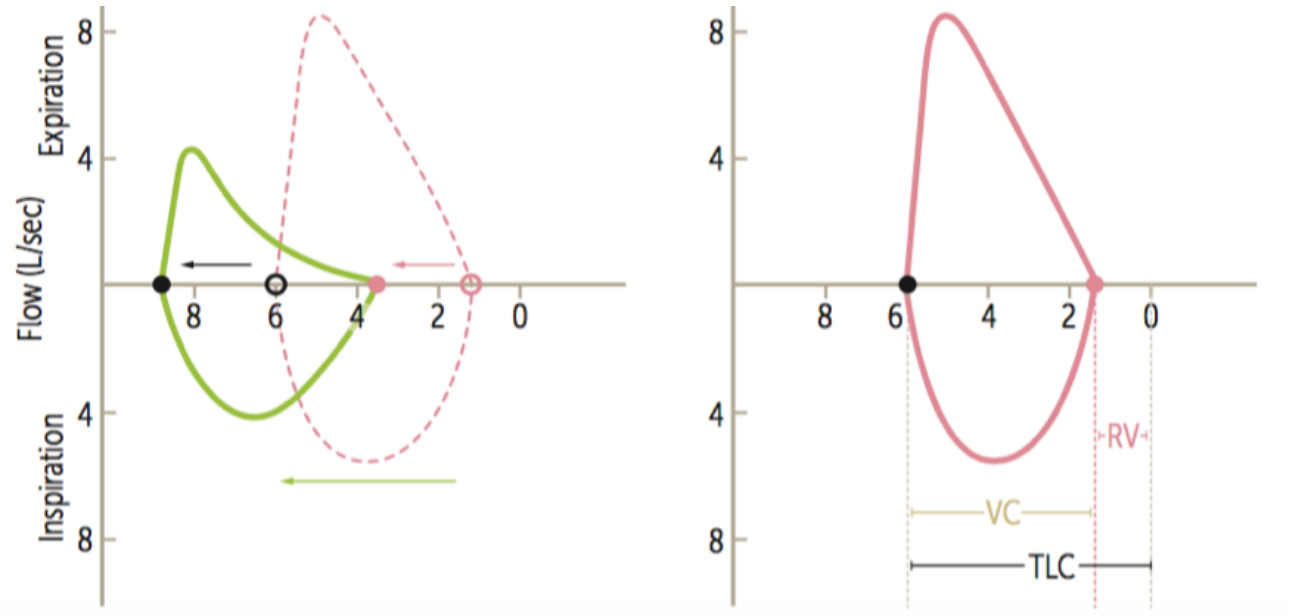
Griffith University Medicine Society
Gold Coast, Australia

Year 1 Peer Based Learning 2018
Respiratory System

Please note – this learning resource has been produced by the GUMS Academic Team. It is possible that there are some minor errors in the questions/answers, and other possible answers that are not included below. Make sure to check with other resources.

Scenario 1:

Stem 1: Toby Acco is a 70 year old male. He has a 20 pack year history of smoking. His flow volume loop is shown below; normal is shown for reference.



Question 1: Define pack year

Number of packs of cigarettes smoked per day by the number of years the person has smoked

Question 2: What is the difference between a lung 'capacity' and 'volume'

A capacity = 2 or more lung volumes totaled together

Question 3: Define the lung volumes/capacity shown on the graph at the right

VC = vital capacity = maximum volume of gas that can be expired after a maximal inspiration
RV = residual volume = air remaining in lung after a maximal expiration
TLC = sum of all volumes - volume of gas in lung after a maximal inspiration (IRV + TV + ERV + RV)

