the fact that advanced age and concomitant medical co-morbidities will compound the impact of a NSAIDS related GI complication as this category of patients poorly tolerate anaemia, shock, anaesthesia and surgery. Gender, alcohol intake and smoking have no influence on the risk profile. The risk does not progressively diminish in patients on long term NSAID usage.

The fundamental principals to practical management

- The most important and fundamental question is whether NSAIDs are indicated in the first place. NSAIDs have antipyretic, analgesia and anti-inflammatory activities. NSAIDs should not be used primarily as antipyretics and analgesics unless these are secondary to an inflammatory condition.

- If NSAIDs are deemed unavoidable, they should be used at the lowest possible dose. The safest, most economically acceptable drug should be chosen.

Doses may be minimised by the use of alternative / synergistic therapy to achieve adequate analgesia[13,14,15,16](Table 1).

Pharmacologic
- narcotics
- calcitonin for osteoporotic pains
- colchicine for gout
- DMARDS for rheumatoid arthritis

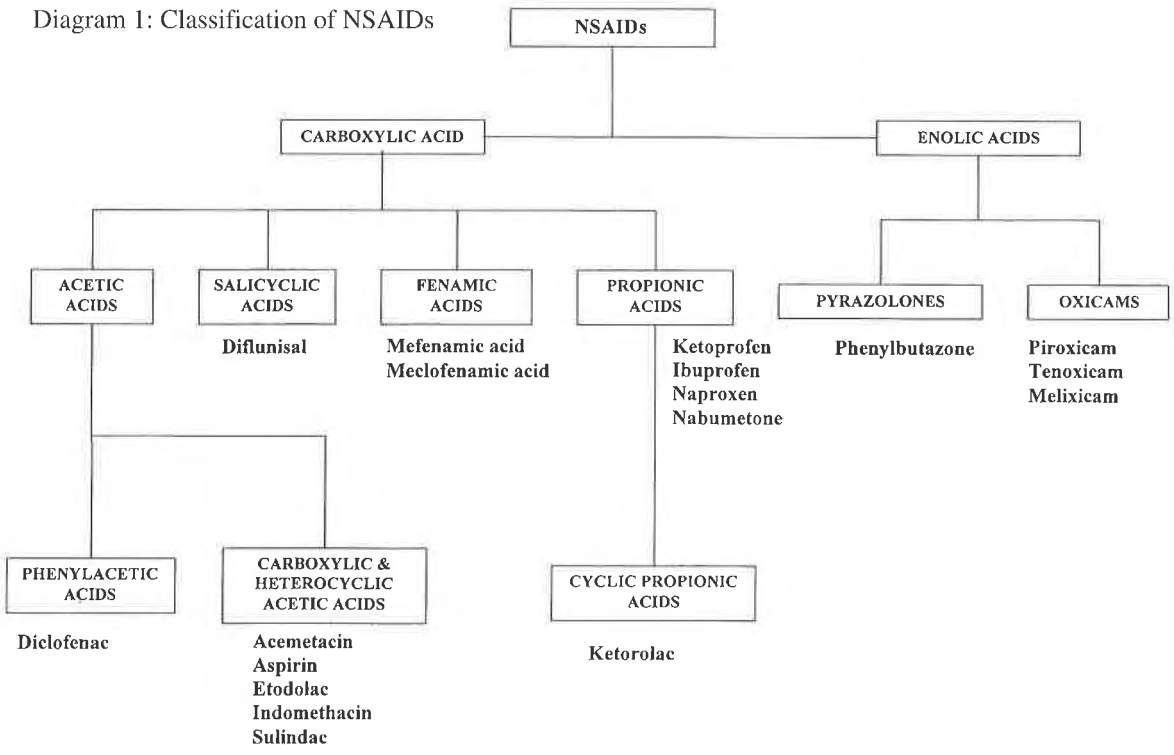
Non pharmacological
- physiotherapy
- acupuncture

Psychosocial therapy
- antidepressants
- counseling

Table 1: Alternative/Synergistic Methods of Analgesia

A large number of chemical compounds come under the classification of NSAIDs (Diagram 1) and the safety profile of each compound differs significantly (Table 2)[17,18]. The safety profile is not determined by chemical class but is instead dependant on (1) drug pharmacokinetics and (2) pattern of COX receptor inhibition[19,20]. Enteric coating offers no protection.

Diagram 1: Classification of NSAIDs



Drug	Garcia Rodriguez	Langman et al
Ibuprofen (<1500mg)	2.9	2.0
Diclofenac	3.9	4.2
Naproxen	3.1	9.1
Ketoprofen	5.4	23.7
Indomethacin	6.3	11.3
Piroxicam	18	13.7
Azapropazone	23.4	31.5

Table 2: Relative Risks of Gastrointestinal Bleeding on Different NSAIDS

Favourable characteristics are specific or preferential COX II inhibition, absence of enterohepatic recirculation, pro-drug and short half life.

- NSAIDs associated dyspeptic symptoms often resolve spontaneously and respond to simple measures like antacids and mild acid suppressive therapy. Therefore, in low risk patients receiving short term NSAIDs treatment, further investigation is not usually indicated. However, in higher risk patients requiring long term, high dose therapy, endoscopy or barium studies should be performed to assess if there is significant pathology which may necessitate a change in therapeutic strategy. Empirical treatment in this high risk category of patients will mask the symptoms of underlying gastroduodenal ulceration, lull one into a false sense of security, place the patient at risk of developing life threatening complications and lose valuable baseline information that is critical to the effective long term management of the patient.

- If an ulcer is diagnosed while the patients is on NSAIDs, the drug should, where possible, be temporarily terminated and the ulcer healed before reintroduction, if at all. If indicated, alternative interim therapy should be instituted. Duodenal and gastric ulcers heal at different rates. Healing rate is also ulcer size dependant and varies according to the drugs used to treat the ulcer. In the best scenario, using a proton pump at adequate doses, duodenal ulcers may be expected to heal in 2-4 weeks, while gastric ulcers should heal by 4-8 weeks.

- When NSAIDs are initiated for the first time to high risk patients or when it is reintroduced to patients who have developed ulcers or its complications while on NSAIDs, further measures must be taken to reduce the risk of subsequent complications. Concomitant use of a gastroprotective drug should be considered.

  What are these gastroprotective drugs[21]? Antacids and sucralfate do not offer protection. H2 antagonists offer protection against duodenal ulcers. Only Famotidine at 40mg BD has been shown to offer additional protection against gastric ulcers. Proton pump inhibitors offer protection against both duodenal and gastric ulcer and are also the only drugs which can achieve ulcer healing in the presence of ongoing NSAIDs treatment albeit at a slower healing rate. Prostaglandin E1 analogues offer effective protection against gastric ulcers but very commonly causes diarrhoea, a dose dependant and problematic pharmacological effect of the drug. While gastroprotective drugs significantly reduce the risk of developing ulcers on NSAIDs, the protection is not absolute. The efficacy of protection is dose dependant so proper dose selection is important.

Role of Helicobacter pylori[22]

Helicobacter pylori (Hp) is an independent and non synergistic cause of peptic ulcer disease. There is, therefore, no justification to check for Hp status with the aim to eradicate it prior to instituting NSAIDs in unselected patients. However, as it is a known cause of recurrent gastroduodenal ulcers, patients with a past history of Hp associated peptic ulcer disease should have Hp eradication prior to instituting NSAIDs. If found to be Hp positive for the first time in the course of evaluating for NSAID gastropathy, it is also considered good clinical practice to eradicate it.

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Cost benefit considerations

NSAIDs are one of the most commonly prescribed drugs worldwide. Indiscriminate first line use of the newer generation, improved safety profile NSAIDs will markedly increase health cost. If, as an added measure of safety, gastroprotective drugs are concomitantly administered, the overall cost of treating a patient will further escalate. However, one needs to consider the cost of treating a patient with NSAIDs complication (hospitalisation, ICU care, blood transfusion, therapeutic endoscopy, surgery, post operative care and recuperation and loss of work hours). The medico-economic equation is therefore more complex than a simple drug for drug cost comparison. The key to effective use of the newer generation NSAIDs and gastroprotective drugs is accurate patient risk stratification. Unfortunately, we are as yet unable to perform this predictably. While we can clearly identify patients belonging to the lowest and highest of risk groups, the majority fall into a mid strata and often cannot be identified until a complication has intervened.

The check list when considering NSAID therapy

1. Is there a valid indication to starting NSAIDs or are there alternative therapeutic modalities?
2. Are there features to indicate high risk patient profile?
3. Do I need to check for Helicobacter pylori and pre-existing ulcers before starting NSAIDs?
4. Which NSAID should I start and is there justification for a more expensive safer profile NSAID and concomitant use of gastroprotective drugs?
5. If symptoms develop, has the patient developed an ulcer and do I need to investigate?
6. If an ulcer is diagnosed, can I terminate NSAIDs for the time being and heal the ulcer first?
7. What other medication can I substitute in place of the NSAIDs if it is a critical medication?
8. When can I restart NSAIDs, if ever?

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Bone Health in the Menopause

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The commonest bone disease in the menopausal years is osteoporosis. Osteoporosis is a chronic, progressive and systemic bone disease with both a quantitative and qualitative component. The quantitative part is the reduced bone mass while the qualitative part is deterioration of the microarchitectural anatomy of bone. Both of these contribute to bone fragility and an increased susceptibility to bone fractures. It is traditional to link hip fractures, vertebral fractures and wrist fractures with osteoporosis but almost all types of fractures in the elderly are due in part to osteoporosis.

Why and How Does Osteoporosis Develop During the Menopausal Years?

Bone is an active tissue and throughout a person's life, the process of bone formation and resorption occurs all the time but the rate varies with one process being more active than the other.

1. A young girl builds much more bone than she loses.
2. From the teen years to the mid 30s, there is a slight edge towards build-up of bone. Peak lifetime bone mass is achieved at about age 35. After this age, women start to lose more bone than in forming it - the net loss is about 1% of bone mass per year.
3. With the onset of menopause there is a sudden depletion of estrogen and this leads to about 3-5% of bone loss annually for a period of 5 to 10 years.
4. By the age of 60 years, a woman can lose more than 25% of her bone mass.
5. After the age of 60, bone loss shows a little in women but continues till the end of life.
6. By the age of 70, some women have 50% of the bone mass of women of 35 years of age.

