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Editorial

The Hong Kong Thoracic Society and the American College of Chest Physicians (Hong Kong and Macau chapter) successfully held an Autumn Respiratory Seminar in November 2007. This one-day programme is comprehensively reported in this issue and we have also included the highlights of clinical summaries presented in the “Interactive Grand Round” session of the Seminar.

The Hong Kong Lung Foundation Fellowship awardees have contributed in “Practical Corner” section in this issue and their articles are very useful and interesting. The “Special Event” section reported our members’ participation in the recently held “Sleep Disorder Symposium” in Hong Kong and “Asian Pacific Society of Respirology Conference 2007” in Australia.

Last but not the least, may I remind you that the Annual Scientific Meeting of HKTS/ACCP will be held at the end of March 2008. This is going to be an excellent opportunity for us to meet one another and update our knowledge in Respiratory and Critical Care Medicine. Further details will be sent to you in due course.

Instruction to Contributors
We welcome contributions from invited guests and members of the Hong Kong Thoracic Society and the American College of Chest Physicians (Hong Kong and Macau Chapter). Articles should be prepared with suitable word processing software (eg Word 2000®). Figures, table, pictures and photo-micrographs should be saved in the same file. Please do not use the auto-indexing features. The file could be sent either by e-mail or by post (on a floppy disc or CD) to the Chief Editor. Please indicate to the Chief Editor if the material has to be returned after the editing process. The article would be printed in the same way as it is submitted. The accuracy of the materials published is the responsibility of the contributors. The contributors must ensure that the materials submitted do not infringe copyright.

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The opinions expressed in this newsletter are those of the author/s and do not necessarily reflect the official policies of the Hong Kong Thoracic Society, American College of Chest Physicians (Hong Kong and Macau Chapter), the institution with which the author(s) is/are affiliated, or the publisher.
The Hong Kong Thoracic Society and the American College of Chest Physicians (Hong Kong and Macau Chapter) jointly organized the Autumn Respiratory Seminar (ARS), which was successfully held in November this year at the Hong Kong Convention and Exhibition Centre.

More than 400 participants attended this one-day meeting on the 25th November. A wide scope of interesting and practical topics was delivered by a distinguished panel of local and overseas speakers.

The ARS began with a symposium of “Interstitial Lung Diseases”. It was followed by a concurrent symposium consisting of one section of “Chronic Obstructive Pulmonary Disease” together with another one on “Ethical Issues & Palliative Care in Respiratory Medicine”, and both were well attended by the audience.

Following a lunch symposium on “Advances in Pharmacological Treatment of COPD” given by a well renowned expert from UK, Professor Wisia Wedzicha, we had an afternoon session of “Interactive Grand Round”. A total of four interesting cases were presented, one of which was selected from the various well presented summaries of previous Society’s clinical meetings. The audience was very enthusiastic throughout the entire section and has learned a lot of useful knowledge from the chairmen, Professor Lam Wah-kit, Dr So Shun-yang, Dr Christopher Lai and Dr Chan Hok-sum, during the case discussion led by them.

The ASM ended with a symposium on a very popular topic recently: “Interventional Pulmonology”, including “Update on Endobronchial Ultrasound” and “The Clinical Role of Medical Thoracoscopy/ Pleuroscopy”.

Dr K Tsang in the “Opening Remarks”  
The audience attending the symposium
From left to right: Prof G Raghu and Dr C Yeung on the stage, and Dr K Tsang presented the souvenir to the guest speaker Dr M Wong

From left to right: Prof G Raghu, Dr C Yeung, Dr M Wong and Prof D Hui in the “Q&A” section of “Interstitial Lung Diseases” symposium

Left: Prof JA Wedzicha and Dr F Ko in the symposium of “COPD”
Right: Drs KS Lau, D Tse and KS Chan in the symposium of “Ethical Issues & Palliative Care in Respiratory Medicine”
(From left to right): Dr Calvin Yeung (radiologist) and the four chairmen Dr SY So, Prof WK Lam, Dr C Lai and Dr HS Chan leading the discussion in the section of “Interactive Grand Round”

Four presenters of the “Interactive Grand Round”: Dr H Cheung (upper left), Dr G Law (upper right), Dr J Ngan (lower left) and Dr SW Yan (lower right)
Mr Wong was admitted for his fourth episode of blood stained sputum in November 2006. He was an 84-year-old ex-smoker and ex-drinker. He enjoyed good functional status. He had known history of chronic obstructive pulmonary disease, hypertension with mild renal impairment, ischaemic heart disease with heart failure and Parkinson’s disease. His previous three episodes of haemoptysis were in March, June and August of 2006. They were associated with worsening of cough, increase sputum production, mild shortness of breathe. He did not have fever, chest pain or constitutional symptom. There was also no relevant travel, contact or clustering history. Physical examination revealed inspiratory crepitation over his right lower chest. Right lower lobe consolidation was present in CXRs in all the four episodes. A course of Amoxicillin + Clavulanic acid (Augmentin) and Transaminic acid were commended in all the episodes and his symptoms did subside for several weeks or even months. An interval CXR in April also showed the right lower lobe consolidation was resolved partially (Figure 1a & b). However, the subsequent CXRs revealed persistent consolidation over the right lower lobe (Figure 1c, d & e). His baseline blood tests including complete blood picture, liver and renal function test were all unremarkable. His sputum was negative for bacterial culture, acid fast bacillus and cytology all along.

