Queen Mary
University of London
Barts and The London

INTEGRATED CLINICAL STUDIES TUTOR GUIDE

MBBS YEAR 3

BARTS AND THE LONDON SCHOOL OF MEDICINE AND DENTISTRY

COMMUNITY BASED MEDICAL EDUCATION DEPARTMENT (CBME)

Table of Contents

1. INTEGRATED CLINICAL STUDIES TUTOR GUIDE

The Year 3 Integrated Clinical Studies curriculum is designed to enable medical students to develop a patient centred approach to their studies. Complementary hospital and community placements provide students with opportunities to practice their clinical skills and learn about important medical and surgical conditions. Students are allocated to three systems based firms in 9 week rotations:

- Cardiorespiratory and haematology (CR3)
- Gastrointestinal, surgical and oncology (Met3A)
- Renal, endocrine, ENT and infection (Met3B)

Overview of Year 3 GP Teaching 2017-8

1. The GP3 module (formerly CR3 and MB3)

Students will attend once weekly teaching during their Met 3B term, but this teaching will incorporate both Met 3B and CR3 themes. This change reflects previous student's requests to be placed on longer attachments with the same practice, and also reflects the integrated nature of learning across systems in Primary Care. Please see module handbook for further information.

2. Change in format to MA3 teaching week to previous years.

Students based in and around London will attend two consecutive days in GP, spend one day with a District Nurse and spend one day undertaking central teaching in the medical school. Students on outer placements will spend four full days in GP with a focus on MDT working. This change has been made to highlight the increasing role of the MDT in primary care. Further information will be supplied both by email and on QMPlus website.

The community sessions are intended to integrate and supplement the clinical exposure and teaching received within the hospital setting. Therefore it is not expected that students will have achieved all of the stated year 3 learning objectives (curriculum coverage) within their general practice sessions, rather by summation of their learning experiences throughout the year.

The sessions provide students with an opportunity to practice history taking and clinical examination with patients seen in an environment which enhances the integration of general medicine and surgery. There will also be an opportunity to provide a varied experience of care in the community as students are with the practice for the whole day.

Students will attend placements with logbooks where observed history-taking, examinations and clinical skills can be signed off. In the GP3 block, they will also need to complete 2 Case-Based Discussions (To be emailed separately) in GP and these also need to be signed off. Finally in the GP3 logbook there is a new 'continuity' exercise to be completed, where the same patient is followed up on a number of occasions over the weeks of placement.

They will be assessed by you at the end of a rotation for attendance, attitude and professionalism as well as the communication skills, knowledge and clinical skills they have demonstrated.

2. CONTACT LIST FOR YEAR 3

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3. CURRICULUM COVERAGE

Overarching Learning Objectives

The overall Year 3 student learning objectives fall into two main categories:

- Clinical and communication skills

The clinical and communication skills objectives will cover all systems.
Students will have seen examination of the systems demonstrated, had some limited feedback and limited practice in these examinations from years 1 and 2 of the curriculum. They have also been assessed on some examination skills in the year 1 and 2 OSCE exams (Objective Structured Clinical Examinations). At the beginning of the third year, students have a comprehensive clinical skills course, with some feedback from tutors. Students should rarely need to be taught an examination procedure from scratch-- instead revision, feedback and repeated practice should be given priority.

By the end of the third year students should be able to:

· Obtain an accurate and comprehensive history – both for diagnosing disease and understanding the patient's experience of illness.
· Perform a full physical examination in a competent manner.

All students will also have a Year 3 Logbook pertinent to each system containing all the clinical and practical skills that they are required to either witness or be assessed as competent**. NB** It is not expected that students will cover all tasks in their logbooks during the GP placements. They will gain experiences during the hospital placements also.

- Core curriculum knowledge: (cardio-respiratory, metabolic systems and some neurology)

Core systems-based material in the third year will mainly relate to **cardio-respiratory, metabolism systems** and certain **neurological** topics such as stroke. In the relatively few days in general practice it will obviously not be possible to cover all the topics comprehensively. It is the student's responsibility to supplement their community experience with theoretical knowledge from reading-up on diseases and finding opportunities to gain appropriate hospital experiences while on their firms.

And include additional pertinent primary care topics:

- Presentation of Malignancy in Primary Care

Students will spend most of year 3 in the hospital setting where cancers are suspected or diagnosed at referral. This day is important to demonstrate how signs and symptoms of malignancy may present in the community. The more vague and undifferentiated presenting symptoms of malignant disease could be discussed. This will be a good opportunity to integrate examination of the systems and to review the 'general parts' of the examination. This day is designed to be held prior to the students' attachment at a hospice- the organisation of which is done centrally by the school.

- Palliative Care in the Community

Discussion of the support and care available for the patient and their family at the end of life will provide an introduction to a half-day visit to a hospice. Students will need an opportunity to share previous experience and perhaps the use of discussion of a suitable case, from your patient list to highlight the issues involved, would be a mechanism to achieve this. This will be an opportunity for students to understand the role of primary care health professionals, specialist nurse practitioners and voluntary support groups in the care of patients dying at home.

Students will be taking part in a “goldfish bowl” experience at the hospice together with discussing use of emetics in palliative care. They may need some support and opportunity to ask questions or raise concerns prior to this visit.

- ENT in Primary Care

Students will have some formal lectures on ENT in their fourth year, so this will be an introduction for most. It is an opportunity to obtain a flavour of basic ENT topics in a clinical setting and would lead to discussion of infections in the head and neck.

4. CONTENT OF TEACHING SESSIONS

The **content of the teaching sessions** will vary from practice to practice, and with the availability of different types of patients. However we recommend you include a balance of the following activities:

Medical interviews
Taking histories from patients either individually or in pairs; preferably observed by tutor or other students so the student gains feedback.

Explanations to patients
Practising communication skills with patients by giving explanations concerning their condition or its management. E.g. a student has looked up the drug treatment of asthma and then explains the way inhalers work to a patient.

Role plays
This is a good way for students to gain confidence and the role-player may also benefit from empathising with the patient’s perspective.

Examining patients
Either focusing on a particular system or a complete examination. Feedback from tutors and other students is important. Initial sessions may require some tutor demonstrations and assisted student examinations.

Visiting patients at home
Using visits to patients' homes to explore coping mechanisms and difficulties in activities of daily living when long-term conditions exist.

Examining other students
This can be useful to re-enforce normal examination findings and to increase student confidence before they examine patients. Care needs to be taken to ensure students are kept in same-sex groups for this and that students have a right to decline being examined if at any time they feel uncomfortable.

Practical skills
Such as urinalysis, blood pressure measurement and spirometry

Presentation skills
Listening to students presenting histories or examination findings.

Student led tutorials
Asking students to prepare a topic to present to the group for the next session.

GPs as role-models
Observation of the doctor modelling history taking or examining a patient. (However GPs should not always combine teaching with a consultation workload).

Student directed and tutor directed learning
Students will need to cover a large proportion of the curriculum through private study. While they are with you there will be many triggers for their learning. Tutors can encourage and direct this learning, by asking students to look up something specific for the next week, or recommending reading. Some useful references are given in the tutor handbook for each system –based day.

Case based discussions
These are discussions based on clinical cases. They allow the tutor to examine clinical decision-making and the application or use of medical knowledge. It also allows for discussion of the ethical and legal framework of practice. Apart from the two mandatory GP3 Case Based Discussion (where tutor notes will be emailed separately) the cases are there as a learning resource that can be used by the tutors as they see fit.

4.1 SAMPLE TIMETABLE FOR A TEACHING BLOCK

Suggested Timetable for Met3A module.

Students will spend two consecutive days in practice, then two days with District Nursing and receiving central teaching on nutrition & MDT

Please note that during their week with you all students have Wednesday afternoons off for sports, and Wednesday morning half-day for their SSC work.

	Monday	Tuesday	Wednesday	Thursday	Friday
9.00 – 11.00	FIRST STUDENT GROUP Introduction to the practice with one hour sitting-in in pairs or singly	Sit in (in pairs or singly) on surgery with GP or nurse	SSC	SECOND STUDENT GROUP to repeat activities or alternative relevant	
11.00 – 13.00	Meet the practice team. Tutorial with GP tutor (s) to set learning outcomes for the module, ground roles, contact details etc. Debrief of Case Based Discussion (CBD) case that you have asked the students to prepare for day 1.	Tutorial with GP tutor to prepare to meet patients in the afternoon	SSC		
14.00 – 16.00	Home visit to relevant patients followed by debrief at the practice with GP tutor	Take a history and examine (in pairs) a patient with relevant GI condition in practice (if possible with some supervision from GP tutor so feedback can be given)	Sports afternoon		
16.00 – 18.00	Prepare and then present history and examination of one patient they have seen on placement. Feedback from GP tutor and peers.		Sports afternoon		

GP3 Example Timetable

Please note that the longitudinal nature of the placement allows students to become increasingly independent (e.g. student-run clinics) and also gives the opportunity to experience increased MDT working. If mutually agreeable, it also means that students have increased opportunity to become involved in audit work. Students usually attend practices in groups of 4 and these activities can be done in pairs.

Week	Morning Session	Afternoon Session
1	Introduction to Practice & Team	Sitting in with GP (2 students) Home visit (2 students)
2	Tutorial/Feedback Home Visit General Nurse Clinic	Sitting in with GP (2 students) Home visit (2 students)
3	Sitting in with GP (2 students) Home visit (2 students)	Audit time
4	Tutorial/Feedback Home Visit Student-run Clinic	Specialist Nurse Clinic
5	General Nurse Clinic	Accompanying MDT members
6	Tutorial/Case Based Discussion Student–run clinic	Sitting in with GP (2 students) Home visit (2 students)
7	Sitting in with GP (2 students) Home visit (2 students)	Audit Time
8	Tutorial/Feedback Home Visit	Specialist Nurse Clinic
9	Sitting in with GP (2 students) Home visit (2 students)	Final Assessments & Sign-off

9. Barts and The London School of Medicine and Dentistry

5. LEARNING RESOURCES

Frequently Asked Questions about Case-Based discussions for Year 3 Community Based Teaching Placements

1. What is a case based discussion?

It is a discussion centred on a patient case. It allows the tutor to examine clinical decision-making and the application or use of medical knowledge. It also allows for discussion of the ethical and legal framework of practice. The formats builds upon the Year 1&2 Problem Based Learning (where students generate their own learning outcomes from a given case) and also introduces students to the format used up to and into postgraduate training.

2. Why were the case based discussions introduced?

In terms of CBD's we get excellent feedback from the students and the tutors. It enables students to discuss the MDT nature of general practice and ethics of treatment.

3. How many case based discussions do I need to do with the students?

During their GP3 block, students are required to do the Haematuria and Diabetes Case Based Discussions during their Primary Care block. These are mandatory and their logbooks will need to be signed off by yourselves to confirm they have done this. A tutor guide is with expansion notes is emailed separately at the beginning of the block- **DO NOT GIVE COPIES OF THE ANSWERS TO THE STUDENTS.**

Students will also be undertaking case-based discussion during their CR3 and MA3 terms but these are undertaken in the hospital settings, and can be found in the relevant student handbooks.

For MA3 GP week for London students, in their central teaching day on nutrition and MDT working, students will be working through some cases that have been put up in the Year 3 GP area of QMPlus. If you are teaching students based outside London you may choose to use these cases in tutorials.

The optional CR3 themed Case Based Discussions listed on the following pages have a strong overlap with those being discussed in the hospital; however they are common conditions and there would be no detrimental effect in covering the topics again in the primary care setting with a more community based focus on the discussion of the management.

GP3 Cased based discussions are currently under development and you will be alerted when they have been released. If you would be keen to be involved in their development then please contact the Year 3 GP module lead.