It is therefore important to remember that menopause is a critical turning point for a woman's bones. Without the protective effects of estrogen, bone mass is lost rapidly after menopause. Estrogen affects bone mass in 2 ways :-

- Directly through estrogen receptors in bone by reducing bone turnover and bone loss.
- Indirectly by increasing intestinal absorption of calcium and by renal calcium conservation.

Why Should We Worry About Bone Health in Menopouse i.e. Why Should We Worry About Osteoporosis?

The first reason is that the "osteoporosis crisis" is big and getting bigger. The realization of the enormity of the problem prompted a full report on osteoporosis to be tabled in the European Parliament in June 1998.

Statistics confirm that the problem of osteoporosis is equally pressing in our part of the world. In Singapore, only 6% of the population was over 65 in 1990 (200,000 people) but by 2030, the proportion will have risen to 17% (800,000 people). The majority of these older people will be women. In Japan 25% of women over the age of 50 are afflicted with osteoporosis. In Hong Kong, the prevalence of vertebral fractures in elderly women was found to be 30%.

The second reason for worrying about osteoporosis is that it is a silent epidemic. It usually progresses without obvious outward signs - sometimes for decades - until the sufferer experiences a fracture. This silent epidemic is a public health problem with far reaching medical, economic and social implications.

The following table shows the number of hip fractures in Singapore in 1997

Hip Fractures in Singapore (1997)

60-69 yrs	Number of cases	263
	Female	169
	Male	94
> 70 yrs	Number of cases	1428
	Female	1057
	Male	371
Total number of cases		1691
Female		1226 (> 72%)
Male		465 (< 27%)

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A study carried out in Alexandra Hospital in 1995 to assess the outcome of 280 hip fractures in Singapore showed that

- 26% of patients died within one year of having a hip fracture
- of the survivors, 9% became bed-ridden and 24% became wheelchair bound

In this study, the cost of treating one hip fracture in a restructured hospital was estimated to be about $7000. Therefore, the cost of treating 1691 cases of hip fracture that occurred in 1997 would have amounted to at least $12 million. This is expected to double in the next 20 years.

Although hip fractures are the most serious fractures caused by osteoporosis, the commonest osteoporotic fractures that occur are vertebral fractures and many of these do not come to the attention of physicians. Vertebral fractures can cause significant complications including back pain, height loss and kyphosis. Postural and height changes caused by kyphosis may limit activity including bending and reaching and their cosmetic effects may erode self-esteem.

Multiple thoracic fractures may result in restrictive lung disease. Lumbar fractures may alter abdominal anatomy leading to constipation, abdominal distension, reduced appetite and premature satiety.

Hip and vertebral fractures can also cause psychological problems especially depression as patients grapple with pain, physical limitation and lifestyle changes. The high morbidity and consequent dependency associated with these fractures can strain interpersonal relationships and social roles for patients and their families.

Another important fact to keep in mind is that the presence of one vertebral fracture is an indication of a 3 to 5 fold higher risk of getting another vertebral fracture in the next 5 years regardless of the level of BMD (bone mineral density). The presence of a wrist fracture doubles the risk of getting a hip fracture and triples the risk of getting a vertebral fracture.

Thus, the presentation of one osteoporotic fracture should alert the clinician that it is important to treat not only the fracture but the underlying condition of osteoporosis as well.

Diagnosis of Osteoporosis

We should consider the possibility of osteoporosis and fracture risk in all post-menopausal women, based on the presence of risk factors as enumerated in Table II.

Table II

Risk Factors For Osteoporotic Fractures

Risk Factors

Non-modifable	Modifable
* Early natural or surgical menopause before age 45 years	* Corticosteroid use (equivalent to Prednisolone >7.5 mg/day for more than 6 months)
* History of previous osteoporotic fracture	* Other drugs e.g. sedatives, anticonvulsants
* Family history (especially maternal hip fracture)	* Endocrine causes (e.g. hypogonadism, hyperparathyroidism, hyperthyroidism)
* Slender body build	* Other illness, e.g. liver disease, malabsorption, rheumatoid arthritis
* Asian or Caucasian	* Frequent falls
	* Low calcium intake
	* Immobility/sedentary lifestyle
	* Chronic tobacco use

BMD Testing

This is recommended for :-

- postmenopausal women under age 65 who have one or more additional risk factors
- all women aged 65 and older regardless of additional risk factors
- peri- and postmenopausal women who present with fractures.

We have to keep in mind that BMD only measures the bone mass (the quantitative aspect) and therefore does not tell us about the qualitative aspect of bone but BMD testing can be used as a guide to initiate treatment and monitor the progress of the condition.

Initiation of Treatment

The guidelines are summarized in Table III

Table III

Initiation of Treatment

* Advise all patients to have adequate calcium and vitamin D intake
* Recommend regular weight bearing exercise
* Suggest initiating therapy to reduce fracture risk in women with BMD T scores below – 2 in the absence of risk factors and in women with Tscores below - 1.5 if other risk factors are present
* Consider all women who present with vertebral, hip or other osteoporotic fractures as candidates for osteoporosis treatment.

Drug Therapy

1. **Hormone replacement therapy**
   Epidemiological studies of HRT indicate a 50 - 80% decrease in vertebral fractures and a 25% decrease in non-vertebral fractures with 5 years of use and an anticipated 50 to 75% decrease in all fractures with 10 or more years of use.

2. **Bisphosphanates** - randomized double blind controlled studies using alendronate indicate a 50% reduction in fractures of the vertebrae, hip and wrist. This medication is therefore a powerful option for those who are unwilling or unable to take HRT and yet meet the BMD criteria for treatment.

3. **SERMS** - (Selective Estrogen Receptor Modulators) - the best example of this class of drugs is raloxifene. This is another alternative treatment for osteoporosis. It reduces the risk of vertebral fractures by 40-50%.

4. **Calcitonin** - polypeptide hormone that inhibits bone resorption and also has an analgesic effect. It can be given intranasally and is especially useful in elderly women with vertebral fractures because of its analgesic effect

5. **Vitamin D derivatives** e.g. Calcitriol - this promotes calcium absorption but serum calcium levels have to be monitored when this drug is administered.

Based on the effectiveness for the treatment of osteoporosis and its other potential benefits for post-menopausal health (i.e. cardio-protection), HRT provides the greatest benefit relative to cost. However, this has to be weighed against the fact that it increases the relative risk of breast cancer by 2.3% per year of use after 5 years.

If we keep in mind that one out of every two white post-menopausal women will experience an osteoporotic fracture at some point in her lifetime and that this may also apply to our local population, then it behoves us as clinicians to counsel post-menopausal women to consider HRT and offer guidance in weighing its risks and benefits. If the patient is unsuitable or unwilling to take HRT, then other treatment options can be offered to the patient. The treatment has to be individualized for each patient.

Conclusions

- Osteoporosis is a common and expensive Disease
- Bone densitometry and potentially, bone markets, are important in the management of women with or at risk of osteoporosis
- HRT is a mainstay of osteoporosis prevention and treatment
- HRT should begin as soon as possible after menopause
- Preliminary data suggest tht elderly should also respond to HRT
- If patient is unwilling or unsuitable for HRT, then to try other drugs for preventing and treating osteoporosis

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Common Foot Disorders in Children

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Foot problems are common referrals to the Paediatric Orthopaedic Clinic. Most of them are benign and require reassurance of the parents. Club foot, calcaneovalgus foot and congential vertical talus are rare disorders which require management by an orthopaedist.

Between 10 and 18 months of age most infants become independent walkers. If a child is not walking by the age of 18 months, suspect retardation or cerebral palsy.

Three common problems dominate the scene - angular deformities of the leg, torsional problems and flat feet.

1 Torsional Problems

The commonest one is in-toeing. When taking the patient's history pay attention to these particular points:

1 Advice of family and friends – many mothers are made anxious by grandmothers or friends pressing a once-used brace on her.

2 Effect of torsion on the child? Does it cause him to fall. Usually this is due to incoordination at first.

3 Is it getting better or worse?

4 Position child sits in? If he sits on his feet all the time, spontaneous improvement takes longer.

Physical Examination Usually Reveals the Cause of In-Toeing

Measure the degree of in-toeing by the gait angle when the child is walking. Lie the child prone to determine the site of in-toeing. Assess hip rotation - internal rotation is increased in internal femoral torsion. Look at the back to exclude a neurological cause and assess hip abduction to exclude a hip dislocation.

Examination usually reveals 3 causes:

1 Metatarsus varus: when the twist is in the foot. Commonest cause in child below 18 months. Treatment usually involves observation, simple manipulation or rarely reverse last shoes.

2 Internal tibial torsion. Commonest cause of in-toeing in age group 18 months to 3 years. The twist is below the knee. Most improve spontaneously. If not better by 3 years, Dennis Browne night splints are required for 3 - 4 months.

3 Internal femoral torsion. Commonest cause for those above 3 years. Twist is between knee and hip. Recommend correct sitting position viz. legs crossed. Braces are ineffective. If severe, osteotomy is required after age 10 years.

Out-toeing

Out-toeing is usually due to excessive external femoral torsion. In an older child suspect hypotonia from some neurological cause. If the examination is otherwise normal, warn the parents that walking is frequently delayed – Charlie Chaplin position does not make it easy to balance.

2 Angular Deformities

Ask the same questions as for torsional problems. Find out who is the person who is concerned and if the deformity is improving or worsening.

Rickets and Blount's disease are uncommon in Singapore. The normal pattern is for the child to have bow legs under 2 years of age and this usually straightens itself. Knock-knee or genu valgus is common between 2 and 7 years; the feet usually look flat. Most legs are straight by the teens.

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It is important to monitor the progress by measuring the intermalleolar distance for knock knees and intercondylar distance for bow legs.

In the absence of any disease of the bone these may be safely regarded as self-correcting normal variants. More than 90% of children with angular deformities are otherwise normal and can be expected to enter adult life with straight legs irrespective of treatment. Most require simple observation. If shoe wear is affected, a medial wedge for knock knees and lateral wedge for bow legs can be prescribed. The efficacy of this has not been proven.

If a severe deformity is present in late teenage life, an osteotomy should be undertaken to secure straightening of the deformity.

3 Flat Feet

This condition is very common in children when they begin to walk, particularly if they are fat and have loose joints.

Infants have flat feet. Flexible feet are common in all ages. Ask the child to stand on tip toes; and the arch appears indicating flexibility. This requires no treatment. Foot exercise and shoe therapy are not effective.

Pathological flat feet are seen in cerebral palsy and vertical talus.

