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General Practice and Palliative Care Volume 2 Issue 4 April/May 2008

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The journal welcomes papers on any aspect of health care and medical education which will to be of benefit to doctors in the foundation training grade in the UK or international equivalents. The predominant emphasis in ***Foundation Years Journal*** is on work related to patient safety and in healthcare education.

Volume 2 Issue 4: General Practice and Palliative Care

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Notice

Questions

General practice clinical problem-solving questions

The following are examples of general practice clinical problem-solving questions that you may face in your GP ST stage 2 exam. These questions are designed to test how well you can apply your knowledge. Answers can be found after the questions so answer the questions first then you can assess how well you have done.

Q1 **A 30-year-old female is found supine unconscious/unresponsive from an unknown mechanism. What should you do first?**

A. Perform a head tilt/chin lift manoeuvre to open the patient's airway
B. Ventilate the patient using a Bag-valve-mask
C. Place an oxygen mask on the patient at 15L
D. Perform a jaw-thrust manoeuvre
E. Maintain an IV line

Q2 **A 72-year-old man is brought to A&E by the carer in his nursing home. He has a past medical history of dementia. His carer tells the doctor that he was very pleasant previously, but now he is very agitated and often attacks the caregivers. He hears voices and believes that people want to kill him.**

Past medical records show the following problems:
Alzheimer's dementia, Diabetes type 2, Hypertension, CVA, COPD
Current medications:
Valsartan (Diovan), Frusemide (Lasix), Metoprolol, Insulin and Aricept

Which of the following is the most probable diagnosis?

A. Acute exacerbation of Alzheimer's disease
B. Bipolar disorder
C. Acute psychosis in demented patient
D. Elderly abuse
E. Opioid abuse

Q3 **A suicidal risk assessment is done. Which of the following features are prognostically worse if it is associated with suicidal ideation?**

A. Euphoric feeling
B. Visual hallucination
C. Auditory hallucination
D. Living with the family
E. Religious preoccupation

Q4 **The patient was recovering well with the treatment and was discharged. But after a few weeks suddenly he deteriorated and was admitted with high fever, muscular rigidity and tachycardia.**

Which of the following is the most likely diagnosis?

A. Septicaemia
B. Pulmonary embolism
C. Malignancy
D. CVA
E. Malignant neuroleptic syndrome

Q5 **Microalbuminuria:**

A. Can occur in the absence of diabetes
B. May be undetectable by normal urinary stick tests
C. Is a strong predictor of development of overt diabetic nephropathy
D. May vary during the 24h period
E. Is clinically often silent

Q6 **In advanced renal disease:**

A. Large amounts of protein may be lost in the urine
B. There is a progressive decline in renal function that is irreversible
C. Changes in blood renal parameters are often present
D. Persistent nausea and lack of appetite may be present
E. Neuropathy and cardiovascular complications directly correlate to the extent of renal dysfunction

Q7 **Microalbuminuria can be detected by the following measures:**

A. Normal urine stick tests
B. Twenty-four hour urine collection
C. Timed overnight urine collection
D. Changes in serum albumin concentration
E. Spot urine albumin:creatinine ratio

Q8 **False positive results for microalbuminuria can commonly be seen in the context of:**

A. Poor glycaemic control
B. Extreme physical exertion
C. Urinary tract infection
D. Treatment with antibiotics
E. Prolonged supine posture

Q9 **The following are recommended investigations for patients with a new diagnosis of microalbuminuria:**

A. Fasting glucose and/or Hb1Ac
B. CT abdomen
C. Abdominal ultrasound scan
D. Twenty-four hour Holter tape
E. Lipid profile

Q10 **In hypertensive patients:**

A. AIIRA is not licensed to be used alone
B. AIIRA results in significant hyperkalaemia
C. Losartan should always be the first-line antihypertensive drug of choice
D. Combining an AIIRA and ACEI results in better blood pressure control
E. Losartan reduces fatal and non-fatal stroke more than β-blockers in hypertensive patients with LVH

Q11 **In patients with heart failure:**

A. Only valsartan has been shown to be of significant benefit
B. Patients should be treated with either an ACEI or an AIIRA
C. β-blockers should be discontinued if an AIIRA is introduced
D. All trials have shown a significant mortality benefit in treatment with AIIRAs
E. AIIRAs are more effective in younger patients

Questions

Q12 **What percentage of all strokes are believed to be due to atrial fibrillation?**

A. 0–5%
B. 5–10%
C. 10–15%
D. 15–20%
E. 50%

Q13 **Which of the following drugs used to treat atrial fibrillation is a positive ionotrope?**

A. Sotalol
B. Flecainide
C. Amiodarone
D. Diltiazem
E. Digoxin

Q14 **You see an 85-year-old woman in clinic who has been noted by her GP to be in atrial fibrillation. A 24 h tape shows you that her heart rate varies between 70 and 150 bpm. What combination of drugs should you start her on?**