Figure 1a (on the left): CXR in March 2006
Figure 1b (on the right): CXR in April 2006 showing partial resolution of the right lower lobar consolidation
Further enquiry revealed he had choking occasionally. Oral examination also revealed poor oral hygiene with the presence of multiple dental caries.

Further work-up included a contrast CT thorax, which showed right lower lobe consolidation and a calcified lesion in his right lower bronchus (Figure 2).

We proceeded to fibreoptic bronchoscopy. It showed an obstructing mass in the bronchus intermedius, with a necrotic-like surface and surrounding inflammed mucosa (Figure 3). Culture of bronchial aspirate yielded several bacteria, including Enterbacter/Serratia species, Coagulase negative Staphylococcus, Alpha-haemolytic Streptococci, Neisseria species and Prevotella species. Cytology, acid fast bacillus and fungal culture were negative. Histology of the biopsy from the mass in bronchus intermedius found acute inflammatory changes and several sulphur granules (figure 4a, b & c). Overall, the clinical picture and histological finding were compatible with endobronchial actinomycosis. The calcified lesion in bronchus intermedius may be a foreign body or a broncholith. Both are known to be associated with endobronchial actinomycosis.
He was treated with 4 weeks of IV Penicillin G, followed by oral Amoxicillin. Some advice was given by the speech therapist to decrease the risk of aspiration. Besides that, root extraction was arranged by the dental surgeon. The mass in bronchus intermedius was found much reduced in size during a reassessment FOB 3 months later.

Actinomycosis is a chronic infection caused by Actinomyces spp. (not fungus), which is an anaerobic-to-microaerophilic, gram positive filamentous rod. Associated or risk factors of endobronchial actinomycosis includes age of 30-50 year old, male (M:F = 2-4:1), underlying respiratory disorders (such as COPD and bronchiectasis), alcoholics, poor oral hygiene, dental & facial disease and aspiration. Actually most of these were present in Mr Wong.

Diagnosis can be confirmed by Gram stain of pus, culture of pus or biopsy specimens or demonstrating sulphur granules in pus or biopsy specimens. Sulphur granule is the pathological hallmark of actinomycosis. Macroscopically, they are ~0.1-1 mm yellowish particles. Microscopically, they are actually conglomerates of filamentous actinomycete microcolonies surrounded by tissue reaction material.

Treatment usually consists of prolonged course of Penicillin for 6-12 months.

The second case presented by Dr Grace Law of Kwong Wah Hospital is one of the previously presented cases in the Clinical Meeting of Hong Kong Thoracic Society. For details, please refer to HKTS Newsletter: Sep/Oct 07, Volume 17, Number 3, Page 4-10.
Case History

A 47-year old man, with past history of left pneumothorax over 20 years ago, suffered from ankylosing spondylitis for 10 years. Infliximab 800mg every fortnight was started since August, 2005 for treatment of ankylosing spondylitis. His baseline Mantoux test was negative but the chest radiograph, which had been taken before commencement of infliximab, showed a small opacity over the left upper lobe peripheral region (fig 1).

He developed haemoptysis 3 days after receiving the third dose of infliximab at the end of September 2005. There was no systemic symptom. Chest radiograph showed new consolidation at the right upper lobe and static size of the left upper zone opacity (fig 2). Blood tests revealed normal white cell count and raised ESR. Sputum smears for acid fast bacilli (AFB) were negative for three consecutive days and bacteria culture was negative. Infliximab was stopped. Flexible bronchoscopy was performed and bronchoalveolar lavage was negative for AFB smear and bacterial culture. A course of augmentin was given with no clinical or radiological improvement. Fine needle aspiration of left upper lobe consolidation revealed granulomatous inflammation but Ziehl-Neelsen stain and Grocott stain were negative for acid fast bacilli and fungus respectively. Empirical anti-tuberculosis treatment including rifampicin, isoniazid, ethambutol and pyrazinamide were started in early December, 2005.
Chest radiograph taken after 2 months of treatment showed resolved right upper lobe consolidation, fibrotic change of right upper lobe and the left upper lobe nodule was static in size (fig 3). However, sputum for fungal culture grew scedosporium apiospermum (fig 4,5). Flexible bronchoscopy was repeated and AFB smear and fungal culture were negative in bronchoalveolar lavage. Computed-tomography (CT) guided fine needle aspiration showed degenerated hyphae only. Patient still experienced haemoptysis intermittently but there was no sign of other organ involvement. Voriconazole was not started in view of localized disease and potential drug interaction with rifampicin.

