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RESPIRATORY MEDICINE

- Upper Respiratory Tract Infections
- Bronchial Asthma
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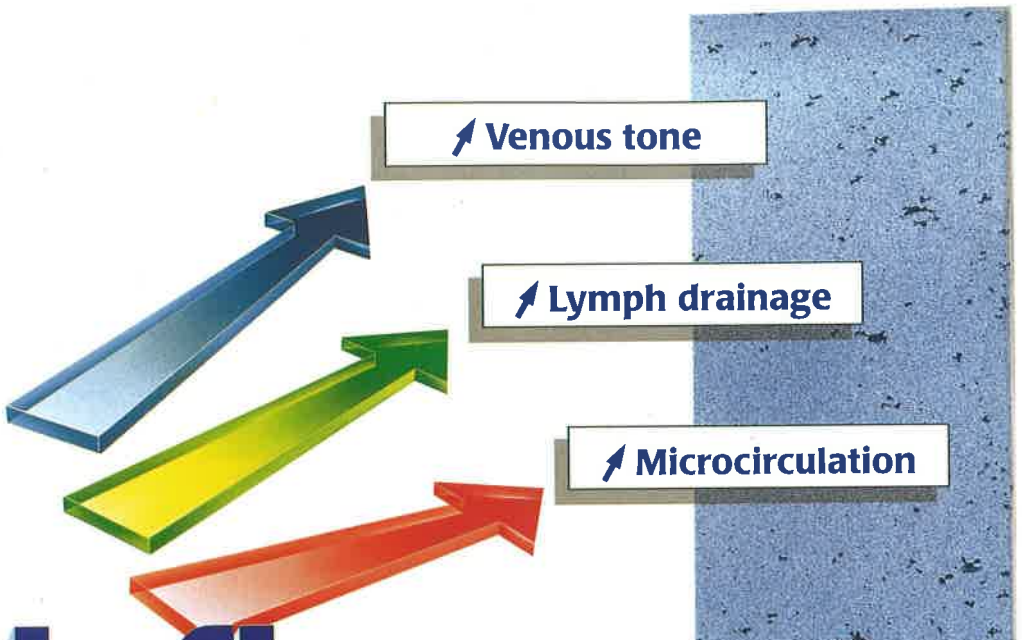

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THE 'BREAD AND BUTTER' OF FAMILY PRACTICE

Upper respiratory infections (URTIs) have long been regarded as the 'bread and butter' of Family Practice. This is so because patients with symptoms suggestive of URTI usually seek consultation with family physicians, and a sizeable proportion of a family doctor's daily patients are diagnosed to have URTI.

In a survey conducted by Emmanuel et al[1] in 1993, URTIs formed 38% of all 'sick visits' to family physicians, far exceeding the next most common condition, which was hypertension (6%). In children up to 14 years of age, more than half of the total consultations (53%) were URTIs. This decreased as the population aged and, in individuals 60 years and older, URTIs constituted 13% of all consultations, after hypertension (22%) and diabetes mellitus (14%).

Managing patients with URTI is an everyday affair for family physicians. Yet, much of the knowledge and skills required for this cannot be obtained from the textbooks. How do family doctors approach this common clinical problem, and how are management decisions made? Most will cite guidelines learnt from other family physicians, and through personal experience. Even with these, there is nothing clear-cut or absolute in the diagnosis of URTI, as well as in its management. How does one decide if the oropharynx is 'injected' or 'not injected'? (If one were to look at asymptomatic throats, many would have the appearance of being 'injected' as well.) How does one decide if the throat infection is viral or bacterial, short of doing a throat swab? Is the presence or absence of fever, or the degree of it, any help at all? What about the colour of sputum, the presence of cervical lymph nodes, the size of the tonsils?

Ultimately, the decision-making process is empirical, and based on a combination of symptoms and signs. What is important to the patient is the severity of symptoms, as well as the degree to which these symptoms incapacitate him or her. However, even these cannot be standardised. It is not useful for doctors to quantify the amount of nasal discharge per day, or the number of times a person coughs or sneezes in an hour, and classify the condition into mild, moderate or severe, such as is possible with bronchial asthma, in terms of the frequency of attacks, and the degree of decrease in peak flow measurements. A person with what is considered 'mild' symptoms may feel that he/she cannot work at all, and another who coughs non-stop may consider this a minor irritation. Besides the difference in individual levels of tolerance, the *perception* of severity of symptoms depends on a host of other factors such as the nature of work, sickness benefit, social events, etc. To complicate the matter further, some patients may complain of symptoms that they are not experiencing and use these as 'tickets of entry' for other matters. Doctors' management decisions will, therefore, depend in part on how much importance their patients attach to the symptoms which they are experiencing or complaining of, and also on whether they are able to uncover the patients' hidden agenda, if any.

In practice, the management of URTI by family physicians is anything but standardised or 'according to the textbooks'. Ideally, to differentiate between bacterial and viral infections, it is recommended that throat swabs be taken. However, in reality, throat swab results can only be available after several days. Does it mean, therefore, that the patient should be asked to wait for several days before the doctor decides what to do? Not only will the patient *not* wait so long, he/she will also think that the doctor does not know his work. In hard reality, the patient

may 'go see the doctor next door'. Thus the conflict between the ideal and the practical, and the dependence on empirical guidelines. Family physicians should not, and need not, make apologies for being practical, as long as the decisions that they make are sound judgementally, and are made with the interests of the patients in mind.

Another issue of contention in management is the need for symptomatic treatment. In Singapore, it is common for family physicians to prescribe cough and cold preparations for the relief of URTI symptoms. In fact, most of these medications can be bought over the counter, or upon recommendation by pharmacists. In certain countries, it is not the practice for such medication to be prescribed, especially for children. The reasons cited include it being better for the patients to expectorate the phlegm than to have the cough suppressed, the efficacy of certain medications such as mucolytics is not proven, etc. It is felt that more emphasis should be placed on patient education, and when patients understand the self-limiting nature of the condition, they would 'bear with it' for those few uncomfortable days, and not expect to be given a prescription for symptomatic relief. However, I would like to be an advocator for the judicious use of symptomatic treatment. Having experienced the inconvenience and discomfort (not to mention the social handicap) of having a runny nose or coughing all the time, and the relief that comes with the use of antihistamines and cough suppressants, I am of the opinion that patients should not suffer unnecessarily, and have their sleep disturbed. This is with the caveat of taking greater care when prescribing in certain circumstances, such as in children or patients with asthma, among others.

Finally, the issue of sick certification is also one that is much discussed. In the 1993 survey, medical certificates issued for URTI constituted 46% of all medical certificates issued, even though URTIs formed only 38% of all consultations. When do patients need sick certification, and when is it not necessary? If necessary, how many days do they need it for? What if the patient is in an occupation that would be risky for him/her and others if he/she is not mentally alert? It is an irony that while we tell patients that their illness is due to viral infection, that they will get well with adequate rest, and yet we are reluctant to give more than one or two days of medical leave. How long does it take for one to recover from a viral infection? Can one work even if one is not completely well? The answer to the first question is an educated guess, and to the second is 'yes, it depends ...' It depends on so many things besides the purely medical facts, such as the nature of the job and the management policy of the employers, the patient's perception and concerns, the past experience of the doctors with 'genuine patients' and 'malingerers', to mention a few.

Thus it can be seen that while URTIs are often dismissed as mild, simple to manage, not worthy of more than a paragraph in a medical textbook, in actual fact there are many unresolved issues, much controversy and uncertainty. There is a need for more research into the practical aspects in the management of URTI. This should be mulit-disciplinary, involving not only the clinicians, but also the microbiologists, pathologists, the psychologists and the sociologists. The essence of Family Practice is that, not only is the biomedical aspect of illness important, the psychological and social aspects surrounding an illness episode are equally (and at times more) important in the total management of the patient.

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Dr Hong Ching Ye

UPPER RESPIRACTORY TRACT INFECTIONS

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SUMMARY

About 95% of all acute respiratory infections involve predominantly the upper respiratory tract. Upper respiratory tract infections are commonest in children and the average child has between 10 and 12 infections per year. 90% or more of respiratory infections are due to viruses.

Keywords: *colds, pharyngitis, virus, streptococcus, symptomatic*

INCIDENCE

About 95% of all acute respiratory infections involve predominantly the upper respiratory tract. They are commonest in children. Between 2 and 5 years, the average child has between 10 and 12 infections annually, with 2 peaks, one at 18 - 24 months reflecting the beginning of socialization; the other between 4 and 5 years, when the child is commencing kindergarten and school. The adult number of 3 to 4 per year is not reached until 11 or 12 years.

Infections primarily affecting the upper respiratory tract, i.e. nasal passages, paranasal sinuses, pharynx, tonsils and middle ear are classified as upper respiratory tract infections (URTIs). Most respiratory illness, however, affect the upper and lower portions of the tract simultaneously or sequentially; others predominantly involve specific portions of the respiratory tree.

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UPPER RESPIRATORY TRACT INFECTION

This includes 4 different disorders:

1. Colds (coryza)
2. Sinusitis
3. Acute throat infection (pharyngitis and tonsilitis)
4. Otitis media.

The clinical manifestations of illness occurring in any patient depends on the interaction of three factors:

1. The infecting agent
2. Host factors
3. Environmental factors.

Infecting Agents

90% or more of respiratory infections are due to viruses, while bacteria are responsible for some upper and lower respiratory tract infections; their exact role in causing diseases is often difficult to determine.

Respiratory syncytial virus is the principal cause of bronchiolitis in infancy. It is also responsible for other middle and lower respiratory tract infections. With successive exposure there is

Table 1: Pathogens Responsible for URTIs

Viruses
- Respiratory syncytial
- Parainfluenza types 1, 2, 3
- Influenza types A, B
- Rhinovirus
- Adenovirus

Bacteria
- Beta-haemolytic streptococcus
- Streptococcus pneumoniae
- Haemophilus influenzae
- Mycoplasma pneumoniae

usually a progressive decrease in the severity of the illness. In the older individuals, the organism may be associated with mild apyrexial URTIs.

With parainfluenza viruses, epidemics occur. These viruses can produce a wide variety of illnesses from mild pharyngitis, laryngotracheobronchitis to severe pneumonia and are the major cause of croup.

Influenza B causes mild outbreaks of influenza while influenza A causes pandemic influenza. At risk are those patients with chronic lung disease and the elderly, but occasionally young adults may suffer overwhelming infections complicated by influenzal or streptococcal pneumonia, myocarditis and encephalitis. In children, the viruses play a minor role.

Rhinoviruses are primarily responsible for the common colds. It is believed that rhinovirus may precipitate acute exacerbations of chronic bronchitis and may occasionally cause pneumonia in infants.

Adenoviruses may cause pharyngitis and conjunctivitis in adults, severe bronchitis in children and rarely severe pneumonia in infants.

Bacteria are responsible for only about 5% of infections, but these tend to be more serious ones. Group A beta-haemolytic streptococcus is the only cause of bacterial pharyngitis and tonsilitis. *Haemophilus influenzae* causes acute epiglottitis. It is also an important cause of otitis media and occasionally pneumonia. Mycoplasma pneumoniae, in younger children, causes coryzal illnesses and pharyngitis and also bronchitis.

Host Factors

Host factors are of the greatest importance in determining the pattern of illnesses. The most serious respiratory disease occurs in the first 3 years of life. The overall incidence is maximum between 2 and 5 years. A substantially higher incidence of lower respiratory tract infections is found in boys under the age of 6. In some patients with frequent, recurrent respiratory tract infections, it is possible to demonstrate an abnormality in immune function of phagocytes or lymphocytes. There is a definite relationship between increased frequency of lower respiratory tract infection and premature babies with hyaline membrane disease.

Environmental Factors

There is a strong and proportional correlation of most severe lower respiratory infections with the quality of maternal care, parental smoking habits, breast feeding, family size and the lower social economic status.

CLINICAL MANIFESTATIONS

The Common Cold

The onset is sudden with fever (39 to 40 degrees Celsius), irritability and sneezing. Nasal discharge soon begins, quickly leading to nasal obstruction and interference with feeding in infants who are nose-breathers. Signs of moderate respiratory distress may occur. During the first 2 or 3 days the eardrums are usually congested and fluid may accumulate behind the drum. A few infants may vomit and have diarrhoea. The febrile phase may last from a few hours to 3 days and may recur with purulent complications.

In older children and adults characteristically the initial symptoms are dryness, irritation in the nose and throat. Within hours, sneezing, chilly sensations, muscle aches, a thin nasal discharge and sometimes cough follow.

Nasal obstruction leads to mouth breathing, and

this, through drying of the mucous membranes of the throat, increases the sensation of the soreness. The acute phase lasts from 4 to 10 days.

Complications occur due to the invasion of paranasal sinuses and other portions of the respiratory tract by bacteria. The cervical lymph nodes may enlarge and suppurate. Otitis media is frequently seen in infants. Complications in the lower respiratory tract such as laryngitis, bronchitis and pneumonia occur much less frequently, but again are more common in infants. Purulent sinusitis occurs more frequently in older children than in infants.

Treatment is symptomatic; adequate fluid intake, paracetamol, clearing of the nose with cotton bud, instillation of nasal vasoconstrictor, a highly humidified environment can be helpful in babies with nasal obstruction. Oral antihistamines and pseudoephedrine have not be shown to be of benefit. There is no evidence that antibiotics limit the duration of coryzal symptoms nor reduce the likelihood of secondary bacterial infection. However, the presence of a purulent nasal discharge does not signify secondary bacterial infection; it may just be a late feature of a viral infection.

Sinusitis

The paranasal sinuses, an extension of the nasal passages, become involved in URTIs. The sinuses contribute to the production of mucoid and purulent nasal discharge and when the drainage is obstructed, secondary infection ensues.

Accurate diagnosis of sinusitis requires the presence of impaired sense of smell, nasal obstruction and purulent rhinorrhoea as well as facial pain. Special investigations are generally not helpful. Coronal computerised tomogram (CT) should probably be done by an ENT specialist in surgical planning rather than diagnosis.

Most cases of acute bacterial sinusitis are due to *Streptococcus pneumoniae* and *Haemophilus influenzae*. Anaerobic organisms such as *Streptococcus pyogenes*, and *Bramhamella catarrhalis* can occur in about 20% of cases. In chronic sinusitis, anaerobic organisms account for about 50% of cases.

Complications of sinusitis are potentially fatal, but fortunately rare. Orbital complications range from periorbital cellulitis, to orbital abscess with rapidly ensuing blindness. Intracranial complications include meningitis, abscess formation and cavernous sinus thrombosis.

The choice of antibiotic is directed against the most likely pathogens. Amoxycillin with clavulinic acid or cefuroxine for 2 weeks would be effective in most cases.

Acute Pharyngitis

Pharyngeal involvement is part of most URTIs and is also found with various acute generalised infections. The term actually refers to conditions in which the principal involvement is in the throat.

The disease is uncommon in children under one, the incidence then increases to a peak between the 4th and 7th years and continues to occur throughout thereafter.

Acute pharyngitis is generally caused by viruses. The only common bacteria other than the diphtheria bacillus are group A Beta-haemolytic streptococci, which account for 15% of cases. Bacterial pharyngitis is most prevalent in children between 4 and 13 years.

Viral pharyngitis is generally a disease of relatively gradual onset, with fever, malaise, anorexia and moderate throat pain. Hoarseness, cough and rhinitis are also common. Even at its peak, pharyngeal inflammation is slight, though occasionally it is severe and small ulcers may form on the soft palate and the posterior pharyngeal wall. Exudates may appear on the lymphoid follicles of the palate and tonsils. Cervical lymph nodes are usually moderately enlarged and may or may not be tender. Extensive exudates on the pharynx, tonsils and soft palate are typical of infectious mononucleosis.

Streptococcal phraryngitis begins with headache, abdominal pain and vomiting in children. The fever may be as high as 40 degrees Celsius.

The throat becomes sore and there are tonsillar enlargement, exudation and pharyngeal erythema. Tender cervical lymphadenopathy occurs early. Fever may continue for 4 days.

Viral and streptococcal pharyngitis are usually indistinguishable clinically. Conjunctivitis, rhinitis, cough and hoarseness occur rarely with streptococcal pharyngitis and the presence of 2 or more of these signs or symptoms suggests viral infection. If the child is under the age of 4, streptococcal pharyngitis is unlikely.

Treatment is to relieve symptoms, prevent complications and to eradicate the infecting streptococcus. If streptococcal infection is thought likely, oral penicillin for 10 days is the standard treatment and a full 10 days is necessary if streptococci are to be eradicated. For patients who do not commit to a full oral course, a single injection of 'bicillin' (a mixture of benzathine penicillin, procaine penicillin and benzole penicillin) can be a painful alternative.

With viral infections, the complication rate is very low, although purulent bacterial otitis media may occur. In debilitated children both viral and streptococcal infections may lead to large, chronic ulcers in the pharynx. With streptococcal disease, peritonsillar abscess occasionally occurs, as do sinusitis, otitis media and rarely meningitis. Since acute glomerulonephritis and rheumatic fever may follow streptococcal infections, it is desirable to re-examine children with proven streptococcal disease within 2 to 3 weeks after illness.

Mesenteric adenitis is occasionally associated with pharyngitis of either viral or bacterial origin.

Acute Otitis Media

The middle ear cavity, an extension of the upper respiratory tract, is often affected in URTIs.

Acute otitis media has the following clinical features: earaches (unilateral or bilateral), deafness, inflamed tympanic membrane, sometimes with a purulent discharge. Constitutional disturbance of varying severity may be present.

Younger children are susceptible due to their immature eustachian tube and immune system. With an URTI, the eustachian tube and middle ear become inflamed, a negative pressure develops in the middle ear due to eustachian tube blockage and the tympanic membrane retracts. This retraction produces pain. If the pressure persists a middle ear effusion then develops and the child becomes deaf. When the effusion becomes secondarily infected with bacteria, acute suppurative otitis media results.

The major pathogens are *Streptococcus pneumoniae* and *Haemophilus influenzae*. The condition is for most children self-limiting and stops spontaneously once the eustachian tube and immune mechanisms have grown up with the child.

Regular auto-inflation (blowing against pinched nostrils with the mouth closed) can help to improve eustachian function. When nasal disease with oedema exists due to infection or allergy, vasoconstrictor or steroid nose drops can be used. Antibiotics should be used in painful otitis media with effusion. The drug of choice is amoxycillin in a 5-day course. When the effusion is purulent, a 14-day course is required. Antibiotic drops such as suframycin, gentamycin or chloramphenicol have been shown to shorten the period of drainage and promote early resolution of infections. Recurrent episodes of acute otitis media without effusion should be considered for specialist referral. Inserting grommets will effectively cure the symptoms and the disease.

ENT specialist referrals are also indicated in cases of recurrent suppurative otitis media or recurrent otitis media with or without effusion despite adequate treatment, associated 25 to 30 dB hearing loss, the presence of speech or language delay, or the presence of persistent perforation of tympanic membrane with or without the suspicion of cholesteatoma. Mastoiditis is a surgical emergency.

PREVENTION

Respiratory infections are a major cause of morbidity and mortality especially in the very young and the very old. They cause considerable anxiety to parents and the substantial health cost

in terms of medical expenses, school absenteeism and work loss to the community.

With the exceptions of epidemic strains of Influenza A, *Bordetella pertussis* and *H. influenzae* type B, effective immunisation against the common and more serious respiratory infections does not seem to be a practical possibility in the near future. The large number of viruses involved and the incomplete understanding of the immunological processes that result from infection with respiratory viruses are the major setbacks.

Because of the ubiquity of the common cold, it is not possible to isolate children and the infirm from this condition. Since complications may be relatively serious, attempts should be made to protect them from contact with potentially infected persons, particularly other children. Avoidance of large gatherings is advisable.

Modification of environmental factors, cessation of smoking, improvement of maternal care by health education perhaps give greater opportunity for prevention.

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MANAGEMENT OF LOWER RESPIRACTORY TRACT INFECTIONS IN GENERAL PRACTICE

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INTRODUCTION

Lower respiratory tract infection (LRTI) includes acute bronchitis, pneumonia and infective exacerbations of chronic obstructive airway disease (COAD). The differences between individual susceptibility, bacterial aetiology and treatment outcome differs between a healthy subject and one with co-existing illness. The diagnosis of pneumonia can be difficult as typical signs and symptoms as described in the textbooks may not always be present. Missed diagnosis and delayed hospital referral can result in increased morbidity and mortality.

DIAGNOSIS OF LRTI

Acute bronchitis is often a complication of upper tract infection. The cough is often productive, and there may be associated wheeze and dyspnoea. The chest X-ray (CXR) is clear.