A. Amiodarone & warfarin
B. Digoxin & warfarin
C. Amiodarone & aspirin
D. Sotalol & aspirin
E. Verapamil & warfarin

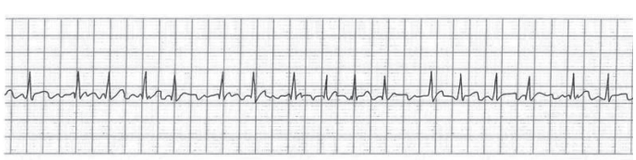
Q15 **Which of the following patients is most likely to stay in sinus rhythm following d/c cardioversion?**

A. A 45-year-old man with 'lone' atrial fibrillation
B. An 82-year-old woman with mitral regurgitation
C. A 76-year-old man with mitral stenosis
D. A 58-year-old man with dilated cardiomyopathy
E. A 77-year-old woman with thyrotoxicosis

Q16 **The best treatment for a distressed patient with atrial fibrillation, a pulse rate of 180, a blood pressure of 70/40 and signs of cardiac failure, is:**

A. D/C cardioversion
B. IV amiodarone
C. IV sotalol
D. IV digoxin
E. IV flecainide

Q17 **A 70-year-old woman was presented with palpitations for the past few hours. An ECG at that time showed the following picture:**

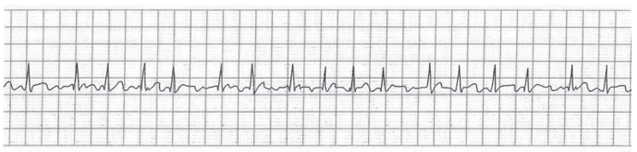


ECG – Long Lead II

Which of the following is true about the ECG?

A. The ventricular rate is in the range of 90–110/min
B. The rhythm is irregular
C. The rhythm is supraventricular in origin
D. The duration of QRS is prolonged
E. P waves are clearly visible

Q18 **Her ECG shows the following picture:**



ECG – Long Lead II

What is the most probable diagnosis?

A. Sinus tachycardia
B. Atrial flutter with 2:3 heart block
C. Atrial fibrillation
D. Paroxysmal supraventricular tachycardia
E. Ventricular tachycardia

Q19 **A 74-year-old man was presented with intermittent chest pain at rest. Which one of following clinical findings would most strongly suggest that the pain was due to myocardial ischaemia?**

A. Associated dyspnoea
B. Coexistent claudication
C. Past history of cigarette smoking
D. Radiation of pain to the jaw
E. Relief of pain by sublingual nitrate

Q20 **A 76-year-old man was admitted with chest pain and his ECG showed minor ST changes. Which of the following enzymes would be the first to rise following a myocardial infarction?**

A. LDH
B. CK-MB
C. AST
D. Troponin I
E. Myoglobin

Q21 **A 67-year-old male presents with an acute inferior myocardial infarction. There were no contraindications to thrombolysis and he received streptokinase with good resolution of ECG changes. Three days later his physical examination was normal, with a blood pressure of 134/76 mmHg. Blood results revealed total cholesterol of 4.8 (normal < 5.2). Which one of the following drugs would not reduce his future morbidity and mortality?**

A. Nifedipine
B. Aspirin
C. Atenolol
D. Simvastatin
E. Ramipril

Q22 **Which of the following is not a well-recognised side effect of ACE inhibitors?**

A. Renal failure
B. Cough
C. Hyperkalaemia
D. Angioedema
E. Diarrhoea

Questions

Q23 **Which of the following drugs has not been shown to improve mortality in patients with congestive cardiac failure?**

A. Carvedilol
B. Spironolactone
C. Ramipril
D. Bisoprolol
E. Frusemide

Q24 **Which of the following drugs will reduce the efficacy of the oral contraceptive pill (OCP)?**

A. Omeprazole
B. Cimetidine
C. Sodium valproate
D. Phenytoin
E. Erythromycin

Q25 **Which of the following is a cause of weight gain?**

A. Malnutrition
B. Hypothyroidism
C. Dieting
D. Depression
E. Anorexia

Q26 **Which of the following is a cause of weight gain?**

A. Malnutrition
B. Hypothyroidism
C. Dieting
D. Addison's disease
E. Anorexia

Q27 **What percentage of the female population suffer from hyperthyroidism?**

A. 2%
B. 5%
C. 10%
D. 15%
E. 20%

Q28 **A 40-year-old woman presents with ophthalmoplegia, lid lag and exophthalmos. What is the single most likely cause?**

A. Addison's disease
B. Graves' disease
C. Hashimoto's thyroiditis
D. Toxic multinodular goitre
E. Plummer's disease

Q29 **Which of the following is not a cause of acute pancreatitis?**

A. Autoimmune disease
B. Scorpion venom
C. Hypocalcaemia
D. Hyperlipidaemia
E. Trauma

Q30 **Acute pancreatitis is a recognised side effect of which of the following drugs?**

A. Amiodarone
B. Lithium
C. Digoxin
D. Azathioprine
E. Paracetamol

Q31 **Which of the following is least essential when investigating a suspected case of acute pancreatitis?**

