

CME Respiratory medicine SAQs (86419): self-assessment questionnaire

Author: Laura Tanner

SAQs and answers are ONLINE for RCP fellows and collegiate members

The SAQs printed in the CME section can only be answered online to achieve external CPD credits. Any comments should be sent in via email only: clinicalmedicine@rcplondon.ac.uk

Format

SAQs follow a best of five format in line with the MRCP(UK) Part 1 exam. Candidates are asked to choose the best answer from five possible answers.

The answering process

- 1 Go to www.rcplondon.ac.uk/SAQ
- 2 Log on using your usual RCP username and password
- 3 Select the relevant CME question paper
- 4 Answer all 10 questions by selecting the best answer from the options provided
- 5 Once you have answered all the questions, click on Submit

Registering your external CPD credits

Carrying out this activity allows you to claim two external CPD credits. These will be automatically transferred to your CPD diary, where you can review the activity and claim your points.

- 1 A 25-year-old intravenous drug user was admitted to hospital with a heroin overdose. He was given naloxone in the emergency department with good effect and brought round to the acute medicine unit. On review he was found unconscious.

Aterial blood gases:

pH	7.25
pCO ₂	8.1 kPa
pO ₂	8.7 kPa
sHCO ₃	24 mmol/l
aHCO ₃	28 mmol/l
base excess (BE)	+1 mmol/l

What do these arterial blood gases show?

- (a) Respiratory acidosis with compensation
- (b) Acute respiratory acidosis

- (c) Type 2 respiratory failure
- (d) Metabolic acidosis
- (e) Metabolic acidosis with compensation

- 2 A 65-year-old lady with severe chronic obstructive pulmonary disease (COPD) was admitted to the acute medical unit. Her exercise capacity was reduced to 20 metres. She had increasing breathlessness and cough with purulent sputum. Chest radiograph showed right basal consolidation.

Aterial blood gases:

pH	7.25
pCO ₂	9.3 kPa
pO ₂	10.8 kPa
sHCO ₃	30 mmol/l
aHCO ₃	36 mmol/l
base excess (BE)	+6 mmol/l

What do these arterial blood gases show?

- (a) Chronic, compensated type 2 respiratory failure
- (b) Acute respiratory acidosis
- (c) Acute on chronic respiratory acidosis
- (d) Acute on chronic respiratory acidosis with supplemental oxygen
- (e) Metabolic acidosis with supplemental oxygen

- 3 A 30-year-old man had a history of 48 hours of vomiting. Blood tests reveal normal renal function but a potassium of 2.2 mmol/l.

Aterial blood gases:

pH	7.47
pCO ₂	6.7 kPa
pO ₂	11.0 kPa
sHCO ₃	33 mmol/l
aHCO ₃	36 mmol/l
base excess (BE)	+10 mmol/l

What do the arterial blood gases show?

- (a) Metabolic alkalosis, no lung disease
- (b) Metabolic alkalosis with impaired oxygenation
- (c) Respiratory acidosis with metabolic compensation
- (d) Metabolic acidosis
- (e) Acute respiratory acidosis

- 4 A 30-year-old male presented with recurrent respiratory infections. Sputum was repeatedly positive for *Staphylococcus aureus* and *Haemophilus influenzae*. Chest radiograph shows bronchiectasis. Genetic testing for cystic fibrosis (CF) showed a R117H mutation of the cystic fibrosis gene (class IV defect).

How does a class IV genetic defect affect the cystic fibrosis transmembrane conductance regulator (CFTR) protein?

- Cystic fibrosis transmembrane conductance regulator (CFTR) reaches the apical membrane but is not activated and is non functional
 - There is reduced synthesis of normal CFTR
 - Nonsense and frameshift mutations lead to premature termination codons resulting in a lack of protein production
 - The mutation results in misfolded CFTR that is then degraded in the endoplasmic reticulum.
 - The mutation results in reduced CFTR conductance at the cell surface
- 5 With regards to new cystic fibrosis transmembrane conductance regulator (CFTR) 'modulator' therapy which statement is true?
- They have been shown to be of benefit in all class defects
 - For the most common F508del mutation a 'potentiator' and a 'corrector' are more effective when given in combination
 - They are not of benefit in patients whose CFTR protein is non-functional
 - They are administered by inhalation
 - They are more effective in more severe phenotypes

- 6 A 75-year-old man presented with breathlessness on exertion. He smoked 40 cigarettes a day and had previously worked in the shipyards. He had a cough productive of a small amount of phlegm in the mornings and no chest pain. Chest radiograph showed a well delineated opacity.

Spirometry:

forced expiratory volume (FEV1)	1.10 l (50% predicted)
forced vital capacity (FVC)	2.10 l (89% predicted)
ratio	60% predicted
carbon monoxide transfer factor (TLCO)	56% predicted
transfer coefficient (KCO)	58% predicted

Computed tomography (CT) of the thorax showed bilateral, discrete, calcified pleural abnormalities with the diffuse pleural thickening.

How should this patient be managed?