Pneumonia is an acute illness with cough (which may produce purulent sputum), fever and the presence of new infiltrate(s) on CXR. These features may not be present all at the same time.

In infective exacerbations of COAD, the CXR changes may be subtle: the diagnosis then depends on the history: increased cough, increased sputum production or dyspnoea. Fever is often absent in the elderly.

The patient with LRTI may be previously healthy or have co-existing illness (Table 1). Those who are young and previously healthy can often be managed on an outpatient basis, whereas elderly patients and those with co-existing illness would benefit from early referral to hospitals, because disease progression can be extremely rapid in some cases with a fatal outcome within 24 hours[1,2].

Causative Organisms

The infecting organisms can be identified only in

Table 1: Characteristics of Hosts that may Influence Treatment and Outcome

CHARACTERISTICS	EXAMPLES
1. Age	
2. Co-existing chronic pulmonary diseases	chronic bronchitis, emphysema, bronchiectasis, cystic fibrosis
3. Co-existing systemic illness	diabetes mellitus, renal failure, chronic alcholism, chronic steroid usage
4. Immuno-compromised state	hematogenous malignancy, chemotherapy, post-transplant, acquired immune deficiency syndrome
5. Institutionalised	old folks home, army camp

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50-70% of patients with LRTI (Table 2)[1,3,4]. Viruses account for most cases of acute bronchitis and a smaller number of patients with pneumonia. Viruses damage the respiratory epithelial defence mechanisms, thus predisposing the subject to bacterial superinfection, especially staphylococcal infection, which can be rapidly fatal.

Table 2: Common Infecting Organisms in LRTI

	ORGANISM	*EXAMPLES*
1.	Viruses	influenza virus, parainfluenza, adenovirus, respiratory syncytial virus
2.	Bacteria	Strept. pneumoniae, Mycoplasma, H influenzae, Staph. aureus, M.catarrhalis, Chlamydia
3.	Mycobacterium tuberculosis	

Although the incidence of pulmonary tuberculosis (PTB) has declined over the past 20 years, *Mycobacterium tuberculosis* is still a common cause of community-acquired pneumonia (CAP) in Singapore, accounting for 10-20% of identifiable organisms[3]. Of the bacterial infections, *Strept pneumoniae* is the commonest, followed by *Mycoplasma pneumoniae, Haemophilus influenzae, Staph aureus* and *Moraxella catarrhalis*. Sporadic cases of *Pseudomonas pseudomallei* infection are seen, mostly in diabetics, often with multi-organ involvement and the mortality rate is high despite appropriate antibiotic treatment.

Legionella infection is uncommon, accounting for 1-5% of CAP cases that are admitted into hospitals. *Chlamydia pneumoniae* is recently recognized as a significant pathogenic organism, but its detection by serology is difficult, as positive reactions may be due to other non-respiratory chlamydial infections.

Patients with underlying lung diseases, especially bronchiectasis, suffer from episodic infective exacerbations. Apart from the common infecting organisms (Table 2), they are at a higher risk of being infected by Gram-negative organisms such as *Klebsiella pneumoniae* and *Pseudomonas aerogenosa.* They may also have mixed infection with anaerobes. These community-acquired Gram-negative organisms are often sensitive to multiple antibiotics such as co-trimoxazole and second generation cephalosporins, unlike the nosocomial Gram-negative bacterial pneumonia, which are often multi-resistant.

As the number of patients who are immunocompromised increases, we are also seeing more unusual organisms, such as fungus, cytomegalovirus (CMV) and *Pneumocystis carinii* (PCP) infection. This group of patients require urgent referral to hospitals as early diagnosis and appropriate treatment is vital.

Clinical Signs

Patients with LRTI present with a cough, sometimes productive; there may be associated dyspnoea, fever and chest pain. The elderly may be more ill from constitutional symptoms such as anorexia, lethargy and dehydration.

Patients with uncomplicated acute bronchitis usually do not have abnormal clinical signs apart from occasional rhonchi. In pneumonia, the classic signs of lobar consolidation, such as bronchial breath sound, increased vocal resonance and localised inspiratory crackles, are seen in less than 25% of cases. A pleuritic rub may be present.

Atypical Pneumonia

In elderly patients and in those with mycoplasmal, chlamydial and legionella infections, the clinical presentations are more often "atypical", with non-respiratory symptoms dominating (Table 3). Lung signs are usually non-localising and, if present, do not correlate with CXR findings.

Treatment with appropriate antibiotics in these patients results in rapid resolution of symptoms and radiological changes, though relapses can occur if treatment is stopped prematurely.

Clinical Investigations

Chest radiography (CXR) is indicated for all

Table 3: Extrapulmonary Features of Atypical Infection

ORGANISM / SYSTEM	*CLINICAL FEATURES*
Mycoplasmal:	
• gastrointestinal tract	anorexia, vomiting, diarrhoea, hepatitis
• musculoskeletal	myalgia, arthralgia, polyarthritis
• blood	cold hemagglutinins, haemolytic anemia
• skin	macular rash, erythema multiforme
• others	myocarditis, bullous myringitis, aseptic meningitis
Legionellosis:	
• gastrointestinal tract	abdominal pain, watery diarrhoea
• central nervous system	confusion, stupor, peripheral neuropathies
• renal	proteinuria, haematuria
Chlamydial:	
• usually mild or subclinical illness with no specific clinical features	

patients suspected of pneumonia; especially if there are localised lung signs, haemoptysis or evidence of effusion[2,5]. A parapneumonic effusion may be present in up to 40% of bacterial pneumonias. Ideally both postero-anterior and lateral views should be requested for, in order not to miss left lower lobe lesions that are situated behind the cardiac shadow.

Actively expectorated sputum specimens from the lower respiratory tract should be collected and processed immediately for Gram-smear and bacterial culture, as well as smear for acid-fast bacilli (AFB) and culture for *Mycobacterium tuberculosis*. Sputum collection should preferably be done before starting antibiotics. However, up to 40% of patients will be unable to produce satisfactory samples, and treatment should not be delayed while awaiting samples. Induction of sputum production may be achieved by nebulizing the patient with 3% hypertonic saline. Result of sputum induction can be improved when coupled with chest physiotherapy.

Any pleural fluid collection in a patient with pneumonia should be aspirated promptly[6]. The aspirate is to be examined for bacteriology, biochemical data and cell counts, in order to identify the group of patients with high risk of developing an empyema. Pleural aspiration is best done with image-guidance.

Full blood count including a differential white cell count is helpful in identifying patients who are more septic, noting that elderly patients may not be able to generate a leucocytic response.

Serological tests for mycoplasmal infection and legionellosis are indicated in suspected atypical infections. Two samples of blood specimen should be taken 2 weeks apart and a 4-fold in IgG titre will suggest recent infection. As the results cannot be obtained rapidly, the diagnosis and treatment of atypical pneumonias are usually based on clinical assessment. Serological tests for chlamydial and viral infections are costly and time-consuming; they are usually reserved for research purposes. Anti-HIV antibody testing and a CD4 count should be done if there is clinical suspicion of acquired immune-deficiency syndrome (AIDS) or the patient has risk factors for HIV infection.

HOSPITAL ADMISSION CRITERIA

There is a group of patients who will require early hospital referral and admission (Table 4)[1,5]. Elderly patients, especially those with concomitant illnesses, may not have fever at presentation. They may even be hypothermic. Subtle signs like tachycardia, tachypnoea and dehydration should be sought for. As they can deteriorate rapidly and unexpectedly, in-patient observation and management can be life-saving.

TREATMENT

Patients with acute bronchitis may require only rest and symptomatic treatment. However, they need to be educated and reassured that an antibiotic is often unnecessary as the commonest cause is viral.

Table 4: Adverse Factors that Suggest Need for Hospital Referral

CHARACTERISTICS	*EXAMPLES*
Patient factors:	
• age	older than 60 years
• co-existing illness	COAD, diabetes mellitus, renal failure, ethanol abuse, cancer
• risk factor for aspiration	strokes, altered mental status
Clinical signs:	
• respiratory	respiratory rate > 30/min, cynaosis
• general	hypotension, confusion,
Social:	
• poor home care facility	

Table 5: Antibiotic Recommendations

ANTIBIOTICS	*EXAMPLES*
Young adults not requiring in-patient therapy:	
• oral macrolide	erythromycin, clarithromycin, azithromycin
Adults above 60 years but without other factors:	
• beta-lactam with beta-lactamase inhibitor	Sultamicillin
• extended spectrum oral macrolides	clarithromycin, azithromycin
Patients with co-existing illness:	
• oral second generation cephalosporins	cefuroxime
• beta-lactam with beta-lactamase inhibitor	Sultamicillin
• extended spectrum oral macrolides	clarithromycin,

The choice of antibiotic therapy in CAP is simple if the infecting organism is known. However, treatment of CAP in General Practice is often empirical, based on the patient's clinical picture and a knowledge of common infective organisms[1,2,3,5]. A young patient with fever, dry cough and multiple constitutional symptoms such as headache, earache and diarrhoea is more likely to have atypical pneumonia such as mycoplasmal infection; whilst another young patient with abrupt cough and fever, rusty sputum or pleurisy is likely to be suffering from pneumococcal pneumonia. A patient with a recent history of viral URTI, or a diabetic patient with poor skin condition, may be predisposed to *Staph aureus* infection.

The guidelines described in Table 5 for the empirical treatment of CAP may be appropriate for the ambulatory patient, while awaiting results of sputum Gram-smear and culture, blood cultures and serological tests.

Role of New Antibiotics in the Treatment of LRTI

A plethora of orally active antimicrobials, all actively promoted by the manufacturers to be effective in CAP, have emerged recently[7,8]. Knowledge about these new oral agents is important in order to make an informed decision regarding the safest, cheapest antimicrobial with the most narrow spectrum appropriate for each patient.

Macrolides

Newer macrolides (Table 6) have been developed to overcome the gastrointestinal side effects that are well known to erythromycin-users. They also have longer elimination half-lives allowing for longer dosing intervals, thus improving patient compliance.

Azithromycin and clarithromycin have superior intracellular penetration property, hence more effective against intracellular organisms, including some of the atypical mycobacteria. They have extended spectrum of activity against *H. influenzae* and *Staph aureus*, though a higher dose (e.g. clarithromycin 500 mg bid) may be required.

Table 6: Macrolides*

	DOSAGE (DURATION)	COST/ TABLET	COST/ COURSE
Erythromycin	500 mg qid (14 days)	$ 0.15	$ 16.00
Erythromycin ES	800 mg bid (14 days)	$ 0.50	$ 28.00
Clarithromycin	250 mg bid (14 days)	$ 3.30	$ 92.40
Azithromycin	500 om (3 days)	$ 5.21	$ 31.26

* Possibility of nausea and vomiting, abdominal pain and diarrhoea; these side effects are less severe with newer macrolides;

* Drug interactions are supposedly less frequent with the newer macrolides.

Beta-lactams

Oral cephalosporins such as cephalexin and cefuroxime are more stable to the beta-lactamases. The choices are myriad but they all have similar spectrum of activity. The new generations boast less frequent dosing (o.m. or b.i.d.) and increased activity against Gram-negative organisms, but they are more costly and also less active against streptococci. They are probably useful in COAD with infective exacerbations where there are often mixed organisms present, and should not be prescribed for CAP in a young and previously healthy patient.

Quinolones

Oral fluroquinolones (cipropfloxacin, ofloxacin and perfloxacin) are especially active against the Gram-negative bacteria. The twice daily dosing interval is also convenient. However they are NOT recommended for office use in CAP. They should be reserved for hospitalised ill patients with proven Gram-negative sepsis, and for patients with nosocomial pneumonia. They have poor *in vivo* activity against *Strept pneumoniae* and anaerobes, and resistance rapidly develops if they are used indiscriminately.

Response to Treatment

A clinical response is defined as deffervecence of fever, reduced cough and sputum production, and improved dyspnoea. If parenteral therapy is used, it should continue till patient has been afebrile for more than 24 hours before switching to oral treatment[1,2]. The total course of antibiotic therapy is usually 7-14 days; longer duration is required in the presence of abscess and empyema. The antibiotic regime may need to be altered if there is no clinical improvement after 3 days or if the patient deteriorates rapidly. Treatment of complicated parapneumonic effusions and pulmonary tuberculosis is considered separately.

Radiographic resolution is slow (4 -6 weeks) in comparison to clinical response. The timing of "follow-up" CXR is therefore highly controversial. In general, a CXR can be repeated in about 2-4 weeks if patient is well and responding to treatment. If the patient remains unwell, or new signs have developed (clinically more lobes are involved, appearance of effusion or haemoptysis), an earlier repeat film is indicated. Where tumour or tuberculosis is suspected, a medium-term (8 weeks) follow-up CXR should be done; by then the sputum mycobacterial culture results will also be available for review.

Adjunctive Therapy

For patients with pneumonia, inspissated secretions are common due to associated dehydration as a result of poor fluid intake, fever and rapid mouth breathing. Careful rehydration is therefore essential, and oral fluid is better as it allows a patient to be ambulated and avoids thrombophlebitis.

Mucolytic agents have no proven benefit in the treatment of pneumonia and should not be prescribed freely. Intravenous bromhexine, oral and nebulized N-acetylcystein have showed anecdotal response in some patients. Chest physiotherapy and nebulized saline may be more helpful in those patients with difficulty in expectorating. Cough suppresant may be necessary if the cough disturbs the patient's sleep at night.

Anti-pyretics may be useful if the high temperature is distressing to the patient. It also provides temporary relief for myalgia. Throat gargles may help, albeit short-term, with the

soreness caused by excessive coughing.

Patients with underlying obstructive airway diseases should receive regular bronchodilator therapy. Occasionally, a short course of steroid treatment (7-10 days) is helpful. Supplemental oxygen is useful if the patient is not in hypercapneic respiratory failure. Ambulation is always actively encouraged to prevent additional hypostatic pneumonia.

Preventive measures such as influenza and pneumococcal vaccines in selected individuals, and smoking cessation in COPD are useful in reducing the incidence of CAP and its related morbidity and mortality.

COMPLICATIONS OF LRTI

Patients with LRTI should be reviewed at 3-5 day intervals to assess the response to therapy and to exclude possible complications. If clinical response is slow, or when there is clinical deterioration, the patient should be carefully re-evaluated (Table 7). A change of antibiotic or hospital referral may be indicated, especially if loculated empyema, proximal tumour obstruction or bronchiectasis are suspected.

Severe CAP can progress to respiratory failure requiring mechanical ventilation. Acute myocaridal infarction and heart failure may also be pericpitated by infection in elderly patients and patients with co-existing diabetes, hypertension or renal failure.

CONCLUSIONS

The mortality rate is low for patients with LRTI that respond to outpatient therapy. For those requiring hospital admissions, it can go up to 25%. Older patients and patients with multiple adverse factors (Table 4) are at an increased risk of dying from CAP if not managed promptly and adequately. Knowledge of locally prevalent organisms and their sensitivity help in the selection of the most appropriate empiric antibiotic and thus improve outcome. The choice of antibiotic is important as it affects patient's compliance: o.m. or b.i.d. dosing encourages compliance, while expensive medications will lead to some patients purchasing inadequate supply or underdosing. A close rapport between the physician and the patient, advice on compliance and the need for a repeat visit will help to improve the success rate of out-patient treatment for CAP.

Table 7: Causes of Treatment Failure in CAP

CAUSE	*CLINICAL MANIFESTATION*
Incorrect diagnosis	pulmonary oedema, pulmonary embolism, organising pneumonitis
Incorrect antibiotic	PTB, fungus, multiresistant Gram-neg organisms
Unrecognised underlying disease	HIV, tumour
Unrecognised complications	empyema, abscess, superinfection
Unrecognised infection elsewhere	septic arthritis, endocarditis, etc

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MANAGEMENT OF ACUTE BRONCHIAL ASTHMA IN ADULTS

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Summary

Acute exacerbations of disease in asthmatics can be effectively treated at the nearest outpatient clinic. The family doctor should be familiar with the clinical features of severe airways obstruction and also be able to identify high risk patients for more intensive treatment and monitoring. Prompt diagnosis and adequate treatment aborts the attack and prevents further progress to a more severe stage of the illness. Treatment is directed at (1) the bronchoconstriction and (2) the airway inflammation. The best bronchodilation is achieved with a maximal dose schedule of an inhaled beta-2 agonist drug. The administration of beta-2 agents may have to be repeated at close intervals in acute severe asthma. The most effective anti-inflammatory treatment for asthma is systemic corticosteroids. Oral administration of steroid is adequate and a one to two week course should be started in the clinic for most patients. Prophylactic medication with long term inhaled steroids should be considered for most patients who experience severe or frequent exacerbations.

Keywords: *Acute severe asthma, beta agonists, corticosteroids, clinic.*

INTRODUCTION

Acute spontaneous exacerbations of airways obstruction occur with variable frequency among asthmatic patients. While acute exacerbations are potentially life threatening events, the majority of patients will respond promptly to medical treatment and do not require hospital admission. Any delay in starting effective treatment may allow progression to a more severe stage of the illness and increase the risk of a fatal outcome. Most acute attacks of asthma can and should therefore be diagnosed and treated by the nearest doctor which, in Singapore, would usually be the neighbourhood general practitioner or polyclinic doctor. The primary attending doctor would also be the best person to identify the patients who are at high risk for fatal asthma for more intensive treatment, closer follow up and early hospital referral.

In recent years there has been wide international consensus on the management strategies for bronchial asthma with publications of comprehensive guidelines by thoracic societies from different countries. In this article I will describe a management approach to acute severe asthma in the outpatient clinic in accordance with published guidelines and appropriate for the Singapore context.

PATHOPHYSIOLOGY OF ACUTE ASTHMA

Understanding the underlying mechanisms of disease is the key to planning rational treatment in acute asthma. Acute exacerbations of asthma are episodes of progressive breathlessness, wheeziness, cough and chest tightness which

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may wax and wane over hours to days. The primary abnormality is contraction of airway smooth muscle causing diffuse airways obstruction. The airways are also narrowed by influx of inflammatory cells, vascular congestion, oedema, hypertrophied submucous goblet cells and abnormal mucus secretion into the lumen with organization into mucus plugs. There might also be gross injury to the airway lining stratified columnar epithelial cells with extensive areas of desloughing and the sloughed epithelium contributing to the luminal plug. The increased airway resistance results in air-trapping behind occluded peripheral airways and hyperinflation. Increased work of breathing is incurred by breathing against high airway resistance and at high lung volumes. This increased respiratory work load contributes to the sensation of dyspnoea in acute asthma. The abnormality of ventilation is accompanied by impairment of gas exchange function. Mismatching of ventilation and perfusion results in respiratory failure which progresses from Type I (hypoxaemia with normal or low CO_2 tensions) in mild/moderate disease to Type II (hypercapnic) in severe airways obstruction.

The airway smooth muscle contraction may be relieved within minutes to hours following treatment by bronchodilator drugs. By contrast, the other abnormalities of the airways such as inflammatory cell infiltration, vascular hyperaemia, oedema, epithelial damage and mucus plugging do not respond to bronchodilator drugs. Moreover, these inflammatory changes may persist for days to weeks and account for persistent airways obstruction, chronic respiratory symptoms and airway hyperactivity which predispose the patient to early and frequent relapses.

The objectives in the treatment of acute asthma are therefore twofold: (1) to abort the **brochospasm** for rapid symptom relief and (2) to control the **airway inflammation** and prevent relapse.

DIAGNOSIS AND ASSESSMENT OF ACUTE ASTHMA

During an acute exacerbation of disease most patients will present with the typical symptoms and signs of acute asthma. There is however, **poor correlation** between the perception of symptoms by patients, detection of physical signs by the doctor and the actual severity of airways obstruction during an acute asthma episode. There is no single symptom or sign which best detects disease severity. The assessment of acute asthma is based upon a composite picture of recent history of asthma including self medication, current level of symptoms including time and nature of last administered medication plus physical signs and objective measurements of the severity of airways obstruction.

Acute exacerbations of asthma are usually preceded by prodromal respiratory symptoms for a variable period. These symptoms arise from exposure to triggering factors in the environment which may include aero-allergens, viral upper respiratory infections or drugs. The initial symptoms may be very mild and localized to the oropharynx such as nasal stuffiness, sneezing and throat irritation followed by a persistent cough. The symptoms of persistent airways obstruction then arise with wheezy dyspnoea, chest tightness and more coughing. Most patients would at this stage attempt to relieve their symptoms by the use of quick acting bronchodilator drugs – usually inhaled beta agonists via metered dose inhalers. Failure to attain symptom remission with self administered drugs or the perception of a more than usually severe episode would then prompt a visit to the nearest clinic or emergency department.