A. Urine dipstick
B. Abdominal USS
C. ECG
D. ABG
E. Bone profile

Q32 **Important aspects of the management of acute pancreatitis include all of the following except:**

A. Urinary catheter insertion
B. Antibiotics
C. IV fluids
D. Daily weights
E. Keeping the patient NBM

Q33 **The most appropriate analgesia for a patient with severe pancreatitis is:**

A. Morphine
B. NSAIDS
C. Aspirin
D. Codeine
E. Pethidine

Q34 **The following are all recognised complications of acute pancreatitis except:**

A. Renal failure
B. ARDS
C. Pancreatic necrosis
D. Hypoglycaemia
E. Thrombocytopenia

Q35 **Which of the following is true regarding hepatitis C infection?**

A. Sequelae include developing gallstones
B. Eighty-five per cent of those infected become chronic carriers
C. Hepatocellular cancer is not a risk
D. The risk of contracting it from a needlestick injury is 10%
E. Breastfeeding is contraindicated in mothers who have hepatitis C

Q36 **You are doing a 'flu' clinic, with one injection given after another. Finally, your last patient leaves the room. You are about to enter the injection batch number/expiry date on your computer when you notice that the vial has no label on it. It was in the box with the other flu injections, it probably was an influenza vaccine but you are not sure. The patient is a frequent attender and has lots of health anxieties already. What do you do?**

A. Keep a note of the patient's name and see if she comes back with any side effects
B. Document everything on the computer, should the patient come back with problems
C. Ring the patient, apologise and ask her to monitor any side effects
D. Put the vial quickly in the sharp's bin, you are 99% sure it was a flu jab
E. Ring the supplier

Questions

Q37 **A 40-year-old solicitor is admitted to A&E with a 1 day h/o worsening diplopia, blurring of vision and shortness of breath. This was preceded by diarrhoea and vomiting. He was otherwise fit and well, and had to rely on pre-cooked meals due to his demanding lifestyle. On examination he was agitated, pupillary responses were sluggish with poor abduction of eyes, he had difficulty in swallowing his saliva and there was generalised reduction in tone and power (3/5) with diminished reflexes. Systemic review and blood tests were normal. The most likely diagnosis is:**

A. Tetanus
B. Botulism
C. Rabies
D. Cerebral abscess
E. Organophosphate poisoning

Q38 **Lesions of the descending tracts produce all of the following clinical signs except:**

A. Hypertonicity
B. Paralysis
C. Exaggerated knee jerk
D. Ankle clonus
E. Fasciculations

Q39 **What is NOT a function of the third cranial nerve?**

A. Elevation of eyes
B. Adduction of eyes
C. Dilatation of pupil
D. Constriction of pupil
E. Changing the shape of the lens

Q40 **Humphrey visual field examination from a man with a sellar chiasmal mass will show what kind of a visual field defect?**

A. Right homonymous hemianopia
B. Left homonymous hemianopia
C. Bitemporal hemianopia
D. Enlargement of the blind spot
E. Complete visual loss in the right eye

Q41 **A patient's palatal movements are being tested. On saying 'Ah', the right side of the palate is lower than the left, and the uvula is also deviated to the left. The patient has a lesion of the:**

A. Right vagus nerve
B. Left vagus nerve
C. Right glossopharyngeal nerve
D. Left glossopharyngeal nerve
E. Right hypoglossal nerve

Q42 **Which blood vessel supplies the motor and sensory speech areas of the brain?**

A. Right middle cerebral artery
B. Left middle cerebral artery
C. Left posterior cerebral artery
D. Right posterior cerebral artery
E. Right anterior cerebral artery

Q43 **An obese 34-year-old woman presents with headaches, raised CSF pressure on manometry, and abnormal fundi. What visual field defect will she have?**

A. Right homonymous hemianopia
B. Left homonymous hemianopia
C. Bitemporal hemianopia
D. Enlargement of the blind spot
E. Complete visual loss in the right eye

Q44 **A 35-year-old woman presents with a 2-month history of difficulty in walking and weakness in her left leg. Her symptoms are gradually deteriorating and she has fallen down a few times. She has been having tingling in her left arm for 2 weeks. She has had an episode of optic neuritis in the past. On examination she has pyramidal signs in her left leg, and is ataxic on tandem walking. The rest of the neurological and systemic examination is unremarkable. The most likely diagnosis is:**

A. Acute disseminated encephalomyelitis
B. Multiple sclerosis
C. Vitamin B12 deficiency
D. Motor neurone disease
E. Tabes dorsalis

Answers

Answers to general practice clinical problem-solving questions

A1 A

The cardinal rule of managing an unconscious patient is ABC:
A = Clear airway
B = Ensure patient is breathing
C = Check whether patient has a pulse and BP

A2 C

The features are suggestive of acute psychosis.

