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Paediatic respirology training in Hong Kong: where are we heading?

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This first issue of the newsletter marks an important milestone of the development of our society and provide an opportunity for the council to update its members on the development of our subspecialty training. Since the Hong Kong College of Paediatricians has announced its plan to launch subspecialty accreditation several years ago, our Society has been active in having paediatric respirology recognised as a subspecialty in the College. A Working Group on Post-graduate Training was formed under the Council and we have taken every opportunity to tap on the expertise of internal leaders in this field. The Forum on Paediatric Respirology Training during the 2004 IPRAIC Meeting in Hong Kong provided the most unique and invaluable opportunity for us to learn from the rest of the world. However, at the end of the day, we will have to decide a training programme that is suitable for Hong Kong. I believe the task of developing and implementing a training programme is particularly challenging for a society like Hong Kong which is not as well developed and resourceful (in terms of richness of clinical materials and research activities as most developed countries) and yet with a high standard of expectation. The task however, will be easier if we are right in two critical areas: a) what type of paediatric respirology specialist do we need and b) what are the critical elements for success and making sure that they are in place. The rest I believe can be further fine tuned as we go along.

What type of specialist do we need?

I, same as many others, believe that we need to train up paediatric respirology specialists who will be able to tackle a full range of paediatric respirology problems, some of these will be rare and complex. We need such highly skilled specialists to render services to our children so that children with such respiratory problems can be well taken care of, just like their counterparts in well developed countries. However, the requirement for such type of specialist is probably small and limited to perhaps one or two special centres in Hong Kong. These centres will need referrals from all the paediatric departments and also from the private sector to development and maintain a high level of expertise. Perhaps a great majority of children, especially those with common diseases like asthma or pneumonia will continue to be cared for by other paediatricians, who may well have completed the respiratory training programme but have settled down in places other than the specialised centres mentioned above. If this is going to be the case, the training programme will need to be designed to produce these types of products, and trainees should be fully aware of their career options.

What are the critical elements for success?

For the training programme to be successful, the following intrinsic and extrinsic factors are of vital importance. Efforts should be made to ensure that they are in place:

a) Its end products must meet the needs of our society, i.e. the needs our patients, paediatric departments and the private sector. It is perhaps easier said than done and requires careful thinking.
b) Sufficient resources to be given to training centres to ensure a high quality of training.
c) Stability of training staff should be ensured or the programme may collapse if the key persons leave the programme for one reason or another. This may mean obtaining commitment from the training departments to have such vacancies filled by qualified trainers in respirology.
d) Sending trainees to training should not mean significant loss of manpower strength, unless compensated.
e) An effective referral system for complex cases ought to be put into place to ensure concentration of cases and a high level of activities.
f) And lastly, and perhaps the most important pre-requisite is the determination and conjoint effort from the entire paediatric community of Hong Kong to make it a success.

It is hoped that this short article will stimulate the thinking of our learned members and the author will be obliged for any suggestions or comments on these issues.

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Allergic rhinitis and asthma : two ends of one airway

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Asthma is a worldwide health problem causing significant morbidity and mortality. Hong Kong has witnessed a significant increase in the prevalence of asthma over the past two decades. It is timely that the Society released the childhood asthma treatment guideline for Hong Kong in May this year and a more detailed account of the guideline is published in the current issue of the Journal. Complicating the picture is the often co-exisiting allergic rhinitis in the same patient. Up to 80% of asthma patients have concomitant allergic rhinitis. On 17th April 2005, a forum on management of paediatric asthma and allergic rhinitis was held in Prince of Wales Hospital, Hong Kong with support from this Society, the close relationship of these two diseases were emphasized. The "One Airway" concept, meaning allergic inflammation often affects both the upper and lower airway, as proposed by the WHO's allergic rhinitis and its impact on asthma (ARIA) guidelines was discussed.

The characteristics of asthma include the following findings: recurrent cough, wheeze or shortness of breath. Clinical signs are often absent between attacks. Although in chronic asthmatics, there may be Harrison's sulci and an increase in chest AP diameter from air-trapping. Allergic rhinitis is a condition characterized by sneezing, which is frequently paroxysmal, profuse clear watery rhinorrhoea and nasal obstruction leading to mouth breathing. Recurrent cough, especially on lying down or rising, is a common feature in allergic rhinitis children. Clinical signs include pale and swollen nasal mucosa, post-nasal drip and allergic shiners. Approximately 20% of allergic rhinitis cases are accompanied by symptoms of asthma. In severe cases, it has been shown that sufferers of allergic rhinitis are more likely to exhibit shyness; depression, anxiety, fearfulness and fatigue than those without the condition. Allergic rhinitis is classified as either intermittent or persistent. Persistent means symptoms are present more than 4 days a week and lasting more than 4 weeks a year.

For children with concomitant allergic rhinitis and asthma, monitoring with asthma and allergic rhinitis diary would be helpful to quantify the control. Identification of allergens and avoidance of the offending allergens would be helpful. This identification could be done by either skin prick test or radioallergosorbent test (RAST). Management of asthma in children is covered in the asthma guideline published in the current issue. For those with mild persistent asthma and intermittent allergic rhinitis with mild symptoms, preliminary evidence suggested that montelukast was effective for controlling both diseases. Further studies are required.

For those with persistent allergic rhinitis, treatment with topical nasal corticosteroids and/or systemic anti-histamines would be advised. Intranasal corticosteroids are effective in controlling all symptoms of allergic rhinitis. Satisfactory control can be achieved in over 90% of patients. The overall symptom control is superior to any other monotherapy. The effect of intranasal corticosteroids on the early and the late phase depend on the duration of treatment before exposure. Therefore, for maximal effect, it should be given regularly and commenced before expected exposure to allergen. Studies have shown a consistent reduction in the early phase reaction after 1 to 2 weeks of treatment. This may be related to the lowering of histamine release. A once daily dose is usually sufficient in most cases. A twice daily regime may be necessary in severe cases and during exacerbations. The aim of therapy is to use the minimal dose necessary to control symptoms. Several synthetic nasal corticosteroids are

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available for use in young children like mometasone for over 2 years old and budesonide for over 6 years old. Studies have shown that intranasal corticosteroids have no effect on the hypothalamic pituitary adrenal axis if used in recommended dosages because of the limited systemic bioavailability. However, growth suppression in children has been reported in some studies with the use of intranasal corticosteroids. The clinical importance of these findings in terms of final adult height is not presently known.