4 The Adolescent Foot

Heel Pad Pain

Heel pad pain is a common complaint. Hard shoes, heavy landing and flat-feet are causative factors.

Treatment involves prescribing a heel pad or injection of corticosteroid.

Stiff Painful Foot of Tarsal Coalition

Pain over the outer side of the foot and stiffness of the foot should raise suspicion. The foot is stiff and inversion makes the peroneals stand out. X-rays or CT scan indicating the bridging of two bones in the hindfoot characterises the condition. When discovered early, between age of 10 and 14, the bar is excised. After this age, arthrodesis is more likely.

Over-Riding 5th Toe

Shoes are uncomfortable until surgical correction is undertaken.

Bunions and Hallux Valgus

Usually girls who wear narrow shoes experience pain over the bunion and request for surgery. This should be avoided in the adolescent. Wider shoes are the answer. Rarely, in marked hallux valgus, is an operation considered.

High Arches (Pes Cavus)

The high arch is accompanied by curly toes. Most of the weight is taken along the outer border of the foot. Walking and shoe fitting becomes difficult. The condition is due to weakness of small muscles in the foot causing overaction of the long tendons in the foot. Fifty per cent of cases are associated with a neurological cause. The ankle jerks are lost in Fredreich's ataxia and Charcot-Marie-Tooth disease. A tethered condition may be associated with a dimple in the back, unilateral cavus and X-ray showing widened interpredicular distance.

Treatment may involve correcting a potential remediable cause in the spine. If no cause is found, increasing deformity requires plantar release. Tendon transfers, toe straightening and even arthrodesis may be required eventually in progressive cases.

Most foot disorders in children require reassurance of the parents and simple observation. It is important to identify selected cases which will require referral to an orthopaedist.

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Office Endometrial Sampling: an Understanding of its Uses and Limitations

Fong Y

Introduction

In our daily clinical practice, we often encounter the need for histological assessment of endometrial tissue to rule out malignancy in women with irregular menstrual flow. The gold standard has been, and still is, endometrial tissue obtained from a dilatation and curettage (D&C). The sentiments in recent years has been that too many D&Cs are being performed for the myriad of menstrual complaints that women present with; on the other hand, the concern of occult endometrial malignancy presenting as menstrual irregularities cannot be ignored without some form of endometrial assessment in most of these women. Endometrial sampling serves to fill this gap in the diagnostic work-up[1] by allowing the clinician to obtain small but adequate samples of tissue for histology without the need for general anaesthesia or an invasive procedure like D&C.

Indications for Endometrial Sampling

The following are some of the common indications for endometrial sampling:

(1) Post-menopausal vaginal bleeding
(2) Abnormal uterine bleeding
(3) History of endometrial hyperplasia
(4) Uterine enlargement (non-gravid) associated with menstrual disturbance
(5) Prior to starting HRT
(6) Glandular cell atypia on Pap smears
(7) Risk factors for endometrial carcinoma
(8) To determine the effects of HRT/monitor the effects of oestrogen therapy
(9) Endometrial dating (for IVF patients)

Most of the women who fall into the first 7 categories listed above would have presented with some form of abnormal uterine bleeding. If the presenting picture is highly suspicious, for example significant post-menopausal bleeding (not spotting) on more than one occasion, intermenstrual bleeding for several consecutive months, irregular bleeding in those with a history of endometrial hyperplasia, it may be advisable for these women to undergo a hysteroscopy and D&C rather than endometrial sampling; the reason for this will be discussed later. Similarly, women with polycystic ovary disease, those who have a history of breast carcinoma or who have been on tamoxifen for several months, are also at higher risk of developing endometrial carcinoma, and may also benefit from a D&C instead of endometrial sampling alone.

Pap smears taken well before or after menses should show only squamous epithelium. The presence of glandular epithelial cells on such smears may suggest excessive shedding of cells from the endometrium. In these situations, it would be wise to examine the endometrial tissue to exclude hyperplasia or malignancy that may have given rise to these abnormal Pap smear results, especially if they are persistent.

In clinical trials involving the use of oestrogens (for example HRT products), endometrial samples are often obtained at various stages of the trial to assess the extent of endometrial stimulation as well as the incidence of hyperplasia, as part of the safety profile evaluation of these products.

Contraindications to Endometrial Sampling

Endometrial biopsies should not be undertaken when there is evidence of:

(1) Ongoing pregnancy
(2) Pelvic inflammatory disease
(3) Cervicitis

In addition, the usual antibiotic prophylaxis for bacterial endocarditis should be provided when performing endometrial sampling in women with valvular heart disease, as the vagina has a large number of commensal bacteria which can infect the endocardium.

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Other relative contraindications to endometrial sampling include an uncooperative patient (eg. tense, anxious patients), stenosis or distortion of the cervix (eg. after a cone biopsy of the cervix), or during menstruation (the yield would be mainly blood with very little endometrial tissue).

Performing an Endometrial Sampling

The following steps should be taken in sequence when performing an endometrial sampling:

(1) Obtain informed consent.
This would include a description of the steps the clinician would take during the procedure. In addition, the patient must understand that she may experience some mild pelvic discomfort or pain, usually similar to the mild menstrual cramps that she would experience on the first 1 - 2 days of menses. The risk of post-procedural endometritis must also be explained; this may present as lower abdominal cramps, fever, foul-smelling vaginal discharge, or even as uterine tenderness (commonly presenting as dyspareunia). Hence, it is common for clinicians to prescribe antibiotic prophylaxis following the procedure. Another commonly encountered problem is the failure to obtain adequate tissue for histological interpretation. This is especially common if the sampling is performed just after a menstrual flow, in post-menopausal women where the endometrial lining is generally thin, or if the vacuum suction produced in the sampling catheter was inadequate. Patients must be warned of this possible problem, and of the possible need to repeat the procedure or even a formal hysteroscopy, D&C should the abnormal bleeding persist.

(2) Pre-medication
Pre-medication with analgesics may be desired or even essential if the patient is known to have a low pain threshold, or is likely to experience significant pain. This may consist of oral non-steroidal anti-inflammatory drugs (NSAIDs) 30 to 60 minutes before the procedure. There is no need to make the patient fast overnight in preparation for an endometrial sampling.

(3) Pelvic Examination
A vaginal examination to take note of the size and position of the uterus, the presence of masses like fibroids, cervical abnormalities like stenosis, presence of cervicitis or vaginal infections, would be helpful in assessing the likelihood of a successful endometrial sampling.

(4) Endometrial sampling
After inserting a bivalve speculum, the external cervical os should be cleaned with a cotton swab containing some chlorhexidine or providone iodine. The uterine cavity is then measured using a uterine sound; this will help in assessing the depth to which the sampling catheter can be safely inserted without causing perforation. If the cervical os is tight, a tenaculum applied to the anterior cervical lip to give some counter-traction will be very helpful.

The sampling catheter is then inserted into the uterine cavity up to the measured length of the cavity. Depending on the type of catheter used, a vacuum is then created in the catheter; if a "Pipelle" type of catheter is used, then the inner stylet is withdrawn to create the vacuum.

The external catheter tubing is then rotated clockwise, and at the same time gradually withdrawn from the uterine cavity in a spiral manner. This ensures that tissue is obtained from different parts of the uterine cavity.

Once totally out of the uterus, the inner tubing is pushed back into the external catheter, thus expelling the sampled tissue into a container of formalin. The procedure can be repeated another 1 to 2 times if the amount of tissue sampled from the first attempt is inadequate.

The procedural steps are summarized in Figures 1 to 5[2].

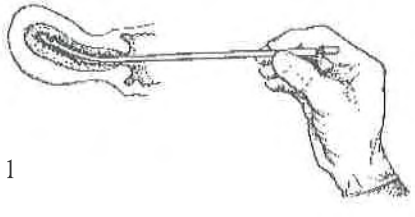

Figure 1

The Singapore Family Physician

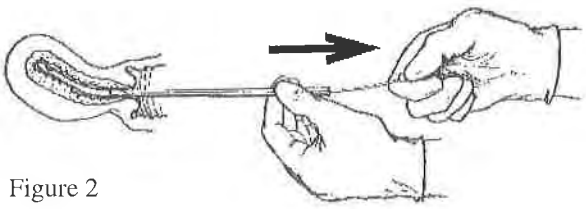

Figure 2

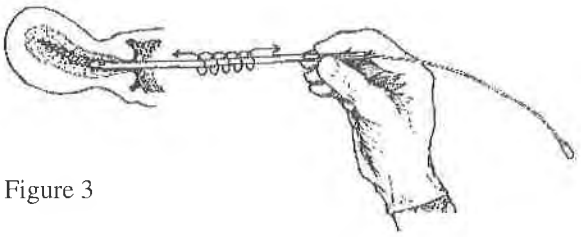

Figure 3

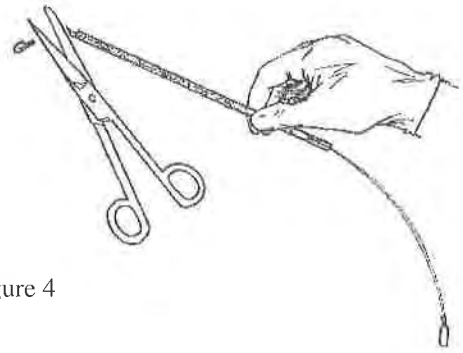

Figure 4

Figure 5

After the Procedure

Women are almost always able to return to their usual activity after an endometrial sampling procedure. Analgesia will usually consist of oral NSAIDs for 1 to 2 days, together with some antibiotic prophylaxis, for example doxycycline. Very rarely, they may require a day's rest if the cramps are bothersome, or if they are involved in strenuous activity. Any vaginal bleeding after the procedure is usually light, and will cease spontaneously after a day or two.

All women should be advised to look out for signs of complications, namely heavy bleeding per vaginum, persistent severe abdominal pain despite analgesics, foul-smelling vaginal discharge or fever. The presence of these signs would suggest lower genital tract infection or uterine perforation, and will necessitate intervention.

Practical Considerations

It is important to realize from the beginning that any endometrial sampling procedure must be preceded by an ultrasound scan of the uterus and pelvis. It has been shown that combined ultrasound scans and endometrial sampling gives a pickup rate for endometrial carcinoma of 60% to 80%[3,4]. In addition, ultrasound scans may also show the presence of endometrial polyps, fibroids or ovarian cysts which non-directed office biopsies without imaging would have potentially missed in almost 20% of cases[5]. Such scans should be done by an operator familiar with pelvic scans as the accurate measurement of the endometrial thickness is crucial, particularly in women with post-menopausal bleeding where an endometrium of 5mm or more is highly suspicious of malignancy.