A subgroup of patients may experience predominantly **nocturnal symptoms** during an exacerbation. They are woken up at 2-4 am by cough and/or wheeze but are asymptomatic in the daytime. Some of these patients may not seek treatment until their symptoms persist into the daylight hours. All asthmatics should be questioned about their quality of sleep and warned that any sleep disturbance due to respiratory tract symptoms for more than one or two nights is unacceptable and an indication for increasing their medication.

In the initial assessment of a patient with acute asthma the doctor should look for specific features of severe obstruction (Table 1) in addition to the evaluation of usual symptoms and chest auscultation. The peak expiratory flow rate (PEFR) is an objective index of airways obstruction and should be an integral part of the

Table 1: Features of Severe Asthma

1. Too wheezy / breathless to complete a sentence in one breath
2. Respiratory rate > 25 /min
3. Pulse rate > 110 / min
4. Use of accessory neck muscles
5. Diaphoresis
6. Inability to lie supine
7. Peak expiratory flow rate < 50% predicted or best

Table 2: The High Risk Patient

1. Previous near-fatal asthma#
2. Using 2 or more beta-2 Metered Dose Inhalers per month*
3. Exacerbated while on oral steroids
4. Poor perception of severe airways obstruction
5. Poor compliance
6. Psychiatric illness

Near Fatal Asthma
   1. *Intubated and mechanically ventilated*
   2. *Seizure of syncopal attack from severe hypoxaemia*
   3. *Hypercapnic respiratory failure*

* *Excessive use of beta-2 agonists via a metered dose inhaler is an independent risk factor for fatal and near-fatal asthma. It is unclear if beta-2 medication is a casual factor or merely a marker of asthma*

overall assessment of asthma severity. The best of three reproducible PEFR should be noted down and expressed as either percentage of normal predicted value or the patient's personal best. The assessment should be repeated following each round of treatment. Failure to improve promptly after adequate initial treatment is indicative of status asthmaticus. A silent chest, frank cyanosis and obvious fatigue or exhaustion are signs of impending respiratory collapse.

The High Risk Patient

Asthma is a potentially lethal but eminently treatable disease. The patient who is at risk to die from asthma may be identified by certain features in his/her clinical profile (Table 2). A patient with any one of these features should be treated with extreme caution and intensive prophylactic anti-inflammatory medication instituted at the first sign of trouble. The threshold for referral to hospital and starting systemic corticosteroids should also be lowered for these patients. They should not be discharged from the clinic before arrangements for close follow up have been firmly established.

MANAGEMENT

Bronchodilator Treatment

The cornerstone in the treatment of acute severe asthma is the administration of bronchodilator drugs. There are three classes of bronchodilators, namely (1) beta-agonists, (2) theophyllines and (3) cholinergic antagonists. Beta-agonists are the choice drugs in treating acute asthma. The main therapeutic effect of beta-agonist drugs in asthma is to engage the beta-2 adrenergic receptors on airway smooth muscle and induce relaxation. They have a more rapid onset of action and greater peak effect in improving lung function than either theophyllines or cholinergic antagonists.

The immediate objective in the treatment of acute severe asthma is to achieve maximal bronchodilation and symptom relief in the shortest time. This can usually be attained by the prompt administration of maximal dosages of a beta-agonist (Table 3). Beta-agonists are best administered via the **inhalation** route. The most convenient way to deliver inhaled beta-agonists in the clinic is via **wet nebulization** from an electrically powered air compressor. While supplementary oxygen in desirable , it will not be needed for the majority of patients who respond rapidly to initial treatment. Unless a complete remission is experienced with the first dose, inhalational therapy should be repeated two to three times in rapid sequence at ten to fifteen minute intervals to achieve a maximal effect. The patient should be carefully reassessed after each round of inhalation to decide upon the next one. Equally effective bronchodilation and clinical outcomes have been reported in patients with emergency room asthma treated with beta agonists delivered via **metered dose inhalers with a large volume spacer** and even **dry powder** preparations via an efficient turbulent flow device (**Turbuhaler**). Repeated, sequential

Table 3: Bronchodilator Treatment

The following regimens are equally effective:	
INHALATION (TOPICAL)	
1. Wet aerozolization *Salbutamol solution 1 ml (5mg)*	Repeated 2 times
2. Metered dose inhaler with spacer *Salbutamol solution (100 mcg) 5-6 doses*	Repeated 4-5 times
3. Dry powder: via Turbuhaler *Terbutaline (500 mcg) 5-6 doses*	Repeated 4-5 times
PARENTERAL	
Subcutaneous Adrenaline: 0.5 ml (1:1000)	Repeated 2 times

dosing at close intervals (10 to 20 minutes) is the key to prompt symptom relief in the treatment of acute severe asthma.

Parenterally administered sympathomimetic drugs are also effective in acute asthma. **Adrenaline injected subcutaneously** was the "gold standard" treatment of acute severe asthma for many years. The newer sympathomimetics with greater beta-2 specificity can be administered intravenously. The parenteral route is not more effective that inhalational therapy. All sympathomimetic drugs, even the beta-2 specific preparations, produce a greater degree of systemic side effects such as cardiac and nervous system stimulation when given parenterally than by inhalation. The parenteral regimens should be used in the treatment of severe asthma only when the inhalational route is not effective or unavailable.

Oral beta-2 agonists are not effective in acute asthma and should not be prescribed.

Theophyllines (oral or parenteral) and anti-cholinergic drugs (inhalation) have much less bronchodilatory effects than beta-agonists and should NOT be used as first line drugs for acute asthma. It is debatable if either theophyllines or anticholinergics provide any additive effect to a maximal regimen of conventional beta agonists in acute severe asthma. They incur additional inconvenience, costs and side effects all of which probably argue against their use in a busy clinic practice. If however, the patient is already on maintenance treatment with a slow release oral theophylline preparation, this should be continued during an acute exacerbation.

Bronchodilator drugs relax airway smooth muscle, reduce airways obstruction and provide short term symptomatic relief. They do not however reverse the persistent airway inflammation, structural remodelling and hyper sensitivity associated with bronchial asthma. These airway changes persist long after the acute symptoms have subsided and account for the chronic disability and high risk of relapse in bronchial asthma.

Corticosteroid Treatment

Corticosteroids are used in the treatment of bronchial asthma primarily for their anti-inflammatory activity. The therapeutic effects of corticosteroids involve modification of nuclear transcriptional factors which regulate the expression of a large number of genes responsible for the control of inflammation and beta-2 adrenergic receptor function. There is a six to eight hour time lag between the administration of steroids and clinically significant improvement in acute asthma. Corticosteroid should therefore be administered as early as possible during an asthmatic episode to avoid excessive delays in response. Steroids sustain the initial improvement achieved by bronchodilators and, with continued use, will prevent subsequent relapse. Steroids taken orally are as effective as injections and this is the preferred route for a clinic practice. Inhaled steroids at doses used for conventional maintenance therapy are not effective for acute asthma and should not be used. Nearly all patients who suffer an acute asthma episode should be prescribed a course of oral steroids. Systemic corticosteroids should be taken for at least a week in order to control the exacerbation of airway inflammation associated with an acute asthmatic episode. A typical regimen would be: Prednisolone 30 mg in the clinic followed by 20-40 mg per day for 7 to 10 days with no "tailing" period. A short intensive course of oral steroids with prompt cessation is much safer than a small dose of the drug taken over a long period with half-hearted attempts at gradual withdrawal. Inhalational steroid therapy

should be phased in early on during the course of intensive treatment to facilitate a smooth transition from systemic to topical medication. Any patient who needs oral steroids for longer than 2 weeks or more frequently than say once in 6 months should be referred to a specialist for further evaluation.

Symptomatic and Adjunctive Treatment

Anti-tussive medication, nasal decongestions and anti-histamines may be prescribed for symptom relief. These drugs however do not influence the disease outcome and patients should be reminded not to depend on them to the exclusion of specific anti-asthma treatment.

Antibiotics should not be prescribed for treatment of an asthmatic attack. Many patients will complain of sore throat, fever and cough with yellow phlegm during an asthmatic exacerbation. This rarely represents a bacterial infection and should not be treated as such. Viral or aero-allergen exposure can cause fever and throat irritation while eosinophilic granules in the sputum may appear yellow.

There is no evidence that mucolytic agents can improve sputum clearance in clinical asthma and they should not be used.

Deployment and Referral

All patients who present to the clinic with acute asthma should receive immediate bronchodilator treatment. Even the most severely ill ones with impending respiratory failure should be treated while awaiting the arrival of an ambulance. No patient should be rushed off to the hospital before commencing treatment.

The initial bronchodilator treatment is usually completed in about 20 - 30 minutes and should be followed by an assessment of the clinical response. This re-assessment includes evaluation of subjective response, a physical examination and PEFR measurement. Depending upon the outcome of initial treatment, the patient might be (1) deemed fit for discharge, (2) treated with another round of bronchodilators or (3) referred to the nearest hospital for admission. There are no universally accepted guidelines on eventual categorization and deployment of patients following emergency treatment of asthma. This depends mostly upon the severity of the presenting attack and its response to intensive bronchodilator treatment. Most patients respond promptly after one or two rounds of bronchodilator drug treatment and may be discharged from the clinic.

The few patients who show no perceptible improvement after treatment or deteriorate further have status asthmaticus and should be managed in an emergency room or intensive care area. Some patients who fail to improve with inhalational therapy may respond to a parenterally administered sympathomimetic drug. Thus, while awaiting the arrival of the ambulance, the administration of subcutaneous adrenaline injections in patients with status asthmaticus may be appropriate.

PROPHYLAXIS

The primary objective of long term treatment in bronchial asthma is to prevent acute exacerbations and preserve a normal life style. Regular inhalation of corticosteroids is the most effective prophylaxis in asthma and should be considered in all patients who experience severe and/or frequent episodes of acute asthma. A single episode per month may be an appropriate indication for starting long term inhalational steroid therapy. There is a wide variety of topical steroid preparations including the appropriate drug delivery modalities for bronchial asthma. A more extensive discussion on prophylactic management is beyond the scope of this article.

CONCLUSIONS

Acute severe exacerbations of bronchial asthma can and should be effectively managed at the primary care doctor's clinic. Most patients will respond favourably and rapidly to inhalation of beta-2 sympathomimetic drugs. Those patients who are discharged from the clinic after successful treatment should be started on a course of oral prednisolone. Prophylactic medication should be considered for all patients to prevent relapse and improve quality of life.

The family physician is a key player in the management of acute asthma and should therefore be familiar with the salient features of its diagnosis, assessment, treatment and prevention.

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(Available from the author upon request)

AMBULATORY MANAGEMENT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Summary

The course of chronic obstructive pulmonary disease (COPD) is usually progressive and the airflow limitation largely irreversible. There are, however, reversible components. Intensive treatment with bronchodilators, corticosteroid, home oxygen, bronchial hygiene, breathing retraining and exercise conditioning may reduce the symptoms, improve the effort tolerance, prevent acute exacerbation and prolong survival. We must also not forget the psychological disturbance and the emotional needs of the patients.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic lung disease generally considered as composed of chronic bronchitis and pulmonary emphysema. Chronic bronchitis has been defined in terms of hyper-secretion of mucus[1] and pulmonary emphysema defined on a pathological basis[1]. Most patients who have chronic obstructive pulmonary disease have a combination of chronic bronchitis and pulmonary emphysema but their relative severity varies considerably from one patient to the next.

The course of COPD is one of deterioration and the survival is closely related to the initial level of functional impairment e.g. the forced expiratory volume in 1 second (FEV_1)[2,3]. Other prognostic factors include resting pulse rate, clinical or electrocardiographic evidence of cor pulmonale, blood gases abnormality and transfer factor (diffusing capacity)[4]. Prognosis is also related to the rapidity of progression of functional impairment during the first few years of observation[4,5]. The mean rate of decline in FEV_1 varies from 50 to 80 ml per year in various series. This is higher than that observed in normal subjects (24 ml/year). COPD could be summed up as a chronic, slowly progressive airway obstructive disorder resulting from some combination of pulmoanry emphysema and irreversible reduction in the calibre of the small airways of the lung.

The hallmark of the definition of COPD is irreversibility in contrast to the other disorders of airflow limitation such as asthma. As a result, patients and doctors alike often think that nothing much can be done. Patients are often discharged from follow up at hospitals' outpatient clinics with recommendation that patients receive oral bronchodilators such as salbutamol and/or theophylline from general practitioners or polyclinics. However, most experienced clinicians are convinced that conscientious care by a skilled physician or by a multidisciplinary team can minimise disability, prevent acute exacerbation, reduce hospitalisation, and avoid some of the early deaths which

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may result from complications of COPD.

The airflow limitation in COPD is largely irreversible. However, most patients do have some degree of reversible airway obstruction especially at the time when they are first seen by a physician. They often show some improvement in ventilatory function when first placed on a therapeutic regime. Following any improvement, most signs and symptoms tend to show gradual deterioration. The exceptions are cough and sputum production which frequently improve especially if smoking is discontinued. The rate of decline in ventilatory function such as FEV_1 is also slower if the patient stops smoking.

It may be possible to estimate the degree of airflow limitation by physical examination, but pulmonary function testing is necessary to quantify the physiological abnormality. Spirometry including a maximum flow volume loop is a valuable screening method and essential to the long term management of COPD. More complete testing such as lung volumes, transfer factor and arterial blood gases may be able to better define the extent of physiologic derangement. They are useful especially in the initial assessment. Simple peak expiratory flow rate (PEFR) as measured by a peak flow meter is helpful as a monitor during the follow up but is inadequate in the initial assessment.

PULMONARY REHABILITATION

We often think of pulmonary rehabilitation as purely chest physiotherapy. It has been defined by the Committee on Pulmonary Rehabilitation of the American College of Chest Physicians in 1974 as "an art of medical practice wherein an individually tailored multidisciplinary program is formulated which, through accurate diagnosis, therapy, emotional support and education, stabilises or reverses both physio- and psychopathology of pulmonary diseases and attempts to return the patient to the highest possible functional capacity by his pulmonary handicap and overall life situation". It should be viewed as the total management of the patients.

Patient Education

Careful and detailed patient education is crucial to the successful management of patients with COPD. The patient and his family members must understand lung structure and function in simple terms, the disease process itself, its pathogenesis and the goals and specifics of therapy. The first step in any therapeutic regime for COPD is the advice to stop smoking. Addiction to cigarette smoking is both physical and psychological. The most important factor in the success of smoking cessation is the subject's own determination. Nicotine patch may aid in dealing with the withdrawal symptoms, a consequence of physical dependence on nicotine.

Pharmacological Agents

Bronchodilators

Most COPD patients do show some increase in FEV_1 after a single dose of inhaled bronchodilator. The response in some patients may be as great as that seen usually in asthmatic patients. The airflow limitation in COPD is not as irreversible as is generally perceived. The next step after smoking cessation in the management of COPD is, therefore, the addition of bronchodilators. These include the anticholinergics, beta-adrenergic agonists and the methylxanthines.

Atropine, which is an anti-cholinergic agent, is an alkaloid of the plant *Atropa belladonna*. It has been known to be used by inhalation of the smoke of the burning leaves in India since the seventeen century. However, because of its marked side effects such as the drying of bronchial secretions, bronchial irritation, suppression of ciliary activity, tachycardia, urinary retention and psychotropic effects, it has had very little use. There has been a revival of interest in anti-cholinergic agents after the investigation of the role of the parasympathetic nervous system in bronchial obstruction[6], and after the introduction of ipratropium bromide, a quaternary ammonium congener of atropine, in the 1970s. Vagal tone has been found to be a major determinant of resting airway tone. Increased vagal tone under pathological conditions results in bronchoconstriction. Anticholinergic agents inhibit this vagally mediated bronchoconstriction by competing with acetylcholine for muscarine receptors on bronchial smooth muscle. Quaternary ammonium compounds have low lipid solubility. They are poorly absorbed systemically and do not cross blood

brain barrier. Comparative studies of ipratropium bromide with beta-adrenergic agonists have suggested that ipratropium bromide results in more bronchodilation in the COPD patients but less in the asthamatics[7]. It has been recommended as the first line bronchodilator in the treatment of COPD. It can be administered through a metered dose inhaler (MDI) or by a nebuliser. The usual MDI dose varies between 0.04 mg to 0.08 mg (ie 2 to 4 puffs) three to four times a day. The onset of significant effect occurs in 15 to 30 minutes. The peak effect is reached between 1 to 2 hours. The duration of action is about 4 to 6 hours. The patient should be cautioned not to expect the rapid onset seen in beta-adrenergic agonists.

Beta-adrenergic agonists are probably the oldest known therapy for breathing disorders. Ma Huang prescribed by ancient Chinese is an ephedrine like medication. There is a long line of beta-2 receptor selective agents to choose from nowadays. The commonly used ones in Singapore include salbutamol, terbutaline and the long acting salemeterol. Salemeterol is available here only in the inhaled form, whereas the first two may be given by intravenous, oral or inhaled routes. Inhaled route is preferred because of its effectiveness and because it causes less side effects. Metered dose inhalers are the most frequently used inhaled delivery system. They have the advantages of convenience, portability and low cost. However, they contain chlorofluorocarbon (CFC) which may affect the ozone layer of the atmosphere. Effective use depends heavily on the correct technique which requires a fair amount of coordination on the part of the patient. There is now a new MDI which does not contain CFC as the propellent. Those who are unable to master the technique of using a MDI may be given a spacer device to be used with the MDI, or a dry powder device such as a diskhaler or a turbuhaler. Nebulisation is not recommended for routine regular use. The dose delivered is much higher. There is 5 mg of salbutamol in 1 ml of Ventolin respiratory solution whereas there is only 0.1 mg in 1 puff of the MDI. Tachyphylaxis may occur in those given high doses chronically. Oral formulations are no more effective than the inhaled ones. They also cause more side effects. They should be limited to a selected group of patients who are unable to use any of the inhaler devices.

It is rational to combine the rapid action of the beta-adrenergic agonist with the sustained action of the anti-cholinergic agent. It must be realised that in most patients, the combination does not result in greater bronchodilation than that possible with optimal doses of either drug by itself. However, the optimal dose to achieve maximal bronchodilation by either drug may cause intolerable side effects. In such a situation, combining the two drugs may achieve maximum achievable bronchodilation at lower and better tolerated dose levels.

The use of methylxanthines as bronchodilators dates to the late 19th century. The preparations used at present are anhydrous theophylline, which is short acting, and its sustained release preparations. There is no pharmacologically rational basis for using the various "salts" of theophylline. The main difficulty of using theophylline is its narrow therapeutic margin. The therapeutic range is between 10 to 20 ug/ml. Mild toxicity may be seen when the serum theophylline concentration exceeds 20 ug/ml and potentially more serious toxicity when the level is greater than 35 ug/ml.

There are many factors that may affect the pharmacokinetics of theophylline and thus its serum concentration. They include age, sex, circadian rhythm, diet, arterial pH, pregnancy and various disease states such as congestive cardiac failure, liver dysfunction, fever, pneumonia and influeza immunisation. It also interacts with many drugs such as erythromycin, cimetidine, oral contraceptives, beta-adrenergic blockers, phenytoin, isoniazid, benzodiazepeines and tobacco. When a patient is prescribed a theophylline, a steady state blood level should be measured after 3 to 4 half lives. Once stabilised, the level should be measured yearly and with any change in patient's condition.

The pharmacological effects of theophylline are relaxation of bronchial smooth muscle, stimulation of mucociliary clearance, inhibition of mediator release, reduction in pulmonary arterial pressure, increase in diaphragmatic contractibility, suppression of capillary permeability oedema, stimulation of the central nervous system and respiratory centre, cardiac

inotropic and chronotropic effects, peripheral vasodilatation, mild diuresis, stimulation of gastric acid secretion and inhibition of lower oesophageal sphincter tone and induction of gastro-oesophageal reflux. Theophylline is a weak bronchodilator. It is uncertain which of its possible actions are important for its clinical effects in COPD. The recommended therapeutic blood level is based only on its bronchodilator effect. There is evidence to suggest that, especially in the older patients with COPD, the best clinical results are achieved with slightly subtherapeutic doses of theophylline combined with aerosol beta-adrenergic agonist and anti-cholinergic agents.

