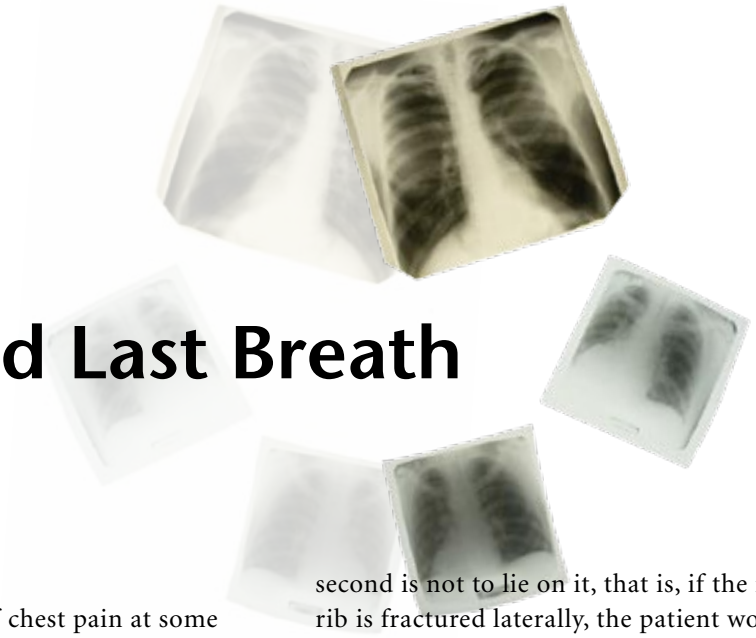
By Professor Chee Yam Cheng, Editorial Board Member



Third and Last Breath

CHEST PAIN

All of us will complain of chest pain at some point in our lives. The chest is always moving air in and out of our lungs. It cannot remain immobile. Even in sleep the chest is moving. So pain can arise from the chest wall. The parietal pleura, joints, ribs, muscles of the chest have pain-sensitive nerves. The neurovascular bundle in each rib space runs in the groove at the inferior margin of each bony rib and almost meets its fellow from the opposite side in the midline. But it never crosses the midline. So the dermatomes and myotomes have well defined innervations arising from the thoracic section of the spinal cord with exit points slightly higher than their corresponding thoracic vertebra (because the spinal cord and conus end at the first lumbar vertebra). So the twelfth thoracic nerves exit the spinal canal at about the level of the eighth thoracic vertebra.

Localised chest tenderness points to local pathology at that rib, rib space or the parietal pleura beneath. So pleuritis is painful, especially so on deep inspiration. Severe prolonged coughing can fracture ribs even if they are not osteoporotic. If there is pain and dysaesthesia in a dermatome distribution, it may be the start of herpes zoster (also known as shingles). It is a myth that should this "snake" (in Chinese) encircle the body, the patient will die. The virus lives in the dorsal root ganglion of the nerve and not in the spinal cord and it is rare to get transverse myelitis from herpes zoster.

With localised chest pain, the body will adapt in two ways. The first is to "splint" the chest cage by breathing less (shallow breathing) plus slower breaths. The patient may even put his hand on the affected site to prevent its motion while the rest of the chest tries to move normally. The second is not to lie on it, that is, if the right sixth rib is fractured laterally, the patient would prefer to sleep lying on his left side. It is not common for a pneumothorax to present as chest pain because the rupture is of the visceral not paretal pleura.

While pleuritis (inflammation of the pleura) may be of infective origin, for example, viral infection or bacterial pneumonia leading to a subsequent pleural effusion, the pain tends to resolve spontaneously when an effusion develops. So with easier lubrication (and loss of pleural rub) between the two pleural surfaces provided by the pleural fluid, the patient may breathe easy again. With our aging population, another important diagnosis not to miss is pulmonary embolism with pulmonary infarctions. There may be pleuritis to start with followed by a bloody effusion (which you will not confirm on plain CXR without sticking a small needle in but if it is really bloody, the fluid may not be aspirated at all). Certain viral illnesses may result in pleurodynia alone without lung infection in which case lung auscultation and plain CXR will show no abnormality.