A3 CE

Psychotic symptom with suicidal ideation is an ominous sign. Three types of psychotic symptoms are particularly harmful:

- Auditory hallucination commanding suicidal acts
- Thoughts of external control
- Religious preoccupation

A4 E

Malignant neuroleptic syndrome is a rare, potentially fatal idiosyncratic reaction occurring in response to neuroleptic drug therapy like haloperidol. It is characterised by: hyperthermia, fluctuating conscious level, muscular rigidity and autonomic dysfunction with pallor, tachycardia, labile blood pressure and urinary incontinence.

A5 ABCDE

Microalbuminuria is a common discovery in a large proportion of patients with diabetes. Most patients would have had microalbuminuria for a while before a diagnosis is made. Microalbuminuria strongly predicts deterioration to overt diabetic nephropathy, which in turn dictates symptoms. Microalbuminuria is not unique to diabetes and can be detected in a number of common conditions such as urinary tract infections, severe exercise, contamination, prostatic disease and other chronic renal problems. There is a diurenal variation in urinary albumin secretion with the highest levels being at night. Microalbuminuria can be detected with sensitive second generation urine sticks. Normal urine stick tests will remain negative.

A6 ABCD

Extensive damage to the glomeruli results in a loss of both homeostatic and endocrine functions of the kidney (e.g. production of erythropoietin). As a result, there is a progressive rise in blood urea, creatinine and potassium levels. In patients with ESRF lack of erythropoietin leads to anaemia. The progression in the glomerular filtration rate appears to be irreversible once it reaches 0.6–2.4 mL/min. Patients with ESRF have multiple complications including cardiovascular disease and neuropathy. However, the severity of complications does not directly correlate with the extent of renal dysfunction. Non-specific symptoms such as nausea, vomiting and lack of appetite are common in advanced renal disease.

A7 BCE

There are a number of techniques available to detect microalbuminuria. These include spot urine:albumin ratios and 24 h or timed urine collections. More recently, second generation stick tests have been developed that have a good correlation with immunochemical measurements of albumin. Plasma albumin levels remain normal in microalbuminuria. Total protein and albumin levels only change with more advanced renal disease and proteinuria.

A8 ABC

A number of conditions can result in false positive tests for microalbuminuria. A number of common conditions are listed below:

- Urinary tract infection
- Contamination during menstruation
- Strenuous exercise
- Prostatic disease
- Other renal disease
- Prolonged upright posture

A9 ACE

Diabetes with/without microalbuminuria may have been present for a long time prior to diagnosis. Therefore, the patient may have been exposed to increased risk of developing a number of complications including cardiovascular problems, retinopathy, advanced renal disease and diabetic neuropathy. The initial screening tests must aim to identify potentially reversible factors. A recommended list of initial investigations includes:

- Biochemical profile
- Estimation of creatinine clearance (24 h urine collection)
- Assessment of diabetes and its control (fasting glucose and Hb1Ac)
- Fasting lipid profile (total cholesterol, LDL, HDL and triglycerides)
- Regular blood pressure checks and if necessary 24 ambulatory blood pressure monitoring
- Other renal investigations must be guided by the clinical picture (e.g. renal tract ultrasound) and specialist opinion must be sought

A10 E

Hypertension is one of the major indications for the use of AIIRAs. AIIRAs are as effective as other conventional agents in the control of blood pressure. They can be safely combined with other class of antihypertensive agents to achieve target blood pressure levels. However, there is little evidence to demonstrate that addition of an ACEI in the context of hypertension confers extra benefits. AIIRAs are well tolerated with few side effects. Hyperkalaemia is uncommon but can be a serious complication. Regular renal function monitoring is recommended. Trials have demonstrated that the

Answers

AIIRA losartan in comparison to atenolol can significantly reduce cardiovascular morbidity and mortality for a similar reduction in blood pressure. However, despite the benefits of AIIRAs the first choice of agent for treatment of hypertension must be individually tailored for every patient.

A11

The results from trials investigating the role of AIIRAs in heart failure are conflicting. The results range from no benefit to a significant fall in mortality. In the major trials patients were optimally managed before the introduction of an AIIRA. One of the trials demonstrated a trend towards increase in mortality when combining an AIIRA and a blocker in the context of heart failure. However, the changes were not statistically significant. There is currently no evidence to suggest that β-blockers should be discontinued in preference to an AIIRA. Some trials have demonstrated that AIIRA exerts their effects regardless of age, sex and LVEF.

A12 E

About 15–20% of all strokes are believed to be due to atrial fibrillation.

A13 E

Digoxin is the only drug in the list above that is a positive ionotrope and a negative chronotrope; therefore, it is useful in patients with impaired cardiac function.

A14 B

In this elderly woman, rate control is the best management option. This can be quite safely done with digoxin, as long as a digoxin level is checked after oral loading. Verapamil is very good at slowing down the ventricular rate in AF, but it is a negative ionotrope whereas digoxin is a positive ionotrope. If not contraindicated, she should be on warfarin to minimise her stroke risk.

A15 A

Lone AF is AF with no obvious cause in the absence of any other cardiopulmonary abnormalities. It is the most likely to respond to cardioversion.