Corticosteroids

COPD patients do not respond to corticosteroids as dramatically as the asthmatics do. Moreover, the response rate is much lower and ranges from 17 to 29%[8]. Corticosteroids may have serious side effects when given on a long term basis. It would be ideal if we could limit its use to only those who would respond. Although there is evidence to suggest that those patients who have features similar to those seen in asthma, such as elevated IgE and eosinophilia, are more likely to get a good response from corticosteroids, there is no reliable predictor to identify clinical responders. The only accurate way is a clinical trial. Patients' sense of well being while on corticosteroids may be due to its euphoric effect. Therefore, an objective measurement is desirable. The usual trial involves giving the patient 30 to 40 mg of oral prednisolone per day for 2 to 3 weeks and measuring his or her FEV_1 of PEFR before, during and after the treatment. There is still no agreement as to the level of increase in the measured parameter that constitutes a positive response. While some clinicians accept 20% improvement in FEV_1 as indicative of a positive response, others insist on 30% as the cut off point. If there is significant response, prednisolone should be tapered to the lowest dose that maintains pulmonary function at its highest level along with subjective symptomatic relief. If the patient reports no relief of his symptoms and there is no improvement in the spirometric indices, the corticosteroid should be stopped. This is particularly so in those patients who appear to have predominantly pulmonary emphysema as assessed clinically, radiologically and by lung function measurement. Pulmonary emphysema is unlikely to respond to corticosteroids on theoretical grounds.

It is debatable regarding what to do with those patients who have symptom relief but no objective spirometric improvement. They report an increase in their effort tolerance and this is often susbstantiated by observation by their family members. It is difficult to ascribe such improvement to be purely an euphoric effect of the corticosteroids. Spirometry measures the lung function at rest. It does not truly reflect the pulmonary reserve or the exercise tolerance of the patients. A better test would be the cardiopulmonary exercise test to measure parameters such as maximum oxygen consumption ($VO2_{max}$). However, it is not easy to get old and breathless COPD patients to get on the treadmill or the ergometer. My own practice is to treat them in the same way as the responders.

Inhaled corticosteroids have a central role in the treatment of asthma. They are not as effective in COPD. But their use has a sparing effect on the oral cortiscosteroids especially in the cortico-steroid responders.

Antibiotics

Antibiotics are not indicated in COPD except during acute exacerbation. Exacerbations are often initiated by viral infection but there are usually secondary bacterial infections. The usual bacteria involved are *Haemophilus influenzae*, *Streptococcus pneumoniae* and *Mycoplasma pneumoniae*. The initial antibiotics given should cover these common pathogens.

Mucolytics and Expectorants

There is little evidence to support the routine use of such agents. Some trials in Europe indicated that oral N-acetylcysteine may reduce the frequency or duration of clinical exacerbations.

Vaccinations

Influenza vaccination confers a protection rate of 60 to 80%. Annual immunisation is recommended in the temperate countries as the outbreak of

influenza is often seasonal. It is difficult to determine the timing of vaccination in the tropics as there is no clear cut seasonal variation of influenza epidemics. The efficacy of pneumococcal vaccine in the COPD patients has not been convincingly established.

Oxygen Therapy

A major abnormality in COPD patients is chronic hypoxaemia. This results in tissue hypoxia and its consequences. It also causes pulmonary vasoconstriction, pulmonary hypertension, salt and water retention and subsequently cor pulmonale. By alleviating chronic hypoxaemia, supplemental oxygen has been shown to improve intellectual function, minimise memory loss, improving depressive symptoms, reduce pulmonary arterial pressure, prevent or minimise cor pulmonale, reduce dyspnoea and improve effort tolerance. Two major controlled clinical trials, the North American Nocturnal Oxygen Therapy Trial (NOTT)[9] and the United Kingdom Medical Research Council (MRC) Trial[10], have established the effectiveness of long term home oxygen in selected patients with advanced COPD in improving survival with increasing use of oxygen. The patients in these 2 trials had very low FEV_1, hypoxaemia, hypercapnia and clinical evidence of cor pulmonale. However, the use of long term home oxygen should not be restricted to patients with such severity of COPD. Although it has not been shown to increase the survival of patients with less severity, it does not necessarily mean it cannot do so. Even if it does not prolong the life of the patients, it improves the quality of their lives.

Domiciliary oxygen can be given by means of oxygen cylinder or oxygen concentrator. The latter is more convenient and cost effective in the long run. There are portable liquid oxygen systems which allow the patients to avoid being house-bound. They are more expensive. The idea of being independently mobile while carrying a small oxygen cylinder has not really caught on yet locally.

Bronchial Hygiene

Impaired clearance of retained secretions is a severe problem in many patients with advanced COPD. The technique of bronchial hygiene involves inhalation of bronchodilators to improve air flow limitation. This is followed by inhalation of moisture and expulsive coughing to remove mobilised secretions. Postural drainage may be useful in a minority of patients. It is still uncertain which is the most effective method of delivering the bronchodilators, i.e. by metered dose inhaler, nebuliser or intermittent positive pressure breathing (IPPB).

Breathing Retraining and Exercise Conditioning

Breathing retraining stresses slow abdominal diaphragmatic breathing with exhalation against pursed lips. This has been known to relieve dyspnoea and improve breathing efficiency. The amount of ventilation required to transport oxygen across the lungs is reduced by pursed-lip breathing. Leaning forward can relieve dyspnoea during the stress of exercise as it offers mechanical advantages to the respiratory muscles. A simple breathing exercise training device has recently gained favour. It allows the patient to breathe for a short period against increasing resistive loads. The aim is to strengthen the inspiratory muscles. Respiratory failure in advanced COPD often results from inspiratory muscle fatigue.

Bicycle riding, treadmill walking and ordinary walking have their own proponents. However, considerable evidence indicates that exercise training is largely task specific and performance is related to the method of exercise. It is therefore far more valuable for patients to be able to walk normally or climb stairs than to ride a bicycle or walk on a treadmill. Exercise training has been shown to improve effort tolerance although it does not change the lung function as measured by spirometry. Portable oxygen delivery system can increase the effort tolerance further during exercise.

Management of Neuropsychiatric Abnormalities

COPD is not strictly a cardiopulmonary disorder. The associate neuropsychiatric abnormalities are receiving increased attention. They contribute to the morbidity of COPD and should be dealt with in the total management of the patients.

Neuropsychological Impairment

COPD patients may suffer from disturbances of the central nervous system as a result of hypoxaemia, hypercapnia and acid-base imbalance. There may be impaired attention, short term memory, abstracting ability, motor skill and perceptual motor ability. Respiratory failure especially with carbon dioxide retention may be associated with headache, confusion, somnolence, tremor, asterixis, myoclonus, papilloedema and extensor planter response. Correction of hypoxaemia through long term oxygen treatment appears to improve brain function.

Psychosocial Disturbances

As with patients suffering from any serious chronic disease, patients with COPD tend to be depressed and withdrawn, and to avoid social interactions. They need emotional support from their family members and the health care givers. There is some indication that comprehensive rehabilitation programmes which are psychologically sensitive can reduce affective disturbance, facilitate independence and generally improve the perceived quality of life.

CONCLUSION

The total management of COPD is team work. It not only involves the chest physician, family physician and health care givers, but also the family members whose emotional and physical support is crucial. Though intensive treatment may not slow the disease progression or prolong survival in many of the patients, it improves the symptom complex, decreases exacerbation, reduces hospital stays and improves the quality of life.

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RESPIRATORY EMERGENCIES

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Summary

The goal of this article is to highlight the salient diagnostic principles and consensus practical approach in management of some common disease entities that can present as respiratory emergencies. Although many aspects of management mentioned can only be realised in a hospital or emergency room setting, it is hoped that this article will serve to give a didactic review of the current thoughts and approaches.

Respiratory emergencies can occur in a variety of settings, at home, in the community and in the hospital. The goal of this article is not to provide an exhaustive account of all respiratory emergencies. Rather, the aims are to emphasize salient diagnostic principles and the consensus practical approach in management. A selected list of specific common disease entities that can present as respiratory emergencies are discussed in this article. Although many aspects of management mentioned can only be realised in a hospital or emergency room setting, it is hoped that this article will serve to give a didactic review of current thoughts on common respiratory diseases that may present as emergencies.

EXACERBATIONS OF ASTHMA AND LIFE-THREATENING ASTHMA[1]

Exacerbations of asthma (asthma attacks) are episodes of progressively worsening shortness of breath, cough, wheezing, or chest tightness, or some combination of these symptoms. Respiratory distress is common. Exacerbations are characterised by decreases in expiratory flow that can be quantitiated by FEV_1 or PEFR.

Exacerbations usually reflect either a failure of long-term management or exposure to a trigger. The severity of asthma exacerbations may range from mild to life-threatening. Deterioration usually progresses over hours or days, but may occasionally occur rapidly over some minutes. Morbidity and mortality are most often associated with underassessment of the severity of the exacerbation, inadequate action at the onset of the exacerbation, and undertreatment of the exacerbation.

The primary therapies are the repetitive administration of inhaled short-acting beta-2 agonist and the early introduction of oral or parenteral corticosteroids, if needed. The immediate aims are to relieve airflow limitation as quickly as possible, relieve hypoxaemia, and restore lung function to normal as soon as possible. Crucial to the success of treatment of exacerbation is close monitoring of the patient's response to treatment with serial lung function in addition to pulse rate, respiratory rate and symptoms. Special vigilance should be paid to 4 categories of patients:

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- current use of or recent withdrawal from systemic corticosteroids
- hospitalization or emergency care visit incurring the use of nebulised bronchodilator and systemic corticosteroids for acute asthma in the past year
- psychiatric disease or psychosocial problems
- noncompliance with asthma medication plan.

Bronchodilators have always remained the mainstay of treatment for acute asthma. Until the past 10 years, subcutaneous adrenaline, 0.5 to 1.0 ml of 1:1000 s.c. was the first-line medication for the emergency room treatment of acute asthma. Aminophylline was often administered at the same time, given as a single bolus 5-6 mg/kg dose, followed by an infusion at a rate of 0.5 mg/kg/hour, to a total of 500 mg to 750 mg in 24 hours.

The current bronchodilator regimen for the emergency treatment of asthma is unanimously nebulised salbutamol in a dose of 2.5 to 5.0 mg (0.5 to 1 ml of salbutamol respiratory solution added to 3 mls of normal saline) repeated 30 mins later, up to a maximum of three doses if necessary. This is followed by 4 hourly administration in the first 24 hours in a hospitalised patient. Ipartropium bromide 125-250 ug (10-20 drops) is often added to the salbutamol/saline nebulisation mixture. Aminophylline is now used less frequently according to the clinical preference of the doctor in attendance. It is usually only added if the efficacy of the nebulised regimen is viewed to be inadequate.

Terbutaline sulphate (125 mg) 0.25 ml or salbutamol 250 ug (0.5 ml) by subcutaneous on intramuscular injection is infrequently used when the nebulised route is considered unsatisfactory due to lack of patient corporation. Intramuscular injection of adrenaline 0.5 to 1 ml (0.5 to 1 mg) is now reserved for anaphylactoid asthma and also when the patient is deteriorating despite the initial bronchodilator therapy. The plan for the management of an asthma attack at home is shown in Figure 1, and that for hospital based care in Figure 2[1].

Figure 1: Management of an Asthma Attack: Home Treatment*

Assess Severity

Cough, breathlessness, wheeze, chest tightness, use of accessory muscles, suprasternal retractions, and sleep disturbance. PEF less than 80% of personal best or predicted.

↓

Initial Treatment

Inhaled short-acting $beta_2$-agonist up to three treatments in 1 hour.

(Patients at high risk of asthma-related death should contact physician promptly after initial treatment.)

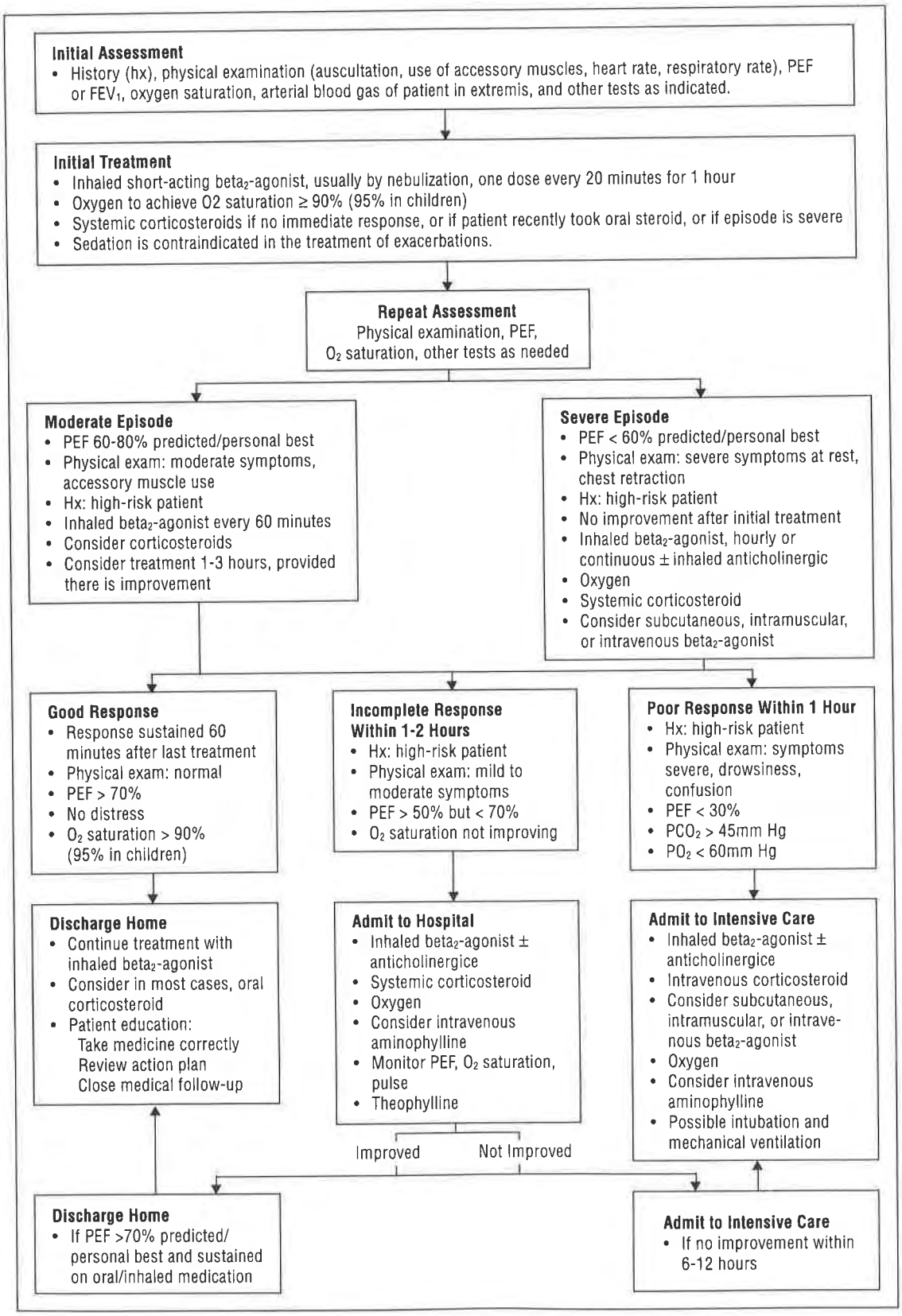
↓

Response of Initial Treatment is ...

GOOD if ...	**INCOMPLETE** if ...	**POOR** if ...
Symptoms subside after initial $beta_2$-agonist and relief is sustained for 4 hours. PEF is greater than 80% predicted or personal best.	Symptoms decrease but return in less than 3 hours after initial $beta_2$-agonist treatment. PEF is 60- 80% predicted or personal best.	Symptoms persist or worsen despite initial $beta_2$-agonist treatment. PEF is less than 60% predicted or personal best.
ACTIONS:	**ACTIONS:**	**ACTIONS:**
• May continue $beta_2$-agonist every 3-4 hours for 1-2 days	• Add oral corticosteroid	• Add oral corticosteroid
• Contact physician for followup instructions.	• Continue $beta_2$-agonist	• Repeat $beta_2$-agonist immediately
	• Consult physician urgently for instructions.	• Immediately transport to hospital emergency department

** Based on Global Strategy for Asthma Management & Prevention. NHLBI/WHO Workshop Report, 1995.*

Figure 2: Management of Exacerbation of Asthma: Hospital-Based Care*



** Reproduced from Global Strategy for Asthma Management & Prevention. NHLBI/WHO Workshop Report, 1995.*

Corticosteroid therapy in acute asthma is now given more liberally than in the past when it was initiated only for a severe attack. The current practice is to administer it together with the standard first line treatment of nebulised bronchodilator for all cases of moderate to severe acute asthma. This treatment consists of intravenous hydrocortisone 200 mg every 6 hourly over the first 24-28 hours. At the same time oral treatment is also started with prednisolone at a dose of 30 mg per day to be progressively reduced and withdrawn over a period of 10 days.

PNEUMOTHORAX[2]

Pneumothoraces are classified as spontaneous or traumatic. Spontaneous pneumothoraces are either primary when they occur in previously healthy individuals or secondary when they occur as a complication of underlying lung diseases. Underlying chronic bronchitis and emphysema (COPD) account for the majority of secondary spontaneous pneumothorax. Traumatic pneumothoraces are either iatrogenic or noniatrogenic.

The common symptoms of dyspnoea and chest pain and the signs of tachycardia and abnormal chest findings are usually mild in primary spontaneous pneumothorax. In comparison, the symptoms in secondary pneumothoraces are more severe because the primary reserve is already compromised by underlying lung pathology. Most pneumothoraces are closed. A tension pneumothorax occurs when a check-valve mechanism exists, allowing air to enter the pleural space during inspiration but not exit during expiration. The pleural pressure will be positive during expiration and often during inspiration. The clinical manifestations are severe: rapid laboured breathing, cyanosis, and respiratory distress, cardiovascular instability characterised by tacycardia and hypotension, and signs of a pneumothorax.

Management of Pneumothoraces

Tube thoracotomy should be performed in all secondary spontaneous pneumothoraces, traumatic pneumothoraces, and pneumothoraces occurring in mechanically ventilated patients. Other forms of pneumothoraces require tube throascotomy only if the pneumothorax is:

- large (>40%)
- associated with significant symptoms or arterial blood gas abnormalities
- progressively enlarges
- does not respond to simple aspiration.

Tension pneumothorax is an uncommon event, but prompt action is needed as it is immediately life-threatening. Treatment of a tension pneumothorax should not await radiographic confirmation. In the presence of cyanosis and hypotension, immediate treatment should be given by the insertion of a wide bore needle attached to a three-way stopcock and a 50 ml syringe partially filled with sterile saline.

Not uncommonly, a persistent air leak remains after 5-7 days of chest-tube drainage. This is due to a bronchopleural fistula and can be definitively repaired by either open thoractotomy or thoracoscopy with suturing or resection of the fistula and scarification of the pleura. Prolonged chest tube drainage is preferred in patients whose operative risk is increased by significant underlying lung diseases or other medical problems.

MASSIVE HAEMOPTYSIS[3]

Haemoptysis is the expectoration of blood originating from the lower respiratory tract (trachea, bronchi, or lung parenchyma). It ranges from blood-streaked sputum to massive, life-threatening haemoptysis. The definition of massive haemoptysis is somewhat arbitrary, ranging from 100 - 600 ml of blood in a 24 hour period. The prognosis depends on the rate of blood loss. Massive haemoptysis usually complicates severe underlying lung disease, adding to the difficulty of patient management.

The most common causes of massive haemoptysis are tuberculosis, bronchiectasis, mycetoma, bronchogenic carcinoma and lung abscess. The diagnostic approach of massive haemoptysis should be directed toward determining the site of bleeding in order to provide rational management. A chest radiograph may show a lung parenchymal source of bleeding. Bronchoscopy is the appropriate

first step in the evaluation of massive haemoptysis. In the past bronchography has been used to visualize bronchiectasis. This is now achieved with the less invasive computerised tomography. For visualization of peripheral lesions, angiography of both bronchial and pulmonary arteries may be diagnostic and define the anatomy of the lesion.

The initial management of a patient with massive haemoptysis should be done simultaneously with the initial diagnostic steps. The urgency of evaluation and therapeutic intervention is determined by the rate of bleeding. Because asphyxiation is the usual cause of death, immediate steps involve the control of the airway. Simultaneously, volume replacement and mild sedation with partial cough suppression should be instituted. Blood should be drawn for measurements of haematocrit, coagulation profile and for typing and cross matching. Coagulation abnormalities should be corrected. Emergency consultations should be initiated for consideration of medical management, invasive radiology or thoracic surgery. Surgery is indicated on an elective basis for the definitive management of anatomically circumscribed lesions. Surgery in such patients may be contraindicated or complicated by poor underlying lung function or by the presence of dense fibrous adhesions. Over the past decade, bronchial artery embolization has proved to be an alternative to surgery that is effective in controlling haemorrhage and preventing recurrent haemorrhage in some conditions. This procedure has made a significant therapeutic impact on the management of massive haemoptysis.