- He should be followed up in chest clinic with serial CT every 3 months
 - He should be given smoking cessation, management of chronic obstructive pulmonary disease (COPD) and reassurance with regards to pleural plaques
 - He should be referred to the surgical team for video-assisted thoracoscopic surgery (VATS) biopsy
 - He should be advised to seek compensation from government-based scheme
 - He should be followed up with serial chest X-ray (CXR) due to 25% risk of mesothelioma
- 7 A 75-year-old man presented with gradually progressive dyspnoea and chronic cough. He is a smoker and had previously worked for 20 years in the shipyards in the 1940s where he was a pipe lagger. On examination he is clubbed with bibasal crackles.

Spirometry:

forced expiratory volume (FEV1)	2.23 (84% predicted)
forced vital capacity (FVC)	2.30 (82% predicted)
ratio	89%

Computed tomography (CT) of the thorax showed bibasal, peripheral fibrosis in a usual interstitial pneumonia pattern and pleural plaques.

What is the most likely diagnosis?

- Idiopathic pulmonary fibrosis
- Asbestosis
- Requires lung biopsy to secure diagnosis
- Lung cancer
- Sarcoidosis

- 8 A 45-year-old woman presented with a 4-day history of leg weakness and paraesthesiae. She had a recent diarrhoeal illness. During her admission she complained of breathlessness on lying flat and nursing staff report she appeared confused and drowsy in the mornings.

What is the best way to assess for respiratory muscle weakness (RMW)?

- Peak expiratory flow rate morning, afternoon and evening
- Pulse oximetry
- Arterial blood gas sampling
- Lying and standing spirometry with spontaneous nasal inspiratory pressure (SNIP), maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP)
- Monitor clinically looking for reduced chest expansion, weak cough and morning headache

- 9 In the same patient as question 8, lying and standing spirometry revealed a vital capacity of less than 1 l. She had also started to develop problems with speech, swallowing and aspiration.

What should be the next step?

- Provide non-invasive ventilation on the ward
- Involve the intensive treatment unit (ITU) for potential invasive ventilation
- Chest radiograph to exclude aspiration pneumonia
- Antibiotics for treatment of aspiration pneumonia
- Arrange further tests of respiratory muscle function, spontaneous nasal inspiratory pressure (SNIP), maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) for first thing in the morning

- 10 A 72-year-old man with idiopathic pulmonary fibrosis (IPF) presented as an emergency to the admissions unit with progressive worsening of breathlessness. He had had a diagnosis of idiopathic pulmonary fibrosis (IPF) for 2 years with steady decline of his lung function. Previous palliative care discussions have taken place and his wish

was to be at home during the end stage of his disease. He has been treated with oral prednisolone but had continued to deteriorate and required 10 l of oxygen via CPAP to maintain saturations of 90%. He is now in comatose.

What is the next step in this man's care?

- (a) Refer patient to the intensive treatment unit (ITU) for intubation and invasive ventilation
- (b) Continue continuous positive airway pressure (CPAP) indefinitely
- (c) Remove CPAP and give morphine and midazolam for symptoms
- (d) Transfer home immediately
- (e) Chest physiotherapy

CME Cardiology SAQs

Answers to the CME SAQs published in *Clinical Medicine* December 2013

Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10
(c)	(e)	(a)	(d)	(a)	(c)	(d)	(b)	(b)	(c)

Please note, the answer for Q3 should be '(a) Digoxin monotherapy would be reasonable for rate control', not beta blocker as printed.

Books

RCP history and heritage series

Founded in 1518, the Royal College of Physicians is the oldest medical college in England, and has built up its collection of books, manuscripts and portraits over almost five centuries. The history and heritage series of monographs are written by doctors and others interested in the history of medicine, and serve to illustrate significant aspects of the RCP's past and its collections.

Sir Clifford Allbutt: scholar and physician by Alexander G Bearn

Sir Clifford Allbutt was an immensely influential physician and scholar. As well as playing a prominent role within the RCP he was a founding member of the Medical Research Council and became president of the BMA.

Allbutt was not only the inventor of the short clinical thermometer, he was also responsible for the introduction of the ophthalmoscope, weighing machine and microscope to the wards. His investigations led to improved treatment of arterial disease.

This monograph is a celebration of a man who, more than 80 years after his death, remains an example of an outstanding professor of medicine and an advocate for science. ■

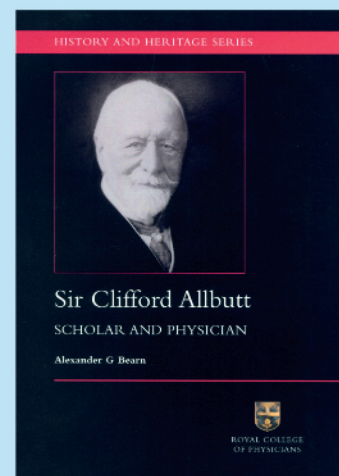
Published: March 2007 **ISBN:** 978 186016 302 9

Price: £10 UK, £12 overseas (inc post and packing)

To order: Tel: +44 (0)20 3075 1358 (8.30am – 4.30pm)

Online: <http://bookshop.rcplondon.ac.uk>

Email: publications@rcplondon.ac.uk



**Royal College
of Physicians**

10% discount for fellows and members

Quote the reference *Clinical Medicine* when making your order