If chest pain is not localised to one side or the other but is central around, and behind the sternal area, then acute coronary syndromes need exclusion as first priority the older the patient is. Retrosternal chest pain can be due to tracheitis (especially if there is persistent dry cough), mediastinal emphysema, aortic dissection or oesophageal dysfunction including reflux. These are the more serious diseases to be excluded.

AUSCULTATION

There are two parts to this; one with patient breathing in and out through an open mouth and two, with the patient saying 99 to elicit vocal

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resonance. In both, the use of the stethoscope is essential. It is a true remark that the information gained is the result of what is between the two ear pieces of this instrument. Left lying on the table, it serves no purpose; left hanging over one's neck also serves no purpose.

From ancient times, limited observation on lung sounds had been made by listening at a distance from patients to such phenomena as wheezing or stridor. Direct application of the ear to the chest increased the number of types of sounds that could be related to diseases. The science of auscultation, however, began with the invention of the stethoscope by Laennec. This instrument allowed him to describe the major categories of lung sounds that form the basis of the modern classification. He revolutionised the practice of medicine by "altering both the physician's perception of disease and his relation to the patient. In particular, the stethoscope drew the physician into the private world in which signs were directly communicated to him from the patient's body."

At the beginning of the 19th century, diagnoses were made principally on the patient's verbal account of his illness and observations with the unaided senses. The stethoscope reoriented physicians to methods for more objective diagnoses, and it periodically fell into disrepute for precisely this reason. The clear superiority of the chest X-ray (X-rays were discovered on 8 November 1895 by Professor Wilhelm Roentgen in Wurzburg, Germany) in diagnosing early pulmonary tuberculosis and lung cancer led to views by prominent chest physicians such as: "the stethoscope is largely a decorative instrument in so far as its value in diagnosis of pulmonary diseases is concerned. Nevertheless it occupies an important place in the art of medicine. Apprehensive patients with functional complaints are often relieved as soon as they feel the chest piece on their pectoral muscles."

BREATH SOUNDS

To most physicians, "breath sounds" mean the respiratory noise heard through the stethoscope on the chest wall. Noisy breathing as a sign of disease was recognised by Laennec (1819) who carefully distinguished it from wheezing and other adventitious sounds. Sounds generated during breathing are a good guide to the state of the airways and lungs in health and disease. Crackles are crepitations and rhonchi are wheezes.

The breathing of a healthy person at rest is barely audible at the mouth while in chronic bronchitis or asthma, it can be heard at a distance of several feet. This barely audible breath sound in normals is probably generated by turbulent air flow in the proximal bronchi and trachea. In the patient with chronic bronchitis or asthma with airways obstruction, the intensity of this noise is greatly increased. The loudness of this inspiratory sound correlates well with the severity of expiratory obstruction measured in airways resistance; forced expiratory volume is one second (FEV1) or peak expiratory flow rate (PEFR). The inspiratory breath sounds at the mouth are paradoxically quiet in patients with emphysema because here, there is no intrinsic narrowing of the bronchi, and the severe expiratory airflow obstruction is caused by dynamic compression of the bronchi due to loss of elastic properties of lung from alveolar destruction.

If there is confusion about terminology, you are not to be blamed. The word "crepitations" in British use seemed to be virtually equivalent to the American "rales". So a Committee of the International Lung Sounds Association met in 1976 to standardise the terminology and reduce confusion. This was adopted by the American Thoracic Society (ATS) in 1977. The separation of adventitious sounds into "continuous" and "discontinuous" is used with further differentiation of these two major classes of sound. So although Laennec described four common lung sounds, the ATS has translated his original terms into coarse and fine crackles (discontinuous sounds) and wheeze and rhonchi (continuous sounds). Coarse crackles or crepitations are loud, low in pitch; fine are less loud, of shorter duration, and higher in pitch. Wheeze is a continuous sound longer than 250 milliseconds, high pitch with dominant frequency of 400 Hz or more (also known as sibilant rhonchus); rhonchi is lower pitched with dominant frequency about 200 Hz or less (also known as sonorous rhonchus).