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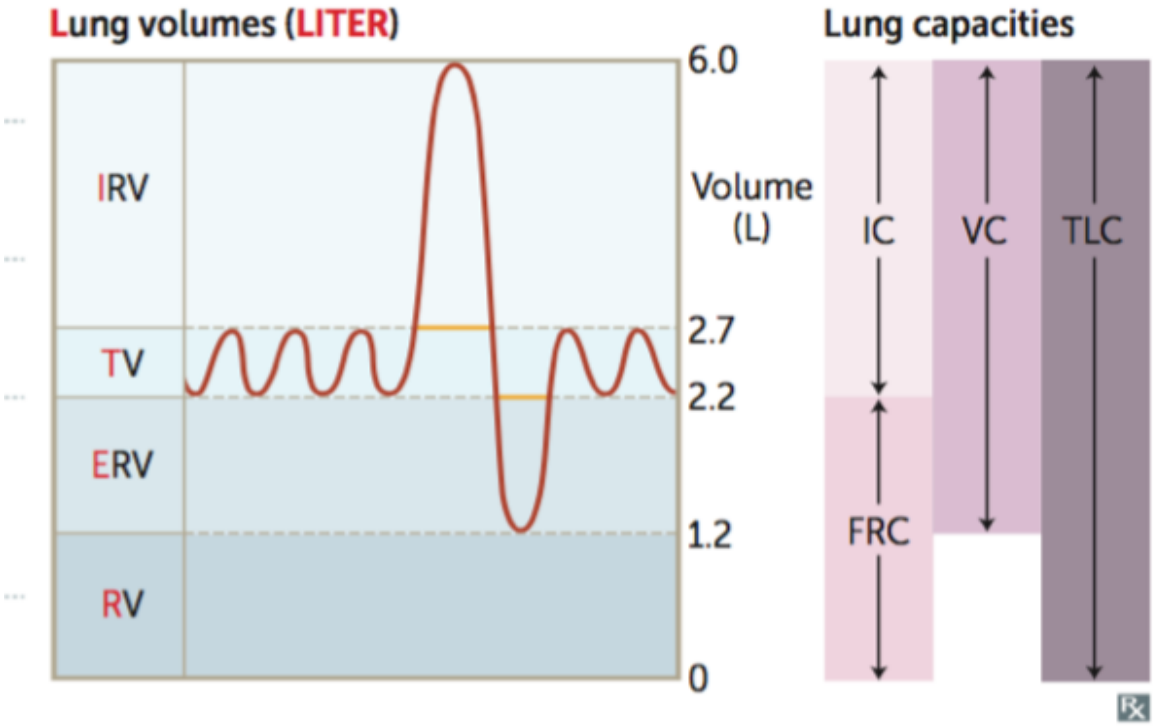

Question 4: State the pattern of lung disease Toby has and how RV and TLC from question 3 have changed. State the underlying mechanism for this.

Obstructive disease. RV and TLC have both increased (RV goes from 0 to about 4 now; TLC goes from 0 to 8). The mechanism for this is air trapping

Question 5: Summarise the main spirometry/lung volume/capacity findings in obstructive disease and restrictive disease by completing the below table:

	Obstructive Disease	Restrictive Disease
RV	increased	Decreased
TLC	Increased	Decreased
FRC	Increased	Decreased
FEV1:FVC	Decreased	Normal or increased

(discuss how this ratio is changed with the students: in obstructive FEV1 drops more than FVC —> decreases ratio; in restrictive there is low FEV1 and low to very low FVC —> normal or increased ratio)

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Question 6: What is the most likely diagnosis? Define this disease

COPD - broad term for chronic bronchitis and emphysema. Explain that even though these are two different diseases, because the major risk factor for both is smoking, they commonly occur together - this is why the blanket term 'COPD' is given

Question 7: Contrast the pathophysiology mechanism of obstruction in chronic bronchitis and emphysema

Both are caused by smoking (which acts as an irritant).

Chronic bronchitis. It results mainly from hypertrophy and hyperplasia of bronchial mucinous glands (mainly) and also goblet cells. This leads to increased mucous production leading to mucous plugging – this is an obstruction. Smoking also causes shortened and immotile cilia, which further decreases ability to clear mucus.

Emphysema. Smoking causes inflammation in the airways, leading to production of proteases via inflammatory cells such as macrophages and neutrophils. These proteases (eg, collagenases, elastases) cause destruction of elastin and collagen, which are responsible for elastic recoil of the lung. Normally, when air leaves the lungs, there is negative pressure which acts to collapse the airways, however the elastic recoil of the lungs prevents this from occurring. In emphysema, the loss of elastin leads to:

- *Collapsing airway walls on expiration (due to negative pressure) leading to air-trapping*
- *More compliant (stretchy) alveoli on inspiration, increasing lung volumes*

There is also degradation of fibrous septa in the lungs, leading to alveoli collapsing into larger alveoli, decreasing the surface area for gas exchange to occur.