5.1. STUDENT CASE-BASED DISCUSSION HANDOUTS FOR CR3

Case1: Transient Ischaemic Attack

A 65 year old male infrequent attender presents with his wife to his GP surgery having had a 1 hour episode of slurred speech during lunch. There was no reported facial or limb weakness. The patient reports that the symptoms came on without warning and that though he could understand and communicate with his wife, his words did not seem to come out properly.

He is not on any regular medication but is a lifelong smoker (20 cigs a day since the age of 14) and an occasional drink in the pub on weekends.

On examination there is no noted cranial nerve deficit. His BP is 148/88 with a regular pulse of 78bpm. The rest of the neurological exam was normal.

Learning objectives:
1. Definition and clinical diagnosis of a TIA
2. Differential diagnosis and how a TIA differs from a stroke
3. Acute management of a TIA
4. Investigations, referral and secondary prevention for a TIA

Case 2: Asthma

A 19 year old girl presents to the GP surgery with a 3 month history of a persistent night time cough. This can wake her from her sleep and is non-productive. She also reports that when she climbs the stairs at her underground station she cannot easily catch her breath. This has been particularly worse over the winter. She doesn't recall any history of any breathing difficulties but does suffer from mild eczema which she treats with emollients. She is an occasional smoker on evenings out and otherwise fit and well with no known allergies.

On examination she looks well with a height of 164cm and a weight of 58kg. Her respiratory rate is 18 breaths per minute. There are no clinical signs of anaemia and no clubbing. Auscultation reveals a slight generalised wheeze with no consolidation. She has a peak expiratory flow rate of 310 L/min.

Learning objectives:
1. Recognise common presenting symptoms of asthma
2. Investigation and diagnosis of asthma
3. Taking and interpreting a peak flow reading
4. Management of asthma using the step wise approach
5. Inhaler use and technique
6. Stopping smoking, treatment options available in the community

Case 3: CR3 Anaemia

A 70 year old woman presented to her GP with a 6 months history of increasing tiredness and shortness of breath on climbing a flight of stairs. On direct questioning she had no other complaints, was not taking any medication and had a normal mixed diet. On examination the only signs elicited were pallor, a smooth tongue (atrophic glossitis) and soreness at the corners of her mouth (angular stomatitis).

Blood Test	Patient's Results	Normal Values
Haemoglobin	8.2g/dl	11.5-15.5 (f)
Mean Corpuscular Volume	67fL	80-96
White Cell Count	7.8x109/L	4.0-11.0
Platelet Count	453x109/L	150-400
Reticulocytes	2.1%	0.5-2.5
ESR	13mm in 1 hour	<20
Blood Film	Microcytic	
Serum ferritin	5 mcg/L	14-150 (f)
Serum vitamin B12	240 ng/L	160-925
Serum folate	5.5 mcg/L	3.0-15.0
Red Cell folate	270 mcg/L	160-640
Renal and Liver function	Normal	

Learning Objectives.

1. Differential Diagnosis of anaemia.

2. Relevant Investigations done by GP.

3. Diagnosis of Iron deficiency anaemia.

4. Referral and further investigations for iron deficiency anaemia, the urgency of investigating iron deficiency anaemia in the elderly.

5. Explain to patient how a colonoscopy and a gastroscopy are done.

Case 4: CR3 Shortness of Breath

A 65 year old retired London docks worker presents to his GP with an 8 week history of progressive shortness of breath on exertion of 6 months duration. Prior to this he had been able to walk his dog for more than a mile but now could only manage 100 to 200 yards before resting. He had also noticed a slight non-productive cough for the same period.

He had been a smoker of 20 cigarettes per day from the age of 18 until 2 years ago when he stopped after having suffered a myocardial infarction. There was no past history of asthma. Over the years he had suffered a few episodes of bronchitis when he had developed a cold in the winter months, but this had improved since he stopped smoking. On examination he was not breathless at rest but had bilateral finger clubbing and there was no anaemia, cyanosis or lymphadenopathy. Examination of the chest revealed dullness, reduced vocal resonance and absent breath sounds over the lower one third of the right chest. There were no signs of heart failure. Abdominal and neurological examinations were normal.

Learning Objectives.

1. Definition and clinical diagnosis of a pleural effusion.

2. Differential Diagnosis of a pleural effusion; causes of transudate versus exudate.

3. Investigations and management of for a pleural effusion.

4. Mesothelioma. Definition, cause, investigations, treatment options and prognosis.

5. Compensation regarding Mesothelioma and asbestos exposure.

6. Death Certification of patients with Mesothelioma and the need to refer to the coroner.

Case 5: CR3 Persistent Cough

A 55-year old woman presented to her GP with a history of increasing cough, shortness of breath and sputum production for the last three years. On questioning it emerged that she was producing about half a cup of sputum per day, mainly in the morning. Over the last three months she had noticed that the sputum had been greenish in colour, occasionally streaked with blood, but without frank haemoptysis. In her past medical history she suffered from whooping cough at age 3. She remembered developing colds in the winter that' went to her chest' and required several courses of antibiotics. She had smoked 25 cigarettes per day for the last 30 years. There was no past history of asthma.

On examination, her blood pressure was 150/80, pulse was 80/minute. There was no anaemia, jaundice or cyanosis. Auscultation of the chest revealed coarse crackles bilaterally at the bases with some scattered wheeze. There was no peripheral oedema. Heart sounds were normal.

Learning Objectives:

1. Differential Diagnosis of the above chest symptoms; COPD, asthma, Pulmonary TB, lung ca etc.

2. Investigations.

3. Chronic Obstructive Pulmonary Disease, definition, causes, investigations, treatment (medical and psychosocial) and prognosis; versus asthma.

4. Bronchiectasis, definition, cause, investigations, treatment and prognosis.

5. Explaining to a patient they have COPD/bronchiectasis.

6. Stopping smoking, treatment options available in the community.

Case 6: CR3 Too much swelling

A 78 year old woman presents to her GP with a 4 week history of increase shortness of breath at rest and on further questioning she also mentioned about increased bilateral leg swelling. The GP has been seeing her regularly due to a previous myocardial infarct several years ago. On examination, she has bilateral pitting oedema below the knees, pulse rate was 110/min, irregularly irregular, BP was 160/95, JVP was 6 cm above the sternal angle, apex was in the 6th ICS along the anterior axillary line and there were fine crackles in both lung bases.

The GP arranges some investigations and refers her to be reviewed by a specialist. Further medications are started and she is discharged back into the community being followed up by the community nurse specialist. Unfortunately her problem slowly gets worse. Even on optimum medical treatment she is still short of breath and unable to leave her home. She gets little sleep having to sleep downstairs in a chair.

Learning Objectives:

1. Likely diagnosis and differential Diagnosis.

2. Recognise the signs of heart failure.

3. Investigations done by the GP and Hospital.

4. Cardiac failure (LVF and bi-ventricular), definition, cause, treatment and prognosis.

5. Multidiscipline approach to care of patients.

6. Palliative stage of treatment and who and what support can be given

5.2 TUTOR GUIDE FOR CR3 CASE BASED DISCUSSIONS

CR3 CBD TUTOR GUIDE

CASE 1: TRANSIENT ISCHAEMIC ATTACK

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1 Definition and clinical diagnosis of a TIA

Definition: (Patient.co.uk)

A transient ischaemic attack (TIA) is a temporary inadequacy of the circulation in part of the brain (a cerebral or retinal deficit) that gives a clinical picture similar to a stroke except that it is transient and reversible. Hence TIA is a retrospective diagnosis. The duration is no more than 24 hours and a deficit that lasts longer than 24 hours is defined as a stroke. The majority are less than 30 minutes.

Clinical diagnosis: Is usually made on the basis of clinical history as the symptoms have often resolved. A TIA may last anything from a few minutes to 24 hours. The usual duration is about 10 to 15 minutes. Onset is over a few minutes. There may be changes in behaviour that are best described by a third party. The clinical features will depend upon the part of the brain that becomes ischaemic:

Carotid territory (80%)

Symptoms are usually unilateral and most often affect the motor area, causing unilateral weakness, affecting an arm, leg, or one side of the face. There may be dysarthria. There may be sensory symptoms in the same areas. If Broca's area is involved, there will also be difficulty with speech, called Broca's dysphasia. This produces inconsistent and unpredictable errors, usually substitution, with spontaneous speech containing fewer errors. There may be *amaurosis fugax* (fleeting loss of vision), a unilateral loss indicative of retinal ischaemia, usually associated with emboli or stenosis of the ipsilateral carotid artery.

Vertebrobasilar territory (20%)

If the ophthalmic cortex is involved there will be a homonymous hemianopia that may present purely as ignoring one side of the visual field. There may be bilateral blindness. There may be hemiparesis, hemisensory symptoms, diplopia, vertigo, vomiting, dysarthria, dysphagia, or ataxia.

Global symptoms by themselves (unsteadiness, dizziness, syncope) are rarely due to TIA.

2 Differential diagnosis and how a TIA differs from a stroke

Differential diagnosis

- Before there is full recovery it is impossible to differentiate from a stroke.
- Intracranial lesion (tumour or subdural haematoma). Beware of diagnosing TIA if there has been loss of consciousness, or convulsion.
- Todd's paralysis:
    - Follows a seizure and is characterised by a temporary, usually unilateral, paralysis.
    - It may also affect speech or vision and usually resolves within 48 hours. The cause is unknown.
- Todd's paresis (transient weakness of a hand, arm, or leg after partial seizure activity affecting that limb) is less severe and more common than Todd's paralysis.
- Syncope due to cardiac arrhythmia.
- Giant cell arteritis (temporal arteritis) has a very high ESR; there is often thickening and tenderness of the temporal artery and monocular, temporary visual impairment is a frequent presentation.
- Migraine, or migrainous aura.
- Retinal or vitreous haemorrhage.
- Focal epileptic seizure.
- Labyrinthine disorders.
- Transient global amnesia.
- Psychological disorders (including hyperventilation).
- Metabolic disturbance - e.g., hypoglycaemia

TIA vs. Stroke

TIA's are a transient and reversible process that last less that 24hours in duration – symptoms persisting beyond this timeframe change the diagnosis to a stroke.

3 Acute Management of a TIA

As per NICE guidance (2008):

Start daily aspirin (300 mg) immediately.

4 Investigations, referral and secondary prevention for a TIA

Who to refer to and when:

Patients with the following should be referred to hospital immediately and admitted for further investigation:

Two or more TIAs in a week

TIA whilst on warfarin (CT scan needed to exclude haemorrhage)

ABCD2 score more than 4 (suggests high risk of an early stroke)

Scoring System for Risk of Stroke after TIA (ABCD2 Score)

Age: Age >60	1 point
Blood pressure: BP>140 systolic and/or >90 diastolic	1 point
Clinical features: Unilateral weakness Speech disturbance without weakness Other	 2 points 1 point 0 point
Duration of symptoms >60 minutes 10-59 minutes <10 minutes	2 points 1 point 0 points
Diabetes: Presence of diabetes	1 point

Everyone else should be referred for urgent outpatient assessment. If you are unsure, then discuss this with hospital colleagues.

THE ROYAL COLLEGE OF PHYSICIANS (RCP) GUIDELINES RECOMMEND:

- All patients with a TIA should be seen by a specialist in neurovascular disease (e.g., in a specialist neurovascular clinic or an acute stroke unit).
- People with a suspected TIA should be assessed as soon as possible for their risk of subsequent stroke by using a validated scoring system such as ABCD2 (as above).