Second generation antihistamines are lipophobic with a large molecular size and possess an electrostatic charge. They generally do not cross the blood-brain barrier and exhibit little CNS effects. One of the major advantages of second generation antihistamine is its non-sedating or low sedating effects. The longer duration of action also means infrequent dosing which makes adherence to treatment easier. Other advantages include preferential binding to peripheral H_1 receptors and minimal antimuscarinic, adrenergic and anticholinergic effects. Examples of second-generation antihistamines include loratadine, cetirizine and fexofenadine. Loratadine and cetirizine are currently available for treating children less than 12 years of age. Antihistamines are more effective if occupation of H_1 receptors occurs before histamine is released; consequently the maximum benefit from antihistamine is achieved with prophylactic treatment before exposure to allergen. Antihistamines are less efficacious than topical corticosteroids. By combining with a decongestant or a topical corticosteroid, efficacy may be enhanced. Alternative treatments for allergic rhinitis included sublingual immunotherapy for those with monoallergen and acupuncture.

Conclusion

Childhood asthma and allergic rhinitis often occur together in the same child. Compared with asthma, allergic rhinitis severity is often under-diagnosed and untreated leading to unnecessary suffering. As allergic rhinitis often aggravates asthma control and symptoms of allergic rhinitis overlap with that of asthma, it is important to treat both diseases.

References are available on request.

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Upper airway obstruction in children

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Introduction

Upper airway obstruction in children is one of the most challenging acute emergencies, which requires urgent management in order to prevent fatal outcome. Severe upper airway obstruction accounts for 3.3% of all admission to paediatric intensive care unit (PICU).[1] There are many causes of upper airway obstruction that can be further classified as acute or chronic causes (Table 1).

In this review, the characters of children who are admitted to PICU with severe upper airway obstruction will be discussed further. Difficulties of intubation, indications and complications of tracheotomy will be discussed afterwards. Angioedema, bacterial tracheitis, acute epiglottitis and croup will be discussed with details separately. Laryngomalacia will also be discussed because it is one of the most common causes of stridor in infant. Patients with other anatomical abnormalities of the upper airway will not be discussed in this review, as most of them are under cared by the surgeons rather than paediatricians.

PICU admission with severe upper airway obstruction

There is marked heterogeneity in the causes of upper airway obstruction that requires PICU admission. According to studies conducted in Malaysia[1] and London,[2] congenital causes account for 6-23%, which include laryngomalacia, vascular ring, subglottic haemangioma, laryngeal cyst and web of pharynx. Acquired causes account for 77-94%, which include infection and anatomical problems. Viral croup is the most common diagnosis and it accounts for about 30% to 50% of all PICU admissions. Acute epiglottitis is extremely rare in Asia and also not common in Western countries. Bacterial tracheitis and subglottic stenosis are the most likely diagnosis requiring ventilation, as these patients are usually more sick and ill. Anatomical causes include tracheal compression, subglottic granuloma, subglottic stenosis and foreign body are other causes of PICU admission.

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Table 1. Acute and chronic causes of upper airway obstruction

Cause of upper airway obstruction	
• **Acute**	• **Chronic**
• Acute laryngotracheitis	• Laryngeal: Laryngomalacia, Subglottic stenosis/hemagioma, Vocal cord paralysis, Laryngeal web, Cyst (posterior tongue, aryepiglottic, subglottic, larygoceles, laryngeal cleft), laryngeal papillomas
• Acute epiglottitis	
• Suppurative tracheitis	
• Laryngeal foreign body	
• Diphtheria	
• Acute Angioneurotic oedema	• Trachea: Vascular ring, Tracheal stenosis, trachemalacia
• Retropharyngeal abscess	

Difficulty in intubation will be encountered in 43%.[1] However, tracheostomy is not common among children who required PICU admission. Regarding the prognosis, non-survivors have a higher Paediatric Risk of Mortality (PRISM) II score although the outcome is generally favorable. The conditions are similar in Hong Kong according to our experience in a teaching hospital (unpublished data).

Intubation

Difficulty in intubation will be encountered in half of the cases during intubations of severe upper airway obstruction patients. There are some characteristics of patients that we should pay special attention during intubation in order to facilitate the procedure. Delay in intubation in those critical ill patients could have very serious complication and even fatal.

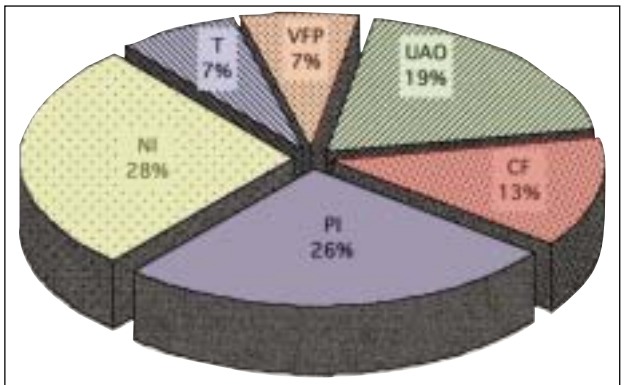
Pierre-Robin syndrome, Treacher-Collins syndrome, Goldenhar syndrome and mucopolysaccharidosis (Hurler, Hunter, Maroteaux-Lamy) are commonly associated with significant cranio-facial abnormalities, and these patients may have problems during intubation. They have micrognathia, relative macroglossia, hypoplasia of the facial bone, macrostomia and even short immobile neck. Mucopolysaccharidosis has excessive intra-lysosomal accumulation of glycosaminoglycans that causes generalised thickening of soft tissues. All these features make the procedure of intubation more difficult.

