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Clinical Anatomy Series- Lower Respiratory Tract Anatomy

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ABSTRACT

As discussed in the previous article of this series,[1] the necessity for a greater understanding of anatomy has never been more pertinent.[2,3] This paper continues on from the cardiac anatomy covered in the last issue by staying in the thorax as the lower respiratory tract is reviewed and placed in a clinical context relevant to undergraduates and junior doctors.

Chest Wall and Diaphragm

It is difficult to discuss the anatomy of the respiratory system without first considering the chest wall and diaphragm, given the crucial role of these structures in respiration. The thoracic wall consists of skeletal and muscular components, extending between rib 1 superiorly and rib 12, the costal margin and the xiphoid process inferiorly. There are 12 ribs; seven 'true' ribs that articulate with the sternum via costal cartilage and five 'false' which do no not connect anteriorly. On the inferior aspect of each rib is a subcostal grove which contains a nerve, artery and vein – i.e. a neurovascular bundle.[4]

Between the ribs are the intercostal muscles that and allowing for alterations in thoracic volumes. These consist of three layers – the external, internal and innermost intercostal muscles. The external layer aids inspiration, whilst the internal and innermost contributes to expiration.[4]

The diaphragm is a thin muscular sheet that defines the lower limit of the thorax. It gains attachment at the xiphisternum, costal margin and upper lumbar vertebrae. Multiple structures pass through it, including (from anterior to posterior) the inferior vena cava, oesophagus, aorta, azygous vein and thoracic duct. It is innervated by the phrenic nerve (anterior rami of cervical spinal nerves 3, 4 and 5). Arising in the neck, the left and right phrenic nerves pass over anterior scalene muscles deep to the carotid sheath, enter the thorax posterior to the brachiocephalic veins and descend over the heart within the fibrous pericardium before piercing the diaphragm. Upon contraction, the diaphragm flattens to increase the volume within the thorax and allow for inspiration. In addition, sternocleidomastoid and the scalene muscles of the neck act as accessory muscles of respiration, which are utilised typically during respiratory distress.[4]

Clinical relevance

Chest drain insertion is a commonly performed procedure in the management of pleural effusion, pneumothorax and empyema. To do so, a chest tube is inserted typically at the 4th or 5th intercostal space in the mid-axillary line and passes through the intercostal muscles and parietal pleura into the pleural space. During this procedure it is important to direct the incision and drain to the superior aspect of the rib (e.g. incise over superior border of 6th rib when passing through 5th intercostal space) otherwise damage can occur to the neurovascular bundle lying in the inferiorly located subcostal groove.[5] Importantly, this can produce a haemothorax due to vessel injury.[6]

As mentioned, both the intercostal muscles and diaphragm are vital for respiration. Therefore, any impairment of their function can be life threatening. The intercostal muscles are innervated by intercostal nerves T1 – T11[4] and as such, a cervical spinal cord lesion

would be required to affect all nerve roots. However, in conditions such as Guillian-Barré syndrome, myasthenia gravis and motor neurone disease, where weakness and paralysis can occur in multiple areas, there can be a significant reduction in respiratory function, potentially leading to type 2 respiratory failure (i.e. low pO2 and high pCO2).[7] Given the long route of the phrenic nerve from C3-5 in the neck, it can be damaged at many points along its course to the diaphragm. Common causes of injury include cervical trauma (such as vertebral fracture[8] and during forceps delivery of the newborn[9]), and malignancy in the neck and thorax,[10] particularly where the nerve lies anterior to the lung hilum.[4]

Pleural Cavities

The lungs lie within the pleural cavities. Each cavity, found lateral to the mediastinum, is lined by pleura – a layer composed of mesothelial cells and connective tissue. The pleura are subdivided into parietal and visceral components; the parietal adheres to the chest wall while the visceral covers the lungs, with a relative vacuum between the 2 layers known as the pleural cavity or space. A small volume of pleural fluid is present in this potential space between the two layers that acts to lubricate movement between the pleura during respiration. Inferiorly, below the lung bases, spaces exist between the parietal pleura adherent to the chest wall and diaphragm, known as the costodiaphragmatic recesses. These recesses expand during expiration as the lung bases move superiorly.[4]

Clinical Relevance

In the pleural space, there is the potential for excess air (pneumothorax) or fluid (pleural effusion) to accumulate. The causes for a pleural effusion to develop are multiple, and the composition of the effusion can be split into transudates and exudates, reflecting different aetiologies (**Table 1**). Transudates have a low protein level (<25g/L) and generally arise due to increased venous pressure or hypoproteinaemia. For example, an increase in left ventricular diastolic pressure occurs in left sided heart failure, producing a rise in pressure within the pulmonary veins and capillaries.[7] In contrast, exudates exhibit a high protein level (>35g/L) and occur as a result of increased capillary permeability, typically secondary to infection, inflammation or malignancy.[11]