<u>High risk pts (ABCD2 score of ≥4)</u>

Patients with suspected TIA who are at high risk of stroke should receive:

- Aspirin or clopidogrel (each as a 300 mg loading dose and then 75 mg daily) and a statin started immediately.
- **NB**: clopidogrel is not licensed for the management of TIA and therefore the National Institute for Health and Care Excellence (NICE) and the British National Formulary (BNF) recommend aspirin plus modified-release dipyridamole.
- Specialist assessment and investigation within 24 hours of onset of symptoms.
- Measures for secondary cardiovascular prevention introduced as soon as the diagnosis is confirmed, including discussion of individual risk factors.

People with crescendo TIA (two or more TIAs in a week), atrial fibrillation or those on anticoagulants should be treated as being at high risk of stroke even if they may have an ABCD2 score of 3 or below.

Low risk pts (ABCD2 score of ≤3)

Patients with suspected TIA who are at low risk of stroke should receive:

- Aspirin or clopidogrel (each as a 300 mg loading dose and then 75 mg daily) and a statin.
- **NB**: clopidogrel is not licensed for the management of TIA and therefore NICE and the BNF recommend aspirin plus modified-release dipyridamole.
- Specialist assessment and investigations as soon as possible, but definitely within one week of onset of symptoms.
- Measures for secondary prevention introduced as soon as the diagnosis is confirmed, including discussion of individual risk factors.

People who have had a TIA but present late (more than one week after their last symptom has resolved) should be treated as though they are at a lower risk of stroke.

Patients with TIA in atrial fibrillation should be anticoagulated in the TIA clinic once intracranial bleeding has been excluded and if there are no other contra-indications.

Other risk factors

In addition to enquiring about the nature of the event, there are a number of other matters in the patient's history that require examination:

Has this happened before?

Has there been recent surgery, especially on the heart or carotids?

Has there been a previous stroke or any coronary heart disease (CHD)?

Is hypertension being treated?

Is there known diabetes?

Are there any other significant illnesses? There may be a hypercoagulable state or vasculitis such as temporal arteritis.

If it presents in a person much younger than age 60 ask about drug abuse, especially cocaine.

Anyone who has had a single TIA must not drive for one month. The patient must inform the insurance company. Those with multiple TIAs over a short period of time must inform the Driver and Vehicle Licensing Agency and their insurance company. They will probably be barred for three months.

CASE 2: ASTHMA

1. Recognise common presenting symptoms of asthma

As per BTS guidance (2008) the following clinical symptoms make the clinical diagnosis of asthma more likely

- More than one of the following symptoms: wheeze, breathlessness, chest tightness and cough, particularly if:

  - symptoms worse at night and in the early morning
  - symptoms in response to exercise, allergen exposure and cold air
  - symptoms after taking aspirin or beta blockers

- History of atopic disorder

- Family history of asthma and/or atopic disorder

- Widespread wheeze heard on auscultation of the chest

- Otherwise unexplained low FEV1 or PEF (historical or serial readings)

- Otherwise unexplained peripheral blood eosinophilia

2. Investigation and diagnosis of asthma

Spirometry

Two results are important: the amount of air you can blow out in one second (called forced expiratory volume in 1 second (FEV1)) and the total amount you can blow out in one breath (called forced vital capacity (FVC)). Your age, height and sex affect your lung volume. So results are compared with the average predicted for age, height and sex.

FEV1/FVC ratio: A low value indicates that narrowed airways which are typical in asthma (but a low value can occur in other conditions too). Therefore, spirometry may be repeated after treatment. An improvement in the value after treatment to open up the airways is typical of asthma.

Note: spirometry may be normal in people with asthma who do not have any symptoms when the test is done. Remember, the symptoms of asthma typically come and go. Therefore, a normal result does not rule out asthma. But, if symptoms suggest the presence of asthma, ideally the test should be repeated when your symptoms are present.

Assessment with a peak flow meter

Peak expiratory flow (PEF) measurement is recommended for:

- Diurnal variability of peak expiratory flow rate (PEFR) greater than 20% for at least three days in a week for two weeks is typical of asthma.
- Or improvement in PEF:

- 10 minutes after high-dose bronchodilator through a spacer.

- After a six-week course of inhaled steroids.

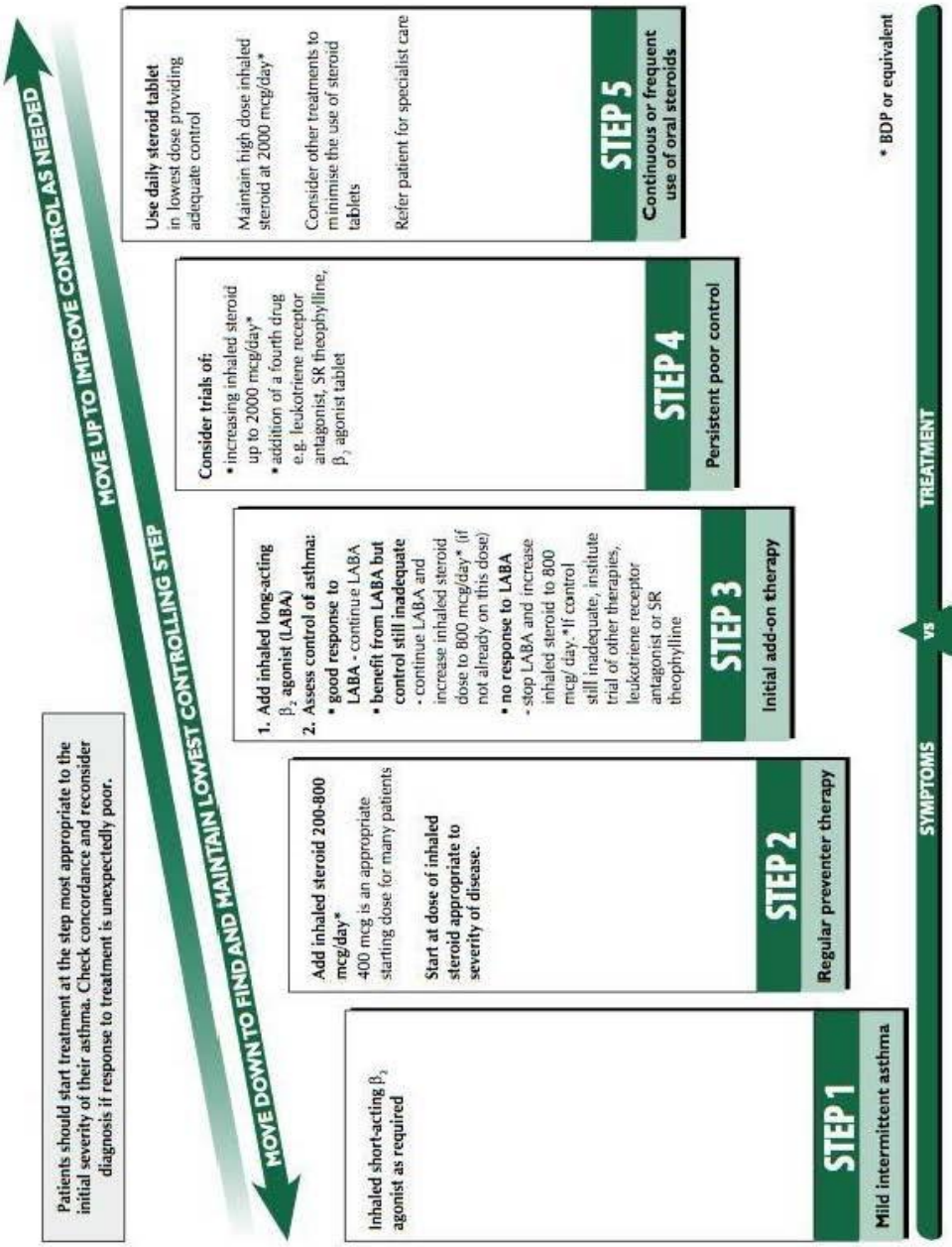
- After 14 days of 30 mg prednisolone.

3. Taking and interpreting a peak flow reading

- The patient can be standing or sitting down.
- Ensure that the marker on the scale is set to zero.
- After a full breath in, the patient should then breathe out with a rapid forced maximal expiratory puff through the mouth and into the meter.
- Repeat to give a total of three readings (maximum pause of two seconds in between) and take the best reading as the result.

Discuss how to plot a peak flow reading on a peak flow chart and its interpretation

4. Management of asthma using the step wise approach



5. Inhaler use and technique

Remove the cap

Shake the inhaler

Breathe out gently

Place mouthpiece between lips

Actuate the inhaler and breathe in slowly and deeply

Hold breath for 5-10 seconds then breathe out

Wait a few seconds then repeat the above process

Replace inhaler cap

6. Stopping smoking, treatment options available in the community

Smoking cessation

– Record a smoking history, including pack years smoked

– All people who smoke, regardless of age, should be encouraged to stop, and offered help to do so at every opportunity.

– Offer nicotine replacement therapy, varenicline or bupropion (unless contraindicated) combined with a support programme to optimise quit rates.

Discuss all the nicotine replacement therapy available and how it is used.

Look up varenicline and bupropion in the BNF to discuss contraindication, side effects and how they are used.

Discuss with the students, NHS Stop smoking quitline, and support groups that are available locally.

CASE 3: CR3 ANAEMIA

1. Differential Diagnosis of anaemia.

Anaemia can be divided in to three main categories: **normocytic, macrocytic and microcytic.**

Normocytic:

Normocytic anaemia occurs when the overall haemoglobin levels are always decreased, but the red blood cell size (Mean corpuscular volume) remains normal. Causes include:

- Acute blood loss
- Anaemia of chronic disease
- Aplastic anaemia (bone marrow failure)
- Haemolytic anaemia

Macrocytic: The MCV is raised.

- Megaloblastic anaemia, the most common cause of macrocytic anaemia, is due to a deficiency of either vitamin B12, folic acid (or both). Deficiency in folate and/or vitamin B12 can be due either to inadequate intake or insufficient absorption. Folate deficiency normally does not produce neurological symptoms, while B12 deficiency does.

- Pernicious anaemia is caused by a lack of intrinsic factor. Intrinsic factor is required to absorb vitamin B12 from food. A lack of intrinsic factor may arise from an autoimmune condition targeting the parietal cells (atrophic gastritis) that produce intrinsic factor or against intrinsic factor itself. These lead to poor absorption of vitamin B12.

- Macrocytic anaemia can also be caused by removal of the functional portion of the stomach, such as during gastric bypass surgery, leading to reduced vitamin B12/folate absorption. Therefore one must always be aware of anaemia following this procedure.

- Hypothyroidism

- Alcoholism commonly causes a macrocytosis, although not specifically anaemia. Other types of Liver Disease can also cause macrocytosis.

- Methotrexate, zidovudine, and other drugs that inhibit DNA replication.

Microcytic: The MCV is low.

Microcytic anaemia is primarily a result of haemoglobin synthesis failure/insufficiency, which could be caused by several aetiologies:

- Haem synthesis defect
- Iron deficiency anaemia
- Anaemia of chronic disease (more commonly presenting as normocytic anaemia)
- Globin synthesis defect
- Alpha-, and beta-thalassaemia

2. Relevant Investigations done by GP.

As specified in scenario

3. Diagnosis of Iron deficiency anaemia.

Iron studies in further blood tests. The blood smear of a patient with iron deficiency shows many hypochromic (pale and relatively colourless) and rather small RBCs, and may also show poikilocytosis (variation in shape) and anisocytosis (variation in size). With more severe iron deficiency anaemia the peripheral blood smear may show target cells, hypochromic pencil-shaped cells, and occasionally small numbers of nucleated red blood cells.

What are the causes of iron deficiency anaemia?

A normal balanced diet will usually contain enough iron for the body's needs. A low level of body iron leading to anaemia can result from various causes. Some are more serious than others, and include the following:

- Heavy menstrual periods

Anaemia is common in women of all ages who have heavy periods. About 1 in 10 women will become anaemic at some stage due to heavy periods.