Inhalation injury makes visualisation of the normal airway anatomy more difficult. Epiglottitis is another

potential life-threatening cause for difficult intubation. Trauma can distort the normal anatomy of the upper airway and make the intubation more complicated. For these patients, an anesthetic approach should be adopted. Atropine pre-medication should be administered to dry up secretions, and oxygen should be given. It is better to use ketamine for sedation for these patients. Muscle relaxants should be withheld until the airway is secured. Intubation should be performed under deep inhalational anaesthesia. Surgical airway should be performed rapidly if the above methods fail.

Tracheotomy

During 1970s, infection such as laryngo-tracheobronchitis and epiglottitis were the common causes for tracheotomy in children. With the popular use of endotracheal intubation, fewer tracheotomies and decannulations were performed. Figures 1 and 2 show the common indications for tracheotomy and the associated decannulation rate. According to Carron et al, the overall decannulation rate is 41%. The overall complication of tracheotomy is 44%, with granuloma formation being the most common.[3] Table 2 shows the common complications associated with tracheostomy. In the same study, the overall mortality rate is 19% (Figure 3), with the vast majority of deaths are due to the child's primary illness. Only 3.6% was known to be directly due to tracheotomy. Most of them have plugging of the tracheotomy tube with resultant respiratory arrests occurring between 0.3 and 30.8 months after the tracheotomy. Misplacement of the tube after operation has been reported.



Primary indications for tracheotomy by grouping. PI=prolonged intubation; NI=neurologically impaired; T=trauma; VFP=vocal fold paralysis; UAO=upper airway obstruction; CF=craniofacial abnormality.

Figure 1. Primary indications for tracheotomy (adapted from reference [3]).

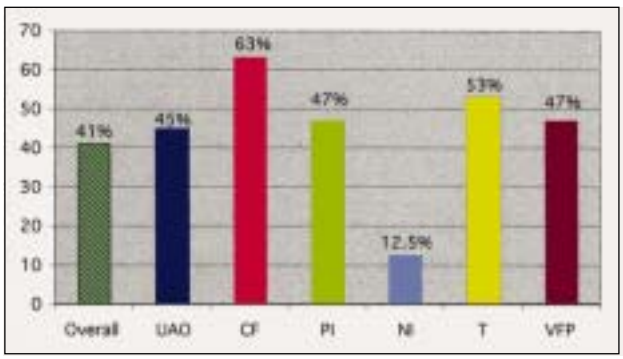


Figure 2. Decannulations rates by group (adapted from reference [3]).

Angioedema

Angioedema is an anatomically limited non-pitting edema that may result in life-threatening airway obstruction.[4] Facial swelling is present in 80% of cases. Other common symptoms include tenderness, dyspnea, dysphagia or hoarseness. Food allergy accounts for about 40% of the causes, and insect bites, infection and drugs are the other common causes. These patients seldom require PICU admission, as the symptoms of these patients usually resolve soon after pharmacological treatment with adrenaline, antihistamine or steroid.

Bacterial tracheitis

Bacterial tracheitis is not a common disease but can be very serious. The clinical presentation is similar to that of severe viral croup, epiglottitis, or foreign-body aspiration. It can range from mild stridor to even

Table 2. Complications of tracheotomy

- Stomal granuloma 20%
- Tracheo-cutaneous fistula 13%
- Tracheal stenosis 2%
- Tube plugging with respiratory arrest 2%
- Accidental decannulation 1.5%
- False passage creation 1%
- Stomal keloid formation 1%

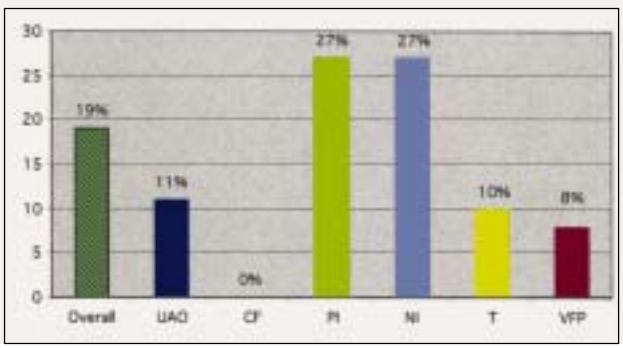


Figure 3. Mortality rates by group (adapted from reference [3]).

cardio-respiratory arrest. The clinical course is generally less acute than that of epiglottitis and also with prodromal symptoms. Patients exhibit high fever, appear more ill, and are less responsive to nebulised epinephrine and other supportive measures than children with viral croup. Drooling and neck hyperextension are uncommon in bacterial tracheitis. Tracheal secretions are copious, thick, and tenacious which persist for 3 to 5 days and even up to 3 weeks. Once these patients are admitted to PICU, 57% of them will be intubated.[5] Intubated patients are usually younger than the non-intubated patients.

The most common pathogens in bacterial tracheitis are *Staphylococcus aureus* and *Haemophilus influenzae*. However, there are reports of bacterial tracheitis caused by *Streptococcus pyogenes*, *Streptococcus pneumoniae*, *Streptococcus viridans*, *Pseudomonas aeruginosa*, *Escherichia coli*, *Moraxella catarrhalis*, non-group A streptococci, and Neisseria spp.

Patients may be extubated safely when their body temperature returns to normal, detection of air leaking around the nasotracheal tube, and when the amount of secretions markedly decreases.

Acute epiglottitis

Paediatric epiglottitis is a serious, potentially life-threatening condition although it is extremely rare in Asia. After Haemophilis influenza (Hib) vaccination in Western countries, the annual incidence of children below 5 years of age decreases from 20.9 per 100,000 in 1987 to 0.9 per 100,000 in 1996.[6] In adults a tendency toward a decrease in incidence is also evidence initially but then rises from a low in 1998 to reach pre-vaccine levels in 2003.[7] Immunisation programme in children may have unexpected effects on the epidemiology of disease.

The presentations of the patients remained static but a decrease in Hib cases (especially the number of ampicillin-resistant organism) are seen. Affected children are, on average, much older after the implementation of universal vaccination programme. Vaccine failures still occur but are rare. Acute epiglottitis should still need to be considered when a child presents with severe upper airway obstruction even after Hib vaccination.