Acute Respiratory Failure

Acute respiratory failure (ARF) is a physiologically defined condition that may result from a variety of disease processes. It may develop over the course of minutes, hours or days in patients with normal lungs or patients with pre-existing disease.

There are two primary types of ARF: failure of oxygenation or failure of ventilation. Both processes may be present in a patient but usually one type predominates. Examples of ARF characterised by predominant hypoxaemia are severe pneumonia, acute respiratory distress syndrome and fibrosing alveolitis. Examples of ARF characterised by predominant hypercapnia are chronic bronchitis and emphysema (abnormalities of the lungs and airways), severe kyphoscoliosis (chest wall abnormality), drug overdose (decreased respiratory drive) and myasthenia gravis (neuromuscular weakness).

One approach to the differential diagnosis of hypoxaemic ARF is based on whether there is coexistent hypercapnia and whether the lung fields on the chest radiograph are "dark" (normal or hyperluscent) or "white" (radiopaque) as listed below (Table 1):

Table 1: Approach to Diseases Causing ARF

ABG	*CXR*	*Diseases*
Normocapnia	"black"	Pulmonary embolism Circulatory collapse Right to left shunt
Normocapnia	"diffuse white"	ARDS Cardiogenic pulmonary oedema Pulmonary fibrosis
	"localised white"	Pneumonia Atelectasis Pulmonary infarct
Hypercapnia	"black"	Status asthmaticus COPD Alveolar hypoventilation Neuromuscular weakness Sleep apnoea syndrome
Hypercapnia	"diffuse white"	End stage pulmonary fibrosis Severe ARDS
	"localised white"	Pneumonia + COPD/drug, or Oxygen induced respiratory depression

The management of patients with ARF consists of:

(i) empirical therapy for ARF and

(ii) specific treatment for the disease which leads to the ARF.

Raising the PO2 to greater than 50 mm Hg is the first goal of therapy. If the patient is alert and cooperative, supplemental oxygen with a repeat

arterial blood gas within 30 minutes and close observation may be adequate. If the patient is stuporous, comatose, or has a decreased gag reflex with shallow respirations, then control of the airway with endotracheal intubation is required. A common example is the approach for correcting hypoxaemia in the acutely ill COPD patient described in the American Thoracic Society statement[4].

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TRANSFERENCE IN PSYCHOTHERAPY

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Summary

The history of transference is outlined briefly from Freudian to post-Freudian contributors. The concept of transference derived from psychoanalysis is traced and its application to modern day individual and group psychotherapy is outlined.

The paper discusses the concept of negative and positive transference, special transferences such as erotic transference, transference neurosis, transference resistance and transference in general practice. The technique of transference analysis using the triangle of insight is described. Transference in Group Psychotherapy including transference dilution or intensification, the bad and good mother group, are also mentioned.

The curative factors in psychotherapy that relate to the transference in supportive and dynamic psychotherapy are brought to the readers' attention.

Keywords: *Transference theory, techniques, management, individual, group psychotherapy*

TRANSFERENCE: DEFINITION

Greenson[1] defined transference as the experience of feelings, drives, attitudes, fantasies and defences toward a person in the present which does not befit that person. It is a repetition of a reaction originating from significant persons of early childhood, and is unconsciously displaced onto figures in the present. It is a form of regression.

Feinchel[2] states that most transference reactions occur in psychotherapy but it can occur outside therapy, implying that all human relationship is a mixture of transference and reality. The person is usually unaware of the transference.

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HISTORY OF TRANSFERENCE

Freud first described transference while treating hysterical patients. He described the technique of analysing the transference as a method of making the patient conscious of the transference, demonstrating that it is an obstacle in therapy, and tracing its origins in the patient's childhood.

In an analysis of a patient called Dora in 1905 Freud[3] described the role and relevance of transference in psychoanalysis. He realised that failure to analyse the transference led to treatment failures or premature termination.

Freud[4] expounded the relationship between transference and resistance. He classified transference into negative and positive transference. Freud[5] coined the term transference neurosis, referring to an intense preoccupation of the patient towards the therapist where feelings towards the therapist are expressed and the patient relives his childhood conflicts in the presence of the therapist. During interpretation,

these intense preoccupations are analysed, and its childhood origins are brought to awareness. Repeated interpretation of the transference neurosis finally brings about its resolution. Freud[5] recommended a blank screen approach by the therapist to encourage the development of the transference. (With further evolution of psychotherapy techniques, the blank screen approach has been superseded by a more transparent approach).

By 1915 he had coined the term "transference love"[6], referring to the patient's erotic feelings towards the therapist. When this happens the therapist was expected to remain abstinent, which meant that the therapist should not react sexually towards the patient. By 1914 he described the concept of the "repetition compulsion", the patient's tendency to act out the transference.

In 1909, Ferenzi[7] stated that transference is not only unique to the psychoanalytic situation but can also appear in the patients' relationships outside therapy. Spitz[8] described the re-enactment of the mother-child relationship. In 1956 Winnicott[9] described how transference could be dealt with when there is inadequate mothering, and Zetzel[10] described the therapeutic alliance. In 1965 Greenson[11] contributed further to the understanding of the separation of the transference (distorted) relationship from the real relationship (therapeutic alliance).

Object relations theorists (Klien 1961[12], Segal 1964[13]) considered transference as arising from projections of good or bad objects from the patients' internal world. In 1946 Alexander and French[14] felt that the transference could be manipulated within a present focus, a view which is not acceptable to contemporary psychoanalysis. In 1965 Greenacre[15] reminded us that the matrix of the transference is in the early mother-child relationship.

WORKING ALLIANCE

The two fundamental relationships that the patient establishes with the therapist is the working alliance and the transference. The working alliance is the real relationship between therapist and patient, although a small aspect of it could be derived from the infant-mother relationship. The working alliance is formed by the patient's motivation for cure, his willingness to co-operate in treatment, his ability to follow instructions and receive insights. The working alliance is formed by the patient's reasonable ego and the analyst's analysing ego (Sterba 1934[16]). Transference analysis and interpretation must be based on a backdrop of a good working alliance.

The working alliance is real, appropriate and starts in the beginning, fluctuating in the middle phase and increasing at the end of therapy, while the transference is unrealistic, inappropriate but truly felt, and mostly appears in the middle phase of psychotherapy. Transference interpretations can only be successful if the working alliance is well established. In neurotic patients the working alliance is established quite easily unlike borderline patients where the main form of psychotherapy is directed towards developing the working alliance.

DYNAMICS OF TRANSFERENCE REACTIONS

When transference develops in a neurotic patient, reality testing may be lost temporarily, the present being misunderstood in terms of the past. Ego regression occurs, though reversibly, but when the therapeutic alliance is restored, the patient will start working on his transference and realise that his past conflicts have been transferred to the here and now of the session. Pre-oedipal and oedipal feelings are re-experienced and defences such as splitting, introjection and denial may be activated. When the regression is of sufficient depth, the Id releases aggressive impulses and the transference becomes negative. In order to mitigate the release of aggression which may threaten the ego, transference resistance develops. This happens when hostile or sexual feelings are appearing. The super-ego may be displaced onto the therapist during regression, and the patient may perceive the therapist as a critical parent.

The psychotherapist has to deal with regression, interpret the transference resistance, handle all affects expressed, and re-establish the working alliance. The scheme of events that take place during transference interventions in psycho-

therapy is summarised as follows:-

- Therapeutic alliance is established as a backdrop.
- Regression of the ego with loss of reality testing.
- Pre-oedipal/oedipal feelings are re-experienced.
- Temporary loss of the therapeutic alliance.
- Defences are activated.
- Therapist intervenes to restore the therapeutic alliance and interprets transference resistance and transference.
- Lifting of regression and re-establishment of reality testing.
- In the absence of therapeutic alliance non-interpretative, supportive interventions are utilised.

"WORKING THROUGH" THE TRANSFERENCE

Freud coined the term "working through", a basic psychoanalytic concept where the therapist points out the transference distortion as many times as possible with as many ramifications as possible, until the patient gains insight.

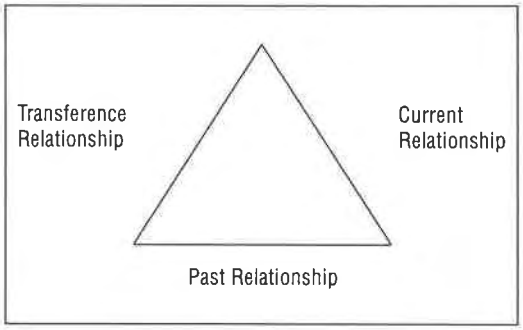
Working through of the transference refers to the analysis of the transference by the therapist so as to make the phenomena conscious. Interpretations are done repeatedly in different situations. However the patient will resist interpretation of the transference as the underlying emotions released may be too threatening to his ego. Therefore "working through" must involve transference interpretation as well as interpretation of resistance to the transference. Within a supportive environment, the patient's distorted impression of the therapist (here and now relationship) must be integrated into his personality.

TECHNIQUES OF TRANSFERENCE ANALYSIS

A useful approach to analysing the transference is the triangle of insight (Figure 1) (Gabbard 1994)[17] which links the current relationship, the transference relationship and the past relationship. The current relationship includes the relationship between patient and others such as parents, friends and marital partners. The transference relationship is the here and now relationship between patient and therapist. The past relationship includes patient's relationship with his parent or caregivers. (However counter transference will be manifested in the therapist's relationship with the patient). The steps involved in transference analysis include the following:-

1. A link is made between the patient's relationship with the therapist (transference material from the here and now) with the patients current relationships outside therapy.
2. The current relationship is linked to the patient's past relationships.
3. Transference relationship is linked to the past relationships.

Figure 1: Triangle of Insight (Gabbard 1994[17])



Although the above sequence of techniques described appears straightforward, considerable experience is required in the timing of interpretations. Therapeutic neutrality must be maintained. The therapist must be able to distance himself from the patient from time to time when he attempts to interpret. He must "strike while the iron is hot", i.e. when the patient has expressed sufficient affect. In borderline patients it may be unwise to interpret while the "iron is too hot".

Therapists must realise that formal training and supervision are necessary before the skills involved in understanding of analysis of the transference can be acquired.

TRANSFERENCE IN GROUP PSYCHOTHERAPY

In psycho-dynamic group therapy, the group itself has an impact on the transference which can be diluted or intensified. Dilution can take place because the negative transference towards the therapist could be displaced towards other members. Transference dilution can also occur when the transference is directed at a co-patient initially and then it becomes intensified when the patient redirects the transference towards the leader as the therapy progresses. Intensification of the transference can occur when the members relate to the group as a whole and members project their primitive object relations onto the group during group regression. Such group regressions can lead to release of powerful affects. Such intensification of the transference is not observed in individual psychotherapy.

Multiple transferences can occur with reactivation of sibling rivalry (sibling transference) during group therapy. The transference to the group as a whole has been elucidated by Scheidlinger 1974[18] and further developed by Kibel 1991[19]. Scheidlinger described the mother group, a primitive state of regression whereby the therapist is viewed as a sadistic mother and the group as a whole is viewed as the idealised mother (note the splitting). The transference towards the group as a bad mother has been described further by Ganzarain 1991[20]. In the special situation where there is a male/female co-therapist pair, Bardon 1996[21], states that patients may work through the paternal and maternal transferences at a more rapid pace compared to a single therapist group.

The three components of transference interpretation in group psychotherapy include the analysis of the transference toward the therapist, member to member, and to the group as a whole. (However the therapist should be aware that counter-transference feelings may be more intense when leading these groups.)

TRANSFERENCE IN GENERAL PRACTICE

In General Practice, the main type of psychotherapy is usually brief and supportive. The therapist may notice the transference but may not interpret. In the doctor-patient relationship, transference contributes partly to the therapeutic relationship while the rest is contributed by the real relationship. The transference which perceives the doctor as an authoritative parent, may interfere with history taking. Bad experiences with previous doctors may also be displaced in current consultations. A friendly communicative style must be adopted during consultations. Psychotherapeutic intervention may be necessary if the transference causes difficulties in obtaining history or in obtaining the patient's co-operation. Counter-transference towards difficult patients (personality disordered, substance abuser or somatising patient) should be noted and prevented from souring the doctor-patient relationship.

In supportive psychotherapy, the transference is *noted* while counter-transference is *monitored*, but depth analysis of transference is *avoided*. A referral to a psychiatrist of psychotherapist should be made if the patient requires formal psychotherapy.

MECHANISM OF CHANGE IN PSYCHOTHERAPY

Most therapists will agree that the analysis of the transference, its interpretation, and its resolution is the basis of cure in psychotherapy. In contrast to psychoanalysis, in long term individual psychotherapy, in-depth analysis of transference neurosis is avoided, the intensity of the transference is less, and a greater emphasis on linking the transference with the current relationship rather than with the past.

Further development in the mechanism of change in psychotherapy shows that other curative factors are involved such as the experience of the therapy itself and the internalisation of that experience as a new relationship (Cooper 1992[22]). The therapist's container functions for the patient's projections (Bion 1962[23], Ogden 1979[24]), the therapist's holding function (Winnicott 1965[25]) and the metabolism and reintegration of projective material in projective identification (Ogden 1988[26]). In group psychotherapy Horwitz[27] observed that structural change depends on the extent of the internalisation process. In

supportive psychotherapy, the therapeutic factors are not so clear and more research needs to be done.

Conclusion

Transference analysis is used in psychoanalysis, but with less intensity in psychotherapy. Analysis of transference has been the main curative factor in psychotherapy but this has led to other interventions such as containment, holding, and processing of projective indentifications, and internalisation of new relationships. Additional dimensions to transference analysis has been provided through group psychotherapy.

A counter-transference – transference for psychotherapy is the most recent addition to the subject of transference. Transference and counter-transference processes have not been sufficiently integrated in clinical settings but now there is a realisation that the two processes are intertwined as seen in the mechanism of projective identification. Techniques of transference analysis require training, supervision and knowledge of theory of psychoanalysis as applicable to Individual and Group Psychotherapy and their application to specific populations and cultural groups. The relevance of counter-transference will be dealt with in the next paper.*

* *The paper "Counter-transference in psychotherapy" will be published in the July-September issue of the Singapore Family Physician.*

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NOCTURNAL ENURESIS – A REVIEW OF TREATMENT OPTIONS

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Summary

Nocturnal enuresis is a common but under-reported paediatric problem. Various aetiological factors have been postulated but there is a lack of consensus as to the exact cause. Nocturnal enuresis is essentially a benign condition, but a thorough history and physical examination are imperative to exclude any pathological causes of bed-wetting. The treatment approach to nocturnal enuresis is controversial. Of the treatment modalities currently available, the alarm system appears to be safe, inexpensive and the most effective. Desmopressin is highly efficacious during the time of administration but the relapse rate is high. Regardless of the therapeutic approach chosen, the family physician must involve both the parents and child in the decision-making process and educate them regarding nocturnal enuresis, thereby dispelling misconceptions and eliminating guilt feelings.

Keywords: *Bedwetting, desmopressin, imipramine, nocturnal enuresis*

"I knew that bedwetting was wicked and outside my control. It was then possible to commit the sin without knowing that you committed it, without warning to commit it and without being able to avoid it." ***George Orwell***

INTRODUCTION

The word enuresis is derived from the Greek word "enourein" which means simply, to void urine. A general definition of the enuretic state is not easy to formulate as norms vary from one country to the next. However, the working definition used by most paediatricians in studies of nocturnal enuresis is: the involuntary voiding of urine on at least 2 nights per month, beyond the age at which bladder control is normally obtained (4-6 years), in the absence of congenital or acquired defects of the urinary tract[1]. This disorder has plagued humans since its original inscription on the Papyrus Ebers in 1550 B.C. (Glicklich 1951)[2]. Treatment modalities since ancient times have included various potions containing animal parts, penile clamps, caustic agents injected into the genitalia, public humiliation and punishment.

Nocturnal enuresis has been identified in all cultures. It is a common problem affecting 15% to 20% of 5-year-olds and there is a spontaneous resolution rate of 15% per year thereafter[3]. Thus, the prevalence decreases to 7% in 10-year-olds and to 1% in adults[4-6]. However, because nocturnal enuresis is still looked upon as something to be ashamed of, prevalence is underevaluated.

Primary nocturnal enuresis accounts for 75% to 80% of all cases and is defined as a child who has never gained nocturnal urinary control. In contrast, secondary nocturnal enuresis is where

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there has been a past history of a continuous dry period of 6 months to a year and it has been associated with a higher incidence of stressful life events[7,8]. Differentiation however between primary and secondary nocturnal enuresis has little impact on the prediction of therapeutic success.

Nocturnal enuresis carries with it a significant emotional stigma and has been associated with emotional and social problems[9-11]. Foxman et al[5] in a population-based study showed that 50% of children and their parents were distressed by the child's enuresis but less than half expressed this concern to the doctor. Moffatt[12] in 1989 reviewed available studies of the psychological impact of the treatment of enuresis and found that treatment of enuresis was appropriate and beneficial – enuretic children treated for bed-wetting improved in relations with their peers and on measures of self-concept, compared to untreated controls.

Although knowledge about the aetiology and treatment of enuresis have grown, expert consensus in these areas remains elusive. This article explores the current concepts on aetiology and reviews the treatment modalities available to the doctor. As the family physician is often the first-line health worker that the family seeks help from, it is imperative that the family physician has a good understanding of the aetiology and treatment options for these children, so that anxious and frustrated parents will not employ physical punishment on their children nor ineffective alternative treatments.

AETIOLOGY

Children with nocturnal enuresis and their parents have been subjected to a wide range of myths but until today, the exact aetiology of nocturnal enuresis remains unclear. Most studies in the past few years have explored possible causative factors including genetics, sleep arousal differences, hormonal problems and bladder capacity.

Genetic Factors

There appears to be a genetic component to enuresis although the mechanisms remain unclear. Many studies have shown that enuretic children often have a strong family history of enuresis[13,14]. Fergusson[15] showed that enuresis in the family history was the strongest predictor for development of enuresis in a child. If both parents had enuresis as children, 70% of their offspring are likely to be affected. When one parent had enuresis, the frequency decreases to 40%. Studies in twins have also shown a strong genetic factor in enuresis[16-18].

Developmental Delay

A commonly accepted theory of nocturnal enuresis proposes a delayed functional maturation of the central nervous system, reducing its ability to inhibit bladder contraction at night. Enuretics are twice as likely as other children to exhibit motor and speech delays, slow growth, and delayed bowel sphincter control, suggesting that nocturnal enuresis may be due to a delay in maturation[19,20].

Sleep Disorders

Sleep arousal differences have been offered as an attractive hypothesis for explaining nocturnal enuresis. Parents of children with enuresis often state that the child sleeps more soundly than the other members of the family. Research in this field has shown conflicting results. Initial studies suggested that enuretic episodes occurred during slow-wave deep sleep[21] but Mikkelsen and Rapoport[22], based on a very extensive study in 1980, stated that nocturnal enuresis was independent of sleep stage and hence, could occur during both deep and light sleep. Recently however, a disturbed arousal threshold has been demonstrated in patients with nocturnal enuresis, which did not allow adequate awareness for inhibition of voiding[23].

Bladder Capacity

Bladder capacity is thought to play a role in the aetiology on enuresis. Starfield[24] showed that enuretic children had a significantly lower functional bladder capacity than their non-enuretic siblings. These results have been confirmed by Zaleski[25] and Jarvelin[26]. Other authors have found enuretics to have normal bladder size.

Antidiuretic Hormone

Humans reduce their urine production during the

night and this has been shown to be due to an increased production at night of the antidiuretic hormone vasopressin. These findings have been verified by investigators in more recent studies by Norgaard and Rittig. This group of Danish scientists has in recent years further delineated the physiology of nocturnal enuresis and discovered that enuretic patients have diminished production of vasopressin during night-time. This has given impetus to widespread promotion of desmopressin acetate (DDAVP) as the therapy for nocturnal enuresis.

Miscellaneous

The discussion about enuresis as a psychological disorder is an old one. Moffatt[31] however, found no evidence of any emotional or behavioural factors in the aetiology of nocturnal enuresis.

Other aetiology factors implicated included constipation[32], upper airway obstruction[33] and even food allergies[34].