Two months later, chest radiograph showed a large cavity at the left upper lobe with multiple soft tissue shadows inside the cavity (fig 6) whereas CT thorax showed evidence of mycetoma formation at the left upper lobe (fig 7). In view of persistent haemoptysis and radiological deterioration, voriconazole 200mg daily was started in June, 2006 and the anti-tuberculosis treatment was changed from isoniazid and rifampicin as maintenance therapy to isoniazid, ethambutol, pyrazinamide for 3 more months. He was referred for surgical treatment of symptomatic mycetoma.
Fig 6 (left): CXR showing multiple soft tissue shadows in left upper lobe cavity  
Fig 7 (right): CT scan showing left upper lobe mycetoma

Left upper lobe lobectomy was performed in June, 2006 but it was complicated by aortic perforation, which was repaired successfully. Histology of the resected tissue (fig 8) showed caseous granulomatous inflammation, destroyed bronchial wall with inflammatory cell infiltration, and fungal mycelium was seen. Culture of tissue grew scedosporium apiospermum. Haemoptysis subsided after the surgery and voriconazole was given for 8 weeks.

Eight months later, patient was readmitted because of dyspnoea, purulent sputum and fever for three days. Chest radiograph showed left hydropneumothorax with soft tissue shadow at the left apical region (fig 9). Patient remained in respiratory failure despite chest drain insertion and required invasive mechanical ventilation. He developed septic shock and succumbed 2 days after admission. Pleural fluid microscopy showed large number of white blood cells and gram negative bacilli, with few gram positive cocci. Culture showed moderate growth of Scedosporium apiospermum.

Fig 8 (left): Resected fungal ball  
Fig 9 (right): left hydropneumothorax with soft tissue shadow in left upper lobe
Discussion

Scedosporium infection is increasingly recognized as a cause of infection in severely ill or immunocompromised patients. Scedosporium apiospermum and Scedosporium prolificans are the two major human pathogens. Scedosporium apiospermum is the asexual form of Pseudallescheria boydii. It can be found in soil, sewage and polluted water. Diagnosis of an invasive mould infection may be made when septate, hyaline-branching hyphae at a 45 degree angle with a single terminal conidia, is identified in a bed of inflammation (fig 5). Aspergillus or fusarium infection also have similar microscopy appearance and a definitive diagnosis can be made by culture. It cause a wide range of pulmonary manifestations, from simple colonization to mycetoma formation and invasive disease, very similar to that caused by Aspergillus spp. Coinfection with tuberculosis and scedosporium has been reported. There was no microbiological evidence of tuberculosis infection in this case but the radiological improvement after anti-tuberculosis treatment suggested underlying infliximab related pulmonary tuberculosis coinfection. Treatment option and duration for scedosporium apiospermum have not been established. It is resistant to commonly used antifungal such as amphotericin B and fluconazole whereas voriconazole has the greatest efficacy against the organism. In our case, there was concern that concurrent administration of rifampicin would decrease the serum level of voriconazole. Treatment failure is common in Scedosporium infection and surgical debridement is encouraged as it is associated with better outcome.

Infliximab is well known to increase the risk of various infections including tuberculosis and fungal infection. This can be explained by the central role of TNF-alpha in development of protective cell-mediated immunity against fungi. Different fungal infections including histoplasmosis, coccidiomycosis, aspergillosis, candidiasis and cryptococcosis have been reported to be associated with infliximab. To the best of our knowledge, this is the first case report of pulmonary scedosporium as a complication in a patient receiving infliximab.

References

Case History and Physical Examination

A young man aged thirty-seven presented himself fifteen years ago with cough since his childhood. He reported frequent bronchitic symptoms with expectoration of whitish sputum. In one of the episodes, he noticed some blood tinge.

On physical examination he had normal body built. There was no clubbing of fingers. Chest examination was unremarkable alongside with other systems. Chest x-ray showed a retrocardiac shadow (Figure 1).

Investigations

Blood tests showed normal haemoglobin. There was no systemic leucocytosis and the differential count showed normal level of eosinophil. Biochemistry for liver and renal function tests was unremarkable. Multiple sputum specimens showed negative findings for cytology examination and no acid fast bacilli were identified. CT thorax revealed a shadow at posterior basal segment of left lower lobe (figure 2).

Further investigation with fibreoptic bronchoscopy showed no endobronchial lesion. Transbronchial biopsy at left lower lobe was unrevealing.

More advanced imaging study with 3–dimensional Magnetic Resonance Angiography (MRA) demonstrated the arterial supply from the descending thoracic aorta to the left lower lobe (figure 3)
Figure 3. MRI with angiography

**Diagnosis:** Sequestration of Lung

**Literature Review**
Please refer to Question 3 of “Practical Corner” section on page 26
To update our knowledge of sleep related breathing disorders and non-invasive ventilation, a symposium was organized by the Hong Kong Thoracic Society and American College of Chest Physicians (Hong Kong & Macau Chapter).

The symposium held on 13th October 2007 at Holiday Inn Golden Mile Hotel was attended by more than 100 participants. The first two talks were elegantly delivered by our two local experts namely Dr Chu Chung-ming on “NIPPV in COPD: From Hospital to Home” and Professor David Hui on “Update on the Cardiovascular Complications and Treatment of OSAS”. It was then followed by the talk from a world renowned expert from Australia, Professor Carmel Harrington. The audience enjoyed the symposium which was ended with a “Q & A” section between the three experts and the audience.