Laryngomalacia

Congenital stridor was first reported in 1853 by two French physicians, Rilliet and Barthez.[8] Sutherland and Lack published the first review of this condition in 1897.[9] In 1942, Jackson first used the term laryngomalacia, coming from the Greek malakia with the meaning of morbid softening of part of an organ. Jackson clearly defined it as softness, flabbiness, or loss of consistency of the laryngeal tissues.[10]

In affected patients, inspiratory stridor is usually present since birth. In some cases, stridor may become apparent few weeks or even months later. Stridor may be exacerbated in some cases by an upper respiratory infection. It is often intermittent and is aggravated when the child is active and crying. On the other hand, symptoms may also be precipitated by supination and head flexion and are relieved by pronation and head extension.

For most of the cases, the symptoms will disappear with time. In 10% of cases, however, upper airway obstruction is so severe that patients develop apnoea or failure to thrive.[11] In these situations, surgery or tracheotomy may be needed.

Croup

Acute respiratory illness caused by inflammation and narrowing of the subglottic region of the larynx is defined as croup.[12] Barking cough, hoarseness, stridor and respiratory distress are the usual presentations. Croup is usually self-limiting and symptoms usually last for 3-7 days. Yet, it still can cause severe upper airway obstructions that result in intubation or even mortality. For those patients with very severe croup, other causes of upper airway obstructions, especially epiglottitis, should be ruled out. If the diagnosis is not certain, patients must be treated empirically as epiglottitis, and intubation should be performed by experienced anaesthetists under deep inhalation induction. Surgical airway should be performed rapidly if the above methods fail. Neck radiograph is usually not necessary for the diagnosis but if taken, it would show the steeple sign (narrowing of the subglottic area) in patients with croup.

The incidence of croup is 1.5-6 per 100 children per year. The admission rate for croup varies from 1.5% to 30%. This wide range of admission rate reflects different criteria for admission. In the United States, croup has been estimated to cause 41,000 hospitalisations annually.[13] The intubation rate is about 0.5-1.5% for those patients admitted to hospital. The re-intubation rate is 5-16%.

Parainfluenza virus type 1 is the main virus causing croup.[14] The parainfluenza virus 1, 2 and 3 can be isolated in all age groups, and altogether account for about 2/3 of all cases. For patients younger than 5 years old, respiratory syncytial virus (RSV) tends to

be isolated more commonly. On the other hand, influenza virus and *Mycoplasma pneumoniae* affect children older than 6 years of age.[15] Moreover, influenza virus tends to cause more serious illness as compared with parainfluenza virus.[16]

Mortality rate for croup is less than 0.5% in intubated patients.[17] According to Sacenkova et al, 92.5% of these cases die of severe pneumonia, 7.5% die of sepsis; 70% of them are 0-2 year old, 75% are boy and nearly all patients had aggravated pre-morbid background.[18] In this study, virus and resistant strains of staphylococci and streptococci (penicillin, ampicillin and cefazoline) could be isolated in the sputum or organ tissues from most of the death cases. The authors concluded that children with croup died of severe pneumonia complications with a low systemic reactivity and high antibacterial resistance.

Treatments of croup include glucocorticoids, nebulised epinephrine, humidification and heliox. Since late 1980s, glucocorticoids have been recognised to provide some clinical benefit for children with croup. Kairys et al published a meta-analysis of clinical trials examining the benefit of glucocorticoids in 1989.[19] The Cochrane Database of Systemic Reviews in 2004 showed that glucocorticoid treatment is effective in improving symptoms of croup in children as early as six hours and for up to at least 12 hours after treatment, and the efficacy was supported by improvement in croup score, decrease in return visit or admission, reduction in length of stay and epinephrine use as an additional intervention.[20] With the introduction of steroid, less croup patients need intubation and intensive care,[21] and the duration of intubation and need for re-intubation also decreased.[22]

Regarding the administration for glucocorticoid, oral route is preferred as this is easily given and the efficacy for oral route is the same as with intramuscular injection. Some children find nebulised therapy distressing. The standard dose for glucocorticoid is dexamethasone at 0.6 mg per kilogram body weight per dose, with a maximum of 10 mg although some authors suggest that lower dose is also effective.

Epinephrine (adrenaline) is a potent agonist of alpha- and beta-adrenergic receptors. Since early 1970s, epinephrine has been used to treat croup. It helps to reduce bronchial and tracheal secretions and oedema. Following nebulised therapy, these effects are noted within 10-30 min and last for about one hour. However, 35% of patients who received epinephrine had a relapse of their symptoms within 2 hours of treatment.[23] Racemic epinephrine, a mixture of equal amounts of dextro(d) and levo(l)-isomer, was traditionally used because this was believed to have fewer side effects and better effectiveness. However, with more published data, racemic epinephrine and levo-epinephrine were found to be the same for their effectiveness and side effect profile.[24] In fact, levo-epinephrine is cheaper and more easily available. Therefore, levo-epinephrine is recommended for treatment of croup instead of racemic epinephrine.

Humidification was a routine therapy in early 1990s. It was believed that this therapy relieved discomfort and prevented the drying of inflamed laryngeal mucosa. However, there is no evidence for these effects. During the therapy, children need to stay inside mist tents. They can become wet and cold. These patients may be quite irritated during isolation. It was also difficult to observe the general conditions of these children in mist tents. With the introduction of steroid therapy, humidification therapy is no longer use as a standard therapy for croup.

Heliox is an experimental treatment. It is a metabolically inert, non-toxic gas that combines helium with oxygen. It has low viscosity and low specific gravity, and these properties allow for greater laminar airflow through the respiratory tract. Heliox is well tolerated, and some studies suggested that it decreased the croup score.

In summary, glucocorticoid reduces croup score, rate and duration of intubation. However, there is no conclusion on the best route and dosage of its administration. Nebulised epinephrine can relieve the symptoms of croup transiently but symptoms will recur following transient improvement. Humidification is no longer a routine treatment for croup. Heliox is still an experimental treatment, and more evidence is needed to support its routine clinical use.

Summary

Severe upper airway obstruction in children requires urgent management. Rapid and effective management can prevent severe complications and even fatality. Tracheotomy is rarely needed. Infections such as croup remain the most common cause of severe upper airway obstruction that requires intensive care management. Acute epiglottitis and bacterial tracheitis are rare, but should be the differential diagnoses in critically ill patients. Patients with angioedema are rarely admitted to PICU, and most of them resolve soon after medical treatment. Laryngomalacia is common but most of them will be resolved without treatment. Steroid is very effective for croup, and much less patients with croup require intubation following the use of steroid treatment.