ORIGIN OF SOUNDS

(1) Normal breath sounds

The site of origin of these sounds remains disputed. Some believe they are generated by turbulent airflow in the large bronchi. Others suggest they are from laminar non-turbulent airflow in small airways of internal diameter 3mm or less close to the alveoli. It seems certain that they are not caused by air entering the alveoli. Laennec believed these sounds were caused by friction of air against the lining of the airways.

The lungs and chest wall act as an acoustic filter narrowing the range of sound

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frequencies audible over the chest to 100-400Hz. Air or fluid in the pleural space increase the acoustic barrier, and the breath sounds become fainter or inaudible over the chest. At the upper margin of a pleural effusion, the thin layer of fluid still permits this transmission of higher sound frequencies, giving the breath sounds a nasal or bleating quality termed aegophony.

The transmission of breath sounds is less at the bases than at the apices. The presence of normal breath sounds virtually excludes severe reduction in FEV1 and reduced breath sound intensity is strongly suggestive of obstructive lung disease.

The respiratory sound heard at the mouth and over the trachea consists of oscillations whose amplitude is evenly distributed over a wide range of frequencies between 200Hz and 2,000Hz. Its loudness follows closely the variations in flow rate, remaining constant throughout inspiration and fading in parallel with the steady falling flow rate duringexpiration. Despite this expiratory decrescendo, the breath sounds remain audible to the end of expiration. By contrast, the breath sounds heard through the chest wall contain a relatively narrow range of frequencies with a maximum amplitude of 200Hz. Above this frequency, the amplitude of oscillations decreases rapidly. Unlike the breath sounds over the trachea, they fade and become inaudible soon after the beginning of expiration. Laennec called these sounds "vesicular" to mark the contrast with "bronchial" sounds heard over the trachea and transmitted to the neck and the interscapular area.

Laennec draws attention to the fact that breath sounds may be abnormally loud at the mouth yet normal or faint when heard through the chest. This discrepancy goes further. The inspiratory sound at the mouth gets louder as air flow obstruction increases, while over the chest, the converse is true. Experiments on models of the bronchial tree show that between turbulent flow in the central airways and laminar flow atthe periphery of the bronchial tree, there lies an intermediate zone extending from the segmental bronchi to about the tenth generation of the airways where the flow pattern is irregular and contains vortices. Discontinuities in gas flow and variations in gas pressure in these bronchi are probably accompanied by oscillations at acoustic frequencies.

(2) Bronchial sounds

The tracheal or bronchial breath sounds heard by listening over the large airways have a different character from vesicular sounds. The sound has a hollow or "tubular" character. Laenaec said that the sound clearly showed that the air was passing through a roomier channel than the air cells. Bronchial sound contains higher frequency components than vesicular sound and the site of these sounds are in airways 4mm or larger in diameter. Presence of these sounds in abnormal locations signifies a continuous infiltration of lung tissue from the periphery 3cm to 5cm inward toward the hilum. Bronchial breath sounds heard over consolidated lung represent improved transmission to the periphery of sounds produced in the large airways. Consolidated lung acts like a continuous accoustically conducting medium that does not attenuate the transmission of sound as does the inflated lung.

Vocal resonance are bronchial sounds accentuated by saying words with a nasal quality, for example, 99. This should not be translated into dialect directly. Rather in Mandarin, it should be the equivalent of one, two, three, the last exhibiting the nasal tone.

It seems probable that bronchial and vesicular sounds each arise from a range of airways rather than from a point source. So breath sounds intermediate between them are called bronchovesicular. "Bronchial" and "bronchovesicular" refer to tubular sounds with a long, loud expiratory phase independent of whether they arise in normal lung or are due to increased transmission of centrally generated sound through peripheral consolidation.

(3) Continuous adventitious lung sounds

Continuous lung sounds are 250ms or more in duration and are usually louder than the underlying breath sounds. Wheezes are not caused by vibration of air as in organ pipes or wind instruments. If they were, the bronchi would need to be several feet long to be able to produce low-pitched wheezes. The wheezes are produced by the vibration of the walls of compressed bronchi and the pitch is determined by the mass and elastic properties of the wall, the tightness of the stenosis and the rate of gas flow though it. The pitch of a wheeze is no guide to its origin through wheezes which are audible at the mouth are probably generated in large central bronchi.