Left: Prof. Carmel Harrington (speaker) and Prof. David Hui (chairman) on the stage
Right: Dr Chan Wai Ming (chairman), Dr Chu Chung Ming (speaker) and Prof. David Hui (speaker) on the stage

Dr Chan Wai Ming presented souvenirs to the guest speakers (from left to right) : Dr Chu Chung Mai, Prof. David Hui & Prof. Carmel Harrington
Asian Pacific Society of Respirology (APSR) held her annual conference in November this year in a beautiful city of the region – Gold Coast of Australia. During the five-day conference, experts from different parts of the world have enlightened and updated us a wide range of interesting and practical topics in Respiratory and Critical Care Medicine.

Many colleagues from Hong Kong also actively participated in the conference. Prof David Hui was both the chairperson and speaker of the Educational Symposium of the APSR. Dr Christopher Lai and Dr Yew Wing-wai delivered talks in the symposia of their expertise, “Asthma & Allergy in the Asia Pacific” and “Multidrug Resistant TB and Extensively Drug-resistant TB: Current Perspectives” respectively. And many of us participated in various Oral Abstract or Poster Sessions.
The Federation of Medical Societies of Hong Kong (HKFMS) has kindly invited the council of Hong Kong Thoracic Society and the American College of Chest Physicians (HK & Macau Chapter) for the first time to join her Annual Dinner 2007.

It has been her tradition in recent years to have her Annual Dinner as an occasion for friends, colleagues and families to get together and welcome the first day of the year. This year the dinner was again held in the joyful day of the year, the New Year Eve, at the Hong Kong Academy of Medicine Jockey Club Building. All the participants enjoyed very much the various programmes, entertainment and cuisine organized by the Federation, and found it precious and enjoyable to put aside all the busy work and duty, and spent an evening with friends and colleagues of different specialties and professions.

Council Members of the Society at the Reception of the Annual Dinner of HKFMS
This section serves to bombard the trainees with questions covering the basic concepts in respiratory medicine. Questions asked will be discussed in the next issue. Specialists and trainers are invited to give their brief discussions. In no way is it meant to be exhaustive or comprehensive, it only serves to highlight important concepts. Specialists are welcome to offer questions (and the discussions) for this section. Trainees are also welcome to give comments, particularly when there is query on what had been published. Please send them to the editor email: soky@ha.org.hk

Questions for the next issue:

1. Percutaneous Tracheostomy
   (from Dr. Thomas JS Lee, Tuen Mun Hospital)

2. "How is the management of lung cancer influenced by the recent advances in bronchoscopy?"
   (from Dr. Chen Wai, Kwong Wah Hospital)

3. The use of conventional transbronchial needle aspiration
   (from Dr Yeung Yiu Cheong, Princess Margaret Hospital)
Review on Endobronchial Ultrasound?

Dr Joan PC Fok
Prince of Wales Hospital/New Territories East Cluster

Introduction

Flexible bronchoscopy was a common procedure performed in the field of respiratory medicine and thoracic surgery in developed countries. In the investigation of lung lesions, it was done as a standard procedure to look for any endobronchial abnormality, to obtain endobronchial and transbronchial biopsy for histological diagnosis. The yield of bronchoscopy was related to size, location and the margin of the lung lesion. For small peripheral lung nodules, they were usually bronchoscopically occult. For lesions < 20 mm in diameter, the diagnostic yield of bronchoscopy under radiographic fluoroscopic guidance was only 11 to 42%.

With the advancement of technology, intervention pulmonology had evolved and broadened the scope of respirology. Intervention Pulmonology was defined as “the art and science of medicine as related to the performance of diagnostic and invasive therapeutic procedures that require additional training and expertise beyond that required in a standard pulmonary medicine training programme.” It included procedures such as endobronchial ultrasound (EBUS), rigid bronchoscopy, endoscopic stenting, transbronchial needle aspiration, imaging-guided thoracic interventions and many others. Their availability depended on resource and expertise.

Endobronchial ultrasound

The use of EBUS was first described in 1990. Reflected echoes were generated when ultrasound waves were transmitted onto various anatomic structures. Electrical signals were transformed from the echoes. It was used for visualizing the tracheobronchial wall and the immediate surrounding structures.

There were two different types of EBUS:
1. endobronchial ultrasound miniprobes (EBUS-MP)
2. endobronchial ultrasound-transbronchial needle aspiration (EBUS-TBNA)

This review would focus on bronchoscopy with EBUS-MP with guide-sheath (GS) for lung nodule biopsy which I had experienced during my 3-month overseas training in Hokkaido University School of Medicine in Sapporo, Japan.

Diagnosing pulmonary lesions

In that centre, the main indication of EBUS-MP was for obtaining histological diagnosis of peripheral intrapulmonary lesions. All patients had computed tomographs (CT) and white-light screening bronchoscopy done as routine investigations. Endoscopically occult lesions would be candidates for using EBUS-MP GS biopsy.