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Journal of Paediatric Respirology and Critical Care

Exercise therapy in the correction of pectus excavatum

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Introduction

Pectus deformities are common abnormalities of the thorax and affect an estimate of 6 to 8 per 1000 children.[1] The cause of pectus deformities is accepted to be an abnormal overgrowth of cartilage between the ribs and sternum that pushes the sternum inward (pectus excavatum) or outward (pectus carinatum) causing a funnel-chest or pigeon-chest respectively. Pectus excavatum is more commonly encountered and it may impact on the pulmonary and cardiac functions in severe cases. Surgical intervention is an accepted practice of intervention for severe pectus excavatum.[2, 3] Other than surgical intervention, exercise is another treatment alternative.[4] Clinically, a number of patients were observed to benefit from exercise training although the long term effect and its physiological benefits are still unknown.

Therapeutic exercises to improve pectus excavatum deformity

In theory, the main question in managing pectus excavatum is how to pull the sunken anterior chest wall outward and forward. The inspiratory muscles that help in pulling up the chest wall should be considered. This includes the scalenus anterior and medius, and the sternocleidomastoid. The serratus anterior and pectoralis minor also participate during forced inspiration. Intercostal muscles help the elevation of the chest wall only when the first ribs fixed and elevated. All these mentioned muscles, however, cannot directly add their pull of force on the lower sternum and sunken ribs, which are commonly encountered in pectus excavatum. Their effect of pull seems limited and mainly affects the upper chest wall.

However, if the upper limbs can be supported by grasping a chair back or table, the sternal origin of the pectoralis major muscles can also assist the elevation process of the chest wall. Pectoralis major is a thick and powerful triangular muscle. Its fibers are inserted by a bilaminar tendon into the lateral lip of the bicipital groove of the humerus. It originates from the sternal half of the clavicle, the anterior sternal surface to the level of the sixth or seventh costal cartilage, the first to seventh costal cartilages, the sternal end of the sixth rib and the aponeurosis of obliquus extenus abdominus. If the insertion of the arm can be put and fixed in an upward stretched position, all these origins especially the latter four can directly and forcibly pull the sunken sternum and infolding ribs up during the muscle contraction. The concept of “reverse origin and insertion” of this muscle action is applied. The direction of pull from the stretched arm position along the sternocostal fibers of the pectoralis major acts effectively on the depressed chest wall. Pectoralis major situated in its mid range of length enables it to recruit the greatest amount of muscle fibers to exert powerful pulling force. With these in mind, it provides the direction for the design of the following suggested exercise program.

With an increase in strength and muscle tone by training, the chest wall deformity may be diminished or at least maintained. In addition, the increase in intensity of training, especially to the anterior chest wall may help to build up larger muscle bulk and a better cosmetic outlook.

Steps to improve the chest wall appearance

In order to improve the chest wall appearance, the patient need to follow the following steps in sequence in their daily exercise program. They are:

1. To increase the mobility and flexibility of the spine and chest wall.
2. To lengthen any tightened and shortened structures.
3. To strengthen muscles in elevating and expanding the depressed chest wall.
4. To restore normal posture.

The first two steps are to mobilize the articulating joints and to lengthen any tight soft tissue around the chest wall so that less impedance will be encountered during the elevation of depressed chest. Exercises of these two steps can, at the same time, serve as a warm-up. They condition the musculo-skeletal in preparing for the following vigorous exercises. These exercises will be done with intensive training to strengthen inspiratory muscles, which are essential in elevating the depressed chest wall. Training of pectoralis major, especially the sternocostal fibres, will be of utmost importance. With better chest wall mobility and muscle tone after these preparatory steps, the patient will be conditioned to learn the postural correction exercises. It is important that the patient must persist in performing these exercises.

Suggested exercise program

Mobilizing and stretching exercise

1. *Forward arm stretching in pone kneeling*
   The patient is positioned in an inclined prone kneeling position with hands stretching forward and

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supported by wall bar (about 2 to 3 feet high from ground) (Figure 1). Slowly lower his upper body and press his scapula towards the floor. Experience the stretch feeling at the front axilla and shoulder. Hold 8 seconds (may get a deep breathe and hold to increasingly stretch the chest wall) and release. Repeat for 20 times and 4 sessions per day.
Purpose: Stretch all anterior chest wall muscles especially pectoralis major and extend the upper back.

Figure 1. Forward arm stretching in pone kneeling.

2. *Upper trunk rotation in standing*
The patient is to stand obliquely to a wall. The near hand is put on the wall a bit higher than the shoulder level. The patient's pelvis turns to the opposite side while still leaving the hand fixed on the wall (Figure 2). A stretch is felt at the anterior shoulder and upper chest wall. Hold 8 second, then release and return to the original position. Take a rest and repeat on the other side. Repeat for 20 cycles and 4 sessions per day.
Purpose: Rotation gives the greatest range of movement for thoracic vertebrae allowing stretch to ligaments, muscles and joints around the chest wall in a different direction.

Figure 2. Upper trunk rotation in standing.

3. *Upper trunk side flexion in sitting*
The patient is seated on a chair. Side bend to one side with the opposite hand crossing over the head to another side (Figure 3). A stretching feeling is felt on the other side of trunk. Hold 8 seconds (may get a deep breathe and hold to increasingly stretch the chest wall) and then return to the original position. Take a rest and repeat on the other side. Repeat for 20 times and 4 sessions per day.
Purpose: Similar to the 2nd exercise.

Figure 3. Upper trunk side flexion in sitting.

Strengthening exercise

1. *Weight lifting in stretch supine lying*
The patient is positioned in supine with the upper trunk on a small foam roll around 2 to 3 inches in diameter (if patient can't tolerate, just lie flat). The arms are put in an upward stretched position. The hands should hold on a fixed wall bar (Figure 4) or hardly movable weight about 10 inches from the surface of the bed (pillows may be used to support the weight) (Figure 5). Deeply inspire and exert maximal force in lifting the wall bar or weight. Hold

Figure 4. Lifting wall bar in stretch supine lying (with foam roll).