There are several catheters available in the market for endometrial sampling purposes. The choice of catheters should be based on the clinician's familiarity with them, as well as other considerations like ease of use, availability and cost. There is no conclusive evidence to show that any one catheter is significantly superior to the others in terms of the rate of tissue retrieval[4,6], provided that each type of catheter is used correctly. In general, there is a high degree of overall sensitivity in detecting carcinomas provided adequate endometrial tissue has been obtained[7]. In our experience this may occasionally be difficult in menopausal women, as they tend to have atrophic endometrial lining and poor yield on samplings.

If the suspicion of malignancy is high, then the investigation of choice is still hysteroscopy, EUA and D&C. This procedure provides direct visualization of the entire uterine cavity, and allows for hysteroscopy-directed curettings to be obtained; the likelihood of sampling failure is also less with this method. A delay in the diagnosis of malignancy would be detrimental to the patient's well being, especially as endometrial carcinoma is a potentially curable condition in its early stages.

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The Singapore Family Physician

Managing Common Problems Faced by the Home Bound Elderly

Lee KS

The home bound patient often has multiple medical problems with associated disabilities and social issues. As a family physician providing care, you need to start off with a comprehensive assessment of the patient in all 3 main aspects of care: medical, functional and social. In this article, the general principles in managing some of the common problems will be highlighted.

Common problems faced by the home bound elderly are:

1. Problems related to underlying medical diseases

Faced with an elderly patient that often has more than one medical condition causing morbidity, your focus will be on those conditions for which treatment will improve or prevent deterioration of function. Be wary also of dismissing symptoms as due to old age.

One common example is Parkinson's disease. The predominant features of bradykinesia and stiffness in Parkinson's disease are often missed because of the gradual onset of the disease and the close resemblance to ageing changes. Proper diagnosis and management can improve mobility and function. Another diagnosis often missed is depression. This may present as pseudodementia. Suspect depression if the patient has deterioration of function with no precipitating medical conditions. Other atypical presentations of depression include: agitation, apathy, and disturbed behaviour. A trial of anti-depressants is worth considering when the diagnosis is not obvious. Cataract resulting in poor vision is a good example of a medical condition for which treatment can result in good functional outcome.

Drug related side effects and drug interactions are common in the frail elderly where polypharmacy is often practised due to the increased number of medical conditions. It is important to review medications given for symptomatic relief and to stop them when appropriate. It is also useful to review medications for chronic conditions as some may no longer be necessary. For example, in a patient with end-stage dementia or cancer, giving him anti-hypertensives to treat his hypertension is not likely to make any difference to the prognosis or the quality of life. Management should be aimed at palliation.

Problems related to the side effects of medications are common, so always check the drugs given when a new symptom is presented by your house bound elderly.

2. Problems related to decrease in function e.g. constipation, pressures sores, urinary incontinence

If your patient is bed-bound, preventive measures are important to prevent the complications of prolonged bed rest. Teaching caregivers about regular turning of the patient, use of pressure relief mattress and bowel and bladder care is an important first step.

Chronic constipation with overflow incontinence is a common problem. Often, the presenting complaints may be frequent loose stools or diarrheoa. The patient may also present with acute confusion. A rectal examination to exclude faecal impaction is important. Sometimes the rectum may be empty as the patient has a high faecal impaction. An abdominal examination will reveal masses suggestive of fecolith. Family members may need to be taught to clear the bowels first with a regular enema followed by a regular laxative. If the caregiver has difficulty managing, you may need to refer

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the patient to the Home Nursing Foundation, where the nurses can support and teach the family about bowel care. The condition often requires close monitoring as it often relapses if not properly managed.

Urinary incontinence is also common in the bed-bound elderly. Management requires an understanding of the underlying cause of the incontinence. Often, there are multiple factors contributing to the incontinence. When in doubt, you may consider referring your patient to a geriatrician for an assessment.

The decrease in function of your elderly patient may be improved through rehabilitation. If the patient has not been seen by a rehabilitation team, it may be worthwhile referring the patient to one. Home visits by therapists may be useful as simple advice on home modification and aides can make a difference to the function of the patient.

3. Problems related to stressed caregivers and inadequate social support

Caregivers providing long-term care often have physical and emotional stress. As the doctor providing care, it is important for you to be aware of this. You may offer support through educating the carers about the disease process and you may also be able to offer help by referring them to the appropriate community support services. Community Hospitals in Singapore provide respite admissions for caregivers who need a break from caregiving.

A caregiver who is not coping will have a negative impact on the physical and psychological well-being of the patient. In such situations, you may want to refer the family to a social service agency such as the nearest family service centre.

Editor's Note:

It is with regret and heartfelt sorrow, that I wish to inform all readers that at the time of publication, Dr Lee Kng Swan passed away on 8 May 2000. The College of Family Physicians, Singapore, would like to express its thanks to her contributions to the College's 7th Scientific Conference and the Singapore Family Physician.

The Singapore Family Physician

Test Your Eye-Q (No. 10)
Unilateral Painless Loss of Vision

LPK Ang, KG Au Eong***

A 65-year-old man with a history of hypertension and diabetes mellitus presented with a sudden, painless, reduction in vision in his right eye for 3 days. His best-corrected visual acuity was 6/120 in the right eye and 6/9 in the left eye. A right relative pupillary defect was present. The anterior segment was normal in both eyes. Fig. 1 is a fundus photograph of the right eye.

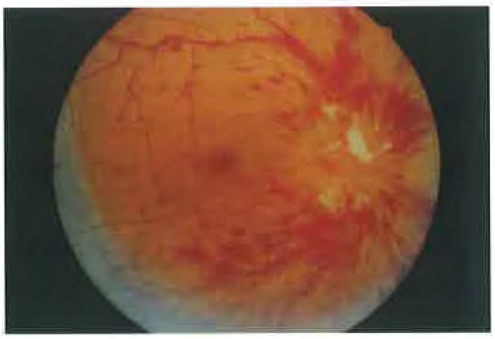

Figure 1

Questions

1. What does Fig.1 show?
2. What is the diagnosis?
3. What are the risk factors for this condition?
4. What are the possible complications of this condition?
5. What is the management of this patient?

Answers

1. Figure 1 shows diffuse flame-shaped retinal haemorrhages in all 4 quadrants of the ocular fundus. The retinal veins are dilated and tortuous. Cotton wool spots are present and the optic disc is swollen.

2. The patient has a central retinal vein occlusion (CRVO) in his right eye.

3. The risk factors for CRVO may be systemic or ocular. The systemic risk factors include increasing age, hypertension, hyperlipideamia, diabetes mellitus, hypercoagulable states (e.g. polycythemia, lymphoma, leukemia, sickle cell disease, multiple myeloma, activated protein C resistance, antiphospholipid syndrome), and drugs (e.g. oral contraceptives). The ocular risk factors include glaucoma, optic disc drusen and periphlebitis (e.g. sarcoidosis, Behcet's disease). Glaucoma is the ocular disease most commonly associated with CRVO.

4. The potential complications of CRVO include neovascular glaucoma secondary to iris neovascularisation, vitreous haemorrhage secondary to retinal or optic disc neovascularisation, chronic macular oedema and ischaemic maculopathy. CRVO may be classified as either ischaemic or nonischaemic. Patients with a visual acuity of 6/60 or worse, a relative afferent pupillary defect, more extensive retinal haemorrhage and multiple cotton-wool spots are more likely to have the ischaemic form of the disease. Ischaemic CRVO has a poorer prognosis and is more likely to develop complications. This patient's CRVO is likely to be the ischaemic form.

5. The management includes a complete ophthalmologic evaluation and a systemic work-up to exclude any predisposing ocular or medical conditions. Any underlying ocular or medical condition should be treated appropriately. In addition, the patient should be followed up regularly to look for the development of chronic macular oedema and neovascularisation of the iris, retina or optic disc. Fundal fluorescein angiography can help to identify areas of capillary nonperfusion of the retina and neovascularization of the optic disc or retina. Panretinal laser photocoagulation is performed if neovascularisation develops to reduce the risk of neovascular glaucoma and vitreous haemorrhage. Chronic macular oedema (3 to 6 months' duration) may be treated with grid laser photocoagulation.

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Book Review

A Textbook of Family Medicine

2nd Edition 1997 *McWhinney*

Reviewed by Gerard Low MH

McWhinney needs no introduction. Neither does his textbook. Except that this is not so much a textbook as the author himself alludes to. It does not cover disease seen in general practice in depth and breadth as in Goroll's Primary Care Medicine, for instance. What he aims to do is to define and conceptualise Family Medicine and he does it magnificently.

In this latest edition, he has maintained the basic structure of the earlier edition, but has added cross references between chapters and case reports in an attempt to integrate this book as a whole. At the end of some chapters, you will find that he has penned additional notes on top of the usual references. A new chapter on alternative medicine reflects the growing need for family physicians to acquaint themselves with this subject many patients already are familiar with.

Ten chapters have been devoted to a comprehensive discourse on basic principles of Family Medicine. I find the chapter on illness, suffering and healing most interesting and compelling. Many important remarks are made on suffering and some are worth reproducing here:

> ... suffering is intensely personal and not by any means synonymous with pain
>
> ... one of the most common errors we make as physicians is to treat pain but ignore other dimensions of suffering

McWhinney reminds us of our important role as healers, and often forgotten role in our highly sanitised and highly technical medical care. As good physicians and healers, we must help patients find their own way through the ordeal of their illness to a new wholeness.

While patient-centred clinical method is emphasised, McWhinney is quick to add the importance of the family and its effect on the patient and his illness. Family physicians must constantly endeavour to seek out vulnerable families, to "be there" in time of crisis, identify "hidden patients" in families and patients who are presenting symptoms of a family problem.

One will find more subtleties and ramifications as one reads along and this is demonstrable of the difficult nature of family practice. For those embarking on a vocation of family practice, do take heart that your work is not less difficult or less fulfilling than your specialist colleagues. Here, McWhinney strongly affirms the significant contribution of family physicians in our very complex medical landscape. He engages head-on with issues, shares with us many insights and equips us with the tools to grow as family physicians.

This book is highly recommended for medical students, health professionals and facilitators and for all family physicians at all stages of their career. If you are a family physician who is weary from your work, feeling directionless and seem to have lost that glow, do wait no longer and get hold of this book. It will prove a most involving and illuminating experience, quite like chicken soup for your soul.