Patient preparation was the same as in flexible bronchoscopy. The EBUS probe was inserted into a GS. The GS-covered probe was inserted to the working
channel of the bronchoscope. For better localisation of the lesion, X-ray fluoroscopy was used so as to place the probe to the closest proximity to the centre of the lesion. EBUS image was produced and the lesion was searched, with repeated attempts, sometimes with the aid of a curette, especially for small peripheral nodules which were not easy to identify. Pulmonary masses had a hypoechoic texture when compared with the surrounding tissue, and had sharply defined borders due to the strong reflective interface produced between the aerated lung and the lesions. After localisation of the nodule, the EBUS probe was removed. The GS was left in-situ. Biopsy forceps and bronchial brush were introduced via the GS to obtain tissue biopsy of the lesion.

As reported by Kikuchi, with the use of EBUS with GS, brushing and transbronchial biopsy, the diagnostic sensitivity in peripheral pulmonary nodules (PPN) of diameter \( \leq 30 \text{ mm} \) and \( \leq 20 \text{ mm} \) were 58.3% and 53.3% respectively. In a prospective trial by Herth with 54 subjects of fluoroscopically invisible solitary pulmonary nodules with mean diameter 22 mm, EBUS-guided transbronchial lung biopsy reached the lesion in 89% of the cases and biopsy established the diagnosis in 70%.11

EBUS GS without fluoroscopic guidance was found to be effective in diagnosing peripheral pulmonary lesions (PPL), especially when they are solid (diagnostic yield 67%), > 20 mm in diameter (diagnostic yield 75.6%), the bronchus leading to the PPL was identified on CT (diagnostic yield 79.2%), located in the middle lobe and lingula. But for PPN \( \leq 20 \text{ mm} \) in diameter, the diagnostic yield was 29.7%.

The additional use of virtual bronchoscopic navigation had been reported to increase the yield and shortened the time for EBUS GS transbronchial biopsy. Diagnostic sensitivities were 44% for lesions < 20 mm in diameter and 91.7% for lesions 20-30 mm in diameter.

Other potential indications of EBUS-MP were as follows:

**Assessing the depth of tumour invasion and bronchial wall.**

**Suggesting the histology of the lesion**
Ultrasound pattern might be used to distinguish between benign and malignant lesions. Homogeneity suggested benign lesions whereas hyperechoic dots, linear arcs and heterogeneous pattern suggested malignancy.

**Lymph node staging**
Lymph node could be detected by EBUS down to a size of 2-3 mm. It had been found that the yield of EBUS-guided TBNA was higher than conventional TBNA (85% vs. 66%). It also spared the risk of invasive procedures such as mediastinoscopy.

The contra-indications and complications of EBUS were that for flexible bronchoscopy. Bleeding was minimal due to protection from wedging by the GS. Pneumothorax had been reported from \(<1\%\) to 4.2 %.

To conclude, EBUS was a safe procedure and was useful in the diagnosis of lung lesions. Skill and patience of the bronchoscopist were essential, especially in localising peripheral small nodules, which might need multiple attempts. ATS stated that EBUS should be reserved for experienced bronchoscopists and it had a long learning curve. I hoped that with advancement in both technology and clinical expertise in the field, EBUS could be more widely practiced and more patients would benefit.
References:

Practical Corner

Influences on Upper Airway Patency in Obstructive Sleep Apnea Hypopnea Syndrome

Dr Jamie CM Lam
Division of Respiratory and Critical Care Medicine, University Department of Medicine, Queen Mary Hospital

Introduction

Obstructive sleep apnea (OSA) is the most common sleep disordered breathing (SDB), affecting 4% and 2% middle-aged men and women respectively. During sleep, patients with OSA experience repetitive episodes of upper airway narrowing and collapse, producing frequent apneic and hypopneic episodes, accompanied by arterial desaturation and sleep fragmentation. There is a growing body of evidence that this cyclical intermittent hypoxia may be the primary trigger for various pathogenetic mechanisms in OSA leading to increased cardiovascular morbidity and mortality. The health implications, of even mild OSA, for both individuals (daytime sleepiness, neurocognitive deficits and increased cardiovascular risk) and the community (automobile and industrial accidents) continue to be increasingly recognized.

Upper Airway Mechanics

The control of upper airway patency has been described as related to the balance of forces acting across the upper airway walls, and the tissues surrounding the upper airway exert a mechanical pressure on the airway wall. Therefore, upper airway patency depends on 2 processes: resistance to the collapsing forces acting on the airway and rapid re-opening of the airway after episodes of obstruction. Previous studies reported that intraluminal pressure required to re-open a closed upper airway was greater than the intraluminal pressure present during closure of the same airway. The difference between these two pressures was ascribed to the force required to overcome “adherence” between the walls of the closed airway. These findings suggested that surface effects due to the liquid lining the upper airway (UAL) exert an influence on upper airway patency. Apart from the action of dilator muscles in determining the balance of forces in the upper airway, there has been another important “adherent” force of the UAL to be determined - the magnitude of the pressure required to separate mucosal surfaces in contact during airway closure.