Table 1: examples of causes for transudates and exudates

Transudates	**Exudates**
Heart failure	Malignancy
Liver cirrhosis	Pulmonary embolism
Nephrotic syndrome	Pancreatitis
Myxoedema	Rheumatoid arthritis

Lungs

Each lung originates from the central hilum that contains a number of key structures including the pulmonary artery, two pulmonary veins, a main bronchus, bronchial vessels, nerves and lymphatics.[4]

The right lung is composed of three lobes that are diveded by an oblique and horizontal fissure. The oblique fissure runs between the lower and middle lobe, and the horizontal fissure runs between the middle and upper lobe. A number of structures are adjacent to the right lung, and indeed leave impressions upon its medial surface. These include the heart, inferior and superior venae cavae, azygos vein and oesophagus.[4]

The left lung is slightly smaller than the right due to the predominantly left sided position of the mediastinum, and is split into an upper and lower lobe by the oblique fissure. Additionally, a tongue-like projection, called the lingula, extends from the upper lobe over

the heart anteriorly. Key structures which lie in close approximation to the medial aspect of the left lung are the heart, aortic arch, thoracic aorta and the oesophagus.[4]

Clinical Relevance

When auscultating the chest, it is important to appreciate the surface landmarks of each lobe. This aids determining which lobe is affected by disease. On the right, the fissures are demarcated as follows:

* Horizontal fissure follows the 4th intercostal space
* Oblique fissure follows a curved line from the T2 spinous process posteriorly to rib 6 anteriorly[4]

The upper lobe is therefore auscultated superior to the 4th intercostal space, the middle lobe between the 4th intercostal space and 6th rib anteriorly, and the lower lobe inferior to the T2 spinous process posteriorly.

For the left lung the oblique fissure follows a curved line from the T2 spinous process posteriorly to the costal cartilage of rib 6 anteriorly[4] The left upper lobe is therefore auscultated superior to T2 spinous process posteriorly and rib 6 anteriorly, and the lower lobe inferior to these points. In addition, the lung apices should be auscultated bilaterally above the clavicles.[12]

As noted, several important structures lie in close proximity to the medial aspects of the lungs. Therefore, any mass arising in this area can quickly involve a number of surrounding structures, and this will clearly affect the clinical picture. Examples of structures that may be affected and resulting symptoms are presented in **Table 2**.

Table 2: structures in close proximity to the lungs, and symptoms arising from their dysfunction[7]

Anatomical structure affected	**Symptom**
Left recurrent laryngeal nerve	Hoarseness
Phrenic nerve	Dyspnoea
Sympathetic plexus	Horner's Syndrome (miosis, ptosis, anhidrosis)
Oesophagus	Dysphagia, postprandial coughing
Pericardium	Palpitations
Pleura	Chest pain, cough
Superior vena cava	Dilated neck veins, plethora, dyspnoea

Bronchial Tree

The bronchial tree begins superiorly with the trachea, which is composed of 'C' shaped cartilage rings with an open posterior wall composed of smooth muscle. This bifurcates into the left and main bronchi at vertebral level T4/5. This is also the level of the sternomanubrial joint (i.e. the sternal angle), the pulmonary trunk, the beginning and end of the aortic arch, and the entry point of the superior vena cava into the right atrium. The right bronchus is both more vertical and wider than the left. These two main bronchi then split into lobar or secondary bronchi that supply a lobe each, and then further divide into segmental or tertiary bronchi to supply one of 10 bronchopulmonary segments in each lung. These bronchopulmonary segments are discrete functionally independent sections of lung that can be removed surgically without affecting adjacent segments. Further subdivisions occur before reaching the level of the bronchioles, the luminae of which are no longer maintained by cartilage.[4]

Clinical relevance

Given the more vertical course of the right bronchus and its wider lumen, aspiration more commonly affects the right lung.[13] Furthermore, the right bronchus first divides into an upper lobe bronchus and the bronchus intermedius. The bronchus intermedius continues for around 5cm before dividing into the middle and lower lobe bronchi, and therefore foreign material or masses which block this bronchus will collapse both the lower and middle lobes.[14]

As mentioned, the bronchioles are not maintained by cartilage. They do contain smooth muscle, however, and as such can constrict (i.e. bronchoconstriction) when stimulated to do so. This mechanism of small airway constriction forms the basis of asthma.[7]

Pulmonary Vasculature

Arteries

The pulmonary arteries arise from the pulmonary trunk and transport deoxygenated blood from the right ventricle to the lungs. The right artery is longer than the left, however both pass through the hilum of their respective lung and branch into lobar, segmental and subsegmental arteries before terminating as capillaries which line the walls of the alveoli.[14]

The bronchial arteries branch from the thoracic aorta and posterior intercostal arteries to supply the lung tissue and pleura. The vessels, carrying oxygenated blood, create an anastomotic network with the pulmonary arteries and veins.[4]