Wheezes are produced when the caliber of the airways is narrowed to the point where its opposite walls touch one another. The best model among musical instruments is the toy

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trumpet with a metal reed whose vibration is responsible for the sound production and whose horn is purely decorative. High-pitched musical sounds are generated in lightly compressed airways of any size. The pitch of the wheeze as stated above is not dependent on the length, caliber or mechanism of airway point of closure but most of all, by the flow velocity through the airway on the point of closure.

It is therefore important to listen over the trachea so that the dampening effect of the chest wall is excluded. Circumstances suitable for the production of continuous adventitious lung sounds are encountered in narrowing of airway caliber caused by bronchospasm, by mucosal thickening or oedema, by the presence of a tumour, phlegm, foreign body or secretions, or by pressure from without by a tumour mass or dynamic airway compression. A forced expiratory manouevre may help produce wheeze.

(4) Discontinuous adventitious lung sounds

Adventitious lung sounds with individual components lasting for less than 20ms are heard as a series of brief explosive sounds. Crackles are produced by explosive reopening of airways during inflation either by the development of a critical pressure gradient across the closed section between the proximal airway and gas trapped distally in the alveoli, or as a result of radial traction on the airway by the surrounding lung tissue as it expands. Late inspiratory crackles are a characteristic finding in patients with pulmonary fibrosis and are usually first heard at the lung bases where the effect of gravity is the greatest and causes airway closure. The quantity of crackles correlates well with the radiological appearances and physiological distribution in this disease, for example, caused by asbestosis or fibrosing alveolitis.

Patients with chronic airways obstruction often have early inspiratory crackles which are easily heard at the mouth. These are usually fewer and of lower pitch than the late inspiratory crackles of fibrosing lung disease.

Airway closure occurs at low lung volumes in normal subjects. Careful auscultation at the bases during inspiration from residual volume in young nursing students revealed the presence of crackles in 63% of them and this rose to 92% when an electronic stethoscope was used. They were rarely heard when the subjects breathed at tidal volume. However, in patients with loss of lung volume due to disease, crackles were heard late in inspiration, when a lung volume of more than 50% of vital capacity (usually between 65% and 85%) had been achieved. More reduction in lung volume and more crackles appeared with oxygen than with air breathing at low lung volumes. This means that reopening of distal atelectatic lung units generates crackles, and that the more units that have collapsed, the more crackles are generated.

CHRONIC BRONCHITIS AND EMPHYSEMA

These patients have noisy chests because of the frequent occurrence of adventitious sounds. Rhonchi that clear with coughing and end-inspiration wheezes are common. In obstructive diseases in general, the intensity of the vesicular lung sounds decreases. A simple bedside technique using lung sounds to detect or assess airflow obstruction requires only a stopwatch and a stethoscope. The patient is asked to take a deep breath and blow it out as fast as possible. The bell of the stethoscope is placed in the suprasternal notch. The duration of audible respiraton (FETo) is timed to the nearest half second. If the FETo is less than five seconds and airflow has stopped, it can be confidently predicted that the FEV1/VC % is greater than 60%. Conversely if the FETo is greater than 6 seconds and airflow continues, the FEV1/VC % is less than 40%.

BRONCHIAL ASTHMA

The most prominent auscultatory feature of bronchial asthma is a high-pitched continous sound called a wheeze. Most but not all asthmatics wheeze during exacerbation of their disease. In early stages, wheezing may be heard over central airways during expiration, and it usually can be detected over the entire chest and in both phases of respiration as the asthma becomes more severe. The absence of wheezing in severe asthma is regarded as an ominous sign, presumably because it results from flow rate so low that wheezes are not generated or from mucus plugging that has severely impaired ventilation. Wheezing has other causes like tumour, foreign bodies, aspiration, pulmonary emboli, congestive heart failure. Clinical asthma can be present without wheezing and so be overlooked.