- Pregnancy

A growing baby needs iron and will take it from the mother. Anaemia is common in pregnant women.

- Poor absorption of iron

Some conditions of the intestine lead to poor absorption of various nutrients, including iron. Coeliac

disease is an example.

- Bleeding from the intestine

Several conditions of the gut can lead to bleeding into the gut. Sometimes this is sudden - for example, after a burst duodenal ulcer. Vomiting or passing blood is then obvious. However, often the bleeding is not obvious. A constant trickle of blood into the gut can be passed unnoticed in the stools (faeces). Conditions causing this include: stomach or duodenal ulcers, colitis, inflammation of the oesophagus, piles (haemorrhoids), cancers of the bowel, and other rare bowel disorders. The patient may have other gut symptoms such as stomach pains, constipation, or diarrhoea. However, in the early stages of these conditions, the patient may not have any symptoms, and anaemia may be the first thing that is noticed. For example, iron deficiency anaemia in an older person may be the first indication that bowel cancer has developed.

- Medication

Some medicines can sometimes cause bleeding into the gut without causing symptoms. The most common example is aspirin. Other anti-inflammatory painkillers such as ibuprofen, naproxen, and diclofenac may also have this side-effect in some people.

- Bleeding from the kidney

A small but regular trickle of blood from various diseases of the kidney or bladder may not be noticed in the urine. However, enough may be lost to cause anaemia.

- Dietary factors

Not eating foods with enough iron is sometimes the cause of iron deficiency anaemia. A restricted diet such as a vegan or a limited vegetarian diet sometimes does not contain enough iron. Traditional diets in some parts of the world contain a high level of chemicals such as phytates and polyphenols. For example, certain types of unleavened breads (such as chapatis) may contain a high level of phytates, and tea can contain a high level of polyphenols. These chemicals interfere with the way iron is absorbed from the gut. So, it can lead to iron deficiency. For example, in parts of India where chapatis are a staple food, iron deficiency anaemia is common.

- Hookworm infection

This gut infection is the most common cause of iron deficiency anaemia worldwide. It affects people living in, and visiting, certain tropical countries. The worm feeds off blood inside the gut.

4. Referral and further investigations for iron deficiency anaemia, the urgency of investigating iron deficiency anaemia in the elderly.

It is important to find the cause of the iron deficiency. The cause may be obvious in some people. For example, anaemia is common in pregnancy, and in women with heavy periods. In these situations, if the patient is otherwise well and have no other symptoms, then no further tests may be needed. However, further tests may be advised if the cause is not clear.

Tests that may be advised include one or more of the following:

Tests to look into the gut to see if there is any internal bleeding. These may be advised even if the patient does not have gut symptoms, especially in older people. The tests may include gastroendoscopy. Checking the rectum and bowel may also be advised. This is

- commonly done by sigmoidoscopy or colonoscopy. It is sometimes done by having a CT scan of the abdomen.

- A specialised blood test for coeliac disease. Sometimes a biopsy of the gut may also be taken to diagnose coeliac disease.

- If the patient has recently been to the tropics, a stool sample may be checked to rule out hookworm.

- Other tests may be advised if the cause is still unclear.

5. Explain to patient how a colonoscopy and a gastroscopy are done.

www.patient.co.uk has excellent leaflets on both procedures to discuss with the students.

Take home message: shortness of breath not always due to a chest or cardiac problems.

CASE 4: CR3 SHORTNESS OF BREATH

1. Definition and clinical diagnosis of a pleural effusion.

Pleural effusion is excess fluid that accumulates in the pleura, the fluid-filled space that surrounds the lungs. Excessive amounts of such fluid can impair breathing by limiting the expansion of the lungs during respiration.

Four types of fluids can accumulate in the pleural space:

• Serous fluid (hydrothorax)

• Blood (haemothorax)

• Chyle (chylothorax)

• Pus (pyothorax or empyema)

2. Differential Diagnosis of a pleural effusion; causes of transudate versus exudate.

Definitions of the terms "transudate" and "exudate" are the source of much confusion. Briefly, transudate is produced through pressure filtration without capillary injury while exudate is "inflammatory fluid" leaking between cells.

The most common causes of transudative pleural effusions in the UK are left ventricular failure, and cirrhosis (causing hepatic hydrothorax), nephrotic syndrome leading to increased loss of albumin and resultant hypoalbuminemia and thus reducing colloid osmotic pressure is another less

common cause. Pulmonary embolisms were once thought to be transudative but have been recently shown to be exudative.

The most common causes of exudative pleural effusions are bacterial pneumonia, cancer (with lung

cancer, breast cancer, and lymphoma causing approximately 75% of all malignant pleural effusions), viral infection, and pulmonary embolism

3. Investigations and management of a pleural effusion.

Once a pleural effusion is diagnosed, the cause must be determined. Pleural fluid is drawn out of the pleural space in a process called thoracentesis. A needle is inserted through the back of the chest wall in the sixth, seventh, or eighth intercostal space on the midaxillary line, into the pleural space. The fluid may then be evaluated for the following:

1. Chemical composition including protein, lactate dehydrogenase (LDH), albumin, amylase, pH, and glucose

2. Gram stain and culture to identify possible bacterial infections

3. Cell count and differential

4. Cytopathology to identify cancer cells, but may also identify some infective organisms

5. Other tests as suggested by the clinical situation – lipids, fungal culture, viral culture, specific immunoglobulins

4. Mesothelioma. Definition, cause, investigations, treatment options and prognosis.

Malignant mesothelioma is a tumour of mesothelial cells which usually occurs in the pleura (80-90% of all cases), but also other sites, including the peritoneum and pericardium. Patients suffering from asbestos-related mesothelioma may be entitled to compensation.

Epidemiology from www.patient.co.uk.

• It is three times more common in men than women. Often presents between the ages of 40 and 70 years.

• Because of the widespread use of asbestos until the end of the 1970's the incidence of pleural mesothelioma has risen for some decades and is expected to peak between 2010 and 2020.

• The lag period between initial exposure and death varies but one study found a median latency of 32 years, with 96% of cases occurring after at least 20 years.

• The annual number of deaths from mesothelioma had risen from 153 in 1968 to 1848 in 2001, and it is predicted to peak at 1950-2450 cases annually between 2011 and 2015.

Clinical features - Malignant mesothelioma should be considered in any patient with a pleural effusion or pleural thickening, especially if a history of asbestos exposure and chest pain is present.

• Chest pain (typically dull, diffuse, progressive and occasionally pleuritic)

• Breathlessness may be caused by a pleural effusion or circumferential pleural thickening.

• Patients may also present with a palpable chest wall mass

• Weight loss, fatigue, fever, sweats; finger clubbing is usually caused by underlying asbestosis

• Pericardial effusion may be caused by local extension in progressive disease. Ascites caused by peritoneal mesothelioma and secondary hydropneumothorax are uncommon but recognised presentations.

• If the tumour has metastasised there may be lymphadenopathy, hepatomegaly, bone pain, bone tenderness, abdominal pain, gastrointestinal obstruction (peritoneal malignant mesothelioma).

Investigations

• Chest x-ray and CT scan: may show a pleural effusion, lobulated or nodular pleural thickening, a pleural mass, rib destruction; other features of exposure to asbestos may also be present.

• Pleural fluid: straw coloured or blood stained. Cytological analysis occasionally leads to the diagnosis but a pleural biopsy is usually required.

• Pleural biopsy: ultrasound or computed tomography- guided percutaneous biopsy.

• Thoracoscopy under local anaesthetic (enabling drainage of pleural fluid, pleural biopsy, and pleurodesis) is becoming increasingly available.

Associated diseases

Inhalation of asbestos fibres can lead to benign pleural disease (pleural plaques, diffuse pleural thickening, atelectasis) parenchymal lung disease (asbestosis) and malignant chest disease (mesothelioma, lung cancer).

Management

• Symptomatic, as cure is only possible with surgery for extremely localised (stage 1) mesothelioma.

Traditional treatment modalities (surgery, radiotherapy, and chemotherapy) have evolved slowly, and there has been little improvement in establishing effective treatments. Neither radiotherapy nor chemotherapy currently improves survival.

Prognosis

• This is difficult to assess because of considerable variation in 'time to diagnosis'.

• Depends on the patient's age, staging information, histology and general 'performance status' at diagnosis, but is generally very poor.

• Median survival is 8-14 months from the time of diagnosis. It is almost always fatal.

5. Compensation regarding Mesothelioma and asbestos exposure.

If the mesothelioma could be due to exposure to asbestos through the patient's work, they may be able to claim compensation.

Claiming compensation can be done in two ways: through benefits paid by the government and/or by suing your employer for the period (or periods) during which you were exposed to asbestos. Your specialist nurse, local Citizens Advice Bureau, a local Benefits Adviser or an Asbestos Diseases Support Group can tell you who to contact and help you through the benefits system.

Further help and information Mesothelioma UK Freephone: 0800 169 2409
www.mesothelioma.uk.com

Mesothelioma UK provides impartial up-to-date information for patients diagnosed with mesothelioma and for their carers.

ADUK (Asbestos Diseases UK) Tel: 0115 927 5108 www.aduk.org.uk

ADUK was set up by those affected by asbestos exposure. It provides help and support to those affected by asbestos exposure.

6. Death Certification of patients with Mesothelioma and the need to refer to the coroner.

Show students a death certificate and in particular the conditions that must be referred to a coroner.

CASE 5: CR3 PERSISTENT COUGH

1. Differential Diagnosis of the above chest symptoms:

• COPD

• Asthma

• CCF

• Pulmonary TB

• Lung ca

• Bronchiectasis

2. Investigations:

• Chest x-ray.

• Sputum Sample.

• Lung function tests.

3. Chronic Obstructive Pulmonary Disease, definition, causes, investigations, treatment (medical and psychosocial) and prognosis; versus asthma.

Chronic obstructive pulmonary disease (COPD) is a general term which includes the conditions chronic bronchitis and emphysema. COPD is the preferred term, but you may still hear it called chronic obstructive airways disease (COAD).

• Chronic means persistent.

• Bronchitis is inflammation of the bronchi (the airways of the lungs).

• Emphysema is damage to the smaller airways and air sacs (alveoli) of the lungs.

• Pulmonary means 'affecting the lungs'.

The term COPD is used to describe airflow obstruction due to chronic bronchitis, emphysema, or both.

What causes chronic obstructive pulmonary disease (www.patient.co.uk)?

Smoking is the cause in the vast majority of cases. There is no doubt about this. The lining of the airways becomes inflamed and damaged by smoking. About 3 in 20 people who smoke one packet of cigarettes (20 cigarettes) per day, and 1 in 4 40-per-day smokers, develop COPD if they continue to smoke. For all smokers, the chances of developing COPD is between 1 in 10 and 1 in 4.

Air pollution and polluted work conditions may cause some cases of COPD, or make the disease worse. The combination effect of occupational exposure to air pollutants and smoking increases the chances of developing COPD.

A small number of people have a genetic (hereditary) risk of COPD due to very rare protein deficiencies that can lead to lung, liver and blood disorders. (The condition is called alpha-1-antitrypsin deficiency). Less than 1 in 100 cases of COPD are due to this.

However, people who have never smoked rarely develop COPD. (Passive smoking remains, however, a potential cause.

Spirometry

The most common test used in helping to diagnose the condition is called spirometry. This test estimates lung volumes by measuring how much air the patient can blow out into a machine. Two results are important: the amount of air that the patient can blow out in one second (called forced

expiratory volume in 1 second - FEV1) and the total amount the patient can blow out in one breath (called forced vital capacity - FVC). The patient's age, height and sex affect their lung volumes. So, the patient's results are compared to the average predicted for your age, height and sex.