Question 8: for the following signs/symptoms, state which is more characteristic of chronic bronchitis vs emphysema and explain why:

- **cyanosis** - *chronic bronchitis: mucus plugging causes a low V/Q ratio (decreased ventilation across the entire lungs) → hypercapnia and hypoxaemia. This is not as much of an issue in emphysema, because the V/Q ratio is more normal or even high (increased ventilation due to increased lung compliance, and 'decreased blood flow' due to reduced surface area with the alveoli)*
- **prolonged expiration with pursed lips** - *emphysema: breathing with pursed lips forces a 'back pressure' into the airways —> ensures smaller airways remain patent*
- **productive cough** - *chronic bronchitis: huge mucus production results in coughing; no where near as much mucus in emphysema (it does occur, just not as much)*
- **wheezing** - *chronic bronchitis: this is the sound of air ossicilating within narrowed airways - the intense mucus plugging causes this.* ***also ask students: name another disease where wheezing is seen and state the mechanism for this: answer = asthma; mechanism = bronchoconstriction AND mucus plugging***

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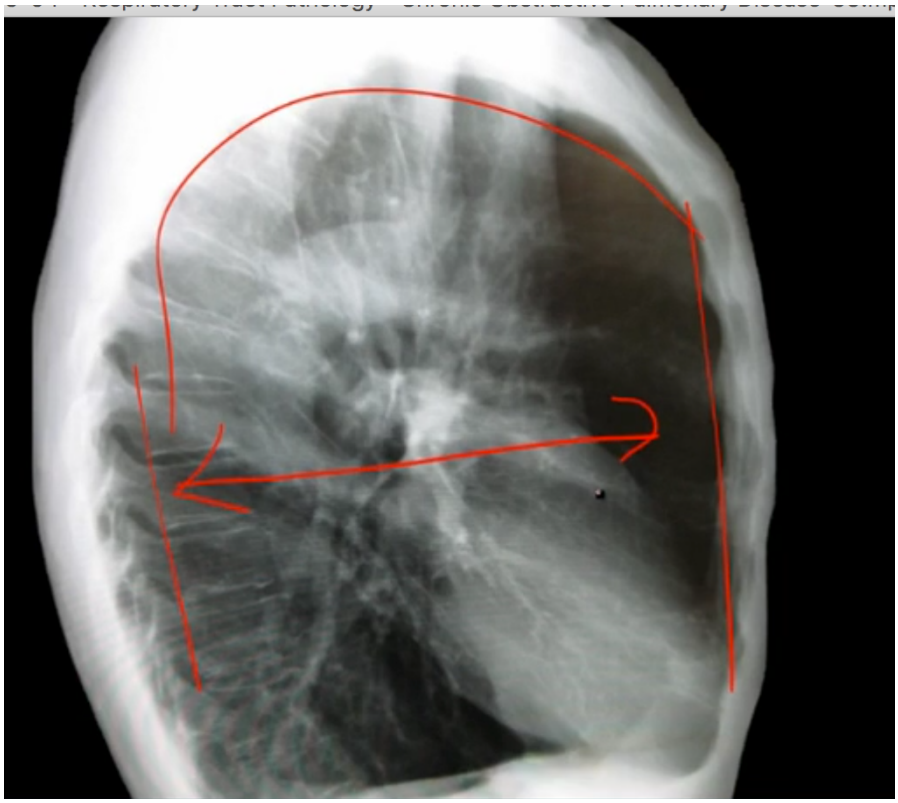
Question 9: What if a patient also presented with liver cirrhosis? Which one of the diseases (chronic bronchitis or emphysema) is more likely to also occur concurrently? State what protein is defective and the mechanism for developing the disease it causes

Alpha-1-antitrypsin deficiency. Causes emphysema. Alpha-1-antitrypsin is an anti-protease. Explain that emphysema is caused by an ***imbalance of proteases and anti-proteases.*** *In smoking, there is increased inflammation —> more proteases released —> number of anti-proteases cannot counteract this. In alpha-1-antitrypsin deficiency, even if the person doesn't smoke, the fact that there are no anti-proteases can't counteract the inflammation that will naturally occur as life goes on —> emphysema*