Figure 5. Weight lifting in stretch supine lying (pillow support the weight).

8 seconds and relax. Repeat 10 times as 1 lot. Take rest then and repeat another 2 lots performing a total of 30 repetitions and 4 sessions per day.
Purpose: By the technique of "reverse origin and insertion", the arms are being fixed and the anterior chest wall is lifted up mainly by the pectoralis major and minor. Maximal force exertion allows recruitment of surrounding respiratory muscles for training. The foam roll under the upper to middle part of the trunk exerts postero-anterior force to the thoracic spine helping in extension, which mobilizes and corrects any thoracic kyphosis . The depressed chest will also be "opened" up facilitating the elevation of the chest wall. Arms, being in a mid-length muscle range, are capable to exert the greatest force to elevate the depressed chest. Tone of pectorlis major is built up for better posture and outlook.

2. *Upper trunk extension in prone lying*
The patient is positioned in prone lying with one or two pillows under the tummy (avoiding the lower anterior chest pressing on the pillow) (Figure 6). The hands are placed behind the head. The feet may be fixed on wall bar. Deeply inspire and extend the upper trunk with arms arching back. Stay and hold 8 seconds and then relax. Repeat 10 times as 1 lot. Take rest then and repeat another 2 lots. Perform a total of 30 repetitions and 4 sessions per day.
Purpose: The strengthened upper back muscles help to balance the improved muscle force of the anterior chest wall muscle. This prevents the development of thoracic kyphosis due to strong anterior muscle pull and keeps a good posture.

Figure 6. Upper trunk extension in prone lying.

3. *Push up*
The patient is positioned in prone lying and both hands are used to push up his body (Figure 7). The level of difficulty depends on the actual ability of the patients (1st level – upper trunk pushed up, 2nd level – whole body pushed up in one piece, 3rd level – push and clap both hands in mid air). Start with the 1st level and when the patient is able to finish the level easily, he may proceed to next level). Repeat 10 times as 1 lot. Take rest and then repeat another 2 lots performing a total of 30 repetitions and 4 sessions per day.
Purpose: The exercise aims at general strengthening of the chest wall. Moreover, the high intensity but low frequency impacting force may be advantageous to stimulate remodeling and shaping of the chest wall deformity. Bone mineralization may also be enhanced.

Figure 7. Push up.

4. *Hands up and down movement behind and by the sides of body (with theraband)*
The patient is positioned in sitting or standing with both arms in a stretched position. Each hand holds one end of a theraband or a spring (resistance should be set at 10 repetitive maximum, RM, i.e. the resistance that one can perform 10 repetitions but no more). Then stretch the theraband and maintain the elbows straight (Figure 8). Slowly put the hands behind and pass by the sides of body and then down below buttock. After 3 seconds rest, the hands slowly go up and along the same track to the starting position. Repeat 10 times as 1 lot. Take rest and then repeat another 2 lots performing a total 30 repetitions and 4 sessions per day.

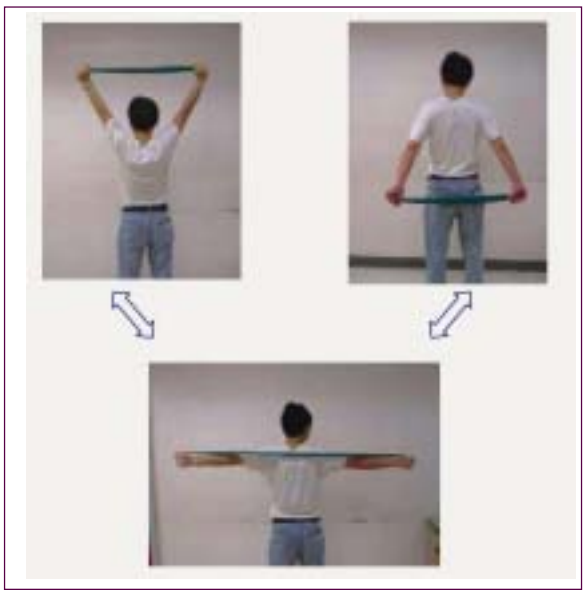

Figure 8. Hands up and down behind and by the sides of body (with theraband).

Purpose: The exercise is used to strengthen the neck, shoulder, upper back and anterior upper chest muscles. It can be treated as a kind of stabilization exercise to the upper thorax.

Postural correction

The above exercises help correct the depressed chest wall deformity and also the thoracic kyphosis. However, as the postural problems of individual patients may have different clinical presentations and causes. It is impossible to have one exercise program to suit all patients for postural correction. It would be best to consult doctors or physiotherapists in postural exercises. On the whole, the exercises should mainly concentrate on realignment of good posture, both static and dynamic. To maintain a sense of good posture in both static and dynamic work is another area to be tackled. Proprioceptive exercise training should also be introduced. In addition, the final postural exercises can act as some cool down activities.

Although some exercises have been suggested, they should preferably be done within the patient's tolerance. The suggested intensity and frequency of treatment should act as a beginning reference. The parameters should be modified whenever the conditions seem necessary. With improvement in exercise performance and effect, patients may increase their treatment frequency and intensity, under the advice from a doctor or a physiotherapist. For young patients, who can't perform these exercises, their parents may help passively to stretch their limbs similar to the described moblising and stretching exercises. For the strengthening exercises, they can try swimming in free-style. The alternate climbing action of both hands is good training for pectoralis major.

Discussion

Performance of the above exercises will see immediate elevation of chest wall. The long term effect of the exercises is unknown. However, some reviews throw light of hope that throughout life, the skeleton is continuously changing to adapt its form and structure to suit their functional needs.[5] Bone growth and maintenance is always a process between osteoblastic and osteoclastic activities. If the former activity is greater than the latter, bone grows. Otherwise bone will be resorbed and the mineral will be redistributed to build up bone against load stress in other areas.[6] This is reflected by the Wolff's law, a principle assuming that mechanical stresses influence the remodeling process of bone and subsequently the structure and strength of bone.[7] All cells participating in the remodeling process have been termed the bone multicellular unit and are thought to proceed through activation, resorption, and formation, during which a quantum of bone is exchanged. The alteration in bone shape was evidenced in a study using immature Holstein bull calves as a model, short-duration but high-intensity exercise to stimulate bone formation and altering bone shape was observed in comparison with the stalled and group-housed calves.[8] Dynamic loads trigger the adaptive response in bone.[9] Whether the shape and deformity of pectus excavatum be improved with the above exercises require further study.