EVALUATION

Although nocturnal enuresis is considered a non-organic disorder, it is imperative that a complete history and physical examination be conducted at the initial visit to exclude any underlying organic disorder.

Important history that should be elicited include the pattern of bed-wetting – whether wetting is confined to the night-time or daytime or both; whether bed-wetting is of recent onset or has persisted since infancy. Any associated symptoms like dysuria, urgency, frequency or any associated bowel incontinence should be documented. Information regarding the family and social background should also be obtained.

Physical examination is directed at identifying any abnormalities in the lumbosacral area for sign of spinal dysraphism and neuropathic bladder dysfunction, and the external genitalia to rule out conditions like meatal stenosis, hypospadias, epispadias and ectopic ureter. In the case of ectopic ureter, urine comes from the introitus continuously without the child having the urge to void and thus, bed-wetting would not just be confined to night-time but would occur in the day as well.

Urinary tract infection and urinary tract malformations are important conditions that need to be excluded by the physician in any child presenting with nocturnal enuresis. As such, a urine microscopy and urine culture are mandatory investigations that heave to be performed in an enuretic child. Only when haematuria, symptoms of urinary tract infection, constant daytime wetting occur can one justify radiologic investigations. An underlying structural abnormality of the urinary tract will not produce isolated nocturnal enuresis. Further investigations, like an intravenous pyelogram or ultrasound should be performed if abnormalities of the urinary tract are suspected based on history and physical examination. When neurologic abnormalities are suspected, urodynamic studies and consultation with the neurologist are indicated.

TREATMENT

The treatment approach to nocturnal enuresis is controversial, largely due to a lack of consensus as to the exact cause of enuresis. Current treatments can be broadly divided into pharmacological and non-pharmacological methods.

Non-pharmacological methods include the use of alarm systems, motivation therapy, hypnotherapy etc., whilst pharmacological methods include the usage of drugs like imipramine and desmopressin. The recent addition of desmopressin to the pharmacologic armamentarium, has been as a result of the work of a group of Danish scientists discovering that enuretic patients have a diminished production of ADH during the night-time. In fact, during the past few years, doctors have been confronted with a considerable amount of publicity related to the treatment of enuresis with desmopressin.

It is important to remember that the benign albeit frustrating nature of the problem of enuresis and its 15% spontaneous annual resolution rate indicates that any treatment modality must carry no risk and only minimal side effects and it should have a cure rate significantly greater than the spontaneous resolution rate before it is considered to be effective.

The following review of the different modalities of treatment aims to address four major

questions. First, how effective are pharmacological treatments compared to non-pharmacological treatments? Second, is there any particular modality of treatment which is the most effective? Third, after the treatment has ended, are improvements maintained? Fourth, what are some of the problems or side effects associated with the different modalities of treatment?

Non-Pharmacological Methods

Numerous non-pharmacological methods have been employed in the treatment of nocturnal enuresis including the alarm system, bladder training, hypnotherapy etc. But amongst the various non-pharmacological modalities, the most commonly utilised is the alarm system.

Alarm Systems

The first enuresis alarm was constructed in 1904 by Pfaundler, a German paediatrician who wanted to alert his nurses when children had wet the bed so that the nurse could rapidly change the bed-sheets. It was then first observed that fewer and fewer children in the ward passed urine during sleep. The enuresis alarm subsequently fell out of use until it was reintroduced during the thirties by Mowrer and Mowrer[35].

The theory of the conditioning effect of the alarm system on the enuretic has been well described by Young[36]. The system works by placing a small sensor in the patient's underwear that sounds an alarm, even when a small amount of urine is released, awakening the patient. The process of urination is then inhibited as the patient awakens. This practice conditions the child to the sensations of bladder filling and trains the child to inhibit bladder contractions before wetting occurs.

Success rates range from 65% to 100% cure after 4 to 6 months of treatment[36]. Turner[37], who compiled 16 published studies, showed an average initial cure rate of 82%. Dische[38] treated 84 enuretic children with the alarm device and within 3 months, 67% were cured and by the end of 12 months of therapy, 92% were cured. The relapse rate was found to be 30% but most relapsers responded to a second course of treatment with the alarm device.

Meadow[39] treated 100 enuretic children with the alarm system – within 4 months of treatment, 85% became dry with the majority in the first 2 months. The relapse rate was 10% and most relapses responded to reinstitution of the alarm treatment. Forsythe and Redmond[40] as well as Schmitt[41] have also reported similar success rates.

The main difficulty with this treatment modality is that its success is highly dependent on co-operation and motivation on the part of the child as well as the parent. Dische[42] found that family difficulties gave a reduced initial cure and an increased relapse rate after treatment. Butler[43] found more failures in children with poor family support and in children resistant to change.

Utilising the alarm treatment definitely puts demands on the family especially siblings sleeping in the same room as the enuretic child and tired parents who have to wake the child up many a time. As such, this modality of treatment requires enthusiasm and support from the treatment team. The problem is compounded by the fact that earlier models of the alarm system were inadequate from the safety point of view, leading to problems like ulcerations, otherwise known as "buzzer ulcers" in some patients[44].

Pharmacological Therapy

Pharmacological therapeutic modalities for nocturnal enuresis are directed at 3 different organ systems. Treatments directed at the central nervous system (sleep patterns) include imipramine, amphetamines and diazepam. Treatments directed at the bladder or the urethral sphincter include parasympatholytics, calcium blockers, prostaglandin inhibitors and alpha-adrenoreceptor stimulators. Treatments directed at the kidney (diuresis) include desmopressin.

Imipramine

Imipramine, a tricyclic anti-depressant, is one of the most commonly prescribed drugs for enuresis. The exact mode of action has yet to be clearly defined. Several theories have been proposed to explain the action of this drug, related to its anti-depressant action, the alteration

of sleep mechanisms and arousal patterns and its anti-cholinergic effects[45]. Puri[46] has also shown that imipramine may also alter the secretion of ADH.

Maclean[47] in 1960 was the first to suggest that imipramine might be useful in the treatment of enuresis, as evidenced by an incontinent adult who was treated for depression with imipramine and became continent as a result. Since then, there have been numerous studies conducted to evaluate the medication. Poussaint and Ditman[48] in a double- blind, placebo controlled study of imipramine in 47 enuretic children showed that treatment with imipramine was superior to placebo and dependent on dosage. The starting dose is 0.9-1.5 mg/kg orally 1-2 hours before bedtime, with 25 mg increment every week if no response occurs within this period. ECG assessment is required with doses over 3.5 mg/kg because of the potential cardiotoxicity of this drug. Optimal duration of treatment is unknown but patients are commonly treated for between 3-6 months and upon cessation, the doses are tapered gradually. Poussaint and Ditman found that there was marked improvement with doses up to 75 mg after 10 years of age and 50 mg below that age. However, only 24% of the children were completely dry off medication.

A low long term cure rate was also observed by Shaffer et al[49], Kunin et al[50] and Blackwell and Currah[45]. Blackwell and Currah in their literature survey reported that almost all double-blind placebo controlled studies showed a very good effect of imipramine. The effect is rapid but disappears when the drug is discontinued. The success rate ranges from 10-15% in the short term but due to frequent relapses when the patients is off medication, there is only a 25% overall cure rate[4,51].

Side effects from treatment are uncommon but they include anxiety, insomnia, headaches, abdominal pain, constipation and loss of weight[49]. However, overdosage can be potentially fatal through hypotension, respiratory complications, cardiac arrhythmias and convulsions[52]. Serious poisonings and deaths have been reported in enuretic patients and siblings[53,54]. The role of tricyclic anti-depressants in treating nocturnal enuresis is declining with increased usage of desmopressin and with concerns over its potential toxicity.

Desmopressin

Desmopressin has been used for many years as an antidiuretic treatment in cases of central diabetes insipidus and was introduced for treatment of nocturnal enuresis in the 1970's in Europe and more recently in the United States.

Desmopressin has for sometime been available for administration intranasally. The recommended starting dose of intranasal desmopressin is 20 μg at bedtime. It should be dose-titrated, starting at 20 μg and increasing to 30 to 40 μg as necessary. There is also a tablet form of desmopressin. The clinically effective dose varies between 100 and 400 μg and a suitable starting dose is 200 μg given at bedtime.

The first studies on desmopressin in the treatment for nocturnal enuresis were performed by Dimson[55] and Tuvemo[56]. Both investigators reported excellent results in about half the number of subjects studied and a satisfactory effect in most of the remaining patients. Dimson studied 20 children aged 6-15 years treated with intranasal desmopressin. The children had not responded to earlier treatments, such as imipramine or the enuresis alarm. Eleven children became dry during the treatment. Four children had 5-6 dry nights per week whilst 5 children did not respond to the treatment. However, all the children relapsed after the treatment. No side effects were noted. Tuvemo conducted a double blind study of 18 children aged 6-12 years. Children were on either 20 μg desmopressin or placebo for 28 days. Results were excellent in 8 children; there was a partial response in another 8. Discontinuation of medication resulted in relapse.

Miller[57] undertook a long term study of children treated with intranasal desmopressin. Forty-six boys and nine girls aged 9-17 years were studied. All 55 children were initially given 40 μg desmopressin for 2 weeks. The criterion for successful treatment was a 50% reduction in the number of enuretic episodes. Those responding in the initial 2 weeks continued their treatment until dryness. It was found that 28 (51%) became completely dry but only half remained dry after stopping therapy.

Terho[58] studied 52 children aged 5-12 years over a period of 12 weeks. With desmopressin, 30% became dry and another 38% halved the number of wet nights. The remaining 33% were considered non-responders. Another part of the study was to evaluate the pharmacological safety of the drug. With continued treatment, all subjects tolerated desmopressin very well and no side effects were found.

Most studies to date have established that desmopressin is efficacious during the time of administration. Equivocal results however are noted after the medication is withdrawn although the majority of trials investigating the parameter were of short duration (less than 5 months).

In the few studies in which desmopressin was administered for longer periods (at least 6 months), results in terms of efficacy and safety have been encouraging. Lehotska et al[59] demonstrated a 70% long term success rate whilst Rittig et al[60] noted an 83% overall reduction in enuretic episodes. Studies are currently underway to evaluate the efficacy of long term desmopressin.

Side effects of desmopressin so far appear few and mild. They include headache, mild abdominal cramps, nausea, nasal congestion and epistaxis. A reversible body weight gain can be seen in some children[61-63]. It is however important to note that cases of water intoxication with hyponatremia and convulsions have been reported[64-67].

Not many comparative trials have been performed. Willie[68] compared the efficacy of desmopressin versus alarm systems and found that desmopressin was effective in the short term in 70% of children whilst there was an 86% success rate with alarm system. There was a quicker response in the desmopressin group but after 6 weeks on treatment, the alarm treated subjects had consistently more dry nights than those on desmopressin. When both therapies were withdrawn after 14 weeks of treatment, the relapse rate in the desmopressin group was significantly higher. Sukhai[69] compared the efficacy of alarm plus placebo with alarm plus desmopressin in a group of 28 children. The question was whether one would see an enhanced response by combining alarm and desmopressin. It was found that the combination of alarm and desmopressin achieved an additional dry night per week compared to the alarm plus placebo.

Others

Many other modes of treatment have been used to treat nocturnal enuresis including anti-cholinergics, musculotropic agents, bladder training, dry bed training, hypnotherapy and even special diets. However, the efficacy of these treatments is still controversial and require further evaluation before they can be routinely recommended.

DISCUSSION

Nocturnal enuresis, in the vast majority of cases, is an essentially benign condition but it causes a considerable amount of distress for the child and parents. Apart from the practical problems of constantly changing and washing bed-sheets, some parents feel that enuresis reflects poorly on their parenting skills or that it is a deliberate act by the child. This results in a stressful home environment and poor parent-child relationships. Interaction with peers also suffers because the child develops avoidance behaviour in order to prevent detection. A study by Meadow[70] showed that after the death of their parents, children perceived wetting their pants as next on the list of the most serious problems that could happen to them. With evidence that continued bed-wetting is associated with increased problems of social and emotional adjustment, the need for intervention is vital.

Regardless of the therapeutic approach chosen, it is imperative to involve both the parents and child in the decision-making process. It is important for the family physician or paediatrician to explain the causes and prognosis of enuresis and provide practical discussion on coping. Educating the family about nocturnal enuresis will dispel misconceptions, eliminate guilt feelings, and hopefully prevent punishment of the enuretic child[71].

Many theories have been expounded with regards to the aetiology on nocturnal enuresis. One of the most publicised theories in recent years is by a group of Danish scientists who have

identified nocturnal polyuria and abnormal secretion of antidiuretic hormone secretion in nocturnal enuresis. However, major questions remain as to why these patients do not wake up in response to the full bladder as is expected and how the alarm system seems to have such high cure rates in children if nocturnal enuresis is simply as a result of abnormal secretion of antidiuretic hormone. Another observation that suggests that urinary volume is only part of the puzzle is that children with sickle cell anaemia, who all have polyuria due to the infarction of the medulla of the kidneys, have an increased but far from the universal incidence of enuresis. Despite the many publicised theories about the aetiology of nocturnal enuresis, the cause remains unclear, although a few authors have postulated a multifactorial aetiology.

In evaluating treatments of enuresis, not only should the efficacy of the treatment be taken into account but, owing to the lack of severe consequence of the condition itself, the risk of treatment must be kept extremely low. In addition, not only the risk of toxicity of the regimen should be considered but also its cost. Treatment should be tailored to the patient's age, motivation and family support.

In recent years, desmopressin has been promoted as the therapy for nocturnal enuresis. Many studies have shown that desmopressin is highly effective in reducing wet nights in children for whom other treatments have failed. However, other studies have shown that it produces complete dryness in a minority and the relapse rate is high once therapy is discontinued. A few studies are currently underway to evaluate the long term efficacy of desmopressin and preliminary results appear promising.

The side effects associated with desmopressin appear to be fairly minor and infrequent. The one that is most feared is water intoxication. In healthy children and adults, this is probably rare but it is more likely to happen in children with problems of osmoregulation e.g. brain-damaged children or those with electrolyte-losing disorders. There are however studies that used lengthy courses of desmopressin to truly ascertain the safety profile of the drug. Clearly, more long term studies are necessary to address this issue.

Another major limiting factor in the usage of desmopressin is its cost. Treatment with desmopressin is expensive relative to other modalities. In Singapore, the cost of using desmopressin is approximately $81-$162 per month if the patient is on the tablet form and is on 200 μg - 400 μg per day. The cost of using the nasal spray is even higher - it costs $110-220 per month using doses between 20 μg - 40 μg per day. In contrast, the price for an alarm device is approximately $150. Imipramine, though less expensive than desmopressin, is fatal in overdose and as such, not a very popular pharmacological modality.

From the various studies reviewed, treatment with the alarm system has been shown to be effective, safe and inexpensive, with no need for systemic medication. However, non-compliance is a major problem. Some children fail to awaken following activation of the alarm, resulting in disruption of the household and rapid discontinuation of treatment.

Despite the problems inherent in the alarm system, review of most of the studies done so far reveals that the alarm system is superior in terms of effectiveness compared to other modalities. The use of desmopressin is best reserved for special occasions e.g. overnight camps, sleep-overs when rapid control is necessary or when the alarm method has failed after a 6-month trial. Desmopressin can also be used to temporarily show the unmotivated child the advantage of becoming dry. Temporary success could motivate the child to obtain a more permanent cure through alarm conditioning.

A problem noted in the review of the various studies is that different criteria have been used not only to select patients for treatments but also to measure outcomes. It is thus recommended that the criteria for defining terms like "success", "drop-outs" and "relapses" be standardised and an excellent reference is the "Minimum Standards of Practice in the Treatment of Enuresis" published by the Enuresis Resource and Information Centre (ERIC)[72]. An initial "success" is defined to be when 14 consecutive dry nights have been achieved within a 16 week treatment period. "Relapse" occurs when there are 2 or more wet nights within 2 weeks of discontinuing treatment and the interval between

an initial "success" and relapse should be stated. These are but some examples of definition of terms. It is recommended that these criteria should be used as standards to compare epidemiological data and response to various therapies.

It must also be remembered that there are many problems and variables in drug trials in enuresis. Confounding variables include patient selection and motivation. Individuals most likely to volunteer for such a trial are those whose enuresis is of greater frequency and who have failed previous treatment modalities. In addition, refractory enuretic patients are most likely to be approved for inclusion by hospital committees responsible for monitoring drug trials.

Additional long term studies are needed to better delineate the role of desmopressin as a therapeutic modality in the treatment of nocturnal enuresis. More randomised trials using alternative treatment modalities for enuresis are imperative due to the growth in the number of interventions available for enuresis and the lack of scientific knowledge available to help select between them.

Most cases of nocturnal enuresis can be successfully managed at the primary healthcare level. But, regardless of the pharmacological agents or other options available, the family physician should never lose sight of his role as an educator and counsellor for the patient and the family. The child and family need to know that they are not alone with the problem. It is important for the physician to emphasise to the parents that ultimately, understanding and positive support are crucial to the successful treatment of nocturnal enuresis.

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QUESTIONS FREQUENTLY ASKED ABOUT CATARACT AND ITS SURGERY

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Summary

Cataract is a major cause of impaired vision in the elderly. Surgery can improve the visual function of most patients with cataract. The family physician often has to answer questions posed by his patients about cataract and its surgery. Patients misinformed by hearsay may shy away from cataract surgery and suffer needlessly from a potentially reversible form of blindness or end up irreversibly blind from lens-induced complications. It is therefore important for the family physician to remain updated on this condition and to provide adequate information to his patients. This article reviews the current management of cataract and answers some frequently posed questions.

Keywords: *Cataract, extracapsular cataract extraction, intraocular lens implantation, lens-induced complications, phacoemulsification.*

INTRODUCTION

Cataract is a major cause of impaired vision in the elderly. Its prevalence increases with age. As the population of Singapore is aging rapidly[1], the number of people suffering from cataract is also on the rise.

Fortunately, cataract in amenable to treatment and good vision can be restored following surgery. Poor vision in the elderly poses a significant handicap and predisposes them to falls and other accidents. Visual rehabilitation reduces the frequency of these accidents and improves the quality of life in these patients. Visual rehabilitation is therefore an important component of the total rehabilitation of the elderly.

The family physician remains the usual initial point of contact between the patient and the medical profession. With increasing literacy in the population, patients are seeking more information about their illnesses. Patients with cataract often pose questions to their family physicians about cataract and its surgery before a referral to an ophthalmologist is made. The family physician therefore plays an important role in providing adequate information to their patients. Patients misinformed by heresay from friends and relatives may shy away from cataract surgery and suffer needlessly from a potentially

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reversible form of blindness. If left untreated for a long time, a cataract may cause complications and result in irreversible blindness.

QUESTIONS

The following questions frequently asked by cataract patients are discussed:-

1. Can my cataract be treated by medication or laser?

There is at present no known medication or laser that can treat cataract. Surgery remains the only treatment modality in which a cataract can be removed[2].

Although several eyedrops are available in the market which supposedly are able to reduce the rate of progression of cataract, their efficacy have not been conclusively proven. Once a cataract has formed and causes visual impairment, these eye drops will not improve vision as they do nothing to the cataract already formed.

A number of lasers are in common use for the treatment of a variety of eye disorders such as diabetic retinopathy, glaucoma and myopia. For this reason, many patients have the mistaken belief that cataract can also be treated by laser. This is not true.

Planned *extracapsular cataract extraction* (ECCE) with intraocular lens (IOL) implantation is at present the most common technique used to treat cataract in Singapore. This technique involves the removal of the anterior capsule, cortex and nucleus of the lens[2,3]. The posterior capsule is left intact to keep the vitreous in the vitreous cavity and to support an IOL in the posterior chamber.

A newer technique called *phaco-emulsification* involves a smaller surgical incision than conventional ECCE. In this method, ultrasound vibrations are used to break apart the cataract, fragmenting it into many pieces. The cataract is then removed using a suctioning device. Visual rehabilitation is more rapid after phacoemulsification because of the smaller incision. The final visual outcome is however, essentially the same as in conventional ECCE.

2. When should I go for cataract surgery?

Cataract surgery may be performed for a number of reasons[2]. The patient's desire to see better is the usual indication for surgery. There is no one particular level of visual acuity in which all patients should undergo cataract surgery. The main consideration for cataract surgery is the patient's visual requirement and this varies from person to person[4]. For example, a librarian with a posterior subcapsular cataract may need surgery when his vision falls to N8 or worse when reading in bright light even though his distant vision is still 6/12. A driver with nuclear sclerotic cataracts which reduce his visual acuity to 6/18 may require surgery although he can still read N5. On the other hand, an elderly patient who does little housework may be happy with fairly poor vision provided he or she can still watch television. In general, cataract surgery can be recommended to a patient when his visual disability interferes with his daily activities, employment or recreation.