Surface Tension (ST) of Upper Airway Lining Liquid (UAL)

When liquid-coated surfaces come into contact, there is a tendency for them to adhere to each other. In the case of two ideally flat surfaces the force holding them together, or the contact adhesion, depends only on the ST of the liquid and the separation between the surfaces. It can be imagined that the smaller the surface separation and the larger the ST, the greater the force holding the surfaces
Adhesion between liquid-coated surfaces is, thus, due to the ST of the liquid, which in turn is ultimately due to the cohesive forces between molecules of the liquid. So, in reality, when the upper airway collapses, liquid lined mucosal surfaces come into contact and lumen diameters approach zero in the contact areas, and the mucosal folding must be present as the airway progressively collapses, and this will result in increased apposition of upper airway surfaces. Therefore, mucosal folds offer conditions where ST is most likely to be active in influencing upper airway collapsibility and re-opening. The formation of liquid bridges within the interstices of mucosal folds may be of particular importance. It should be noted that when the upper airway collapses, mucosal surfaces are completely in apposition, hence, creating the circumstances whereby ST of UAL may have a substantial influence.

The ST of UAL can be measured by a unique surface force measuring apparatus that assesses ST of liquids via measurement of the “pull-off” force required to separate two smooth silica surfaces bridged by the test liquid. Measurement of ST using this approach can be performed using sample volumes as small as around 0.2 microliter. There are different values of ST of UAL in rabbits, anaesthesized humans, and OSA patients. It has been established that ST of liquid samples obtained from posterior pharyngeal wall is similar to that of saliva (54-62 mN/m) and substantially less than that for water (71 mN/m), reflecting the presence of endogenous surfactants in UAL.

Surface Tension Effects on Sleep Disordered Breathing

There is emerging evidence of ST effects on upper airway mechanics and their functional implications for upper airway patency during sleep are substantive. It has been shown that addition of a topical lubricant or surfactant to the upper airway significantly reduced the severity of OSA in humans, and the magnitude of the decrease in apnea-hypopnea index (AHI) correlated strongly with the magnitude of the fall in ST of UAL in recent studies. However, ST of UAL may vary between individuals and it may predispose to or protect from SDB. It is believed that the relationship between the ST of UAL and “altered structural changes and sensorineural defects in upper airway control” should be further examined.

Physiological regulation of ST of UAL

It is still uncertain how the ST of UAL is being regulated. It is crucial to understand the potential pathophysiological processes that lead to an abnormal ST of UAL, this may allow development of therapeutic options that manipulate the physiological control of ST of UAL in OSA.

In awake healthy subjects, ST of UAL is similar to that of saliva. Saliva is predominantly produced from the parotid, submandibular and sublingual glands but 10% arises from accessory glands, including those in the soft palate and pharyngeal mucosa. Saliva is transferred from the oral cavity to the pharynx by swallowing, where it coats the mucosal surfaces and lubricates the passage of food. Saliva contains surface-active phospholipids that result in a low ST (57 mN/m). While this has recognized gastrointestinal benefits, such as reducing adhesion of micro-organisms to oral surfaces, it is postulated that it may have an additional respiratory related benefit.

Salivary flow rates are depressed during sleep and xerostomia associated awakening during the night is commonly encountered, especially in OSA patients. Recent data suggest that rhythmic masticatory activity and
swallowing during sleep promotes salivary production\textsuperscript{19}. It can be imagined that the interaction between salivary flow and swallowing during sleep, provides a mechanism that produces and transfers low ST saliva from the oral cavity to the pharyngeal mucosal wall. Therefore, this process may help to maintain a coating of low ST saliva on the walls of the pharyngeal airway. Other factors may also influence the maintenance of a low ST layer of saliva on the pharyngeal mucosa. One of these is breathing route. A recent study found that as the mucosal surfaces were relatively dry in upper airway with oral breathing in humans, the ST of UAL increased, on the contrary, with nasal breathing, where the mucosal surfaces were comparatively wet and ST of UAL decreased\textsuperscript{22}. It can be concluded that nasal breathing during sleep may be partly controlling the ST of UAL and some protective mechanism may be present with nasal breathing that lowers an already low ST of UAL at a time when the UA is most vulnerable to collapse. Hence, it may well be projected that conditions characterized by increased oral breathing during sleep will lack this protective mechanism, as in OSA patients. However, how the breathing route effect on ST of UAL is mediated is not yet clear, but lowering the ST of UAL may contribute to reduction of the severity of SDB.

Future directions

OSA has become a public-health issue since the last decade. There is increasing data of independent contribution of OSA towards cardiovascular diseases\textsuperscript{2}. Current management regimes for long-term control of OSA are aimed at providing pneumatic (continuous positive airway pressure, CPAP) or mechanical (mandibular advancement splint) support for the upper airway during sleep, and need to be applied every night. Although cumbersome, these therapies can be very successful at reducing the severity of SDB, but there are potential problems of patient compliance, acceptance and resource allocation. There is a growing need to develop less invasive, more sophisticated and more patient “friendly” adjunct therapies, especially preventive therapies that could be applied widely to combat disease progression. There have been increasing number of studies to investigate the dynamic changes of upper airway, and its patency is influenced by upper airway dilator muscle activity\textsuperscript{3}, surrounding extraluminal tissue pressure\textsuperscript{4}, intraluminal pressure\textsuperscript{5} and surface forces of UAL\textsuperscript{6}. It has been demonstrated that the severity of SDB can be reduced by lowering the ST of UAL\textsuperscript{12}. This finding strongly suggests that manipulation of the ST of UAL may offer a complimentary approach to the management of SDB. Further studies are needed to explore the role ST of UAL in control of upper airway patency and the potential for new therapeutic strategies based on the manipulation of ST of UAL, such as a ST lowering agent, would be more acceptable to our OSA patients.