Most studies show that mineralisation of bone can be developed through impacting exercises[10] to increase the bone mass density of the weight bearing bone. There is also evidence that bone developmental changes in bone strength might also be secondary to the increasing loads imposed by larger muscle forces. The results of this study are compatible with the view that bone development might be driven by muscle development.[11] This gives an implication that high intensity loading during exercise design may help to increase mineralization of the bone to maintain a reformed shape.

From the skeletal point of view, bone shaping should be done early before it is mature. Since the ossification of the chest wall begins in utero and continues to approximately the 25th year, and even earlier for the rib as ossification is completed by age 20.[12] Therefore the best training period should be started earlier than this age and childhood is probably the ideal period.

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Childhood asthma treatment guildeline 2005

Hong Kong Society of Paediatric Respirology

Childhood asthma: diagnosis

Asthma is usually diagnosed on the basis of the history of symptoms along with physical examination. In selected cases, measurement of lung function, reversibility of airway obstruction may enhance diagnostic accuracy.

The following are important diagnostic hints of asthma:

History

1. Coughing is the commonest symptom of asthma especially in young children.
2. Wheezing: a common symptom of asthma but the absence of wheezing attacks does not exclude the diagnosis.
3. Breathing difficulty.
4. The chance of asthma increases if the above symptoms occur:
   - recurrently or persistently
   - during or after a cold
   - on exercise
   - when in contact with animals
   - when in contact with pollen or some plants
   - interrupting sleep at night
   - when the weather changes
   - when in contact with some known allergens
   - when in contact with smoke or other irritants
5. A family history of atopy and allergic diseases.
6. Past history of atopic diseases.

Physical examination

1. Signs of airway obstruction:
   - chest hyperinflation
   - obstructed expiration: spontaneous wheeze or forced expiratory wheeze
   - diminished air movement in the chest
   - physical examination may be normal in children with mild asthma
2. Signs of other allergies or complication:
   - eczema, allergic rhinitis, growth failure, sinusitis, chest deformity

Investigations

1. In older children (6 or above) who are able to perform simple lung function testing (peak flow rate or spirometry), demonstrate variable airflow obstruction with diary cards and peak flow monitoring, or use bronchodilator inhalation to demonstrate improvement in spirometry.
2. In younger children, demonstrate immediate improvement of signs with bronchodilator inhalation, or a therapeutic trial of asthma medications to look for improvement over time.
3. Other supportive diagnostic modalities: Exhaled Nitric oxide measurement, skin-prick test, infant lung function measurement if available.

Special note

1. Often a period of observation is required to ascertain the diagnosis.
2. Asthma is a chronic disease and patients have to be reassessed on a regular basis to adjust the prescribed treatment.

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Treatment Guideline for Managing Asthma in Children Older Than 5 years of Age: Treatment

Classify Severity: Clinical Features Before Treatment or Adequate Control			Recommended Medications Required To Maintain Long-Term Asthma Control
	Symptoms/Day Symptoms/Nights	PEF or FEV1 PEF Variability	Daily Medications
Step 4 **Severe Persistent**	Continual Frequent	≤60% >30%	• Preferred treatment: - High-dose inhaled corticosteroids (>800 mcg of budesonide or equivalent) AND - Long-acting inhaled beta$_2$-agonists AND if needed, - Corticosteroid tablets: a short course of steroid may be needed to suppress airway inflammation • Alternative treatment: - High dose inhaled corticosteroids AND either leukotriene modifier or theophylline
Step 3 **Moderate Persistent**	Daily >1 night/week	>60% - <80% >30%	• Preferred treatment: - Low to medium dose inhaled corticosteroids (200-800 mcg of budesonide or equivalent) and long- acting inhaled beta$_2$-agnoists. • Alternative treatment: - Increase inhaled corticosteroids within medium-dose range OR - Low-to-medium dose inhaled corticosteroids and either leukotriene modifier or theophylline
Step 2 **Mild Persistent**	>2/week but <1x/day > 2 nights/month	≥80% 20-30%	• Preferred treatment: - Low-dose inhaled corticosteroids (<400 mcg of budesonide or equivalent). • Alternative treatment options: cromolyn, leukotriene modifier, nedocromil, OR sustained-release theophylline to keep serum concentration of 5-15 mcg/mL.
Step 1 **Mild Intermittent**	≤2 days/week ≤2 nights/month	≥80% <20%	• No daily medication needed. • Inhaled (preferred) or oral bronchodilators may be prescribed and use as needed

Quick Relief **All Patients**	• Short-acting bronchodilator: 2-4 puffs short-acting inhaled beta$_2$-agonists as needed for symptoms. • Intensity of treatment will depend on severity of exacerbation; up to 3 treatments at 20-minute intervals treatment as needed. Course of systemic corticosteroids may be needed. MDI with spacer is just as effective as the nebulizer in most situations of mild to moderate attacks. • **Use of short-acting beta$_2$-agonists >2 times a week in intermittent asthma (daily, or increasing use in persistent asthma) may indicate the need to start long-term-control therapy.**

↓	**Step down** Review treatment every 1 to 6 months; a gradual stepwise reduction in treatment may be possible.
↑	**Step up** If control is not maintained, consider step up. First, review patient medication technique, adherence, and environmental control.