Veins

The pulmonary veins originate at the lung hilum as a superior and inferior vein. These carry oxygenated blood from the lungs to the left atrium. Some bronchial veins also join with these pulmonary vessels.[4]

Lymphatic drainage

The principle route of lymphatic drainage for the body is through the thoracic duct. This extends from vertebral level L2 to the root of the neck. It begins superior to the confluence of several lymph ducts, known as the cisterna chyli, which drains the abdomen, pelvis and lower limbs. The thoracic duct enters the thorax posterior to the aorta through the diaphragm, and traverses superiorly through the posterior mediastinum to the right of the midline. It then moves to the left in the superior mediastinum, lying posterior to the oesophagus, before entering the neck where it is joined by the left jugular and subclavian trunks that drain the left head, neck and upper limb. The thoracic duct then empties into the venous circulation at the junction between the left subclavian and internal jugular veins.[4] A large palpable node in the supraclavicular area where the thoracic duct empties into the venous circulation is known as Virchow's node, and classically indicates abdominal malignancy.[7] Lymphatic drainage from the right side of the head, neck and upper limb enters circulation via the right jugular and subclavian trunks at the junction between the right subclavian and internal jugular veins.[4]

Clinical relevance

Pulmonary embolus (PE) is a common and potentially fatal condition. However, the diagnosis is often delayed due to non-specific symptoms and signs which naturally worsens prognosis.[15] As represented in Virchow's triad, predisposing factors to PE include endothelial injury, stasis or turbulent blood flow, and hypercoagulable states.[10]

Examples of aetiological factors include[16]:

Venous stasis: immobilisation, polycythaemia, dehydration
Hypercoagulability: surgery, antithrombin III deficiency, protein S & C deficiency
Surgery and trauma: particularly orthopaedic and spinal surgery
Pregnancy
Malignancy: particularly pancreatic and bronchopulmonary tumours
Acute medical illness: e.g. inflammatory bowel disease, MI, heart failure[16]

Emboli often arise from the deep veins of the lower limb (i.e. DVT), such as the popliteal and femoral veins, but not exclusively as the upper limbs and right side of the heart represent other potential sites of thrombus formation. The diameter of the pulmonary artery lumen that is occluded will determine the clinical picture, hence the variability of presentation, and this is in part related to emboli size. The embolism can be characterised as central or peripheral depending on the site of occlusion; central refers to the pulmonary trunk, pulmonary arteries and lobar arteries, whereas peripheral indicates segmental and subsegmental arteries.[17]

Occlusion of a pulmonary arterial vessel results in increased alveolar dead space with subsequent hypoxaemia, and an elevated pulmonary arterial pressure that reduces cardiac output due to increased right ventricular afterload. Common symptoms and signs which patients' may present with are described in Table 3. Note that 'small/medium' embolism refers to peripheral vessel occlusion whilst a 'massive' embolism involves occlusion of central arteries. A further subdivision is chronic PE, where patients experience dyspnoea, syncope on exertion, and weakness over weeks and months. Again, it is important to note the variability with which patients present, and not all symptoms and signs are likely to be evident.[10] The modified Wells score can be used to calculate the likelihood of PE.[18]

The diagnosis of PE is primarily made with radiological investigations, however attention should be paid to the arterial blood gas (type I respiratory failure if significant embolism), D-dimer (excludes PE if undetectable) and the ECG (may show evidence of right heart strain: e.g. right axis deviation, right bundle branch block and T wave inversion, however the classical pattern of "S1, Q3, T3" is relatively rare).[10] If a PE is suspected, a chest X-ray (CXR) should first be performed. This may show linear atelectasis or a small pleural effusion, however these are not diagnostic. However, the importance of performing the CXR is to determine which investigation to request next: if the CXR is normal, a V/Q scan should be performed but if any abnormalities are identified on the CXR, a CT pulmonary angiogram should be carried out instead.[19] However, it should be noted that less and less centres are performing V/Q perfusion scans as the availability of CT pulmonary angiograms is improving.

Table 3: common symptoms and signs found in small/medium and massive PE [10]

Small/medium PE	Massive PE
Pleuritic chest pain	Severe central chest pain
Dyspnoea	Pale and sweaty
Haemoptysis	Syncope
Tachypnoea	Tachypnoea
Localiased pleural rub	Tachycardia
Normal CVS examination	Hypotension
	Elevated JVP
	Death

Treatment of PE involves low molecular weight heparin (weight dependent dose) for at least five days, in addition to high flow O_2 and analgesia. Treatment with heparin should not be delayed until after diagnostic imaging in patients with a high suspicion of PE. In pregnancy, different treatment regimes exist, which should be consulted. If there is significant cardiovascular compromise thrombolysis with a recombinant tissue plasminogen activator, such as alteplase, should be considered (in the absence of contraindications) and urgent senior advice sought.[19] It is important to review local guidelines relating to the diagnosis and management of PE, however, to optimise the delivery of care.

Further Reading

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