A cataract may also be removed to prevent possible lens-induced complications. Occasionally, cataract surgery is performed to facilitate visualisation and treatment of ocular fundal diseases such as diabetic retinopathy.

3. Is it true that a cataract can only be operated when it is "mature"?

This is not true. The maturity of a cataract is a *morphological* diagnosis. An *immature* cataract is one in which scattered opacities are separated by clear zones in the lens. A *mature* cataract is one in which the entire lens is opaque. A mature cataract is associated with severe visual impairment and the visual acuity is usually in the range of counting fingers to light perception. Mature cataracts are therefore frequently removed to improve vision and prevent lens-induced complications. Many immature cataracts also cause significant visual impairment and they too should be removed if the patient's usual activities are disturbed by

the decreased vision. Many doctors and patients loosely use the term "mature" to denote a cataract that is causing significant visual impairment and therefore requires surgery.

4. What can happen if I do not operate on my cataract?

Cataracts usually increase in size and density over a period of months to years although some may remain stationary. Long-standing cataracts may cause lens-induced complications.

A cataract may become swollen *(intumescent)* when it imbibes water. An intimescent cataract blocks the pupil and pushes the iris anteriorly. This closes the anterior chamber angles and reduces the outflow of aqueous through the trabecular meshwork. As a result, the intraocular pressure rises acutely causing *phaco-morphic glaucoma.*

A cataract may also become *hypermature* due to leakage of denatured lens proteins through an intact lens capsule. It then becomes smaller and has a wrinkled appearance. Leakage of lens proteins stimulates a macrophagic reaction in the anterior chamber. These macrophages engulf the proteins in the anterior chamber and become swollen. Both the swollen macrophages and protein molecules can obstruct the trabecular meshwork and cause the intraocular pressure to rise. This is called *phacolytic glaucoma.*

An eye with an uncomplicated cataract does not show any sign of inflammation. In *lens-induced (phacomorphic or phacolytic) glaucoma*, the eye becomes red and painful. The patient may also experience nausea, vomiting and a unilateral headache on the side of the complicated cataract. Lens-induced glaucoma is an ophthalmic emergency as the high intraocular pressure can damage the optic nerve and result in irreversible blindness if treatment is delayed. The intraocular pressure has to be reduced rapidly with medication and the cataract extracted as soon as possible.

5. Will my visual outcome be better if I operate on my cataract earlier rather than later?

Before the onset of lens-induced complications, the visual outcome following cataract surgery is not dependent on the density or maturity of the cataract. Cataract removal clears the visual axis of a media opacity and the final visual outcome of the eye is dependent on the health of the rest of the eye as a whole. Barring any intra-or post-operative complications, eyes with different degrees of cataract but are otherwise equally healthy may be expected to achieve approximately the same final visual outcome. Eyes with concomitant ocular pathology such as glaucoma, age-related macular degeneration or diabetic retinopathy may still have impaired vision following cataract extraction because of these disorders.

Eyes with a worse preoperative visual acuity due to cataract may experience a more dramatic visual improvement after surgery compared to those with a better preoperative visual acuity. This is because of the larger difference between the preoperative and postoperative visual acuity. For example, an eye with a preoperative visual acuity of counting fingers will experience a larger improvement of visual acuity than one with a preoperative visual acuity of 6/18 even if both the eyes achieved a final postoperative visual acuity of 6/9. This is why some patients mistakenly feel that it is better to have surgery when the cataract is advanced.

6. What complications can occur with cataract surgery?

A number of complications can occur with cataract surgery[3-5]. Some of them are sight-threatening while the majority are not.

The most feared complication is post-operative infection *(endophthalmitis)*. The incidence of this complication is about 0.1% in most modern centres[5]. The common causative organisms are *Staphylo-coccus epidermidis, Staphylococcus*

aureus, Pseudomonas aeruginosa and *Proteus sp.*. Endophthalmitis commonly presents during the second to the fourth postoperative day. The patient usually has redness, pain and decreased vision in the eye. He may also have oedema of the eyelids, chemosis, corneal haze, a fibrinous exudate or hypopyon in the anterior chamber, vitritis and an absent or diminished red reflex. Occasionally, these features are delayed in onset and less severe. This is likely to occur when the offending organism is relatively less virulent (e.g. *Staphylococcus epider-midis*).

Postoperative endophthalmitis is a devastating complication that can result in blindness. It is an ophthalmic emergency and requires immediate medical attention. Successful treatment depends on the identification and elimination of the offending organism with topical, subconjunctival, intravitreal and intravenous antibiotics and possibly vitreous surgery.

The posterior capsule of the lens may be ruptured inadvertently during cataract surgery. The vitreous may then prolapse forward and this may result in a number of complications including updrawn pupil, uvetis, secondary glaucoma, retinal detachment and chronic cystoid macular oedema. These complications are reduced if the prolapsed vitreous is adequately removed during the operation. In the presence of a *posterior capsule rupture*, there may not be enough capsular support for a posterior chamber IOL to be implanted. In such a case, an anterior chamber IOL may be inserted.

Other complications of cataract surgery include *iris prolapse, glaucoma, cystoid macular oedema* and *retinal detachment.* These complications are best managed by the ophthalmologist.

7. Do I need to be hospitalised for cataract surgery?

Cataract surgery is now commonly performed as day surgery under regional anaesthesia. Patients are usually observed for several hours after the operation before they are allowed home. General anaesthesia may be preferred in selected patients such as those who are anxious, uncooperative or mentally deficient. Day surgery is suitable for healthy patients and patients whose medical conditions such as hypertension and diabetes mellitus are well-controlled. Patients with more serious or multiple medical problems are best operated as in-patients.

8. Can my cataract recur after cataract surgery?

Cataract does not recur following surgery. A condition sometimes known as *after-cataract* does occur in some patients several months to years after extracapsular cataract extraction. It is more commonly known as *posterior capsule opacification*. The posterior capsule of the lens that is left intact to support a posterior chamber IOL can become opacified following cataract surgery to cause a mild decrease in visual acuity. This problem can be remedied by making a defect in the opacified posterior capsule with a Nd:YAG laser (*Nd:YAG posterior capsulotomy*). This laser procedure is relatively simple and painless and is usually done in the outpatient clinic.

9. Will I still require glasses after cataract surgery with IOL implantation?

There is usually a residual refractive error following cataract surgery even if an IOL has been implanted in the eye. Glasses may therefore be necessary to correct this refractive error. The glasses that are used are the usual lenses used to correct low degrees of ametropia and are not the thick convex aphakic glasses that are used when no IOL is implanted. In addition, unlike the normal crystalline lens which can mould itself during accommodation, the IOL cannot change its power and therefore a pseudophakic eye cannot accommodate. Additional glasses may therefore be required for near vision such as during reading.

It must be emphasised that the need for

glasses is dependent on the patient's visual requirements. An elderly lady whose main task is to look after her grandchildren may be extremely happy after a cataract operation that improved her vision from counting fingers to 6/18 and may not wish to wear glasses even though with them she may obtain 6/9 vision.

10. Do I need to clean, service or change my IOL?

This question commonly arises because some patients think that an IOL is similar to a contact lens. An IOL does not require cleaning or servicing. It remains in the eye and does not normally need to be replaced.

CONCLUSION

With Singapore's rapidly ageing population, the prevalence of cataract is on the rise. Cataract surgery can improve the visual function of most patients with cataracts. Patient education is therefore important to allay the fears of patients who may otherwise shy away from cataract surgery and end up irreversibly blind from lens-induced complications.

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A HEARING SCREENING PROGRAMME FOR PRESCHOOL CHILDREN IN A PRIMARY HEALTH CLINIC

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Summary

We studied 510 asymptomatic preschool children, aged four and five years, who attended Toa Payoh Polyclinic for their health surveillance programme. The objective of the study was to obtain the screened out rate of abnormality of ear pathology and hearing impairment in asymptomatic preschool children. The hearing screening package consisted of age appropriate history, otoscopy, audiometry and tympanometry.

The screened-out rate of abnormality was seen in 115 cases or 24.5% in the group. Serous otitis media (SOM) formed 22.5% and eustachian tube dysfunction (ETD) 2.0%. There were no cases of sensorineural hearing loss. The screened-out rate of abnormality by otoscopy was 23.4% (11.2% due to impacted wax; 11.6% due to SOM; 0.4% due to otitis externa; and 0.2% due to foreign body); by tympanometry 34.1% (6.1% ETD; 27.9% SOM) and by audiometry was 10.4% (8.8% failed partially, 1.6% failed totally). Except for 8 cases (1.6%) who were referred to the hospital's Ear, Nose and Throat department, the rest were followed up in the clinic. Using only otoscopy with audiometry or tympanometry gave readings ranging from 7.1% to 17.3 units above the 24.5% which was the final outcome of the screened out rate of abnormality.

Although otoscopy, audiometry and tympanometry, have the potential of detecting all ear abnormalities the package would require a cost effective assessment before it could be recommended for mass screening of preschool children. Until then only those at risk with persistent ear symptoms should be subjected to the total package of tests.

Keywords: *Audiogram, otitis media, tympanogram*

* *Deputy Director*
** *Medical Officer*
*** *Senior Registrar*
Family Health Service
Ministry of Health
Singapore

\+ *Consultant Audiologist*
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INTRODUCTION

The Primary Health Division of the Ministry of Health provides a comprehensive health surveillance programme for preschool children in Singapore. It covers six years of preschool period with ten visits to the polyclinics. The services provided include coverage of immunisation schedule, health check-ups, growth and developmental assessments, and hearing and vision screening[1].

Currently the hearing tests are done as a part of the health surveillance programme in the government polyclinics, and offered free of charge to all asymptomatic preschool children from birth to six years of age. An age appropriate hearing history is taken at all well-child visits. At six weeks to three months of age, the child's response to a loud bell (of about 60 dB to 90 dB) is checked. At six to nine months of age, the Manchester rattle and the Viennatone are used as instruments for free field sound distraction tests. After infancy, no formal hearing checks are offered. But children with ear symptoms have clinical and autoscopic checks. Referrals have been offered to those with a history of delayed language development or suspicious hearing behaviour.

In this paper, we report the findings of the hearing screening of a group of 510 asymptomatic preschool children aged four and five years, who attended Toa Payoh Polyclinic for their health surveillance programme, during a six month period from June to November in 1992. This is a pilot project where a hearing screening package of otoscopy, pure-tone audiometry and tympanometry was used.

In Singapore, as the local family units have shrunk from 4.5 children per family in 1966 to 1.7 in 1991, children have become especially precious[2]. Concurrently the need to identify any abnormality through formal childhood screening programmes has become even more important now than a decade ago. In other developed countries, although serous otitis media was found in up to two thirds of their preschool children, the prevalence of sensori-neural hearing loss of about 50 dB was present only in 1 to 2 per 1000 children[3]. Though local epidemiological figures of deafness in preschool children are not available, figures from studies done in Europe show that the most common cause of conductive hearing loss was found to be serous otitis media[4]. and the prevalence is as high as 50% to 60% in children aged two to six years[5]. To date no documented local study results in preschool children is available.

To address this lack of local epidemiological data on hearing impairment in preschool children, this pilot study was conducted with the objectives of:

i) Obtaining the screened out rate of abnormality in the ear in asymptomatic children aged four to five years, attending the health surveillance programme in a polyclinic and

ii) Identifying the feasibility of screening using a screening package with otoscopy, pure-tone audiometry and tympanometry.

MATERIALS AND METHODS

This study was conducted at the Toa Payoh Polyclinic's Maternal & Child Health Section from 29th June to 30th Nov 1992. All children born in 1988 and 1987, who were aged four or five years on 1st June 1992, and who were registered in the clinic under the health surveillance programme, were selected. Those with known ear and hearing abnormalities, and a history of middle ear surgery were excluded because the primary objective of hearing screening programme was to find the screened out rate of abnormality among asymptomatic children. Among a total of 644 children who were given appointments to attend the clinic for hearing screening, 17 did not keep their appointments. A further five children we excluded from the initial six hundred and twenty seven, as they were not co-operative in their otoscopy, audiometry or tympanometry. These were excluded from the study. One hundred and twelve children failed to turn up after two recall letters for a retest four to six week after an initial finding of the abnormalities in either otoscopy, audiometry or tympanometry. This one hundred and twelve children (17.9%) were considered to be 'drop outs' and were excluded. Our final sample was 510 children.

Procedure

All the 510 children were checked on their age appropriate hearing history and their particulars recorded in a pre-prepared questionnaire. They were then subjected to clinical examination by the polyclinic doctor who also performed the otoscopy, audiometry and tympanometry. The polyclinic doctor had a two-week attachment with the ENT specialist prior to the study to update her knowledge and operational skills in the use of the hearing screening package.

Otoscopy

Otoscopy was then performed on the child, in the usual manner. Assessment of the child with middle ear pathology also included a full examination of the upper respiratory tract, as it is known to affect the hearing status of the middle ear.

Audiometry

The MAICO 25 puretone audiometer was used in this study. The child was told to place a block into a cup on hearing a 4000 Hz pure tone presented at 60 dBHL via the headphones. It was deemed necessary to raise the threshold of hearing intensity from 25 dBHL to 40, 30, and 30 dBHL and the frequencies to 500, 1000 and 4000 Hz respectively, the reason being that the test environment was not sound-proof and this situation although unfavourable, is representative of most polyclinics conducting hearing screening for preschool children.

If the child was unable to do the test due to distractions, or was in an uncooperative mood, another appointment was given to repeat the whole procedure.

Tympanometry

The Welch Allyn Microtym was used. It gives adequate readings to highlight the status of the middle ear. In tympanometry, a probe was inserted into the patient's ear and the pressure in the ear canal varied gradually from +200 mm to -200 mm water. The pressure changes were noted in the form of computer recordings. Specific patterns of these recorded graphs represent the main middle ear pathology e.g. normal, perforated tympanic membrane, eustachian tube dysfunction and serous otitis media. Children with impacted ear wax were treated with syringing before tympanometric readings were recorded again.

Recording of Results

The 'Final Outcome' for children who passed all the 3 components of the screening at the first visit were recorded as 'normal'. Children who failed any one of the screening procedures, due to lack of co-operation, were given appointments to return in a months's time for a reassessment using the screening package. Those with pathology treatable at primary care level, e.g. ear wax, upper respiratory tract infections, otitis externa and otitis media were treated before the retests. If any of the retest results were abnormal after a month despite treatment, the child was then referred to the audiologist. A total of 336 tympanometer recordings together with the children's audiograms and records of otoscopy findings were despatched to the audiologist for her concurrence. Among the 336 cases, 145 children were actually reassessed by the audiologist. The tympanometric results read by the audiologist were recorded as the 'Final Outcome'. If the audiologist deemed it necessary, the case was referred to the Ear, Nose and Throat surgeon in the hospital. However cases with foreign bodies and perforated tympanic membrane were referred to hospital directly. For children referred, the diagnosis made by the ENT surgeon were recorded as the 'Final Outcome'. For cases not referred, the results of the clinic doctor was taken as the 'Final Outcome'.

The data collected were analysed using dBase III and a Statistical Software Statpac Gold 42.

RESULTS

Final Outcome of Screening

One hundred and twenty-five cases (24.5%) failed the screening package because 2.0% had eustachian tube dysfunction and 22.5% had serous otitis media. The screening results of individual components of the screening were cross-tabulated with 'Final Outcome' and compared (Table 1).

Table 1: Otoscopy Findings and the Final Outcome

OTOSCOPY FINDING		FINAL OUTCOME				
		Normal	Eustachian Tube Dysfunction	Serous Otitis Media	Total No	Total %
Normal in both ears		318	7	66	391	76.7
Impacted Wax		30	1	26	57	11.2
Otitis Externa		2	0	0	2	0.4
Serous Otitis Media		34	2	23	59	11.5
Foreign Body		1	0	0	1	0.2
Total	No	385	10	115	510	100.0
	%	75.3	2.0	22.5		

Table 2: Tympanometric Findings and Final Outcome

TYMPANOMETRIC FINDING	FINAL OUTCOME				
	Normal	Eustachian Tube Dysfunction	Serous Otitis Media	Total No	Total %
Normal	297	3	36	336	66.0
Eustachian Tube Dysfunction	20	3	8	31	6.1
Serous Otitis Media	68	4	71	143	27.9
Total	385	10	115	510	100.0

False negative rate = (3 + 36) / 336 = 11.6%
False positive rate = (20 + 68) / (31 + 143) = 88/174 = 50.6%

Table 3: Audiometric Findings and Final Outcome

AUDIOMETRIC * FINDING	FINAL OUTCOME				
	Normal	Eustachian Tube Dysfunction	Serous Otitis Media	Total No	Total %
Normal	358	8	91	457	89.6
Partial	22	2	21	45	8.8
Abnormal	5	0	3	8	1.6
Total	385	10	115	510	100.0

** AUDIOMETRIC FINDING:*

Normal – Passed all first audiometric tests done at the clinic in both ears at 500Hz, 40db; 1000Hz, 30db; 4000Hz, 30db

Abnormal – Failed all first audiometric tests done at the clinic in both ears at 500Hz, 40db; 1000Hz, 30db; 4000Hz, 30db

Partial – Neither "Normal" nor "Abnormal", passed some and failed some tests. 'Unsure' results were taken as 'failed'.

Otoscopy

Otoscopic findings are shown in Table 1. While 391 cases were normal by otoscopy, 318 cases were normal by tympanometry, a difference of 73 cases. While 66 cases had serous otitis media by tympanometry, only 23 had the same by otoscopy, a difference of 43 cases. These 43 cases were missed by otoscopy due to the inexperience of staff in recognising SOM.

Tympanometry

Table 2 illustrates the tympanometry findings. Although 385 cases were normal by tympanometry, in the final outcome 297 were normal. (Hence without tympanometry 88 cases would have been missed). The false negative rate was 11.6% and false positive rate was 50.6%.

Audiometry

The audiometric findings are presented in Table 3. A case was classified as 'normal' if the child passed all first audiometric tests done at the clinic in both ears at 500 Hz, 40 dB; 1000 Hz, 30 dB; and 4000 Hz, 30 dB; 'partial' when they failed some of the screening criteria and 'abnormal' when they failed all tests.

Among the 115 cases with serous otitis media in the final outcome, 91 cases were normal, 21 were partially abnormal, and 3 were totally abnormal. By audiometry only 8 or 1.6% cases had impaired hearing.

Comparison of Audiometric and Tympanometric Findings

The comparison of the audiometric and tympanometric findings are presented in Table 4. While only 329 cases were normal by audiometry, 336 were normal by tympanometry, a difference of 7 cases which may have been missed without the use of audiometry.

Hence the co-positivity was 9% and the

Table 4: Comparison of Audiometric and Tympanometric Findings

AUDIOMETRIC FINDING	TYMPANOMETRIC FINDING				
	Normal	Eustachian Tube Dysfunction	Serous Otitis Media	Total	
				No	%
Normal	329	26	102	457	89.6
Partial	6	4	35	45	8.8
Abnormal	1	1	6	8	1.6
Total	336	31	143	510	100.0

Co-positivity = (4 + 1 + 35 + 6) / 510 = 46 / 510 = 9.0%
Co-negativity = 329 / 510 = 64.6%

Proportion failed the tympanometric test but passed the audiometric test = (26 + 102) / (31 + 143) = 128 / 174 = 73.6%
Proportion failed the audiometric test but passed the tympanometric test = (6 + 1) / (451 + 8) = 7 / 53 = 13.2%

Table 5: Comparison of the Audiometric Findings of the Clinic Doctor and Audiologist

FINDING OF DOCTOR*	FINDING OF AUDIOLOGIST		
	Failed	Passed	Total
Failed	5	51	56
Passed	6	83	89
Total	11	134	145

+ Passed – Passed all audiometric tests done in both ears at 500Hz, 40db; 1000Hz, 30db; 4000Hz, 30db.

** Failed – Failed all audiometric tests.*

conegativity 64.6%. Hence the population that failed tympanometry but passed audiometry was 73.6%; the proportion that failed audiometry and passed tympanometry was 13.2%.

Comparison of Audiometric Findings as Recorded by Clinic Doctor and Audiologist

The results of a total of 145 pairs of readings were pooled and presented in Table 5. For 14 children, audiometry was done by both the polyclinic doctor and the audiologist. The rest of the results were based on audiometric and tympanometric records seen by both. The results were compared and tabulated.