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The Singapore Family Physician

Myanmar Vacation Aka Clinical Skills Short Course: A Trainee's Perspective

Yung C

Background

A clinical skills short course was conducted from 16 – 18 January in Yangon, Myanmar, this year. The aim of this exercise was to upgrade, in the shortest time, the clinical skills of the M. Med. Family Medicine trainees from the Private Practitioner's Scheme. Six such trainees, myself included, dutifully went along, as we needed to have such a course under our belts before being allowed sit for our exam. We were joined by a young M.O.H. trainee, who was a welcome companion, being well versed in the finer points of medical knowledge. This course was the joint effort of the Myanmar Medical Association and the Singapore Medical Association.

Other faculty members for this course besides A/Prof. Cheong were A/ Prof. Goh Lee Gan, Dr Henry Yeo, one of our clinical tutors, SMA Secretary, Dr Wong Chiang Yin and SMA treasurer, Dr Yue Wai Mun. On the Myanmar side were the Professors and consultants from the Yangon General Hospital, the Yangon Orthopaedic Hospital and the Yangon Women's Hospital.

Day One: Sunday 16 Jan 2000

Yangon

We arrived just before noon at the Yangon airport which had a small airfield with only one or two planes. The first lesson was given by A/Prof. Cheong Pak Yean when we stepped out of the airport building and were accosted by the flotsam of porters fighting over our bags. He deftly averted a fight amongst these many eager porters by enforcing some street rules regarding the quantum of tips per baggage. Welcome to Myanmar. Dr Wong Chiang Yin, another seasoned traveller to Yangon, continued the orientation in the bus. He enlightened us on the sights, customs (e.g. how much to tip) and food haunts of Yangon.

Yangon lies to the West of the Ayeyarwady River, the longest river in Myanmar. The Ayeyarwady, which has its source in the Himalayas, divides the country into east and west and Yangon lies in its fertile eastern delta. Although Yangon is the capital city with a population density of about 400 people per square km, it is a quiet town. The city is very flat and has two large lakes. We arrived

The Singapore delegation and their Myamar hosts

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during the cool season, but it was very hot during the afternoons as the beautiful blue skies had hardly any cloud cover. The evenings were pleasantly cool. The population of Myanmar is a veritable mix of various ethnic groups, the majority (69%) being ethnic Bamar. Among the other races are the Shan, Karen Arakanese, Chinese, Indians and Pakistanis. 90% of the population are Buddhists.

Our first stop was the Traders Hotel, arguably the best hotel this side of the Ayeyarwady. This 5 star hotel is partly owned by DBS Land. Service is excellent and the rooms offer a panoramic view of the city. Its central location was a definite plus-point because our tight schedule gave us very little time to shuttle from place to place.

We were given barely enough time to put our luggage down when we were whisked off to the Yangon Orthopaedic Hospital, which vaguely reminded us of the old Woodbridge. There we met for the first time, our fellow Myanmar trainees. These doctors are the pioneer batch of GPs who recently obtained the Diploma in General Practice, the first time that Myanmar has conducted a post graduate course for GPs. We watched eagerly as one by one they streamed into the lecture room. It was like going to a new school and meeting new classmates.

Myanmar Fashion

The lady doctors were so demure. They had their long hair tied up with not a strand out of place. I then understood the reason for the prudish memo the SMA sent us before we left for this course: All ladies with long hair must have it tied up during the course. In fact, during our three-day stay in Yangon, we never met a lady who had wanton flowing tresses. Every lady had her long hair tied back into a ponytail or a severe bun. I was glad I did not rebel against the aforementioned memo directive.

As we are on the subject of fashion, I have to say that we three lady trainees tried not to embarrass Singapore and came suitably attired in business clothes, smart jackets and all (it was supposed to be the cool season). Within one hour, the jackets came off and we were mopping our less then stylish brows. On the other hand, our Myanmar colleagues, both male and female, were looking cool and comfortable in their longyis (Myanmar national dress that looks like a sarong) and black slippers. Some men wear Western style shirts, ties and pants but all the ladies in Myanmar wear ankle length longyis. The three of us girls had the shortest skirts in town that day. The only Myanmese lady that I met who was not in a long skirt was the gym attendant at the hotel whose uniform was a tee-shirt and shorts.

Orthopaedics

Coming back to Orthopaedics, the stark take home message that afternoon was, to quote the Professor Myo Myint, Professor of Othopaedics, Rector of Institute of Medicine 1 and President of the Myanmar Medical Association, "Socio-economic factors are the cause of disease and influence the management of disease." Almost every case presented that afternoon had complications because the patient was too poor to seek treatment early or lived too far away from medical care. The case that touched me was a 12-year-old boy who had a fracture of the femur and osteomyelitis. As he lived in a rural area a few days journey from Yangon, his parents brought him for treatment only after he was not able to walk for 4 months and had developed discharging sinuses over his left thigh. I couldn't help musing that a boy like him should be up and about playing soccer and fighting spiders.

Shwe Dagon

After the intense Orthopaedic session, we were set free to roam the magnificent Shwe Dagon pagoda on Singuttara Hill, an imposing monument that is 2500 years old. This is the most important pagoda in Myanmar, the Land of Pagodas, because Buddha's hair and other sacred relics are enshrined there. The dome of the main pagoda is covered with pure gold plate. Thieves have attempted to enter the main pagoda to steal the precious relics, but none have emerged because of the complicated maze of tunnels within designed by the ancients to thwart such attempts. To tour the whole pagoda complex needs a certain level of aerobic fitness as it is huge and has many steps to climb.

A Point Of Digression

Besides the main pagoda, the other shrines in Shwe Dagon are also ornate works of art decorated with precious stones, intricate carvings, mosaics and gold leaf. No expense is spared to honour the Buddha. Some of these shrines are devoted to special intercessory needs like the wish to have a baby.

Our guide was a Myanmese of Chinese descent who calls himself Michael. He filled us in with the history of Buddhism in Myanmar, various aspects of Buddhist practice and even used a special table to tell us which day of the week we were born on. Buddhists in Myanmar place emphasis on the day of the week that a person is born on because of the belief that each day bestows a certain characteristic on the person. Many also turn vegetarian on the day of the week that they are born.

The pagoda was full of devotees that evening, praying at the various shrines. Despite the number of people present, it was quiet, orderly and an aura of serenity pervaded the place.

Faculty Dinner

Our day ended with a banquet at the Royal Garden Restaurant, a posh Chinese restaurant on a lake. This dinner was hosted in honour of the Myanmar faculty. In typical Asian style, the men were seated at one table and three of us lady trainees at another. I sat next to a very attractive and well-spoken lady. Not only was she gracious and pretty, I found it hard to concentrate on my food as my eyes were distracted by the sparklers on her ear lobes and sternal notch, (my guess is at least 3 carats apiece). I was to find out later that this lady was Professor Cherry, Professor of Gynaecology at the Yangon Women's Hospital, the largest women's hospital in Myanmar. The other ladies at our table were also Professors and consultants in Paediatrics and O&G. We were only humble trainees and they were obviously very senior and highly respected doctors in Myanmar, but they spoke to us graciously and we made polite conversation about the different ways of life in our two countries.

Thanaka

I remarked to our distinguished dinner guests that most Myanmar ladies had lovely skins. Middle aged ladies did not have much melasma. They attributed this to thanaka. Thanaka powder is made from the bark of a small tree. A branch, about 17 cm long is ground on a special grinding stone using water as a lubricant. The resulting paste is smeared on the face and body. Thanaka is ubiquitous in Myanmar. Ladies walk around with thanaka smeared in great circles on their faces. The Myanmese are great believers in the soothing properties of thanaka. It is an astringent as well as a sunscreen, we were informed by Professor Cherry, that for those who don't want to bother about grinding it, it also comes in convenient jars of powder. We nevertheless found thanaka grinding stones every where: by patients' bedsides and in the nurses' tea rooms. In fact, a patient's thanaka paste was forcefully smeared on our faces by Prof. Cheong, a great believer in cultural immersion.

The Professor of Paediatrics informed us that the Myanmese do not accept the practice of sponging to bring down a child fever, so they tell parents to smear thanaka paste on the child's body. This works well in bringing down fever and the doctors attribute this to the water in the thanaka paste. The parents think that it is the cooling properties of the thanaka that is doing the trick. Whatever the reason, this was a great lesson in incorporating a patient's cultural beliefs into modern medicine, something close to the hearts of family physicians.

The three of us lucky girls received a gift of a thanaka branch from the Professor of Paediatrics and a jar of thanaka powder from Professor Cherry. I have since been applying this thanaka daily to my face in the hope of looking as good as Professor Cherry!

Day 2 – 17 January

Paediatrics

Day 2 day began at the Yangon Children's Hospital. Our group was split up and we were teamed with Myanmar GPs. We were made to clerk and present long cases and taken through short cases. Infections that are almost unheard of among children here were common there. We saw children with disseminated TB, cerebral malaria and rheumatic heart disease. My heart went out

to the 9 month old baby who had keratomalacia and rickets due to Vitamin A deficiency because his mother had lactation failure.

The children were all quiet, timid and co-operative. There were none of the over-fed, over-active and precocious children that we find at home. In fact, the boldest children I encountered in Myanmar were the child monks on the street begging for alms.

All of us were deeply appreciative of the patient mothers who allowed us to examine their children and crying babies over and over again.

My group was attached to a very pleasant paediatrician who took us through the short cases. However, he had to compete for my attention with the birds that were flying around freely in the air conditioned room where the tutorial was conducted. I think that these are pets and part of the shrine that was in that room. It was an interesting cultural practice.

General Surgery

General surgery was next on the programme and this was conducted at the Yangon General Hospital. This hospital is about 200 years old and was built by the British. The distinct red brick hospital buildings had many intricate wrought iron balustrades. There was even an old lift with collapsible gates as doors, much like the old lift in the College of Medicine Building before it was renovated. In fact, walking through the corridors of this hospital was like walking through the old Singapore General Hospital and Alexandra Hospital. The surgeons presented cases of disease that had progressed to such a late stage like liposarcomas almost the size of footballs and a lady with carcinoma of the breast who sought treatment only after the tumour had ulcerated through the skin. Once again, I said a silent prayer of thanksgiving that we have such easily accessible health care at home.