A16 A

This patient is in extremis and IV medications would work too slowly. You would need to get an anaesthetist to help you sedate the patient and administer a d/c shock. Obviously, this carries increased risk and should only be done as a last resort. LMWH should be started immediately.

A17 BC

The ventricular rate is in the range of 120–150/min
The rhythm is grossly irregular
Yes the rhythm is supraventricular in origin
The duration of QRS is normal
The P waves are not clearly visible

A18 C

The ECG shows irregular rhythm with abnormal P waves. The features are suggestive of atrial fibrillation.

A19 D

Radiation of chest pain to the jaw should always raise suspicion of myocardial ischaemia.

Nitrates may relieve the pain of oesophageal spasm as well as that of myocardial ischaemia. Dyspnoea has a number of aetiologies, including pulmonary embolism, heart failure and tamponade. Coexistent claudication suggests the presence of peripheral vascular disease, and perhaps coronary disease, but does not confirm an acute coronary syndrome (ACS). A past history of cigarette smoking is a risk factor for development of cardiovascular disease, but again does not confirm an ACS.

A20 E

Although myoglobin is the first protein to be released (it rises as early as 30 min), it is not specific to cardiac muscle and is rapidly excreted, making the time window for its detection short.

A21 A

Aspirin leads to a 12% reduced risk of death and 31% reduced risk of reinfarction in the evidence reviewed by the Antiplatelet therapy trialists also in GISSI studies. Several trials have demonstrated the benefit from long-term treatment with β-blockers, by reducing incidence of recurrent MI, and death from all causes. Numerous trials have shown benefit from ACE inhibitor therapy post-MI in those even without evidence of left ventricular impairment. The 4S (Scandinavian Simvastatin Survival Study) demonstrated a benefit from lowering cholesterol with Simvastatin in patients with coronary disease. There is no evidence to support a beneficial effect of nifedipine in post-MI.

A22 E

ACE inhibitors have proven mortality benefits in patients with ischaemic heart disease and heart failure, but the side effects of the drugs are marked and should not be underestimated. Renal function and electrolytes should be checked regularly to look for evidence of renal failure and hyperkalaemia. Cough can be very irritating and mandate stopping the drug, whereas angioedema can be severe. Diarrhoea is not well recognised, but possible with almost any medication.

A23 E

Frusemide, although useful in symptom control, offers no evidence-based long-term prognostic benefits. All the others have been shown to improve mortality in patients with CCF.

A24 D

Phenytoin is the only liver enzyme inducer. It will increase the breakdown of the pill and hence reduce its effect. PC GRABS is a good way of remembering the liver enzyme inducing drugs:

P – Phenytoin
C – Carbamazepine
G – Griseofulvin

Answers

R – Rifampicin
A – Acute alcohol
B – Barbiturates
S – Sulphonylureas

A25 B

Hypothyroidism may also present with dry skin/hair, constipation, hoarse voice, depression, fatigue and slow-relaxing reflexes.

A26 B

Hypothyroidism may also present with dry skin/hair, constipation, hoarse voice, depression, fatigue and slow-relaxing reflexes. All the others are causes of weight loss. Classically, patients with Addison's disease will be pale, thin and dehydrated.

A27 A

As with hypothyroidism, females are more commonly affected than men by an overactive thyroid. The Whickham survey, conducted in the north of England in the 1970s, found a prevalence of thyrotoxicosis or hypothyroidism of at least 2% in females and 0.2% in males. Long-term follow-up over 20 years showed a mean incidence of 0.8/1000 per year for hyperthyroidism.

A28 B

Lid retraction and lid lag are due to increased catecholamine sensitivity of the levator palpebrae superioris muscle and may occur in any form of hyperthyroidism. Exophthalmos and opthalmoplegia only occur in patients with Graves' disease.

A29 C

Hypocalcaemia is a side effect of acute pancreatitis, hypercalcaemia is a cause. Other causes include gallstones, alcohol, post-ERCP, mumps, hypothermia, emboli, pregnancy and drugs.

A30 D

Other drugs that can cause pancreatitis include steroids, pentamidine, didanosine and mercaptopurine.

A31 A

A urine dipstick will give little information that is necessary in the immediate management of acute pancreatitis. All the other tests will and an ECG will rule out myocardial infarction.

A32 B

Antibiotics for acute pancreatitis are only indicated if concurrent infection is present.

A33 E

Traditionally, pethidine has been the antalgic of choice in managing pancreatitis – morphine is a better analgesic but can cause contraction of the sphincter of Oddi. NSAIDS and aspirin would be contraindicated due to the risk of bleeding. Paracetamol would be inadequate.

A34 E

All of the others are recognised complications of acute pancreatitis. Others include pseudocyst formation, bleeding, DIC, hypocalcaemia, hypoalbuminaemia, shock and death.

A35 B

Chronic infection occurs in 85% of infected cases with 15% clearing the virus. Subsequently, cirrhosis occurs in 20–30% of those with chronic disease.