Question 10: Which disease is more likely to have the following x-ray? Explain



increased AP diameter = ***barrel chest = emphysema,*** *for expansion of the thorax, it is a battle between the chest wall and lung tissue. lung recoil has a tendency to collapse inwards, and the chest wall has a tendency to pull the thorax outwards. because of loss of elastic recoil, the chest wall wins this war —> barrel chest*

- ***Ask what other features might be seen on X-ray:*** *increased lung field lucency and flattened diaphragm – both due to hyperinflation*

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Q11. Which disease is more likely to cause infection as a complication? Explain

Infection is more likely in chronic bronchitis. a general principle in pathology is that if there is an obstruction, you get infection down-stream e.g. give a faecolith causing appendicitis as an example

Question 12: What is the effect of hypoxia on the pulmonary vasculature? Explain in ONE sentence the impact of long-term COPD on the heart and name this condition

Hypoxia causes vasoconstriction. *Give the example of an upper lobar bronchus obstruction. Here, it is advantageous to constrict the pulmonary vasculature, because you divert the blood to the other lobes of the lung and can still get sufficiency gas exchange. Now, nowhere is getting enough oxygen in COPD, so everywhere constricts.*

One sentence: hypoxic vasoconstriction —> increases afterload —> RVH and heart failure - ***cor pulmonale***

Question 13: You administer ipratropium. State the MOA

muscarinic antagonist —> bronchodilation

Question 14: Would you administer supplemental oxygen? Discuss

- It may lead to hypercapnic respiratory failure (Type II respiratory failure) in some COPD patients. This is because, in COPD, patients are able to optimise their gas exchange by hypoxic vasoconstriction. By administering excessive oxygen, this overcomes the hypoxic vasoconstriction, increasing blood supply to areas which are poorly ventilated, creating a V/Q mismatch (low V and high Q).

Stem 2: 10 years later, he presents with distended neck veins, ipsilateral partial ptosis, miosis and anhidrosis.

Question 15: What disease (don't need to be specific) has he most likely developed now? What lobe of the lung is most likely affected? What other structures could also be compressed here and what should you look for?
lung cancer. upper lobe = pancoast tumour = compresses sympathetic chain (e.g. stellate ganglion) and SVC or brachiocephalic veins. also should check for:

- *recurrent laryngeal nerve (hoarseness of voice)*
- *thoracic outlet syndrome: compression of lower trunk or T1 of brachial plexus plus subclavian vessels - intrinsic hand muscle wasting (e.g. thenar, hypothenar wasting -* ***get a picture from google images and show them****), ischaemia, pain and oedema in lower limb from vessel compression*

Stem 3: The patient complains of altered mental state.

Question 16: If these were caused by his lung cancer, elucidate TWO possible mechanisms for this

prompt students with: think about paraneoplastic syndromes

small cell lung cancer —> releases ADH, causing SIADH —> hyponatraemia —> altered mental state
squamous cell clung cancer —> releases PTHrP (Parathyroid hormone-related protein), causing hypercalcaemia —> altered mental state

Question 17: complete the following table, comparing the main features of lung cancers:

also explain that small cell lung cancer is its own category - the others (squamous cell, adenocarcinoma etc.) are all types of non-small cell lung cancer. small cell treated by chemo/radiotherapy; non-small treated by surgery

*give them the following mnemonic to help them remember: **s**quamous cell and **s**mall cell lung cancer are **'s'**entral, caused by **s**moking, have paraneoplastic **s**yndromes and are more common in males. the one thing that is different is treatment. then, adenocarcinoma, is mostly the opposite!*

	location in lung	risk factors	paraneoplastic syndromes	histological features	treatment
small cell lung cancer	central	male smoke	SIADH, Lambert-Eaton syndrome, ACTH	neuroendocrine tumour, poorly differentiated	chemotherapy
adenocarcinoma	peripheral	non-smokers and female smokers	nil	back to back glands, mucin	surgery
squamous cell	central	male smokers	hypercalcaemia	keratin pearls, intercellular bridges	surgery