GP attachment

The last item on the programme for day 2 was our GP attachment. All in all, we felt that this was the greatest eye opener. Dr Myint Oo, a GP with a clinic in a Yangon suburb drove the three of us ladies and Dr Henry Yeo to his clinic, where his charming wife was waiting with refreshments. His clinic is on the ground floor of a two-storey building. Dr Myint runs his clinic in the morning,

Dr and Mrs Myint Oo outside their clinic

afternoon and evening. His waiting room is very small and he has stools lined up on the pavement outside. The consultation room is separated from the waiting room by a curtain, and everyone outside can hear the goings on inside. Nobody seems to mind this. The consultation room could only accommodate an examination couch, some wall shelves and a small desk. Dr Myint examines all his patients on this couch. He does not sit down at all during consultations. His wife is right there in the room with him dispensing medicine. After the patient is seen, Dr Myint writes his prescription in a book and Mrs. Myint dispenses it. She puts all the medicine for one day in one bag. Thus if you are given medicine for two days, which seems to be the usual amount, you are given two bags. Most patients are also treated with injections.

The four of us sat outside in the waiting room observing the patients as they arrived because the consultation room was so small. If there was any thing interesting, Dr Myint would invite us in to have a look. Actually, this turned out to be almost every case.

The Singapore Family Physician

Although the flu was raging in the US, Europe and China at that time, and I was seeing quite a few myself at home, there was not a single case of URTI that evening. What we saw were cases of typhoid, malaria, gonorhoea, diabetes and hypertension. There was a young man who does manual work who came for intravenous glucose because he felt tired. Dr Myint gets such requests from time to time. There were two young men who had multiple purulent discharging sinuses from their foreskins as a result of multiple self injections of a sclerosing agent from Thailand. This is quite a common practice it seems, and the recurrent infections don't seem to put these men off. There were also men with foreskin implants of small beads called "goli". Dr Myint has removed many of them either because of infection or because their partners have become disenchanted with the goli. He keeps them in a tin and gave each of us one as a souvenir. We showed our souvenirs to Prof. Goh the next morning as a "short case". He looked and thought hard but could not make a diagnosis, such is the scarcity of this condition here. (It also was a good feeling to know more that our Professor for once, although on such a dubious subject!)

As we sat in the waiting room, we noticed that the patients arrived in a steady stream. There was no large crowd at any one time. Somehow, the patients seem to know when to come. We also explored the area around the clinic. There was a dental clinic next door that belonged to Dr Myint's sister. His in-laws live in the house behind the clinic. There was a small dining room next door, and half way through the evening, we were invited there to partake of a delicious meal of Mohinga, the most famous dish of Myanmar. This is a dish of noodles in a spicy fish soup, which is garnished with various condiments.

Housecall

One patient that evening was a frail old lady with who spoke fluent English with beautifully rounded vowels. It transpired that this was the old English teacher of Dr Myint and she requested that the good doctor make a house call to see her sister. While waiting for Dr Myint to clear his patients before the visit, she shared her life story with us. She is an ethnic Karen and a Christian. She is 73 years old and her sister is 75. They were both teachers and were previously employed by the Baptist Missions. Now retired, she lives on a small pension and has to depend on her former students like Dr Myint to help her out. She spends her time now taking care of her older sister and teaching little children in the Karen Compound where she lives.

This Karen Compound is like a kampong. Her modest home is a wooden house that she shares with her sister. When we arrived, her sister was in bed. This bed, the kitchen and an old piano were all in a tiny room. An old picture of Queen Elizabeth and her family taken in the 70s hung incongruously from the dusty rafters. "I love the Queen" the lady explains.

The older sister was suffering from body ache and the younger sister was tired of massaging her. All 5 of us doctors could find nothing much wrong with her. My two fellow trainees have a Diploma in Geriatrics and proceeded effortlessly to do a quick mental assessment in English. They concluded that she had Alzheimer's disease and explained this to the younger sister. She immediately exhorted her sister to read the Bible daily as an antidote to her memory loss.

Before we left, we asked the sisters to play the piano for us. Dr Henry Yeo requested that they sing "You are my sunshine". This transformed the two old ladies and they both played the piano and sang this song with gusto. They also sang us a song in Myanmar. Music therapy seemed to rid them of their troubles momentarily.

I must mention that Dr Myint did not charge a cent for treating the two sisters. In fact, he gives discounts quite readily to his patients and runs a free clinic in a poor area.

We ended the evening at a restaurant called Singapore's Kitchen on Strand Road which served Chinese food.

Day 3 – 18 January 2000

Medicine

After our usual breakfast buffet, we were rushed back to the Yangon General Hospital for the session on Internal Medicine. The whole department turned out to welcome us and even the housemen were tasked to make and serve us refreshments, such is Myanmese hospitality.

The format here was the same as for Paediatrics. One of the short cases was a 14 year old boy with Thalssemia Major. He had the typical "chipmunk facies" which our younger colleagues were not familiar with because of the advent of genetic counselling and Desferral therapy in Singapore. Another case that I found interesting, because we are unlikely to encounter it in Singapore, was a man with Beri – beri who had ECG changes that helped to clinch the diagnosis. We were shown the post treatment ECG as evidence of the success of treatment.

Obstetrics and Gynaecology

Our last clinical session was at the Yangon Women's Hospital where our hostess was the lovely Professor Cherry. As in all the other departments that we visited, a good selection of both obstetric and gynaecology patients were waiting for us. Yet again, the patients selected for us were the epitome of patience. The pregnant ladies allowed repeated antenatal examinations and those with pelvic signs did not mind the 20 or so of us observing as their signs were demonstrated during the VE by the consultant. Of particular note was a 28 year old lady with a fungating mass at her cervix. All of us literally cheered when we were told that the histology turned out to be TB. This is a very rare manifestation of TB, even in Myanmar.

After the O and G session, we had the rest of the evening and about two hours the next morning to relax, so we hit the streets.

Chinatown

The streets of Yangon are wide and busy. The cars are reconditioned used cars imported from Japan and not many have air conditioning. The taxis are reconditioned taxis from Singapore, many with their original coat of paint, so you will easily recognise NTUC and TIBS taxis there. Traffic police help direct the heavy down town traffic by blowing their whistles and doing a little dance with their arms. They stand at intervals on the road dividers and wear a uniform of khaki shorts, knee high socks and a 'topi' just like how I remember our policemen used to dress in the 60s.

The low rise buildings that line the streets have interesting facades and are different from the typical shop houses that that line the streets of Singapore and Malaysia. Many were in need of restoration but if you look beyond the grime, you can pick out beautiful architectural details.

Our new friend, Dr Win, was kind enough to lend us her pick-up truck together with the services of her driver. Incidentally, Dr Win received her Ph.D. from NUS for her work on glial cells but has chosen to be a GP in Myanmar. She offered to be our guide that evening in Chinatown so all us, the two Professors included, piled into the pick up. The Chinatown area is full of people shopping and hawking their wares. You can buy anything: clothes, food, medicines like full strength Betnovate cream and even an ice kachang grinder. No one hassled us and no one ever calls out to passers by to buy their wares. Actually, the only "hassle" were the little child monks and their begging bowls.

Food stalls are set up by the roadside just like in old Singapore. People sit on low tables and stools and enjoy a wide variety of offerings, many of which are similar to food we find at home. There was the Myanmese version of satay chelop, where patrons fry skewered morsels of food like intestines, in a big pot of oil. I also saw dumplings (bak chang), yam cake (or kway), sticky Chinese New Year cake (nien kow) and many other Chinese snacks. Drivers have to be careful not to run into the diners. Of course, there were also stalls selling Mohinga. Prof. Cheong waxed lyrical about the soya bean curd (tau huay) that he ate, sitting by the road side.

Our group made one road side vendor of crispy snacks a very happy woman that night. We were

just browsing and wondering whether to buy any when one of us made the first move. That set off a chain reaction and together we must have given her at least US$50 worth of business buying peanut candy and other munchies. We felt sorry for the other vendors around selling exactly the same stuff who did not benefit from our custom. The lady was so pleased with our business, and also with the fact that we could exchange some words in Chinese, that she gave us each a small gift.

After Chinatown, we travelled in the pick-up to Roof Top Seafood Restaurant at Dr Wong Chiang Yin's insistence. This restaurant is on a street lined with bars that had many dolled up young ladies waiting outside for patrons. Roof Top did not provide such services, but true to Chiang Yin's promise, this restaurant did serve very fresh and tasty seafood including eel and turtle. However, the most memorable item that night was the animal stationed at the entrance to the restaurant. We assumed that it was a black guard dog lying unconcerned as we passed by until it stood up on its hind legs. It turned out to be a bear!

We ended the evening with drinks at the Strand, the first hotel opened by the Sarkies brothers in South East Asia 200 years ago. These were the same folks that came to Singapore later and built the Raffles Hotel. We did not get to see much of the hotel's old-world charm as it was dark, but we appreciated the effort that its investors, including DBS Land, put in to restore it. By now, you should have an idea of the business interests that Singapore has in Myanmar. By the way, the Straits Times is available daily at US$3.

Scot market

On the morning of our departure, we were given 1 hour to shop at the famous Scot Market. This has been renamed Bogyoke Aung Sang Market. Many other streets and places have had their British names changed to Myanmar names, especially in honour of their famous generals like the revered Aung San.

This market is a souvenir hunter's paradise. Many of us bought beautiful longyi, silk blouses, and gems. Dr Win recommended us to her friends, a married couple who are both doctors. They had given up medicine to becomes jewellers. We were mesmerised by their jade, rubies and amethysts. Many of us bought some with absolutely no idea of what we were buying but thank goodness they turned out to be genuine and quite good buys.

Departure

Before we knew it, we were on the road back to the airport. After clearing customs and immigration without trouble, we sat in the coffee house and waited for our plane. Being such a small airport, we only had to check in 1 hour before departure. While sipping coffee, the more anxious among us were worried that we might miss our flight as the time of our departure drew near. The waiter sensed our discomfort and told us not to worry as the plane was not ready for boarding. We wondered how he knew and he jerked his head towards the window. One only has to look out to be updated on the latest goings on in the air field. We carried on drinking.

True to name, this trip was a very short course but such an eye-opener to another world and culture which was vaguely familiar being Asian, yet strange in other ways. I came away awarding the people of Myanmar the most gracious people of Asia award for their hospitality and geniality, no matter what walk of life they come from. Would I want to make another trip to Myanmar? Most definitely yes and we trainees are now pressing our Professors to organise another one soon, and hopefully it will not be such a short course.