References:

Pulmonary sequestration (PS) was first used by Pryce\(^1\) in 1946, in which sequestare in Latin means “to separate”. It is defined as a non-functioning lung tissue that is not in normal continuity with the tracheobronchial tree and that derives its blood supply from systemic vessels\(^2\).

Pulmonary sequestration is divided into two types based on their pleural covering, extralobar and intralobar. In extralobar pulmonary sequestration, the bronchopulmonary tissue has a distinct pleura that maintains complete anatomical separation from adjacent normal lung tissue. In contrast, intrapulmonary sequestration is the lung tissue that contained within the normal visceral pleura. The aetiology of sequestration has been controversial and including five groups of proposals: vascular traction, vascular insufficiency, co-incidental occurrence, acquired pathology following infection, and common developmental theory\(^3\). It is a rare abnormality that accounts 0.15%-6.4\% of all congenital pulmonary malformation\(^4\). Intralobar sequestration is more common (75\% of cases) than extralobar type.

**Clinical features**

Extralobar type usually presented at infancy or diagnosed antenatally. There may be co-existing congenital anomalies such as diaphragmatic hernia, lung hypoplasia, congenital cystic adenomatoid malformation, bronchogenic cyst. On the contrary, intralobar sequestration typically presented in early adulthood with chronic cough, mucopurulent sputum production, recurrent pneumonia, or as incidental finding of lung mass on radiograph. Both types usually involve lower lobes with left side being more common in particular.

**Radiologic appearance**\(^5, 6\)

Chest radiograph may find a homogeneous consolidation with irregular margins, uniformly dense mass with smooth or lobulated contours in the posterior basal portion of a lower lobe, or cavitatry and cystic lesions. Focal bronchiectasis, subsegmental atelectasis, reduced lung volume, mediastinal shift and prominent pulmonary hilum have been described. Whilst, pleural effusion is rare (less than 4 \% of cases).

CT not only demonstrates lung parenchymal lesions but also can delineate the anomalous systemic arteries in about 80\% of cases. Helical CT had been reported to facilitate the display of the aberrant artery relative to conventional CT by volume acquisition through slice reconstruction with narrow intervals and multiplanar and 3-dimensional reformatting.

MRI and MRA can evaluate the thoracic aorta and pulmonary vasculature of the sequestration. However, it may not be able to detect the small anomalous vessels.
Retrograde aortography has been the traditional approach in diagnosing pulmonary sequestration by defining an aberrant systemic artery. However, it remained invasive and is becoming less favour with the availability of other imaging modalities.
Sonography is suited for evaluating the prenatal and post-natal chest.

Treatment
Surgical resection as either segmentectomy, or a full lobectomy is the main treatment mode. Radiological delineation of the systemic vascular supply and venous drainage of the sequestration would assist the surgeon in planning a successful ligation of vessels and avoiding inadvertent bleeding.

References
Lung Cancer is a major cause of cancer-related deaths in men and women. However, since the first edition of *Lung Cancer* was published 14 years ago, rapid progress in the biology, prevention, diagnosis, and treatment of the disease has been made. This book consisting of more than 20 chapters provides clinicians with comprehensive, updated and practical information in lung cancer management.

Members of Hong Kong Thoracic Society will enjoy 10% discount when ordering the book(s) under the “Book review column” through McBarron Book Co. 麥伯倫醫護圖書中心

Enquiry and Ordering Tel: (852) 2770 8521 Fax: (852) 2385 6236.
(Free Delivery Service to local hospitals included)

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<td>Florida, USA</td>
<td>44th Annual Meeting of The Society of Thoracic Surgeons</td>
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<td><a href="http://www.jrs.or.jp">www.jrs.or.jp</a></td>
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<td>4-8 October, 2008</td>
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<td>18th ERS Annual Congress</td>
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### Upcoming Clinical Meetings

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<td>Jan 24, 2008</td>
<td>Clinical meeting by United Christian Hospital and Haven of Hope Hospital</td>
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<tr>
<td>March 30, 2008</td>
<td>Annual Scientific Meeting 2008</td>
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<td>May, 2008</td>
<td>Tuen Mun Hospital and Tseung Kwan O Hospital</td>
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### Medical Societies

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<td>ACCP (HK &amp; Macau Chapter)</td>
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<td>American College of Chest Physician</td>
<td><a href="http://www.chestnet.org/">http://www.chestnet.org/</a></td>
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<td>British Thoracic Society</td>
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<td>Canadian Lung Association</td>
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<td>Society of Critical Care Medicine (USA)</td>
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<td>The Federation of Medical Societies of HK</td>
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### Publications

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<td>American Journal of Respiratory Cell and Molecular Biology</td>
<td><a href="http://ajrcmb.atsjournals.org/">http://ajrcmb.atsjournals.org/</a></td>
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<tr>
<td>Asian Medical News</td>
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<td>Thorax</td>
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The Official Journal of ACCP is now on-line but is only available to subscribers (Fellows, members or affiliated members). Trainees are welcome to join at a very privilege rate (US30 per year). Applications should be directed to ACCP (USA) through their trainers (who must be a Fellow of the ACCP). Proof and detail of training (tentative period of training) is required. Any query can be directed to the Secretary, ACCP (HK and Macau Chapter) (see page 1). Browsing of information is available on the ACCP website: http://www.chestnet.org/membership/categories.html

Membership News

♦ As of 25 November 2007, there are 748 Members (200 Ordinary members, 5 Honorary members, 78 Life members and 465 Associate members).