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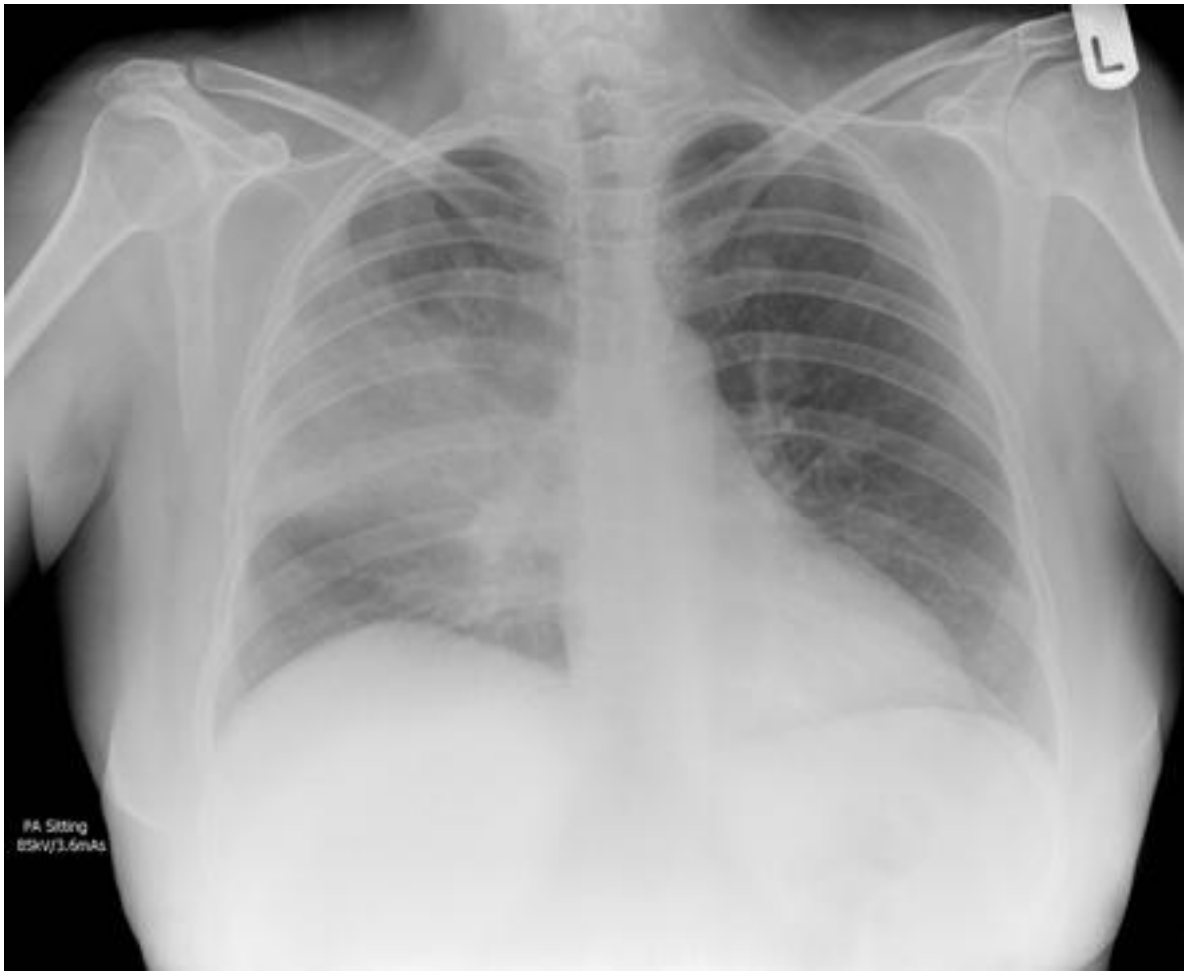
Stem 4: The patient goes onto develop a fever and the following x-ray whilst in hospital:



Question 18: State the diagnosis. What predisposed him to this infection? What is the most common causative organism? What is the treatment for this? What is this patient's prognosis?

Lobar pneumonia

COPD (esp. chronic bronchitis)

S. pneumoniae

Penicillin ***(State strep has no resistance to penicllins)***

Poor ***(state pneumonia is a common cause of death of the elderly in the hospital***

Wrap up: state other important topics for them to study: pleural effusion, pneumothorax other types of pneumonia etc.