51 out of 56 or 91.1% passed audiometric tests

Table 6: Comparison of Results of Abnormalities in Ear Screening of 510 Preschool Children

	Normal		Abnormal		Deviation from Standard
	No	%	No	%	
1. Final Outcome	385	75.5	115	24.5	Standard
2. Otoscopy	318	62.4	192	37.6	+ 7.1
3. Audiometry	353	69.2	157	30.8	+ 6.3
4. Tympanometry	297	58.2	213	41.8	+ 17.3

done at both the polyclinic and at the audiologist clinic. Of these 6.7% passed the audiometric screening at the polyclinic but, failed at the audiologist's centre.

Comparison of Results by Different Equipment

When comparing the results of screened-out rate of abnormalities using otoscopy, audiometry and tympanometry with the gold standard of final outcome, the results with all the 3 equipment done individually gave results above the final outcome. By otoscopy alone the screened out rate of abnormality was 37.6%, 7.1 units above final outcome, by audiometry it was 7.6% or 6.3 units above, and by tympanometry 41.8% or 17.3 units above final outcome (Table 6).

Referral To ENT Specialist

Eight cases were referred to the ENT specialist in the hospital. Of the eight cases, one was reported as normal by the surgeon and seven cases were diagnosed as having serous otitis media.

DISCUSSION

In a comprehensive and efficient health surveillance programme for preschool children, hearing abnormalities would be picked up well before four years of age, in particular, those related to congenital abnormalities and growth defects. These hearing defects are usually sensorineural in type. However the situation may be contrary in asymptomatic preschool children, where one in 12 have signs of past or present

otitis media with conductive hearing defects[7]. Checking of middle ear function at intervals of one to two months may identify those at risk of problems requiring prompt intervention, in order to prevent future developmental problems with speech and behaviour[8,9]. This is because middle ear pathology may develop any time after the initial screening in infancy. Those with unresolved serous otitis media may also develop middle ear fibrosis, adhesions and cholesteatoma[10]. In this pilot project conducted in Toa Payoh Polyclinic, targeted towards children aged 4 and 5 years old who attended the clinic for health surveillance, an attempt was made to discover what proportion had ear abnormalities although they were asymptomatic.

Hearing screening of young children can be difficult and time consuming and often requires repeated assessments in order to confirm the results obtained in an initial assessment. Significant hearing loss can occur due to background noise levels, if no adequate sound proofing is provided in the test environment. No one method of hearing tests is known to detect all ear pathology and hearing abnormalities[7]. A fully comprehensive hearing screening is possible only after appropriate clinical history taking, clinical examination, and competent use of otoscopy, pure tone audiometry and tympanometry. While autoscopy enables the clinical assessment of the status of the ear drum and external ear, puretone audiometry demonstrates the hearing levels, and tympanometry measures function of the middle ear. Although a comprehensive hearing package should include otoscopy, audiometry and tympanometry, the practical difficulties in availability of resources and high costing have been in disfavour for their use in a mass screening programme for children[7,8]. Those children with temporary conductive hearing loss due to serous otitis media experience a self-limiting period of hearing loss. A false positive labelling and referrals of these children would be unnecessary[11]. Until studies of cost effectiveness favour mass screening, the routine use of these equipment would be available only for a selective group of patients.

There were inevitable limitations in this study which were beyond our ability to control. With limited resources available, no additional funds could be obtained for an ideal environment, additional staff or selection of a truly random group of preschool children and a control group from the community. Because the polyclinic staff were using the audiometer and tympanometer for the first time with only a short period of training in the ENT department, their output was different from the audiologist, an expert in the field. This varied level of skills could have influenced the level of accuracy of the readings. Also due to a lack of a sound proof room in the polyclinic, audiometric readings may have been muffled by the external noise levels. In addition, doing puretone audiometry in preschool children needed much more refined skill than testing in adults. These were some of the possible reasons why there were disparities in the audiometric readings of the clinic doctor and the audiologist.

A great disadvantage was that all the 510 cases could not be subjected to examinations by both the polyclinic doctor and the audiologist because the parents thought the cost and the distance were impediments. The tympanometric and audiometric readings of 336 cases could be referred to the audiologist for her concurrence. But only 145 children could actually be assessed by both the polyclinic doctor and the audiologist. This is why the specificity, sensitivity and predictive values of test results could not be evaluated. Despite all these short comings, the information obtained from this study was useful for the knowledge and skills of the staff and the programme planners.

Among ear abnormalities, serous otitis media is most commonly seen in children. Serous otitis media (SOM) is also known as middle ear effusion (MEE), otitis media with effusion (OME) or glue ear[9]. In older preschool children, conductive rather than sensorineural deafness is more common[12] and the majority is due to SOM. The prevalence of SOMs in preschool children show a wide variation in various studies. This is perhaps because different techniques and criteria were used. Bain found the prevalence of SOM to be 3% to 4% in Canadian children aged 5 to 8 years[7]. A Danish study showed the same to be 14.2% in a control group where doctors were not trained to use the tympanometer. Where doctors were especially trained to use the tympanometer

the rate of SOM went up to 25%[13]. Maw reported the prevalence of SOM in children below 6 years as ranging from 20% to 30%[15]. In our study the screened-out rate of abnormality, the majority being SOM, was 24.5% in children aged 4 and 5 years old. This figure seems to be in line with the studies mentioned above.

How closely this rate compares with the national prevalence in the country can only be established through future studies, using experienced staff, standardised equipment and test rooms with adequate sound proofing. Accurately conducted studies would require a strict standardisation of all these variables. It was noted that individual methods of hearing tests produced results above that of the final outcome: i.e. by 7.1% for otoscopy, 6.3% for audiometry and 17.3% for tympanometry (Table 7). The co-positivity of 9% and conegativity of 64.6% in tympanometry further indicate the higher rate of false positive readings attributable to the highly sensitive nature of this equipment. Despite this oversensitivity of tympanometry the early detection of SOM is important in order to pinpoint the proportion of children that may not resolve spontaneously[14-16]. More attention may also be given to detect SOM in children with upper respiratory tract infections[17], those exposed to environmental pollution with passive smoking[18] and those at risk for hearing loss[19]. Although these may sum up to be of a small proportion, it is imperative to refer the unresolved group for specialist intervention[20,21]. In our group eventually only 1.6% with impaired hearing were referred to the hospital where 7 out of 8 cases had serous otitis media and are being followed up. However tympanometry and audiometry can be effectively used in a family medicine practice, where either the doctor or the nurse is specially trained[23,24]. Regular otoscopy, audiometry and tympanometry are useful tools for follow-up of cases with ear problems to enable effective management[25]. This would also facilitate the early detection of new cases of childhood deafness for prompt intervention[26-28]. The primary care and family physicians are in a unique position to do this. Where mass screening is concerned however, known evidence reports that screening for hearing impairment should be done for all high risk neonates and those children with history of impaired hearing, congenital perinatal infections and birth defects and other situations at risk for intrauterine anoxia or hypoxia. So far there has been insufficient evidence that recommends routine audiological testing of all preschool children aged 4 and 5 years. However audiological testing often occurs in a clinical setting where abnormal results can be further verified, referred for on-going audiological assessment and prompt intervention when necessary with the selection of aids, family counselling, educational management and periodic medical evaluation[11].

Table 7: Types of Cases Referred to the ENT Specialist in the Hospital

Case No.	Final Outcome at ENT	Abnormal Screening Findings at Toa Payoh Clinic
1	normal	failed audiogram; otitis externa
2	SOM	otitis externa
3	SOM	SOM; failed audiogram
4	SOM	SOM; wax
5	SOM	SOM; failed audiogram
6	SOM	SOM
7	SOM	SOM
8	SOM	SOM

SOM: Seous otitis media

CONCLUSIONS

A pilot project was conducted in Toa Payoh Polyclinic among 510 asymptomatic preschool children aged four and five years. A screened-out rate of abnormality was 24.5%, of which 22.7% was due to serous otitis media and 2.0% due to eustachian tube dysfunction. Although the rate of SOM in our study seems to be higher than generally accepted, it is worthwhile to conduct further studies to substantiate or disprove our finding. However there is a need to standardise skills of the staff performing the tasks and adequate sound proofing of test environment to establish accurate results. Till then, only preschool children at risk, and those with persistent symptoms should be subjected to this package of otoscopy, audiometry and tympanometry.

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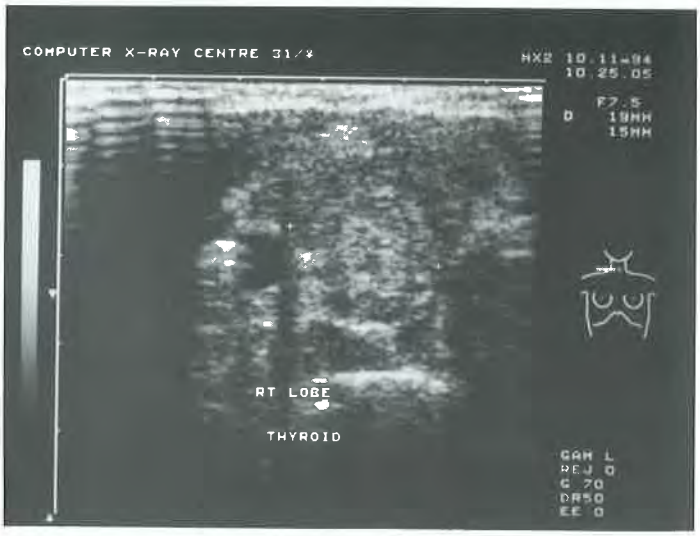
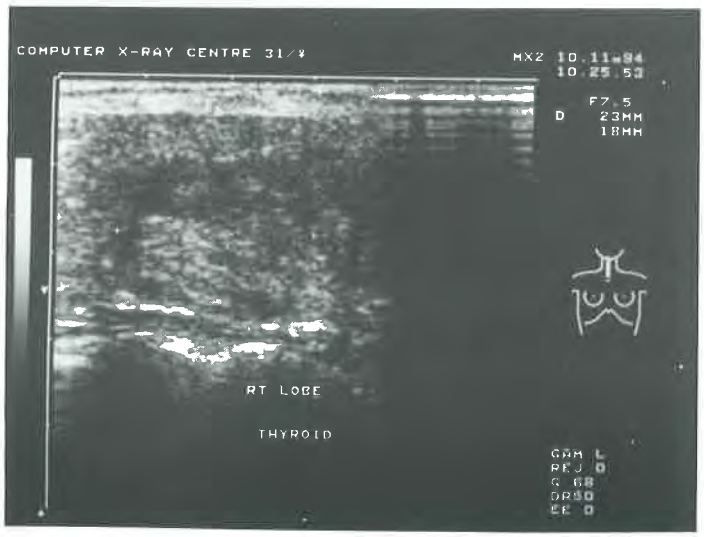
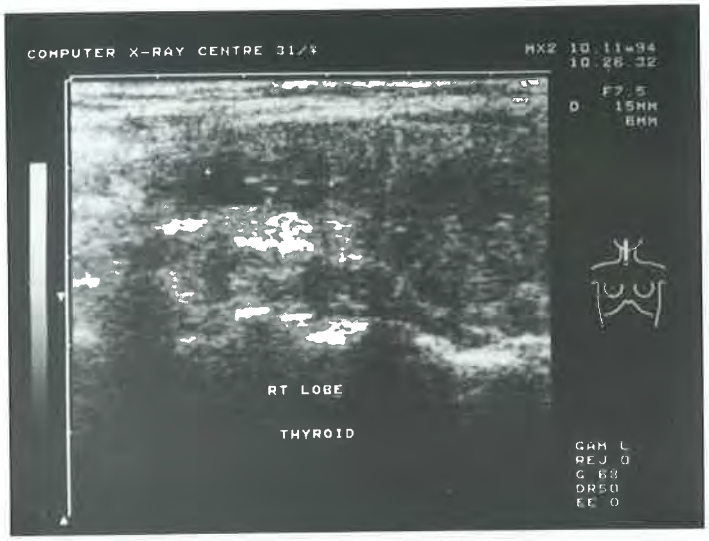
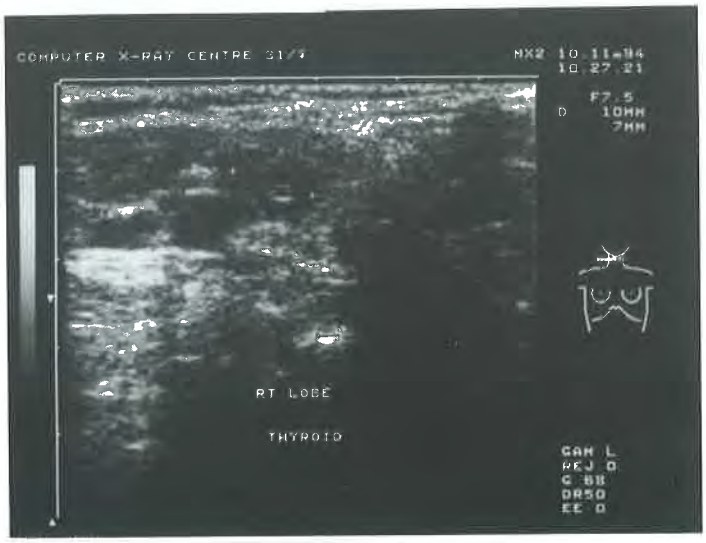
THYROID ULTRASOUND*

A 31 year old female presented with complaint of a swelling in the neck for the past 5 years. Clinical examination revealed a well-defined mass at the lower right side of the neck. There was no retrosternal extension; cervical lymph nodes were not palpable. There were no signs suggestive of thyrotoxicosis. The following is this patient's thyroid ultrasound:









Questions:

1. Describe what is seen on the ultrasound scan.
2. What is the most likely diagnosis?
3. How would you mange the patient?

For ultrasound report, please turn to page 123

ANSWERS

Thyroid Ultrasound – Report

1) Ultrasound shows it to be well defined solid mass sited at lower half of its right lobe. There is another mass, 15 mm x 6 mm demonstrated at its upper pole.

2) Isthmus and left lobe are normal for comparison.

3) Morphologically it is an adenoma. Ultrasound cannot exclude malignancy although there is no clinical support. Most surgeons will proceed to FNB (fine needle aspiration) for cytology. Isotope scan will differentiate hot nodule from cold nodule. 20% of cold nodules may be malignant.

* Clinical history and ultrasound films courtesy of
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THERAPEUTIC NOTES: HYPERURICAEMIA AND GOUT

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ASYMPTOMATIC HYPERURICAEMIA

Hyperuricaemia beyond 9 mg/dl is a risk factor for gout but the risk is only 5 percent per year. Also asymptomatic hyperuricaemia does not have any adverse effects before the development of gout. Drug treatment of asymptomatic hyperuricaemia is therefore not required, but it is prudent to treat correctable causes like high purine intake, obesity, regular alcohol consumption and hydrochlorothiazide therapy.

GOUT

Gout is a clinical syndrome resulting from the deposition of urate crystals in a joint. This results in an acute inflammatory response. Deposition in soft tissues such as cartilage causes no inflammation.

Acute Gouty Arthritis

A choice of three treatments is available. Colchicine or NSAID is the first choice with prednisolone as an alternative.

Colchicine

Colchicine is started with 1 mg initially and followed by 0.5 mg every two hours until abdominal discomfort or diarrhoea appears or a total of 8 mg has been given. Most patients have pain relief by 18 hours. The doses should be reduced by 50% in patients with hepatic or renal disease and in elderly patients.

NSAIDs

Any NSAID will do. Indomethacin, the first of these drugs to be used extensively, provides relief within 4 hours. The appropriate dose is 150 to 300 mg per day, given in three divided doses until the pain resolves. The usefulness of NSAIDs is limited by their side effects, but in general, the risks are greatest in elderly patients, particularly those with renal dysfunction.

Prednisolone

Systemic therapy is indicated only when NSAID and colchicine have been ineffective or are contraindicated. The dosage is 30 to 50 mg per day.

Interval Gout

In a patient with established gout, the acute attacks may be prevented by either colchicine or a NSAID. Colchicine is effective as an oral dose of 0.5 mg to 1 mg per day. The usual dose of indomethacin is 150 to 300 mg per day. Such treatment is necessary while correction of hyperuricaemia is being achieved.

Correction of Hyperuricaemia

If dietary and lifestyle factors are identified, appropriate changes should be made. Drug therapy to correct hyperuricaemia is indicated if the patient has had two or three definite attacks of gout or has tophi and the cause of the hyperuricaemia cannot be corrected or, if corrected, does not lower the serum urate concentration to less than 7.0 mg/dl (420 umol/l). The goal of therapy is to lower urates to 6.0 mg/dl (360 umol/l). A reduction to less than 5.0 mg/dl (300 umol/l) is required for the absorption of tophi. Two classes of drugs are available: uricosuric drugs and xanthine oxidase inhibitors. It is now established that urate-lowering drug treatment should be lifelong.

Uricosuric Drugs

These increase the urinary excretion of urate, thereby lowering the serum urate concentration. The greatest risk of therapy is the formation of uric acid crystals in the urine and deposition in the renal tubules, pelvis or ureter. These risks are reduced by initiating therapy with a low dose and increasing the dose stepwise and by maintaining a high urine volume, especially during the early weeks of therapy. Satisfactory control of hyperuricaemia to 6.0 mg/dl can be achieved with 1g probenicid a day in 60 percent of patients and in 83 percent of patients with 2g per day. The uricosuric effect of probenicid is decreased as glomeraular function declines and the drug has little effect with a creatinine clearance of less than 50 to 60 ml per minute.

Sulphinpyrazone is three to six times more potent than probenicid on a weight-for-weight basis. The initial dose should be 50 or 100 mg twice daily with gradual increments to 200 or even 400 mg twice daily. Alkalination of the urine with 1g sodium bicarbonate three to four times a day reduces the risk of uric acid crystalluria.

Xanthine Oxidase Inhibitors

Allupurinol is the only xanthine oxidase inhibitor in clinical use. It is effective in lowering serum urate concentrations in patients with overproduction or underexcretion of urate or both. The main side effect is hypersensitivity. A severe hypersensitivity reaction to allupurinol leaves a uricosuric drug as the remaining choice.

A dose of 300 mg of allupurinol per day reduces serum urate concentration to normal in 85 percent of patients with gout, and in some patients a dose of 100 mg to 200 mg is adequate. The risk of precipitating acute gout is reduced if therapy is begun with a low dose of 50 to 100 mg per day and increased stepwise during a period of three to four weeks. The dose should be decreased in proportion to the creatinine clearance. The appropriate dose is 100 mg per day in a patient with a creatinine clearance of 30 ml per minute, 200 mg per day in a patient with a creatinine clearance of 60 ml per minute, and 300 mg per day in a patient with normal renal function.

Two percent of patients treated with allupurinol develop a rash. This usually subsides after the allopurinol is withdrawn and may not recur if therapy is resumed with a lower dose. If the rash is an exfoliative dermatitis, which occurs in about 1 in 1000 patients, it is safer not to resume allupurinol after withdrawing it.

Difficult Clinical Problem

The most difficult clinical problem arises when a patient who has tophaceous gout with renal disease becomes sensitive to allupurinol and the sensitivity cannot be corrected with desensitisation. The renal disease is usually mild to moderate but contributes to the development of sensitivity to allupurinol. Such patients should be treated with a maximal dose of a potent uricosuric drug such as sulphinpyrazone, avoidance of diuretic drugs and more vigorous attention paid to the correctable factors promoting hyperuricaemia. If the serum urate concentration does not decline appreciably, the emphasis must be placed on symptomatic therapy rather than corrective therapy.

Further Reading

Emmerson BT. The management of gout. New Engl J Med 1996; 334: 445-54.

GUIDELINES FOR AUTHORS
THE SINGAPORE FAMILY PHYSICIAN

Authors are invited to submit material 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 or articles may be suitable for publication: case reports, original research works, audits of patient care, protocols for patient or practice management and review articles.

PRESENTATION OF 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 three copies of all elements of the article: summary text, references, tables and illustrations. The author should retain a personal copy.

The Title Page

* The title should be short and clear.

* Include on the title page first name, qualifications, present appointments, type and place of practice of each contributor.

* Include name, address and telephone number of the author to whom correspondence should be sent.

* Insert at the bottom: name and address of institution from which the work originated.

The Summary

* The summary should describe why the article was written and give the main argument or findings.

* Limit words as follows: 100 words for major articles; 50 words for case reports.

* Add at end of summary: an alphabet listing of up to 8 keywords which are useful for article indexing and retrieval.

The Text

The text should have the following sequence:

* Introduction: State clearly the purpose of the article.

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

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