♦ To be eligible for Life membership, 3 years of full membership prior to the application is necessary. Please write to the Honorary Secretary (Dr CY Tam, Department of Medicine, Tuen Mun Hospital) and send the letter with a cheque of HK$2,000 to Dr Loletta So, Department of Medicine, Pamela Youde Nethersole Eastern Hospital. Acceptance will be decided in the Hong Kong Thoracic Society council meeting.

♦ For membership renewal, please fill in the application/renewal form (available at http://www.fmshk.com.hk/hkts/member.htm) and send to Dr Loletta So (address as on the form) with the subscription (HK$100/200 for associate/ordinary members respectively), (cheque payable to HONG KONG THORACIC SOCIETY LTD). Members who had their names deleted should re-apply as new members. For enquiry, or checking your membership status, please reach Dr Loletta So by email (soky@ha.org.hk) or fax (852 2515 3182) (Please supply your name and fax number). Apology for not entertaining telephone enquiry.
**Hong Kong Lung Foundation Fellowship**

The fellowship is open to medical practitioners, allied health professionals, scientists, students and others for travelling aboard to engage in research, study and training in order to gain experience in modern methods of diagnosis, prevention and treatment of diseases of the respiratory system. Please note that priority will be given to active members of the Hong Kong Thoracic Society.

The Hong Kong Lung Foundation Fellowship has three types of Awards as specified below:

1. Open to members of the medical profession granting a sum up to HK$50,000 for training of 3 to 9 months and a sum up to HK$60,000 for training of over 9 months.
2. Open to members of the nursing/paramedical profession granting a sum up to HK$30,000.
3. Open to all members of the medical, nursing and paramedical profession granting a sum up to HK$30,000 for attending conference or short training course of 3 months or less.

Hong Kong Lung Foundation Fellowship which opens its application twice a year in June and December. Applicants should submit the application forms to the Hon Secretary of the Hong Kong Lung Foundation, not later than 30th June and 31st December of each year.

Application procedures and application form of Fellowship program can be downloaded from Hong Kong Lung Foundation Website:

http://www.hklf.org/HKLF/hklf_fellows_e.htm

Hon secretary: Dr KS Chan, Pulmonary & Palliative Care Unit, Haven of Hope Hospital, Tseung Kwan O, Kowloon , Hong Kong. Fax: 2703 8799 Email: chanks@ha.org.hk

**Hong Kong Lung Foundation Research Grant**

The Hong Kong Lung Foundation was established in 1996 to nurture advancement in clinical practice in the field of lung diseases in Hong Kong Special Administration Region. As from January 2001, the foundation shall award research grants, on an annual basis, to fund research projects being performed in the HKSAR. This aims to enhance the research culture and standards of local clinicians and health-care professionals in the field of respiratory medicine and related disciplines.

Please refer to the Hong Kong Lung Foundation Research grant regulations, which must be strictly adhered to. The completed application form and other required documents must be returned to the Honorary Secretary of Hong Kong Lung Foundation by 30th November of each year. Email submission is also acceptable and should be sent to: chanks@ha.org.hk.

Application procedures and application form of Research Grant can be downloaded from Hong Kong Lung Foundation Website:

http://www.hklf.org/HKLF/research_grants_0304_e.htm

Hon secretary: Dr KS Chan, Pulmonary & Palliative Care Unit, Haven of Hope Hospital, Tseung Kwan O, Kowloon , Hong Kong. Fax: 2703 8799 Email: chanks@ha.org.hk
The Pneumoconiosis Compensation Board (PCFB) set up a research fund in 1996 with the purpose to support projects that are related to the prevention, diagnosis, assessment of disability and treatment of pneumoconiosis in Hong Kong. Individual or group are invited to apply. Interested parties may visit the website: www.pcfb.org.hk or contact the PCFB at Tel: 2541 0032, Fax: 2541 0211 or Email: contact@pcfb.org.hk.

The Pneumoconiosis Compensation Board (PCFB) has established a training grant to facilitate health-care workers and occupational safety and health personnel to enhance their knowledge and skills in pneumoconiosis. This scheme aims to encourage eligible applicants to attend overseas training programmes or conferences that are related to the topic of pneumoconiosis. A maximum grant of HK$ 100,000 will be allowed for a suitable course longer than 6 months, and HK$ 50,000 for a course of 6 months or less. Interested applicants may contact the Board Secretariat, Trophy Mak at 2541 0032, or contact the PCFB at Tel: 2541 0032, Fax: 2541 0211 or E-mail: contact@pcfb.org.hk.