PULMONARY FIBROSIS

The most striking feature of the chest examination in patients with this disease is the presence of fine crackles. These sounds

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have a distinctive character and have been described as "close to the ear", cellophane or velcro crackles. In mild disease, they are usually confined to end inspiration and are gravity dependent in that they are best heard at the lung bases with the patient upright. Their distribution can vary with change in position In more advanced stages of the illness, these crackles persist, despite position changes and are heard at increasingly higher levels above the bases. They may become pre-inspiratory often with an end-inspiratory accentuation as the disease progresses. An end-inspiratory short wheeze beginning with a single crackle may be associated with these sounds presumably caused by air passing through a newly opened but still narrowed airway. As it opens abruptly, it crackles; as it is still narrowed, it wheezes.

PEAK FLOW METERS

Pulmonary function tests provide objective quantifiable measures of lung function. They are used to evaluate and monitor diseases that affect heart and lung function, to monitor the effects of environmental, occupational and drug exposures, to assess risks of surgery and to assist in evaluations performed before employment or for insurance purposes. Spirometric examination is the most widely used test. Spirometry is the measurement of the movement of air into and out of the lungs during various manoeuvres. Such examination should be readily available and routinely used in medical offices.

Just as the blood pressure measurement is essential to the diagnosis and management of hypertension by providing objective readings in numbers, the peak flow meter, for example, mini-wright, provides a numerical value of the PEFR (peak expiratory flow rate) in L/minute. While not exactly equivalent to the FEV1 (forced expiratory volume in one second), which is usually performed in a lung function laboratory, the PEFR is a reliable bedside surrogate. It is essential in the diagnosis and management of patients with asthma because of evidence that both patients and physicians have inaccurate perception of the severity of asthma that contribute to delays in treatment. Under-estimation of the extent of airflow obstruction is associated with increased mortality in asthma. Spirometry allows this severity of the abnormality to be quantified and the presence of reversible airflow obstruction to be determined. The presence of the latter differentiates asthma (reversible obstruction) from COPD (irreversible obstruction).

MINI-WRIGHT FLOW METER

This handy portable device can be used by patients to monitor their own asthma or COPD status. Readings can be recorded and trends noted. It can indicate when the disease is not responding to current medication and trigger the need to go to hospital or call the asthma nurse for advice. The best of three readings in the upright posture is recorded. Because this test is fully patient-dependent, there must be full effort in forcing expiration rapidly from a deep breath in. There is a reference normogram to determine what a normal value is and this is dependent on gender, age and height of the subject. Non-cooperation by the patient invalidates the result.

Asthmatics not under control will have highly variable readings over a 24-hour period. With stabilisation using appropriate medication, the best PEFR readings on record will rise to normal while the variation in readings will decrease. Normal variation is no more than 10%. As patients age, it is useful to remember that non-smokers tend to lose FEV1 at the rate of 20ml to 30ml per year whereas "sensitive" smokers (those with accelerated decline in FEV1) lose FEV1 at a rate of more than 60ml per year. Serial measurements are necessary to monitor this decline and one value within the normal range is no predictor of the future rate of decline.

In the Framingham Study, decreases in VC (vital capacity) were a better predictor of heart failure than were symptoms. Improvements in VC may also indicate recovery from congestive heart failure.

CONCLUSION

I have reviewed some common symptomatology in patients with respiratory ailments. Also I have emphasised some aspects of basic chest examination revolving around IPPA (inspection, palpation, percussion and auscultation). Each step in this clinical method is fully applicable to chest examination unlike in other systems where some steps predominate in importance. Lastly, I urge all doctors to have in their consultation rooms a peak flow meter. This should form part of the equipment that characterises a good physician's practice, not just a stethoscope and a blood pressure set.

Why the title in these three-article series? It is to emphasise that you are allowed three separate breaths in doing the PEFR measurement and only the best effort counts. You are allowed three chances to demonstrate how good your lungs are. If you aim to be a pilot or buy health insurance, no doubt you will be fully cooperative. However, if the motive is to avoid training or going to work, cooperation may be entirely lacking. ■