THE

COLLEGE MIRROR

Issue: No 1 Jan - Mar 2000
MITA(P) No 385/03/99

FROM THE EDITOR'S DESK

The start of the millenium has witnessed many changes to the CME system. On the national level, there is now an on line system administered by the Singapore Medical Council. Previously, the administration of the CME system was carried out by the College on behalf of the SMC-CME Committee from 1993 to the end of 1999. Judging by the feedback received from College members and other medical practitioners, the administrative transition is witnessing some teething problems. To clarify how we can help our members, read the article on page M5 by the College's CME Committee Chairman.

The College's aim is to make CME more practical and relevant for GPs/Family Physicians. One step to achieving this is exemplified by the 4th Annual Surgical Update followed by the hands-on Minor Surgical Procedures Course over two weekends in March 2000. The latter included the transmission of live surgery demonstrations of various surgical procedures via a two-way audio video link where the participants could interact directly with the surgeons. Both events proved successful with good constructive comments from the course participants (see pages M2 - M4). Dates have also been set for the next surgical courses in 2001.

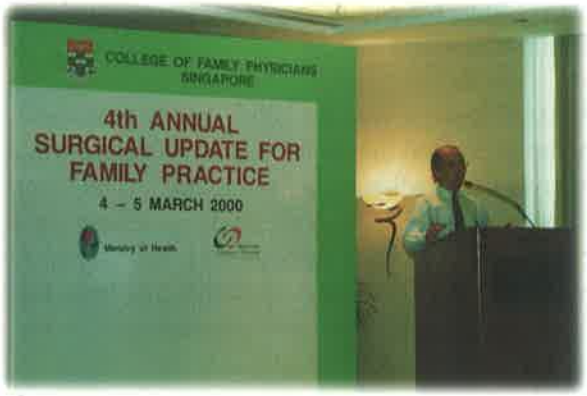
On a separate subject, another milestone will be reached when the College launches its Graduate Diploma in Family Medicine. This is a vocational training certification for primary care doctors to practise Family Medicine at an enhanced level to meet the needs of the child, the adult and the elderly. Full details of the course syllabus can be found on pages M7 - M8.

On the international scene, The WONCA Asia Pacific Regional Conference 2000 will be held in Christchurch, New Zealand in June, and going further afield, the WONCA European Regional Conference will in held in Vienna, Austria in early July. More details are found in the announcements section of this newsletter.

Ms Yvonne Chung

4th Annual Surgical Update

This year's 4th Annual Surgical Update for Family Practice was held over the weekend of 4 - 5 March 2000 at the Grand Copthorne Hotel. This annual CME event has improved in quality and sophistication over the last four years and has developed a reputation of its own, with the enthusiasm of both the teaching faculty and the participants remaining as strong as when it was first launched in January 1997. Some 130 delegates attended the event.



A/Prof Lim Lean Huat, President of the College, welcoming the participants to the Surgical Update.

Participants at the Surgical Update

Day One's theme was Surgical Oncology and the symposia covered cancer programmes in Singapore, genetic and familial aspects of cancer, reconstructive options in surgical oncology, new technologies in cancer management, and cancer screening.

Day Two concentrated on Gynaecologic Surgery and the presentations gave the latest updates and advances in infertility treatment, general gynaecolology and common gynaecological complaints and office procedures.

Dr Paul Tseng (standing) answering questions from the floor with the panel looking on, seated left to right: Dr Arthur Tan, Dr Yu Su Ling & Dr Tan Hak Koon

Four break-out group workshop demonstrations were also held in the areas of suturing and dissection of lumps and bumps; mammogramms, imaging and biopsies; gynaecologic procedures; and endoscopies. Each participant was able to do all 4 workshops on a rotational basis.

The College would like to extend its thanks and appreciation to the organising committee (most of whom have been involved since the very first Annual Surgical Update) the teaching faculty and to the participants, especially those who have supported this event in the past years as well.

We look forward to seeing you all again at the 5th Annual Surgical Update in February 2001 (see the announcement section).

Ms Yvonne Chung

Minor Surgical Procedures Course

The College's inaugural Minor Surgical Procedures Course for Family Physicians, jointly organised with the Singapore General Hospital, was held on 11 – 12 March 2000. This was the follow-up course from the 4th Annual Surgical Update held on the previous weekend. This highly intensive hands-on course which included live surgery demonstrations, was three times oversubscribed. All places were filled up within 10 days of the first announcement going out!

Participants of the course

The organising committee and teaching faculty had put together a progressive and very relevant modular syllabus, comprising practical tips on the appropriate selection and handing of instruments, correct incision and excision techniques, wound closure, knot tying techniques and methods of haemostasis. Given the practical nature of the course, it was limited to 40 participants, which enabled each person to have his or her own set of instruments, knot tying board, a set of anatomically correct tissue models, sutures, a full set of course notes and close tutoring and supervision from the teaching faculty.

Live demonstrations of surgery on patients with the various lesions were transmitted from the Ambulatory Surgery Centre of SGH, to the course participants in the lecture room. Participants were able to view the administration of local anaesthesia, the incision planning, and the dissection and closure performed by the surgeons. They were also able to interact via a two-way audio-video link with the surgeons during the procedures.

The class in action and practicing their knot tying techniques. Dr Cheong Pak Yean (2nd left) receiving personalised tuition from Dr Hong Ga Sze (3rd left)

Dr Colin Song demonstrating one of the knot tying techniques

After the live surgery demonstrations, it was back to the Clinical skills laboratory for the participants to try out the procedures on the skin models.

Judging from the feedback from the participants, the course was enjoy and beneficial. Below are excerpts of some of the comments made:

"Best hands-on course ever attended. Thoroughly enjoyable and useful."

Amidst the concentration, there's time for a smile from Dr Howe Wen Li (1st left)

"Excellent, good show, good interaction, lively!" "Enjoyable, practical. Appreciate the presence of tutors to help and guide-personalised teaching"

"Excellent course. Thank you very much!"

We would also like to thank those participants who gave constructive feedback on how to make future courses even better.

The next Minor Surgical Procedures Course will be expanded to accommodate 80 participants over 2 weekends of 16-17 February and 23 – 24 February 2001 respectively. Watch out for further details to be announced soon!

Ms Yvonne Chung

Minor Surgery Course - A personal account from one of the participants

The Minor Surgical Procedures Course for family physicians held on 11-12 March 2000 was both a visually and clinically enriching experience.

Over two days, the 40 participants were taught various surgical procedures by an enthusiastic team of surgeons from the Singapore General Hospital. The different teaching methods adopted included lectures, demonstrations and hands-on practice on life-like models with pathological lesions.

The highlight of the event was the live-surgery demonstration of excision techniques of sebaceous cyst, papilloma, subcutaneous lipoma, wedge resection of ingrowing toenail and band-ligation and injection of haemorrhoids. We were linked to the operating surgeons in two day-surgery theaters via a 2-way audio video system and were taken step-by-step through the procedures. Questions could also be posted to the relevant surgeons live.

Most of us were so enthralled, we were willing to forgo the lunch break in order not to miss out on the "action" at the other end. Instead, we grabbed our food during the camera change over period from one theatre to the other and ate while being "entertained" on the different ways of doing band-ligation of haemorrhoids!

All in all, the course was a resounding success owed much to the efforts of the organising committee, the sponsors, and the altruism of the patients in the live-demonstration.

Dr Howe Wen Li
Family Physician
Private practice

New CME System

Website address: http://www.smc-cme.gov.sg

The administration of the national Singapore Medical Council – Continuing Medical Education (SMC-CME) programme which was carried out by the College from 1993 to 1999 on behalf of the SMC-CME Committee, was taken back by the SMC on 1 January 2000. This move is in line with the launch of their new on-line national CME system on 22 January 2000. Each doctor who is registered with the SMC is given an on-line account free of charge to encourage more active participation in CME on a voluntary basis.

Since the launch of the new system, the College Secretariat staff have received many enquiries, much feedback and also unjust criticism from members and other CME organisers on the problems that they have encountered under the SMC's new on line system. Some have even gone as far as venting their frustration on the Secretariat staff. It appears that there is still a lot of confusion about the changed role of the College in the new CME system and what the College is able to do. I would like to clarify the situation and how the College can help its members.

College's role

The College continues to play an important and vital role in the accreditation of CME that is relevant to GPs/ Family Physicians. The College is represented in the SMC's Education Committee which now oversees the new national CME programme, and the SMC regularly consults the College CME Committee on such matters.

Attendance records and CME points

The database for the recording of CME points achieved by doctors is kept and maintained by the SMC. Under the new system, doctors who attend CME organised by accredited organisers will have their attendance entered into the SMC's database by the organiser. This is done by entering the doctor's MCR number. Hence, there is a need for the participating doctor to sign the attendance record and give their MCR numbers at the CME events. Once the attendance records are keyed into the database, the number of CME points will be duly accredited by the SMC. Doctors can access the on line system and check the number of CME points they have achieved in the given year.

Doctors who attend overseas conferences and who do self-study will need to electronically key in the CME points themselves. Each doctor who is registered with the SMC will have been issued with their username and password by the SMC to access their on line account. For College members who do not have access to a computer to carry out this exercise, please submit your logsheet, detailing the self-study items, overseas conferences together with your MCR number and personal/password details to the College Secretariat; such information will be treated as confidential. The staff will be glad to provide members with the necessary assistance for this on line data entry.

CME Calendar

The Calendar of CME events can be found in the SMC's website (at the above website address). Doctors can search by specialty, topic or by date. The information contained in the Calendar is derived from organisers submitting their applications **directly** to the SMC for accreditation of their respective events. For this reason, the College no longer produces the monthly CME calendar that it used to do when organisers applied to the College for CME accreditation under the old system.

Further assistance and information

For other information or problems encountered with the on line system, please contact the SMC-CME Helpdesk at telephone 3259032 or 3259155.

Dr Richard Ng Mong Hoo
Chairman, CME Committee
College of Family Physicians, Singapore

Graduate Diploma in Family Medicine

The Gradaute Diploma in Family Medicine (GDFM) is a vocational training certification for primary care doctors. The aim of this programme is to train primary care doctors to practise Family Medicine at an enhanced level to meet the needs of the child, the adolescent, the adult and the elderly.