A value is calculated from the amount of air that the patient can blow out in one second divided by the total amount of air that the patient can blow out in one breath (called FEV1/FVC ratio). A low value indicates that the patient has narrowed airways. The FEV1 compared with the predicted value shows how bad the COPD is.

COPD is divided into mild, moderate and severe groups, depending on the level of airflow obstruction. The airflow obstruction is the FEV1, measured with spirometry.

• Mild (stage 1) COPD is an FEV1 at least 80% of predicted value.

• Moderate (stage 2) COPD is an FEV1 between 50% and 79% of predicted value.

• Severe (stage 3) COPD is an FEV1 between 30% and 49% of predicted value.

• Very severe (stage 4) COPD is an FEV1 less than 30% of predicted value.

Other tests

A chest X-ray may show signs of COPD and can be used to help exclude other serious conditions (including lung cancer). Occasionally, a special CT scan of the chest - high-resolution CT (HRCT) - is needed. A blood test to make sure the patient is not anaemic is often helpful. (Anaemia can lead to breathlessness.) Sometimes a blood test can show changes (called polycythaemia) that suggest you have chronically low levels of oxygen (hypoxia).

NICE Guidelines of Treatment:

In people with stable COPD who remain breathless or have exacerbations despite use of short-acting bronchodilators as required, offer the following as maintenance therapy:

– if forced expiratory volume in 1 second (FEV1) ≥ 50% predicted: either long-acting beta2 agonist (LABA) or long-acting muscarinic antagonist (LAMA)

– if FEV1 < 50% predicted: either LABA with an inhaled corticosteroid (ICS) in a combination inhaler, or LAMA

– offer LAMA in addition to LABA + ICS to people with COPD who remain breathless or have exacerbations despite taking LABA + ICS, irrespective of their FEV1.

Also discussion about home oxygen, mucolytic therapy, use of oral steroids and antibiotics. Also pulmonary rehab courses.

What's the difference between chronic obstructive pulmonary disease and asthma?

Asthma and COPD cause similar symptoms. However, they are different diseases. Briefly:

• In COPD there is permanent damage to the airways.

• In asthma there is inflammation in the airways which makes the muscles in the airways constrict. This causes the airways to narrow. The symptoms tend to come and go, and vary in severity from time to time. Treatment to reduce inflammation and to open up the airways usually works well.

• COPD is more likely than asthma to cause a chronic (ongoing) cough with phlegm.

• Night time waking with breathlessness or wheeze is common in asthma and uncommon in COPD.

• COPD is rare before the age of 35 whilst asthma is common in under-35s.

• There is more likely to be a history of asthma, allergies, eczema and hayfever (so-called atopy) in people with asthma.

Both asthma and COPD are common, and some people have both conditions.

What is the progression and outlook (www.patient.co.uk)?

Symptoms of COPD typically begin in people aged over 40 who have smoked for 20 years or more. A 'smoker's cough' tends to develop at first. Once symptoms start, if the patient continues to smoke, there is usually a gradual decline over several years. The patient tends to become more and more breathless. In time the patient's mobility and general quality of life may become poor due to increasing breathing difficulties.

Chest infections tend to become more frequent as time goes by. Flare-ups of symptoms (exacerbations) occur from time to time, typically during a chest infection.

If the condition becomes severe then heart failure may develop.

Respiratory failure is the final stage of COPD. People with end-stage COPD need palliative care to make them more comfortable and ease any symptoms.

At least 25,000 people die each year in the UK from the end stages of COPD. Many of these people have several years of ill health and poor quality of life before they die. About 8 in 10 men with mild COPD will survive for five years or more after diagnosis, compared with 7 in 10 women. The survival rate is lower in severe COPD. About 3 in 10 men and just over 2 in 10 women with severe disease will survive five years from diagnosis.

Depression and/or anxiety affect at least 6 in 10 people with COPD, and can be treated if recognised.

4. Bronchiectasis, definition, cause, investigations, treatment and prognosis.

Bronchiectasis is an abnormal widening of one or more airways. Extra mucus is made in the abnormal airways which is prone to infection. The main symptom is a cough which produces a lot of sputum. Treatment often includes regular physiotherapy and courses of antibiotics. Long-term antibiotic treatment is needed in some cases. Inhalers are sometimes used. Surgery is occasionally needed. The patient should not smoke as smoking can make things worse. Immunisation against flu and pneumococcus are advised.

What causes bronchiectasis (www.patient.co.uk)?

The cause is often not clear and no cause can be found in over half of cases. An underlying cause is found in about 4 in 10 cases. Some conditions that affect or damage airways can cause bronchiectasis. Examples include the following:

• Severe lung infections such as tuberculosis (TB), whooping cough, pneumonia or measles, which can damage the airways at the time of infection.

• Deficiencies of the immune system.

• Some inherited conditions. For example, a condition called primary ciliary dyskinesia affects the cilia so they do not beat correctly to clear the mucus. Cystic fibrosis is another condition that affects the lungs and causes bronchiectatic airways. Some rare immune problems can cause lung infections and damage to airways.

• Inhaled objects, such as peanuts, can become stuck and block an airway.

• Some diseases that cause inflammation in other parts of the body can occasionally cause inflammation and damage in the bronchi and lead to bronchiectasis. For example: ulcerative colitis, Crohn's disease, coeliac disease, rheumatoid arthritis, systemic lupus erythematosus.

How is bronchiectasis diagnosed?

If your symptoms suggest bronchiectasis then the diagnosis can be confirmed by a CT scan. Widened bronchi seen on a CT scan confirm bronchiectasis. Various other tests may be advised if an underlying cause is suspected.

What is the outlook (prognosis)?

Most people with bronchiectasis (with no underlying cause) have a good outlook. The condition becomes worse in some cases and breathing problems may develop. In a small number of cases the condition becomes gradually worse over time as more and more of the airways become affected.

A life-threatening bleed from a damaged airway may also occur but is rare.

The outlook for people where bronchiectasis is part of another condition depends on the underlying cause.

5. Explaining to a patient they have COPD/bronchiectasis.

Importance of using language the patient can understand and finding out what the patient knows about the disease already.

6. Stopping smoking, treatment options available in the community.

Smoking cessation

– Record a smoking history, including pack years smoked, for everyone with COPD.

– All people with COPD who still smoke, regardless of age, should be encouraged to stop, and offered help to do so at every opportunity.

– Offer nicotine replacement therapy, varenicline or bupropion (unless contraindicated) combined with a support programme to optimise quit rates.

Discuss all the nicotine replacement therapy available and how it is used.

Look up varenicline and bupropion in the BNF to discuss contraindication, side effects and how they are used.

Discuss with the students, NHS Stop smoking quitline, and support groups that are available locally.

CASE 6: CR3 TOO MUCH SWELLING

1. Likely diagnosis and differential diagnosis.

Likely diagnosis is congestive heart failure.

Differentials:

• COPD/asthma.

• Pneumonia.

• Pleural Effusion.

• Anaemia.

• Pulmonary embolism.

• Atrial Fibrillation.

2. Recognise the signs of heart failure.

What are the symptoms of heart failure?

The symptoms that may develop depend upon the type of heart failure the patient has.

Generally, left heart failure causes shortness of breath (breathlessness). The main symptom of right heart failure is swollen ankles and legs. This is due to the build up of excess fluid in the patient's legs. The liver may also become enlarged.

Other symptoms of heart failure (on either side of the heart) can include:

• Tiredness

• Dizziness

• Feeling sick (nausea)

• Constipation

• Loss of appetite

3. Investigations done by the GP and Hospital.

GP tests may include bloods FBC, U+E, TFT, LFT and fasting glucose and cholesterol. They may also arrange a chest x-ray and ECG. The blood test B-type
natriuretic peptide (BNP) is a specific test indicative of heart failure.

Hospital main investigation is an ECHO.

4. Cardiac failure (LVF and bi-ventricular), definition, cause, treatment and prognosis.

What causes heart failure?

Heart failure is not an exact diagnosis. Heart failure is a general umbrella term and may develop as a complication of various conditions. Conditions that causes heart failure affect the ability of the heart to function well as a pump. Heart failure may affect only the right ventricle (right-sided heart failure) or the left ventricle (left-sided heart failure), or both. Conditions that may cause heart failure include the following:

• Ischaemic heart disease (IHD) is the most common cause - in particular, heart failure may develop after a myocardial infarction.

• Diseases of the heart muscle (cardiomyopathy).

• High blood pressure.

• Diseases of the heart valves.

• Diseases of the pericardium.

• Some types of abnormal heart rhythms (arrhythmias).

• Drugs or chemicals that may damage the heart muscle. For example, alcohol excess, cocaine and some types of chemotherapy.

• Various non-heart conditions that can affect the function of the heart. For example, severe anaemia, thyroid disease and Paget's disease.

Sometimes the cause of heart failure is not known.

The following drugs are commonly used to treat heart failure. They will be tailored to the individual person, depending on the cause and severity of the heart failure.

• Angiotensin-converting enzyme (ACE) inhibitors

These drugs prevent a build-up of fluid by interfering with the enzyme angiotensin. ACE inhibitors also have a protective effect on the heart, and may slow down the progression of heart failure.

Drugs in the class of drugs called angiotensin-II receptor antagonists work in a similar way to ACE inhibitors. One may be used instead of an ACE inhibitor if you have problems or side-effects with taking an ACE inhibitor (such as a persistent cough).

• Betablockers

A betablocker drug such as bisoprolol or carvedilol is usually prescribed in addition to an ACE inhibitor. Like ACE inhibitors, betablockers have a protective effect on the heart.

Research studies have shown that ACE inhibitors and betablockers not only help to ease symptoms, but can improve the outlook and extend life expectancy for people with heart failure.

• Diuretics ('water tablets')

A diuretic is commonly needed to ease fluid retention.

Other treatments

As mentioned above, heart failure is not an exact diagnosis, but develops as a complication of various conditions. Other treatments for the underlying condition may be advised in certain cases. For example:

• Treatment to lower blood pressure.

• Treatments to slow down the progression of ischaemic heart disease if this is the cause of the heart failure. For example, lowering a high cholesterol level.

• Surgery to replace or fix a heart valve may be done if a damaged heart valve is the cause of the heart failure.

• A heart transplant is an option in some cases. What is the outlook (prognosis)?

It is difficult to give an outlook (prognosis) for an individual. In general, the more severe the heart failure, the worse the outlook. In many cases, the symptoms remain at a stable level for quite some time (months or years) before becoming worse. In some cases the severity and symptoms become gradually worse over time.

5. Multidiscipline approach to care of patients.

Importance of team work between patients, cardiac failure nurses, GPs and hospital consultants. District nurses and social services may also be useful.

6. Palliative stage of treatment and who and what support can be given.

Heart failure in some cases may be terminal. As the treatments failure to work the patient usually becomes less mobile, has increased swelling of legs and increased breathlessness. These symptoms can be very upsetting for the patient and family. Importance of Cardiac failure nurses, district nurses, GPs, social services and palliative care nurses.

5.3. STUDENT CASE-BASED DISCUSSION HANDOUTS FOR MET3A

THESE CASE BASED DISCUSSIONS WILL NOW BE CONVERED IN CENTRAL TEACHING AT THE MEDICAL SCHOOL BUT ARE INCLUDED HERE FOR YOUR INFORMATION.

1 Painless Leg Ulcer

An 85 year old lady who is the main carer for her husband, (who has dementia), comes to your GP surgery. She presents with an area of skin that has 'broken down', exposing the underlying flesh just above her ankle. It does not hurt. On further examination of her legs she has chronic changes of venous insufficiency.

After explaining she has a venous leg ulcer you refer her to the district nurses for treatment. The district nurse took an ABPI measurement and started compression bandaging. A few months later you are asked by a neighbour to visit her as he is worried she seems very low. On visiting you discover she is still having compression bandage treatment which she has found very uncomfortable. She has stopped going out due to the smell of the bandages even when they are changed twice a week. She feels guilty as her husband has been taken into respite as she has been unable to look after him.