Hepatocellular carcinoma and cryoglobulinaemia are also associated. Needlestick injury has a low risk of transmission of approximately 2% (although this is higher than for HIV) and mother-to-child vertical transmission is about 6%. Breastfeeding is not contraindicated, as there is no evidence that avoiding this decreases rates of mother-to-child transmission – most infants will acquire HCV in utero or in the peripartum period.

A36 C

GMC guidance states "If a patient under your care has suffered harm or distress, you must act immediately to put matters right, if that is possible. You should offer an apology and explain fully and promptly to the patient what has happened, and the likely short- and long-term effects".

This is not a black and white scenario as it is not actual harm but potential harm. You have to weigh up loss of trust if there is a problem and you do not tell her with possible unnecessary anxiety.

A37 B

The acute onset of a generalised neurological syndrome characterised by paralysis is suggestive of an acute infection or toxic effects.

Absence of focal s/s & N blood excludes an abscess.

Tetanus is characterised by tonic seizures in a conscious patient.

Rabies has a protracted onset with features of encephalitis.

Organophosphate poisoning is characterised by strong cholinergic/muscarinic effects and not by a flaccid paralysis.

Spores of *Clostridium botulinum* produce a potent antitoxin under anaerobic conditions (canned foods, pickled vegetables, etc.). It is destroyed by heat, and early stages of botulism include diarrhoea, vomiting and diplopia followed by bulbar paralysis and weakness of limbs. Treatment is with antitoxins and supportive measures.

A38 E

A, B, C and D are features of UMN lesions, which occur when descending tracts are damaged, whereas E will be caused by an LMN lesion.

A39 C

Superior rectus and inferior oblique both elevate the eye, and are innervated by III.

Medial rectus, which adducts the eye, is innervated by III.

The sphincter pupillae is supplied by parasympathetic preganglionic fibres from III which end in the ciliary ganglion. Short ciliary nerves arising from the ganglion convey postganglionic fibres to the sphincter. This muscle constricts the pupil.

The dilator pupillae is supplied by sympathetic fibres. Preganglionic fibres arise from the lateral horn of the spinal cord at the T1 level and end in the superior cervical ganglion. Postganglionic axons reach the dilator by way of blood vessels to the eye.

The oculomotor nerve (III) controls the shape of the lens through its parasympathetic innervation of the ciliaris muscle. The course of the pre- and postganglionic fibres is identical to the supply of the sphincter pupillae muscle. During accommodation when the eyes are used for near vision, contraction of the ciliaris muscle by nerve III makes the lens more convex and adjusts the focal length.

A40 C

A sellar chiasmal mass such as a pituitary adenoma is the most likely cause of a bitemporal hemianopia.

A41 A

The vagus supplies motor fibres to the palate. If there is unilateral damage, the corresponding side of the palate will fail to rise (right, in this case) and the uvula will be pulled towards the normal side (left).

A42 B

The majority of people are left hemisphere dominant, and the middle cerebral artery supplies Broca's area (motor speech) and Wernicke's area (receptive speech).

A43 D

This patient has benign intracranial hypertension, with papilloedema, and the earliest sign of papilloedema on a visual fields test is an enlarged blind spot.

A44 B

Pyramidal weakness in limbs and ataxia, with past history of optic neuritis, in a young or middle-aged woman is highly suggestive of primary demyelination i.e. MS. Relevant investigations include CSF analysis for oligoclonal bands, delayed visual evoked potentials and MRI brain.

Team Working for Foundation Year Doctors

Kilian A. Hynes, Turan S. Huseyin and Brijendra P. Shravat

Introduction

As modern medicine has advanced there have been many changes, none more so than in the area of team working. In the past it was accepted practice for many doctors to work alone, but this is no longer the case. There is a tendency for doctors to develop areas of special expertise and work in teams. There is a multitude of specialist roles that have developed, e.g. falls coordinator, cancer nurse specialist, etc. There have also been great increases in the NHS management team. It is no longer sufficient for a doctor to be a good clinician; they must also be able to work well within a multidisciplinary team.

According to Good Medical Practice[1] there are five essential elements in team working for doctors:

1. Respect the skills and contributions of your colleagues.
2. Communicate effectively with colleagues within and outside the team.
3. Make sure that your patients and colleagues understand your role and responsibilities in the team, and who is responsible for each aspect of patient care.
4. Participate in regular reviews and audit of the standards and performance of the team, taking steps to remedy any deficiencies.
5. Support colleagues who have problems with performance, conduct or health.

We will look at each of these areas in turn.

Respect the skills and contributions of your colleagues

As a foundation year doctor it is very easy to feel underappreciated for the contribution you are making. In order to be understood and appreciated it is often necessary to understand and appreciate the role of others.[2,3] When one does this it may become apparent that other team members make an outstanding contribution and one's burden may not seem so great. Kind words recognising colleagues' contributions will be well received and often reciprocated thus fostering a spirit of harmony and cooperation within the team. The aim would be for it to be a joy to come to work with no task appearing too great.

