By Professor Chee Yam Cheng, Editorial Board Member

One Breath Away

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BREATHLESS

Shortness of breath is a common complaint. It means the patient's breath is short or he/she has inadequacy of breaths or both. To have a clearer understanding of the complaint, ask the patient which phase of breathing is this feeling of inadequacy felt and in which phase, the difficulty. If it is difficult in expiration, then the illness lies in the lower respiratory tract. If it is in inspiration then the problem is at the vocal cords and above in the upper respiratory tract. The airways and lung parenchyma can expand on purposeful inspiration but most of expiration is elastic lung recoil, which can be increased by forced expiration. So if lung elastic fibres are destroyed as in emphysema, then air is trapped and cannot leave, and forcing expiration leads to more airway collapse. Hence the classical sign of purse-lip breathing to retain some positive airway pressure during expiration so that airways do not collapse.

Another complaint is tachypnea. It means breathing faster than the usual adult rate of 14 to 16 breaths per minute. As you voluntarily breathe faster, you are blowing out carbon dioxide. In the hyperventilation syndrome or with anxiety and panic, over-breathing leads to the classical symptomatology of light-headedness, circumoral paraesthesiae, tingling sensation in the hands and feet and carpopedal spasms. Try it; you too can have this experience. This is what mass hysteria and panic is all about. It has occurred in factories where young female workers start to hyperventilate when scared or panic-stricken for whatever reason; they then faint from the light-headedness and injure themselves when they fall.

Why light-headedness? Blowing off carbon dioxide lowers arterial blood levels and the brain blood vessels therefore vasoconstrict. The anaesthetist can beautifully use this to control cerebral oedema in any neurosurgical operation should the brain overflow out of its skull. The other symptoms result from the alkalosis when carbon dioxide levels and hence carbonic acid levels in the blood fall. With alkalosis, ionic calcium levels fall and thus parasthesiae results in the extremities.

What about dyspnea? It means difficulty in breathing and could be a symptom or an observed sign. Again it is very useful to analyse if it is difficulty in inspiration or expiration. Ask the patient to demonstrate if necessary. What about platypnea? Never heard of the term? Well, it means difficulty in breathing when lying on one side – left or right. If there

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is disease in the right lung, which side would the patient lie on when sleeping? Left side, that is, he would be most comfortable lying on his normal side. Why so?

It is all a matter of lung physiology regarding V/Q – ventilation and perfusion. The lung parenchyma exists for gaseous exchange between air breathed into (can be obstructed by narrowed airways) and blood flowing into the alveoli. When sitting or standing upright, the V/Q ratio is greater than one in the lung apices, it then decreases to one somewhere in the mid-portion and is smaller than one at the dependent lung bases where perfusion is greater than ventilation. In a patient with a diseased right lung, the V/Q mismatch is greatest if he lies on his right side. What is the point of sending so much blood to the diseased right lung? But if he lies on his good lung, that is, the left side, there would be better V/Q matching compared to the right lung.

To summarise the breathless story, by properly analysing breathing difficulty, you would have a good idea of whether the upper or lower respiratory tract is involved, whether it is one side or the other and why patients who hyperventilate develop those symptoms and signs.

PHYSICAL SIGNS

I will not go through all the signs but the cardinal ones that could assist in making the diagnosis.

AIR TRAP

The lungs are for gas exchange. The thoracic cage is the pump that helps air into the lungs actively (that is, you can choose to stop breathing) and air out of the lungs (greater by elastic recoil than by forced effort). That is why I pity those with severe emphysema where the patient could breathe in (voluntary effort) but has great trouble getting air out. If little or no air can come out of the lungs, how is the next breath of air going into the lungs? This leads to a barrel chest where air trapping causes the ribs to be almost horizontal, lungs hyperresonant to percussion and diaphragms are pushed way down the thorax into the abdomen. The liver is easily palpable, being pushed down by the diaphragm. The diaphragm is hardly dome-shaped as it should be and must be for efficient action. Instead it is flat or even worse, everted such that the convexity points to the pubis. When this happens, Harrison's Sulci appear at the lower costal margins – a sign that the slips of diaphragmatic muscle are pulling on the lower ribs and cartilages all the while. This action actually brings the lateral sides of the chest closer together – quite the opposite of what mother nature intended with the pump handle action of the ribs when the diaphragm is correctly positioned. So what a chore – when you are breathing in, you are breathing out (because diaphragmatic contraction pulls the chest wall inwards towards each other instead of doing the reverse) and when it is time to breathe out, you cannot breathe out because the airways have collapsed.

INSPECTION

The chest examination is the classic demonstration of IPPA – the well-tested clinical method of applying inspection, palpation, percussion and auscultation. I will not describe the full inspection routine and ritual but just highlight some useful tips.