Learning Objectives.

1. Describe the different types of leg ulcers.

2. What causes leg ulcers.

3. What are chronic changes of venous insufficiency.

4. ABPI measurement.

5. Compression bandaging treatment.

6. Discuss the psychosocial aspects of having an ulcer.

7. How can venous ulcers be prevented.

8. How can we support patients with venous ulcers.

2 Hepatitis

A 23 year old married man presented to his GP having returned early from his travels in the Far East. He was someone who enjoyed the natural things of life like fresh salads, but who eschewed the unnatural – like immunisations. He avoided alcohol but occasionally used recreational drugs. He had been feeling off colour with slight nausea and anorexia for ten days before becoming jaundiced. There had been no abdominal pain. His urine had become dark and contained bilirubin and urobilinogen on testing.

On examination he was jaundiced but there were no stigmata of chronic liver disease. The liver was just palpable below the right costal margin and slightly tender but there was no splenomegaly. Blood tests revealed ALT 1100 U/l, (5-40) and alkaline phosphatase 200 U/l (25 – 100). The patient was concerned about transmission of hepatitis to his sexual partner and the possibility of progression to chronic liver disease.

Learning Objectives:

1. Discuss the differential diagnosis of acute painless jaundice and give the diagnosis here.

2. Consider the various risk factors for hepatitis presented.

3. Compare the biochemical findings in liver function tests between obstructive and hepatic/hepatocellular jaundice.

4. Consider virological tests for viral hepatitis and describe their significance.

5. Consider long term sequelae of viral hepatitis and potential needs for counselling.

6. Ethics around the risk of transmission to his sexual partner.

7. Discuss Immunisations available.

3 Palliative Care - Out of Hours

While you are observing an out-of-hours GP session, a lady phones up the service. She is very distressed about her husband and is requesting an urgent visit. He was diagnosed with gastric cancer last year and underwent treatment. Unfortunately the prognosis was poor and he has been given a few months to live.

His wife is very distressed due to the pain he is in. You decide to visit the patient. Around the bed of the patient are various medications including paracetamol, codeine phosphate, MST oramorph and fentanyl patches.

The patient's wife explains she is unsure which medication to give him when. She also asks 'when will he die?' She explains she is unable to sleep or go to the shops as she worries he may die alone. She worries about having time off work and what to tell his children. The children know he is unwell but she wants to know when she should phone them to say he has only days left, one child lives in France.

Learning objectives.

1. Gastric cancer symptoms cause and treatment.

2. Pain management in palliative patients including pain ladder and strength of fentanyl patches.

3. Side effects of these medication.

4. Other organisations that can be involved to help the patient and family.

5. Answering her questions: when will he die? Worries about leaving the patient? What to tell the family and when?

6. Getting death certificate and funeral organised

4 Dyspepsia

A 55 year old man comes to the GP surgery. He has been suffering from a burning feeling in his upper

abdomen for the last 2 months, particularly at night. He has been using bottles of Gaviscon but when buying his last bottle from the pharmacist she recommended he see a doctor. The Gaviscon does work but he finds it expensive to buy every couple of days.

After further history taking and examination, the doctor believes him to be suffering from dyspepsia. He arranges for a H.pylori test and starts him on a PPI. He has no family history of note. After two months the patient returns feeling better and asking “I think I need an endoscopy to check the cause of the heartburn.”

Learning Objectives.

1. Presentation of dyspepsia.

2. Differential diagnosis of dyspepsia.

3. Initial management and investigations eg H. Pylori testing.

4. Red flags and cancer referrals using two week wait.

5. Treatment and side effects.

6. Endoscopy when needed according to NICE guidelines.

7. Ethics of NICE or any guidelines. NHS funding changes.

5.4. TUTOR GUIDE FOR MET3A CASE BASED DISCUSSIONS

MET3A CBD TUTOR GUIDE

CASE 1: MET3A PAINLESS LEG ULCER

1. What are the types of leg and foot ulcers?

The three most common types of leg and foot ulcers include:

• Venous stasis ulcers

• Arterial (ischemic ulcers)

• Neurotrophic (diabetic)

Ulcers are typically defined by the appearance of the ulcer, the ulcer location, and the way the borders and surrounding skin of the ulcer look.

Ulcers are typically defined by the appearance of the ulcer, the ulcer location, and the way the borders and surrounding skin of the ulcer look.

Venous stasis ulcers

Venous ulcers are located below the knee and are primarily found on the inner part of the leg, just above the ankle.

The borders of a venous ulcer are usually irregularly shaped and the surrounding skin is often discoloured and swollen.

Arterial (ischemic)

Arterial ulcers are usually located on the feet and often occur on the heels, tips of toes, between the toes where the toes rub against one another or anywhere the bones may protrude and rub against bed sheets, socks or shoes.

The base of an arterial or ischemic ulcer usually does not bleed. It has a yellow, brown, grey, or black colour. The borders and surrounding skin usually appear as though they have been punched out. Arterial ulcers are typically very painful, especially at night. The patient may instinctively dangle his/her foot over the side of the bed to get pain relief. The patient usually has prior knowledge of poor circulation in the legs and may have an accompanying disorder, such as those listed in the section, "What causes leg ulcers?"

Neurotrophic (diabetic)

Neurotrophic ulcers are usually located at increased pressure points on the bottom of the feet. However, neurotrophic ulcers related to trauma can occur anywhere on the foot. They occur primarily in people with diabetes, although they can affect anyone who has an impaired sensation of the feet.

The base of the ulcer is variable, depending on the patient's circulation. It may appear pink/red or brown/black. The borders of the ulcer are punched out, while the surrounding skin is often calloused.

Neuropathy and peripheral artery disease often occur together in people who have diabetes. Nerve damage (neuropathy) in the feet can result in a loss of foot sensation and changes in the sweat-producing glands, increasing the risk of being unaware of foot calluses or cracks, injury or risk of infection. Symptoms of neuropathy include tingling, numbness, burning or pain.

IT IS EASY TO UNDERSTAND WHY PEOPLE WITH DIABETES ARE MORE PRONE TO FOOT ULCERS THAN OTHER PATIENTS. THIS IS WHY PEOPLE WITH DIABETES NEED TO INSPECT THEIR FEET AND THEIR SHOES DAILY AND WEAR APPROPRIATE FOOTWEAR. PEOPLE WITH DIABETES SHOULD NEVER WALK BAREFOOT.

2. What causes leg ulcers?

Leg ulcers may be caused by medical conditions such as:

• Poor circulation, often caused by arteriosclerosis

• Venous insufficiency (a failure of the valves in the veins of the leg that causes congestion and slowing of blood circulation in the veins)

• Other disorders of clotting and circulation that may or may not be related to atherosclerosis

• Diabetes

• Renal (kidney) failure

• Hypertension (treated or untreated)

• Lymphoedema

• Inflammatory diseases including vasculitis, lupus, scleroderma or other rheumatological conditions

• Other medical conditions such as high cholesterol, heart disease, high blood pressure, sickle cell anaemia, bowel disorders

• History of smoking (either current or past)

• Pressure caused by lying in one position for too long

• Genetics (ulcers may be hereditary)

• A malignancy (tumour or cancerous mass)

• Infections

• Certain medications

3. What are chronic changes of venous insufficiency?

-Inflammation of the vein (thrombophlebitis).

-Swelling of the foot or lower leg.

-Skin changes over the prominent veins. The possible skin changes are: discolouration, eczema, skin ulcers, or 'lipodermatosclerosis' (hardening of the fat layer under the skin, causing areas of thickened, red skin).

4. ABPI measurement

To rule out poor circulation as a cause, it is usual for a doctor or nurse to check the blood pressure in the ankle and in the arm. The ankle blood pressure reading is divided by the arm blood pressure reading to give a blood pressure ratio called the Ankle Brachial Pressure Index (ABPI). If the ratio is low (less than 0.8) it indicates that the cause of the ulcer is likely to be poor circulation (peripheral vascular disease) rather than venous problems. This is very important to know as the treatments are very different. An ABPI may be checked every six months or so to make sure the circulation to the legs remains good.

5. Compression bandaging

This is the most important part of treatment. The aim is to counteract the raised pressure in the leg veins. This gives the best chance for the ulcer to heal. The common method is for a nurse to put on 2-4 layers of bandages over the dressing. When the bandages are put on, the pressure is put highest at the ankle and gradually less towards the knee and thigh.

The bandages are reapplied every week or so.

A note of caution: when the patient has a compression bandage on the patient should still be able to move their ankle around. Occasionally, the compression is too tight, or it may affect the circulation in the legs. Therefore, take off the bandages if the patients foot changes colour or temperature, or if they have increasing pain.

6. Discuss the psychosocial aspects of having an ulcer

Discussion around pain, smell of ulcer treatment. Patients often have a decrease in confidence. The treatment is long and regular trips to the GP may be difficult.

7. Preventing a recurrence of venous skin ulcers

Venous leg ulcers commonly recur after they have healed. To prevent this, patients should wear a compression (support) stocking during the daytime for at least five years after the ulcer has healed. This counteracts the raised pressure in the veins that causes venous leg ulcers.

There are different classes (strengths) of compression stockings - class 1, 2 and 3.

The higher the class (class 3) the greater the compression.

Sometimes surgery for varicose veins or other vein problems is advised after an ulcer has healed, in order to help prevent a recurrence.

The use of emollients can also help prevent ulcers.

8. Supporting patients with leg ulcers

Important to keep patients informed of the treatment and how long it may take to improve. Providing them with telephone numbers of people they can contact if in difficulty. District nurses visiting patients to do their dressings. Social services helping with meals and carer support.

CASE 2: MET3A HEPATITIS

1. Discuss the differential diagnosis of acute painless jaundice and give the diagnosis here

The differential diagnosis lies between the various causes of acute hepatitis; viral, alcoholic, autoimmune, drug reaction or toxicity. Viral hepatitis is by far the most likely diagnosis in this young man.

2. Consider the various risk factors for hepatitis presented

The risk factors here are travel in the Far East with salad eating (hepatitis A), iv drug use, or sexual promiscuity when risks of hepatitis B and C are greatest. Hepatitis C is a risk for iv drug users and travel is the main risk factor for Hepatitis E. The time course for hepatitis A is demonstrated in the scenario.

3. Compare the biochemical findings in liver function tests between obstructive and hepatic/hepatocellular jaundice

Acute hepatitis causes a massive increase in blood transaminases whereas cholestatic jaundice caused by bile duct obstruction typically results preferentially in a rise in alkaline phosphatase.

4. Consider virological tests for viral hepatitis and describe their significance

IgM antibodies confirm that this is an acute hepatitis A rather than longstanding immunity from a previous attack when IgG antibodies would only be present. In hepatitis B immunity is demonstrated by the presence of antibodies without presence of surface or core antigens in the blood. Infectivity and acute/ chronic disease shows positive antigenaemia. In hepatitis C only antibodies can be detected so that it is not easy to say serologically whether a patient is immune or infective. HCV RNA is used to determine evidence of viraemia.

Characteristics of Viral Hepatitis

	A	B	C	D	E
Virus type	RNA	DNA	RNA	RNA	RNA
Faecal/oral transmission	Yes	No / yes	No/ yes	No	Yes
Blood transmission	Rare	Yes	Yes	Yes	No
Vertical transmission	No	Yes	Yes	Rarely	No
Age group affected	Young	Any	Any	Any	Any
Carrier state	No	Yes	Yes	Yes	No
Chronic liver disease	No	Yes	Yes	Yes	No
Hepatocellular carcinoma risk	No	Yes	Yes	Yes	No

Characteristics of Viral Hepatitis

5. Consider long term sequelae of viral hepatitis and potential needs for counselling

Though it may persist for months Hepatitis A does not cause chronic liver disease. Both hepatitis B and C can, with resulting progression through chronic active hepatitis, cirrhosis and hepatocellular carcinoma formation. Chronic hepatitis B and C carriage present risks to others and demand family screening and advice to prevent their blood contaminating other peoples through cuts and abrasions or blood transfusion. Sex must be protected by a condom with potential for religious/ethical discussion.