Goals of Therapy: Optimal Asthma Control

- Minimal or no chronic symptoms day or night
- Minimal or no exacerbations
- No limitations on activities; no school/work missed
- Maintain (near) normal pulmonary function
- Minimal use of short-acting inhaled beta$_2$-agonist
- Minimal or no adverse effects from medications

Treatment Guideline for Managing Infants and Young Children (5 years of Age and Younger) with Asthma

Classify Severity: Clinical Features Before Treatment or Adequate Control		Medications Required To Maintain Long-Term Control
	Symptoms/Day **Symptoms/Nights**	**Daily Medications**
Step 4 **Severe Persistent**	Continual Frequent	• Preferred treatment: - High-dose inhaled corticosteroids (>800 mcg of budesonide or equivalent) AND - Long-acting inhaled beta$_2$-agonists AND if needed, - Corticosteroid tablets: a short course of steroid may be needed to suppress airway inflammation (2 mg/kg/day)
Step 3 **Moderate Persistent**	Daily >1 night/week	• Preferred treatment: - Low-dose inhaled corticosteroids (<400 mcg of budesonide or equivalent) and long-acting inhaled beta$_2$-agnoists OR - Medium-dose inhaled (400-800 mcg of budesonide or equivalent) corticosteroids. • Alternative treatment: - Low-dose inhaled corticosteroids and either leukotriene receptor antagonist or theophylline.
Step 2 **Mild Persistent**	>2/week but <1x/day >2 nights/month	• Preferred treatment: - Low-dose inhaled corticosteroids (with MDI with holding chamber with or without face mask or nebulizer). • Alternative treatment (listed alphabetically): - Cromolyn (MDI with holding chamber or nebulizer) OR oral leukotriene receptor antagonist.
Step 1 **Mild Intermittent**	≤2 days/week ≤2 nights/month	• No daily medication needed. • Inhaled (preferred) or oral bronchodilators may be prescribed and use as needed.

Quick Relief **All Patients**	• Bronchodilator as needed for symptoms. Intensity of treatment will depend upon severity of exacerbation. - Preferred treatment: Short-acting inhaled beta$_2$-agonists by face mask and space/holding chamber or nebulizer - Alternative treatment: Oral beta$_2$-agonist • With viral respiratory infection - Bronchodilator q 4-6 hours up to 24 hours - Consider systemic corticosteroid if exacerbation is severe or patient has history of previous severe exacerbations • **Use of short-acting beta$_2$-agonists >2 times a week in intermittent asthma (daily, or increasing use inpersistent asthma) may indicate the need to start long-term-control therapy.**

↓	**Step down** Review treatment every 1 to 6 months; a gradual stepwise reduction in treatment may be possible
↑	**Step up** If control is not maintained, consider step up. First, review patient medication technique, adherence, and environmental control.

Goals of Therapy: Optimal Asthma Control

- Minimal or no chronic symptoms day or night
- Minimal or no exacerbations
- No limitations on activities; no school/parent's work missed
- Minimal use of short-acting inhaled beta$_2$-agonist
- Minimal or no adverse effects from medications

Steroid equivalent table:

Estimated Comparative Daily Dosages for Inhaled Glucocorticosteroids						
Drug	**Low Daily Dose (µg)**		**Medium Daily Dose (µg)**		**High Daily Dose (µg)**	
	Adult	**Child**	**Adult**	**Child**	**Adult**	**Child**
Beclomethasone	200-500	100-250	500-1000	250-500	>1000	>500
Budesonide-DPI	200-600	100-200	600-1000	200-600	>1000	>600
Budesonide-Neb Inhalation suspension		250-500		500-1000		>1000
Fluticasone	100-250	100-200	250-500	200-400	>500	>400
Mometasone furoate	200-400		400-800		>800	

Notes:

The most important determinant of appropriate dosing is the clinician's judgment of the patient's response to therapy. The clinician must monitor the patient's response in terms of a combination of clinical parameters and adjust the dose accordingly. The stepwise approach to therapy emphasizes that once control of asthma is achieved, the dose of medication should be carefully titrated to the minimum dose required to maintain control, thus reducing the potential for adverse effects. Actual delivered dose may vary according to the device used for delivery of inhaled steroids.

Reliability of scoring arousals in normal children and children with obstructive sleep apnea syndrome
Wong TK, Galster P, Lau TS, Lutz JM, Marcus CL
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Assessment of the Pediatric Index of Mortality (PIM) and the Pediatric Risk of Mortality (PRISM) III score for prediction of mortality in a paediatric intensive care unit in Hong Kong
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Time-table for HKSPR Clinical Meetings 2005-6 (Revised as of June 2005)

Date	Hospital(s) / Organization(s)	Title(s) of Presentation	Speaker(s)	Chairperson
29 Jun 2005	TMH	A New Treatment for a Newborn with Respiratory Distress	Dr. Siu Luen Yee	Dr. TY Miu
27 Jul 2005	AHNH + NDH	Cases presentation in Asthma	Dr Chow Siu Ngai	Dr. Grace Chan
31 Aug 2005	CMC + OLMH	A child with tracheal stenosis presented as recurrent wheezing	Dr Lam Ping	Dr. WK Chiu
28 Sep 2005	KWH	Chronic Cough in Children	Dr. Eric Chan Dr. PY Chow	Dr. SY Lam

World Asthma Day, 3 May 2005

World Asthma Day (WAD) is a yearly event initiated by Global Initiative for Asthma (GINA) to arouse asthma awareness and care throughout the world. In the years before, WAD activities in Hong Kong was mainly hosted by the Hong Kong Asthma Society (HKAS). This year, your Society has joined hands with Hong Kong Medical Association (HKMA) and HKAS to host a press conference, announcing some joint activities against asthma. The Childhood Asthma Guideline for Hong Kong, produced by HKSPR, will be the theme of a series of lecture to front line doctors in various districts in Hong Kong in the coming year. While HKMA will organize the luncheon lectures, HKSPR will provide the speakers and contents. The Guideline will therefore have a chance to reach doctors working in the frontline, hopefully helping them to deal with childhood wheezing and asthma effectively. Such lectures will also provide a chance for feedback from the doctors so that the Guideline can be made more useful and appropriate for Hong Kong in the future. The first of these lectures were given on WAD to over 250 front line doctors by Dr. Alfred Tam and Prof. Gary Wong at Langham Hotel.

Hong Kong Asthma Society will be hosting a series of lectures, and training workshops for parents and teachers this year, focused on asthma and allergies. These will be very good training for the lay public. HKSPR Medical Advisors will be invited to be speakers of these events.