In the examination, inspection often takes place with the doctor standing on the right side of the patient. Inspection is best done with the doctor at the foot of the patient's bed. Observation is for the equal rise and fall of the thorax and abdomen in synchrony. When the thoracic cage expands on inspiration, the abdominal wall will rise (because the diaphragm is pushed from the thorax into the abdomen). To the astute observer, if this is unequal between the two sides, then there is thoracic pathology on the side that moves less well. This must be so since the whole exercise of the cage is to move air in and out and if it cannot do this, then it has pathology. Of course, in bilateral diseases, the side that moves less has more disease. From the observation of the abdomen, diaphragmatic weakness can be detected should the abdominal wall on that side fail to move up and down as much as on the other side.

Frequently, we fail to note movements of the upper thoracic cage because it is more fixed compared to the lower cage. Therefore focusing on the supraclavicular fossa becomes essential for detection of upper lobe lung disease. Just look at the two concavities. The one that is more depressed is the diseased side. So the lung extends above the clavicle and it is wrong to do IPPA only from below the clavicle downwards on anterior chest examination. If pulmonary tuberculosis is still present with us, most likely the upper lobes are the site of chronic fibrosis.

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In the undergraduate examination, students fail to find scars not because they fail to remove the shirt and the bra but because they fail, after having done so, to look carefully at the sides (a barrel chest has a lot of side under the armpits) and the back of the thorax before lying the patient down again. Looking at the lung volumes which are accessible to clinical chest examination, there is much more available to elicit signs from the posterior rather than the anterior aspect of the chest. So again look at the patient's back during inspection; if positive for signs (for example, surgical scar and drainage masks), there are much dividends when pressed for time.

PALPATION

Clubbing of the fingers is an important clinical sign and there are five stages to it. The earliest is increased fluctuancy of the nail bed and this must be palpated for. There is this feeling of increased sponginess when the nail base plate is pushed inwards towards the phalanges. Clubbing when presents means chronic suppuration is present. Tuberculosis does not usually result in clubbing.

In the thorax, it is essential when detecting shifts of the mediastinum, to locate the tracheal position (upper mediastinum shift) and the apex beat. However, in barrel-chested individuals where air-trapping is large, the apex beat cannot be felt. In bull-necked and short-necked individuals, there may be difficulty locating the trachea. Do you prefer the neck extended or flexed? It should be flexed. Stretching the neck muscles makes it more difficult, so relax them and flex the neck towards the feet. Place the chin upon the chest. Use your right index and middle finger and place them into the suprasternal notch. Feel the tracheal rings in the neck. Then flex the head further and push your finger not backwards towards the cervical spine, but downwards into the thorax, pointing towards the lumbar spine. After all, you want to know whether the upper lobes are pulling or pushing the trachea to one side. So the deeper into the thorax you go, the more likely you will obtain this piece of information.

Vocal fremitus is a poor sign. Vocal resonance is more reliable but lung pathology has to be of significant size/area for abnormality to be detected. The liver edge is palpated to assess liver size and the position of the right hemidiaphragm. Lymph nodes, if enlarged, are significant in the context of malignancy.

The question I often ask students is which thoracic and cervical lymph nodes are directly relevant to the lung and the answer is usually not forthcoming. Axillary lymph nodes do get involved in breast cancer and lung cancer. In the latter, only when the parietal pleura and chest wall have been seeded by malignant cells. So long as the visceral pleura remains clean, then the axillary lymph nodes cannot be involved. Going back to lung embryology, the foregut gives rise to the lung bud which as it divides and subdivides, grows into the mesoderm to form smaller and smaller bronchioles with surrounding alveoli. The limiting membrane to this proliferation is the visceral pleura. The pleural space is the watershed area. The parietral pleura is derived from the mesoderm itself. So if a cancer cell is found in the pleural fluid, it has a choice. It can settle on the visceral or the parietal pleura. If it settles on the latter, then the metastasis would follow the chest wall lymphatics and reside in the axillary nodes. If it settles on the former, it will drain into the lung lymphatics back, tracing the paths the lung bud first took when forming the lungs. That is, it will drain centripetally to the mediastinal nodes, then to the supraclavicular nodes. So in examining the patient, malignancy has spread through several nodal stations before appearing in the supraclavicular fossae nodes (quite unlike the drainage of the parietral pleura reaching the axillary nodes more quickly).

Students tell me instead about the various chains of neck nodes, down the sternomastoid muscle, around the jaw, nape of neck by the trapezius and so on. These are not directly relevant to the chest and thorax. They fail to realise the significance of Virchow's node which is the supraclavicular node on the left in between the two heads to the sternomastoid muscle. It is the last node before lymph drains into the venous circulation and into the right atrium of the heart.

If any of these nodes are palpable and clinically significant, then in terms of cancer spread, it is surgically inoperable for a cure. So finding a hard, enlarged node in the axilla or supraclavicular fossa that is clinically relevant to the lung cancer is a bad prognostic sign. The corollary no node palpated does not mean no metastasis either.

SEMI-CONCLUSION

In the next article, I will discuss cough and the other two clinical methods of chest examination, percussion and auscultation. ■