While working as a foundation year doctor you will get a chance to see how your senior colleagues work and analyse different styles of role model. You should try to look in depth at the full contribution of each person within the team. As you move up the ladder consider areas that you can develop. This reflection can guide you in setting personal objectives. In a large general practice one will find that most doctors will have an area of special interest, e.g. dermatology, child health, practice management, audit, training, etc. In an emergency department one consultant may work predominantly on the shop floor and be highly supportive of juniors but other consultants may not be so visible. It is worthwhile finding out their contribution which may be significant in management, teaching, clinical governance, computerisation or other areas. By looking at your peers in any speciality, it can inspire you to find out what roles you could fulfil in the future.

As well as respecting the contribution of colleagues within your immediate team, you need to appreciate the effort within the greater team of the Trust or the NHS as a whole. The contribution of all staff has to be recognised. For example, without cleaners *Clostridium difficile* and MRSA infection rates would be higher; managers help to police government targets and deliver patient care within budget. You will often hear criticism of GPs, speciality doctors, allied specialities and much maligned managers. One must remember ill-founded criticism of colleagues is contrary to GMC guidance.

It hardly requires mentioning that one should not bully or harass colleagues or discriminate against them on any grounds. However, it is also your duty to confront colleagues who discriminate or bully.[1] In practice it can be difficult to differentiate between bullying and a robust management style. If you think you or a colleague is being bullied it is important to discuss this with the perpetrator as often it will be unintentional and will stop as soon as he or she realises how the offending behaviour is perceived.[4] If this does not succeed you should discuss further with your clinical tutor or the BMA will give impartial advice. If these avenues fail escalation to the Human Resources Department will be necessary and every Trust has a bullying policy which can be enacted. Resolution within the team is ideal as the involvement of outside agencies could damage the team's trust and cohesion that may have taken a long time to build.

Communicate effectively with colleagues within and outside the team

Poor communication is a cause of adverse patient events and duplication of work, e.g. clerking and investigations. When referring a patient either between teams or from a GP to hospital, written and sometimes verbal communication is mandatory. A full clinical summary including the results of relevant investigations should be included. When a patient is seen in hospital outpatients or A&E or discharged from an inpatient bed a letter should be sent to their GP provided they give consent. With shift systems and ward based teams, handover of patients is very important. It can compromise patient care to go off shift without doing so. As a result many doctors feel obliged to stay late which can be a potential cause for team discontent.

Make sure that your patients and colleagues understand your role and responsibilities in the team, and who is responsible for each aspect of patient care

It is important at the start of every job to understand your roles and responsibilities. This can be done by discussing with your predecessor but should be clarified with your supervising consultant. In a situation where you are asked to perform a task beyond your competence think carefully. In the patient's interest it is essential to let your team know you are unable to do a task but attempt to gain this skill by negotiating additional training and supervision particularly while the task is being performed.

As a foundation year doctor you will be responsible for day to day management of inpatients but usually not the overall plan. It is important you communicate any problems to senior colleagues. You should make patients aware that though you are a member of the

team caring for them the final responsibility lies with your supervising consultant and if the patient is unhappy with the care plan the patient needs to discuss this with your consultant. Trying to 'fob off' patients will only lead to difficulties.

Participate in regular reviews and audit of the standards and performance of the team, taking steps to remedy any deficiencies

It is the duty of all healthcare professionals to regularly audit and review the services they provide, continually seeking to improve. This is part of clinical governance and provides quality assurance for patients. As a foundation year doctor you must be involved and you may be required to gather data for audit. The NHS is no longer as hierarchical and though you are junior you should not be afraid to make suggestions to improve patient care.

Support colleagues who have problems with performance, conduct or health

It has been thought in the past that there is a culture of doctors 'sticking together' and 'covering up' for poor performance in colleagues. You must raise your concerns immediately if a threat is posed to patient safety by the performance, conduct or health of a colleague no matter how senior. It is best if you can discuss your concerns with that colleague first to make sure they are well founded. Initially your concerns should be referred in accordance with the policies of your employing organisation (every NHS Trust is required to have a whistle blowing policy). However, if your concerns are not dealt with locally you should inform the GMC or relevant regulatory body. The BMA has an anonymous service for reporting sick doctors.

When reporting concerns they should be well-founded, specific and well thought out. Reporting vague, non-specific concerns or hearsay could be interpreted as bullying or harassment.[4] It would be wise to contact the BMA, GMC or your defence organisation for advice before reporting concerns about a colleague.

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Clinical audit

Understanding Clinical Audit

Khalid Elamin, Raoya Farah and Khaled M.A. Khaled

What is clinical audit?

Clinical audit has been around for a long time and dates to the time of Florence Nightingale who demonstrated that through observing strict sanitary and hygiene rules mortality has dropped from 40% to 2% in injured soldiers. The definition of clinical audit as defined by the Department of Health is that 'Clinical audit is a quality improvement process that seeks to improve the patient care and outcomes through systematic review of care against explicit criteria and the implementation of change. Aspects of the structures, processes and outcomes of care are selected and systematically evaluated against explicit criteria. Where indicated, changes are implemented at an individual team, or service level and further monitoring is used to confirm improvement in health care delivery'.[1] This definition has been endorsed by both NICE and the Healthcare Commission.