6. Ethics around the risk of transmission to his sexual partner

The doctor should encourage the patient to inform his wife. If the wife has been put at risk she needs to be tested. If the patient refuses to tell his wife, it is advised the doctor contact their medical defence union. At present if the disease is seen to be fatal for the patients wife if they contacted the disease i.e. HIV. You are allowed to break confidentiality to check she is tested and aware of the risks.

7. Discuss Immunisations available

Immunisation is available for Hepatitis A and B.

CASE 3: MET3A PALLIATIVE CARE/OUT OF HOURS

1. Gastric cancer symptoms cause and treatment

(www.patient.co.uk).

Stomach cancer is sometimes called gastric cancer. Worldwide it is one of the most common cancers. It is common in Japan and China but is less common in the UK. About 8,500 people develop stomach cancer each year in the UK. Stomach cancer is more common in men than women and tends to occur mainly in older people. Most people who develop stomach cancer are over the age of 60.

Adenocarcinoma of the stomach is the most common type of gastric cancer.

Other types of stomach cancer

There are some less common and rare types of stomach cancer which include:

• Lymphomas.

• Sarcomas. These are cancers which arise from the muscle or connective tissue within the wall of the stomach.

• Carcinoid cancers. These are cancers which arise from cells in the stomach lining which make hormones.

Many people develop stomach cancer for no apparent reason. However, certain 'risk factors' increase the chance that stomach cancer may develop. These include:

• Ageing.

• Pernicious anaemia, which causes a lack of vitamin B12.

• Diet is probably a factor:

- Countries such as Japan where people eat a lot of salt, pickled and smoked foods have a high rate of stomach cancer.

- Eating a lot of fruit and green vegetables can reduce the risk.

• Smokers.

• Long term infection of the stomach lining with a bacterium called H pylori seems to lead to a slightly higher risk of stomach cancer.

• Gender. Stomach cancer is twice as common in men as women.

• If you have had part of your stomach removed in the past for any reason. For example, to treat a stomach ulcer or some other condition.

• Family history. For some cases, stomach cancer may run in the family. However, most cases of stomach cancer do not run in families and are not inherited.

What are the symptoms of stomach cancer?

When a stomach cancer first develops and is small, it usually causes no symptoms. Initial symptoms may include:

• Pain or discomfort in the upper abdomen, especially after eating.

• Indigestion.

• Feeling sick. Some people have a sense of fullness after eating.

• Weight loss and/or loss of appetite.

• Blood in the patient's faeces.

As the cancer grows in the stomach, symptoms may become worse and include:

• The same symptoms as above, but more severe.

• Tiredness.

• Anaemia.

• Dysphagia to food and drink.

If the cancer spreads to other parts of the body, various other symptoms can develop.

How is stomach cancer diagnosed and assessed?

Initial assessment and gastroscopy

Biopsy - to confirm the diagnosis Assessing the extent and spread

What are the treatment options for stomach cancer?

Treatment options which may be considered include surgery, chemotherapy (and sometimes radiotherapy). The treatment advised for each case depends on various factors such as the stage of the cancer and your general health.

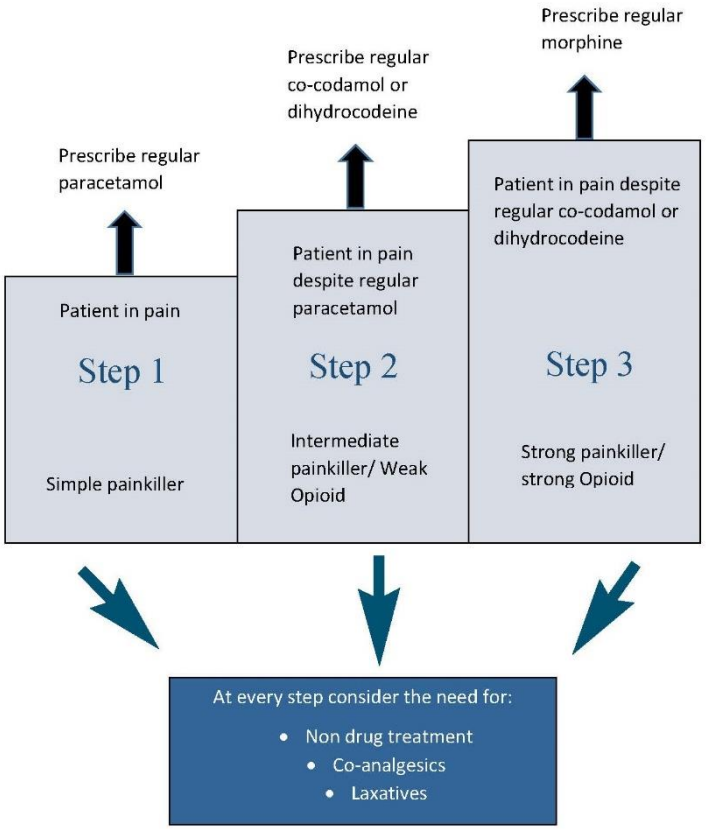
What is the prognosis?

Without treatment, a stomach cancer is likely to get larger, and spread to other parts of the body. If it is diagnosed and treated at an early stage (before growing through the wall of the stomach or spreading to lymph nodes or other areas of the body) then there is a good chance of a cure with surgery. Unfortunately, most cases in the UK are not diagnosed at an early stage.

If the cancer is diagnosed when it has grown through the wall of the stomach, or spread to other parts of the body, a cure is less likely. However, treatment can often slow down the progression of the cancer.

The treatment of cancer is a developing area of medicine. New treatments continue to be produced.

2. Pain management in palliative patients including pain ladder and strength of fentanyl patches



Pain Ladder

Fentanyl Patches - may be suitable for patients who are stable on morphine but are experiencing problems (e.g. drowsiness, constipation, unable to take oral preparation).

Conversion from oral morphine to transdermal fentanyl

Oral 24 hour morphine (mg per day)	Fentanyl patch (micrograms per hour)
50 - 134	25
135 - 224	50
225 - 314	75
315 - 404	100
405 - 494	125
495 - 584	150
585 - 674	175
675 - 764	200
765 - 854	225
855 - 944	250
945 - 1034	275

Conversion from oral morphine to transdermal fentanyl

3. Side effect of medications

Please show the students the BNF and ask them to look up the many side effects of medication used in palliative care. Important to mention constipation, itching, drowsiness and treatment of overdose of morphine.

4. Other organisations

- Macmillan and palliative care team.

- District nurses.

- Social services.

- Informing the out-of-hours team of patient.

- Hospital team of specialist nurses, surgeon and oncologist.

5. Answering questions

Importance of listening to the patient and giving her names and telephone numbers to call for support. Facing the unknown together. Unknown how long he has left but giving broad approaches e.g. months, weeks may help. Explaining the patient will usually sleep a lot more as the time draws nearer but ever patient is different. There is never any need for a patient to be in pain. Important for the carer to look after herself, as the patient would want that. Organisations that will sit with the patient at night or if the wife wants to go out for something. Keep the family informed of the unknown element of their fathers illness, maybe let them make the decision when best to come.

6. Getting will and funeral organised.

Importance of making a will and supporting arrangements for the funeral the patient and family would like.

CASE 4: MET3A DYSPEPSIA

1. Presentation of dyspepsia.

Epigastric discomfort

Fullness or bloating

Excessive flatus

Nausea

Fatty food intolerance

Always ask about family history and medication use.

2. Differential Diagnosis.

• Peptic ulcer.

• Functional (non-ulcer) dyspepsia.

• IBS.

• Atypical gastro-oesophageal reflux disease (GORD/GERD).

• Biliary pain, e.g. gallstones.

• Achalasia.

• Medication-induced dyspepsia.

• Oesophageal spasm.

• Carcinoma of oesophagus or stomach.

• Be aware to rule out cardiac pain.

3. The NICE guideline suggests the following steps:

• Review medications: possible drug causes of dyspepsia include NSAIDs, steroids, calcium antagonists, nitrates, theophyllines, and bisphosphonates. Reduce or stop if possible.

• Offer lifestyle advice, i.e. stopping smoking, more regular meals, ceasing excessive alcohol consumption.

• Antacids are cheap, simple and may be all that is required for relief of occasional symptoms. Most antacids contain a mixture of aluminium hydroxide that tends to cause constipation and magnesium hydroxide that tends to cause diarrhoea.

- Test for H. pylori (carbon-13 urea breath test, stool antigen or laboratory serology) and eradicate if positive.

- Empirical acid suppression (with PPI) - full dose for one month.

Where there has been a satisfactory response at any of the steps above, reassure and return to self-care.

If the patient responds to PPI but then relapses, consider low-dose or intermittent treatment.

If there is no response, consider a prokinetic, e.g. metoclopramide or H2RA, e.g. ranitidine for one month.

Where patients show an inadequate response to treatment, consider other diagnoses, e.g. gallstones and/or referral to a specialist.

4. Urgent specialist referral - 2 week rule

If the patient has dyspepsia at any age with any of the following alarm symptoms:

• Chronic GI bleeding

• Progressive unintentional weight loss

• Progressive dysphagia

• Persistent vomiting

• Iron deficiency anaemia

• Epigastric mass

• Suspicious barium meal

NB: patients aged 55 years or older with unexplained and persistent recent-onset dyspepsia should be referred urgently for endoscopy.

Please show students the 2 week wait referral forms and guidelines for each condition.

5. Treatment side effects.

Please ask the students to look up the side effects of the various drugs involved in treatment in the BNF. Particularly highlighting diarrhoea with the use of PPIs.

6. Endoscopy when needed according to NICE guidelines.

Routine endoscopic investigation of dyspeptic patients is not necessary, but should be considered in patients over the age of 55 where symptoms persist despite H. pylori testing and acid suppression.

However, there has been some dissent over the NICE recommendations, citing the value of early detection of GI cancer and its improved survival rates.

Patients with the following risk factors have a higher risk of malignancy and so lower your threshold for endoscopy referral:

• Family history of upper GI cancer in more than two first-degree relatives.

• Barrett's oesophagitis.

• Pernicious anaemia.

• Peptic ulcer surgery over 20 years ago.

Known dysplasia, atrophic gastritis, intestinal metaplasia.

7. Ethics of NICE or any guidelines. NHS funding changes.

Discussion with students about how NICE guidelines are produced and the economic slant they may have. Further discussion about the limited resources of the NHS and the changes in funding with the recent NHS white paper. Discussion with students about guidelines are just for guidance as patients present in many different ways.

5.5 TUTOR GUIDE MET 3B CASE BASED DISCUSSIONS

Tutor Guide Met 3B Case Based Discussions

MET3B PBL/CBL SCENARIOS

Tutor Notes will be sent out by email to GP3 Tutors.

PBL/CBL number 7 and 8 should be done in primary care.

Hospital based PBL/CBLs:

1. A woman in a coma
2. A woman in shock
3. A dizzy secretary
4. A woman with headaches and sweating
5. A man with constipation and depression
6. A woman with irregular periods

Primary Care based PBL/CBLs:

7. A woman with blood in her urine
8. A man attending for a routine diabetes check

Problem Based Learning Session: If you plan to run the session as a PBL session, you should ideally allow the students to develop their own learning objectives and allow them time to research the objectives and feedback in the following session.