Course Objectives

The GDFM training programme and the examination places due emphasis on clinical diagnostic and management skills in the various clinical disciplines. At the end of the programme, the course participant should be able to:

- describe the knowledge base relevant to General Practice/ Family Medicine that has been detailed in the Graduate Diploma course; apply relevant clinical skills to approach and manage common problems encountered in General Practice/ Family Medicine;
- describe the care of chronic medical conditions (such as diabetes mellitus and hypertension) and the care of the elderly in Singapore; and
- meet the standards prescribed for the Diploma in Family Medicine

Entry Requirements

The candidate must possess the following to be eligible to register for the course a basic degree of the MBBS or equivalent qualification registrable with the Singapore Medical Council one year as a medical officer or general practitioner

Course Organisers and Teaching Faculty

The main teaching faculty will be Family Medicine teachers from the University, public sector healthcare and private sector healthcare who have been involved in the teaching of the Master of Medicine (Family Medicine) degree.

Course Structure

The course is on a part-time basis and planned to accommodate the busy doctor's schedule; the majority of candidates will not need to take leave or time off from their regular work.

The course is to be completed within 24 months. Any candidate who has not completed the course at the end of this period may seek approval for an extension.

Course Programme

The course consists of:

- Distance-learning and workshops (FMTP)
- Tutorials
- Family Medicine Skills Courses
- Elective Short Clinical Courses

Distance learning and workshops (FMTP)

- The Family Medicine content is organised into 8 modules to be covered in 2 years. The modules are the same as the MMed(FM).
- One module is covered in 3 months.
- Four Saturday afternoon workshops are conducted for every module.

Graduate Diploma in Family Medicine

Tutorials

The tutorials provide the micro-teaching and learning opportunities. Each trainee would be attached to a tutorial group of up to 6 trainees, under an appointed tutor who must possess at least a Master of Family Medicine degree or equivalent. One tutorial will be conducted each quarter, corresponding to the topics in the current module of learning.

- The tutorials' focus would be on common and important aspects of Family Medicine in practice.

Clinical and practice skills courses

Three Clinical and Practice Skills courses to emphasise basic clinical diagnostic and management skills essential to general practice will be conducted in the two years. Attendance is compulsory. These are

1. Communication and counselling (6 hours)
2. Clinical family medicine (6 hours)
3. Basic clinical skills (5 days - attachment to hospitals and clinics)

Elective short clinical courses

These short skill courses will emphasise basic clinical diagnostic and management skills essential to general practice. They will be conducted by the clinical departments and credited as courses for the DFM. The course participant is expected to attend at least one elective short clinical course a year in the area of his or her choice.

Log Book

The participants will also be issued with a logbook in which they will be required to log the FMTP modules, the tutorials and the short courses attended.

Course Syllabus

The course syllabus consists of:

Family medicine and whole person medicine

- The principles of Family Medicine
- Care of the child and adolescent
- Care of chronic medical problems and palliative care
- Care of the elderly
- Care of the individual, family and community
- Care of the adult
- Care of the antenatal/ obstetric patient
- Care of the gynaecological patient

Disease management by body systems

- Cardiovascular and respiratory disorders
- Gastrointestinal disorders
- Renal, haematological and haematological disorders
- Psychiatry
- Dermatology
- Musculo-skeletal disorders, emergency medicine
- Neurological disorders, eye, nose and ENT
- Metabolic and endocrine disorders

Practice management

- Confidentiality and medical records
- Dispensing, certification and notification
- Managing the practice
- Computer use and Medical Information System
- Practice issues
- Setting up a practice
- Financial management
- Quality assurance

The first examination is planned for year 2002. The details of the examination will be made known after it has been finalised. Three parts to the examination are proposed:

- 25%: MCQ paper (2 hours)
- 25% Short Answer/Modified Essay Question paper on patient centred and oriented care (2 hours)
- 50%: Skills Assessment by OSCE (Objectively Structured Clinical Examination) (2 hours)

Graduate Diploma in Family Medicine

Course Fee

The course fee is **S$2,900 for the 2-year programme**. This will be inclusive of the cost for the log-book, 8 FMTP modules, tutorials and the 3 Family Medicine skills courses conducted by the College of Family Physicians, Singapore. This does not include the examination fee which is payable separately to the Graduate School of Medical Studies, NUS.

Course details for 2000

The 2-year programme is targeted to start in July 2000. The quota of students will be based on resources available. In the event that the course is over-subscribed, priority will be given to practising Family Physicians and members of the College of Family Physicians, Singapore.

The Relationship of the Proposed Graduate Diploma to the MMed (Family Medicine)

There are doctors who pass the GDFM and wish to further pursue the MMed(FM) qualification. These doctors need not attend the FMTP modules since they have already done so. Other requirements of MMed(FM) will apply.

Administration

CFPS will be the administrator for the course/ training component. CFPS will also co-ordinate promotion of the DFM and admission into the programme. **Applications are now open for the year 2000 batch of entrants to the course. The closing date for applications is 10 June 2000.**

Please send the completed application form to and cheque payment to:

College of Family Physicians Singapore,

College of Medicine Building, 16 College Road #01-02, Singapore 169854.

For full course brochure and any further information please contact

Ms Yvonne Chung/ Ms Katy Chan: tel 223 0606, fax: 222 0204.

Email: rccfps@pacific.net.sg

News to Share

New Arrivals

Congratulations to Dr and Mrs Cheong Seng Kwing on the recent birth of their first child, a girl, Frances.

Welcome to New Members

Welcome to New Members

A warm welcome to the following new members who joined the College in the first quarter of 2000:

Ordinary Membership

Dr Thng Lip Mong	(Private practice)	
Dr Charlotte Yung	(Private practice)	CFPS membership wef 1-4-2000)
Dr Lim Fong Seng	(Government practice)	CFPS membership wef 1-4-2000)
Dr Siew Chee Weng	(Private practice)	CFPS membership wef 1-4-2000)
Dr Soong Chuon Vui, Jovian	(Private practice)	CFPS membership wef 1-4-2000)

Associate Membership

Dr Shiau Ee Ling	(Private Practice)	
Dr Anbumalar Ramiah	(Private practice)	CFPS membership wef 1-4-2000)
Dr Ng Chee Yung	(SAF)	CFPS membership wef 1-4-2000)
Dr Ng Seo Peng	(Private practice)	CFPS membership wef 1-4-2000)
Dr Ong Jin Ai	(Government practice)	CFPS membership wef 1-4-2000)
Dr Phoon Chiew Yong	(Private practice)	CFPS membership wef 1-4-2000)
Dr Siew Wei Fong	(Government practice)	CFPS membership wef 1-4-2000)
Dr Tang Kim Lian	(Government practice)	CFPS membership wef 1-4-2000)
Dr Wong Yu Yi	(Government practice)	CFPS membership wef 1-4-2000)

Announcements

College of Family Physicians, Singapore

5th ANNUAL SURGICAL UPDATE FOR FAMILY PRACTICE

10 - 11 February 2001

Day 1 - Aesthetic and Implant Surgery
Day 2 - Orthopaedic Trauma

organised in conjunction with

MINOR SURGICAL PROCEDURES COURSE FOR FAMILY PHYSICIANS

Hands –on and live surgery demonstration

17 – 18 February 2001 (1st group)
and
24 – 25 February 2001 (2nd group)

Details of the venues, programme, course syllabus and registrations fees will be sent to members in due course

Announcements

Breastfeeding Mothers' Support Group (Singapore)

Seminar for doctors

Supporting the breastfeeding mother through evidence based practice

19 August 2000, 2.00 - 5.45pm, College of Medicine Building

- Supported by the College of Family Physicians, Singapore
- Relevant for GPs/ Family Physicians, paediatricians, and O&Gs in both the public and private sectors

For more details, contact:
***Mrs Wan Siew Kuan**, Chairman, August 2000 Seminar Sub-Committee, **tel: 284 9298**, email: wan2wan@singnet.com.sg*

WONCA Asia Pacific Regional Conference 2000
CHRISTCHURCH, NEW ZEALAND

General Practice into the New Millenium
20-24 June 2000

For more details, contact:
Conference Innovators
PO Box 1370
Christchurch, New Zealand
Tel: +64 3 379 0390, Fax: +64 3 379 0460
Email: wonca@conference.co.nz
Webpage: www.rnzcgp.org.nz

WONCA European Regional Conference 2000
VIENNA, AUSTRIA
2 – 6 July 2000

Patient Care – Values and Trends in General Practice

For more details, contact
Vienna Academy of Postgraduate Medical Education & Research
Alserstrasse 4, A-1090 Vienna, Austria

Tel: +43 1 405 13 8310, Fax: +43 1 405 13 83 23
Email: h.seitner@medacad.org
Internet: http://www.oegam.at

GUIDELINES AND INFORMATION FOR AUTHORS THE SINGAPORE FAMILY PHYSICIAN

Authors are invited to submit articles for publication in ***The Singapore Family Physician*** on the understanding that the work is original and that it has not been submitted or published elsewhere.

The following types of articles may be suitable for publication: case reports, original research, audits of patient care, protocols for patient or practice management and review articles.

PRESENTATION ON THE MANUSCRIPT

The Whole Paper

- Normally the text should not exceed 2000 words and the number of illustrations should not exceed eight.
- Type throughout in upper and lower case using double spacing, with three centimetre margins all round. Number every page on the upper right hand corner, beginning with the title page as 1.
- Make all necessary corrections before submitting the final typescript. Headings and subheadings may be used in the text. Indicate the former by capitals, the latter in upper and lower case underlined.
- Arrange the manuscript in this order: (1) title page (2) summary (3) text (4) references (5) tables and (6) illustrations.
- Send 3 copies of all elements of the article: summary text, references, tables and illustrations. The author should retain a personal copy.
- Their accuracy must be checked before submission.
- All articles are subject to editing.

The Title Page

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

The Summary

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

The Text

The text should have the following sequence:

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

  Drugs must be referred to generically; all the usual trade names may be included in parentheses.

  Dosages should be quoted in metric units.

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

  Do not use patients' names, initials or hospital numbers.
- **Results**: Present results in logical sequence in the text, table and illustrations.
- **Disk & Electronic Production**: If your article is accepted for publication, we may invite you to supply a copy on a 3.5 inch disk, using Microsoft Word software.

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Circulation

The Singapore Family Physician is published quarterly. It is circulated to all Fellows, Diplomate Members, Ordinary Members and Associate Members of the College of Family Physicians Singapore, and to private and institutional subscribers.

The journal is also circulated to all relevant government, professional, medical and academic organisations in Singapore, sister Colleges overseas and to the World Organization of National Colleges and Academies of General Practitioners/Family Physicians (WONCA).

Printed by Prime Packaging Industries Pte Ltd

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