Case Based Learning Session: If you plan to run the session as a CBL session, you may want to use these learning objectives to help structure the teaching.

Please **DO NOT SHARE THESE TUTOR NOTES WITH STUDENTS**

HOSPITAL BASED PBL/CBL

1. A WOMAN IN A COMA

A 67-year old widow who lives alone, was diagnosed with Type 2 diabetes three years ago following referral from Moorfield's Eye Hospital, where she had attended for investigation of deteriorating visual acuity.

When first diagnosed she had been treated by diet alone in an attempt to lose weight. Since this was largely unsuccessful in the first year she had subsequently been treated with *metformin*. She also took *lisinopril* and *simvastatin.*

At recent diabetes clinic review, her weight was 75 kg and height 1.60 m (body mass index 29.3 kg/m^2). Blood pressure was 146/84 mmHg, and she had reduced fine touch and vibration sense in the lower limbs. Glycated haemoglobin was 86 mmol/mol (28 – 42 mmol/mol), cholesterol 5.6 mmol/L, albumin creatinine ratio 8.6 mg/mmol (0 - 3.5).

On his recent weekly visit to his mother, the patient's son discovered her collapsed on the sofa, breathing but otherwise unresponsive, and dialled 999.

Examination:

Clinically severely dehydrated

Glasgow Coma Score 7/15

Capillary glucose >30 mmol/L

Pulse 120 irregular

Plasma glucose	52 mmol/L (4 - 6)
Serum sodium	150 mmol/L (135 - 145)
Potassium	4.8 mmol/L (3.5 - 5.5)
Bicarbonate	23 mmol/L (22 - 28)
Creatinine	208 umol/L (56-97)
Serum Osmolality	362 mOsm/kg (275 - 285)
pH	7.36 (7.35 – 7.45)
pO2	12.0 kPa
pCO2	5.5 kPa
Urinalysis	Glucose +++ Blood Neg Protein + Ketones Neg

She was admitted to the Intensive Care Unit, and urgent treatment was instituted. She required a 6 day stay in ICU where she was stabilised metabolically, and was stepped down to the ward. Once she was eating and drinking, capillary glucose levels were noted to be 13-24 mmol/L despite titration of oral hypoglycaemic medication to maximum doses.

HOSPITAL BASED PBL/CBL

2. A WOMAN IN SHOCK

A 72-year old woman is admitted urgently via A&E. Her daughter states she is normally fit and well, has a past history of Type 2 diabetes (treated with tablets) and hypertension, but no known kidney problems. She has been unwell for 48 hours with fevers and abdominal pain. On the day of admission, she became confused and disorientated and her daughter phoned for an ambulance.

Examination:

Confused

Glasgow Coma Score 9/15

Pulse 110 regular

Blood pressure 84/43 mmHg

Temperature 38.9 ^{o}C

Oxygen saturations 89% on room air

Chest Clinically clear

Abdomen Soft, no organomegaly

No focal neurology

Investigations:

Hb	10.4 g/dl (11 - 13)
White cell count	23.5 (3.5 - 9.0)
Sodium	138 mmol/L (135 - 144)
Potassium	6.5 mmol/L (3.5 – 5.2)
Bicarbonate	16 mmol/L (24 - 28)
Urea	18.7 mmol/L (3.3 - 6.6)
Creatinine	427 umol/L (75 - 95)
Lactate	6.7 mmol/L (0.5 - 2.2)

pH	7.28 (7.35 – 7.45)
pO2	8.9 kPa (On air)
pCO2	4.5 kPa
Urinalysis	Blood ++ Leucocytes + Nitrites + Protein ++ Ketones Neg

CXR Normal ECG – enlarged T waves

She was admitted to the intensive care unit, and required emergency treatment with inotropes, fluids, haemofiltration and subsequently ventilatory support. Over the next 10 days on ICU she was weaned off ventilatory support, but did not pass urine. The nephrologist had a discussion with her and her daughter about the likelihood of her kidney function recovering. Consideration was given for long term renal replacement therapy.

HOSPITAL BASED PBL/CBL

3. A DIZZY SECRETARY

A 34 year old secretary had noticed that she is getting progressively tired, with weight gain, constipation and feeling cold. She also had developed areas of depigmentation on her forearms. Blood tests revealed:

TSH	56.5 mIU/l (0.6-6.0)
Free T4	7.0 pmol/L (9-20)

She was prescribed medication by her GP and was assured that she would rapidly feel better. After two weeks, she noted that she was not feeling any better and indeed began to feel worse. She started feeling nauseated and felt dizzy as she got out of bed in the morning.

Despite this, she continued to work until whilst travelling on the underground in the morning during her commute she collapsed. She was taken to her closest A&E department.

Examination:

Alert, but looks unwell

Looks tanned

JVP not visible

Pulse 108 bpm

Lying BP 104/75 mmHg

Sitting BP 84/60 mmHg

Investigations:

Full blood count	Normal
Sodium	122 mmol/L (135-144)
Potassium	6.5 mmol/L (3.5 – 5.2)
Bicarbonate	16 mmol/L (24-28)

Appropriate endocrine tests were sent. She was treated with emergency intravenous treatment, stabilised and converted to standard oral treatment within three days.

On discharge, she was educated about dose adjustment in the event of illness and was issued with an emergency pack.

HOSPITAL BASED PBL/CBL

4. A WOMAN WITH HEADACHES AND SWEATING

A 48-year-old female accountant is referred to the endocrinology clinic with headaches, sweating and pains in her joints. She is a partner in a firm of accountants. Her two teenage children were doing well at school and the family had no financial or other domestic concerns.

During consultation, she described a number of other symptoms; she had noted her clothes and shoes felt somewhat tighter; she had noticed some problems with her eyesight, and had stumbled into door posts on more than one occasion. Her husband noticed that she had started to move her head from side-to-side when reading the newspaper, and also noted a change in her facial appearance. She complained of pins and needles in both hands, especially at night, and also noticed milk like secretions from her nipples on a few occasions. Her periods were regular but she was taking a combined oral contraceptive pill.

Examination:

Large jaw and hands	
Pulse	100 bpm
Blood pressure	160/95 mmHg
Visual fields	bi-temporal hemianopia with optic atrophy

Investigations:

Serum sodium	139 mmol/L	(135 - 145)
Potassium	4.8 mmol/L	(3.5 - 5.5)
Urea	4.5 mmol/L	(3.3 - 6.6)
Creatinine	86 umol/L	(75 - 95)
Free T4	14.5 pmol/L	(9 – 25)
TSH	0.3 mU/L	(0.4 – 4)
Prolactin	987 mU/L	(<400)
LH	4.5 IU/L	(1-20)
FSH	6.5 IU/L	(5 - 30 pre-menopausal 50 -100 post-menopausal)

An appropriate confirmatory dynamic test was done, and she underwent a dedicated pituitary MRI scan, whereby the diagnosis was confirmed. She was treated with initially medical treatment, followed by surgical resection.

HOSPITAL BASED PBL/CBL

5. A MAN WITH CONSTIPATION AND DEPRESSION

A 69-year old man has noticed increasing symptoms of abdominal pain, constipation and headaches. He attended his GP surgery to complain about these symptoms but his wife also mentioned that she thought that he was depressed. His GP prescribed him some medication for his constipation and recommended paracetamol for his headaches. This helped for a period.

A few weeks later whilst lifting some furniture he developed sudden and severe back pain in the lower lumbar region. The pain was so severe that he called an ambulance that took him to the Emergency Department.

He was examined by the doctors there and investigations were organised, the results of which are shown below:

Calcium	2.90 mmol/L	(2.20 - 2.60)
Phosphate	0.62 mmol/L	(0.80 - 1.50)
Alkaline Phosphatase	290 U/L	(30 -130)
Albumin	39 g/L	(36 – 53)
Urea	7.8 mmol/L	(2.5 - 7.5)
Creatinine	80 umol/L	(55 – 106)
Parathyroid Hormone	15.2 pmol/L	(1.1 - 6.8)
Urinalysis	Blood ++	

Lumbar spine X-ray showed a wedge fracture of L5, and KUB showed a left renal calculus.

He was treated with intravenous fluids and analgesia. He went on to have a DEXA scan indicating significant loss of bone mineral density.

He underwent diagnostic radiological tests and was told by his endocrinologist that operative treatment was required to effect a cure.

HOSPITAL BASED PBL/CBL

6. A WOMAN WITH IRREGULAR PERIODS

A 30 year old canteen assistant comes to see her GP because she and her partner had been trying for a baby for over 2 years without success. On closer questioning she reports irregular scanty periods for the last 18 months. She has a family history of type 2 diabetes with her father and paternal uncles affected. On examination she is noted to be obese with a BMI of 31.4kg/m^2. She has facial acne, hirsutism and central obesity. Her blood pressure is 160/90 mmHg. Investigations are requested and the results are shown below.

Fasting	6.4 mmol/l
Androstenedione (<8)	11.2 nmol/l
SHBG (sex hormone binding globulin) (>30)	12 nmol/l
T4 and TSH normal	
Prolactin normal	

Whilst she is embarrassed by her facial hirsutism, she is most concerned about her infertility and wants to seek treatment for this. The doctor states that she is at risk of developing diabetes, and suggests that she sees a dietitian to makes appropriate lifestyle changes to lose weight. The doctor also suggests she commence *metformin*, and consider fertility treatment to aid her fertility.

HOSPITAL OR PRIMARY CARE BASED PBL/CBL

7. A WOMAN WITH BLOOD IN HER URINE

A 65 year old woman with a long history of cigarette smoking, goes to see her GP complaining of frequency of micturition, dysuria and haematuria.

Urinalysis results:	Blood ++
Protein +	
Glucose -	
Ketones -	
Nitrites ++	
Leucocytes ++	

Her GP starts her on trimethoprim 200mg for three days. He also sends the urine sample to the lab for M,C+S. The report shows E. Coli is grown sensitive to trimethoprim, co-amoxiclav and nitrofurantoin. The GP contacts the patient to ask her to drop off a further sample to check the infection has gone. This sample again grows E. Coli. A course of co-amoxiclav is now given. Unfortunately her urine still grows E. Coli.

She is referred to the local urology services but whilst awaiting an appointment she develops severe left sided colicky loin pain and begins to pass blood in her urine with clots. She attends the local A&E. Following an initial assessment, she is admitted for investigations in order to determine the cause of her loin pain and haematuria.

Investigations requested by the admitting team include urine cytology, blood tests, imaging and a cystoscopy. These investigations reveal a transitional cell tumour of her left ureter. She is counselled about the various treatment options. Given the limited nature of the tumour and the lack of evidence of spread she is offered a left nephroureterectomy.

She has an uncomplicated recovery from her operation and is discharged from hospital 7 days later and remains under follow up with her consultant urologist.

HOSPITAL OR PRIMARY CARE BASED PBL/CBL

8. A MAN ATTENDING FOR A ROUTINE DIABETES CHECK

A 72 year old man goes to see his GP for his routine diabetic check. He was diagnosed with type 2 diabetes 4 years previously when he suffered with excessive thirst and was found to have an elevated glycated haemoglobin.

Since then he has sustained a heart attack and suffers with pain in both calves if he walks for more than 100 metres.

He attends regular appointments at an ophthalmology clinic for poor vision and says he has had laser treatment to his retina for complications of diabetes. At his GP review, it is noticed he has protein in his urine. His blood pressure in clinic is 172/93 mmHg.

Further investigations:		
Serum creatinine-	102 umol/L	(60-110)
Estimated GFR (eGFR) -	47 mls/min	(using the MDRD formula)
Urine albumin:creatinine ratio (ACR)	94 mg/mmol/L	(<2.5)

The GP tells the patient that he has diabetic nephropathy and that his management will consist of good blood pressure and glycaemic control.