Reaching the target sometimes necessitates going through the same process more than once and hence introducing the audit cycle concept which is an integral part of clinical audit. Clinical audit was integrated into clinical governance which was introduced to the NHS in 1993. Full participation in clinical audit by all hospital doctors is now a crucial component of clinical governance.[2]

The government NHS Plan has taken these policies further, with proposals for mandatory participation by all doctors in clinical audit and initiatives to support the involvement of other health care professionals including nurses, midwives and other NHS staff. Annual appraisals of audit results, first introduced in Wales[3], are now generalized to all the NHS.

Why do we carry out audits?

In our striving to provide the best care for our patients, we need a process by which we can gauge our performance and seek further improvement and the tool for this is clinical audit. Clinical audit is an integral part of clinical governance and as outlined above undertaking and participating in clinical audit is now mandatory for all doctors. Clinical audit is now compulsory for summative assessment in Foundation programmes and most Royal Colleges have introduced it into training curricula. It is also an aid to continuing medical education and gives a sense of personal and professional achievement. In addition it may lead to publications and can improve CVs.

Educational benefit from audit

Audit allows a critical review of current best practice on evidence base, highlights the need for specific knowledge, the acquisition of new skills and the development of existing ones. It also improves presentation and communication skills and enables attitudes to be modified when working with others.

How to carry out an audit

Identify the need for change

This may come from a personal experience or concerns raised within the team, department or hospital or a feeling that something could or should have been done better. Identified problems can be categorized into three basic areas:

Structure: This refers to the input of care such as manpower, premises and facilities.

Process: This refers to the provision of care and care pathways, i.e. looking at what is done and how it is done.

Outcome: This refers to the result of clinical intervention.

Setting criteria and standard for clinical audit

A **criterion** is an item of care or aspect of care that can be used to assess quality. The criterion is a written statement. Given below are three examples of criteria relating to an audit in structure, process and outcome, respectively.

All patients requesting an urgent appointment in a GP surgery will be seen that day.

All patients with epilepsy should be seen at least once a year.

All patients on warfarin should have their INR within the recommended limits.

To make the criteria (statement) useful a **standard** needs to be defined. A standard describes the level of care to be achieved for any particular criterion. For example, a standard may state: 98% of patients requesting urgent GP appointments will be seen on the same day; 90% of patients with epilepsy should be seen at least once a year; and 100% of patients on warfarin will have their INR within the recommended limits.

Standards must be set. Bodies like NICE (National Institute of Health and Clinical Excellence), Royal Colleges and other professional regulatory bodies take charge of setting standards.

Data collection

Identify what data needs to be collected, how and in what form it needs to be collected and who is going to collect it.

Assess performance against criteria and standards

Analysing the collected data will allow the identification of any area of care below the predetermined standard. The results can then be used to develop an action plan, i.e. what needs to be done, how it needs to be done, who is going to do it and when is it going to be done.

Achieving and maintaining standards

Without re-evaluating care provision, it is impossible to see if recommendations have been implemented and to what level has care improved. When satisfactory levels of care provision have been achieved it is necessary to make sure these are maintained over time (Figure 1).

Remember: when constructing criteria and standards

- Make unambiguous statements.
- Keep the task focused on the audit project.
- Refer to the literature indicating current practice.
- Choose criteria and standards in line with current practice.
- Ensure the criteria and standards are based on facts.

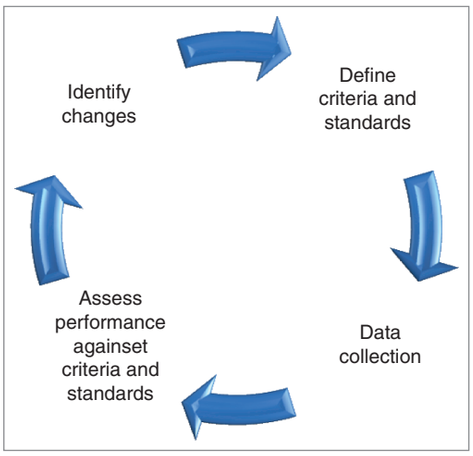

Figure 1: The audit cycle.

Clinical audit is not research

'Research is concerned with discovering the right thing to do; audit with ensuring that it is done right'.[4] Research is about testing hypothesis and creating new knowledge – what works and what does not. It provides the foundations for national and/or local agreement about the kind of clinical treatment and care we should be providing, i.e. it helps to answer the question 'what is best practice?'

Clinical audit asks whether we are doing the things we have agreed we *should* be doing or achieving the outcomes we have agreed we should be achieving, i.e. it answers the question 'are we following the agreed best practice?'

The table below adapted from Madden (1991) and Firth-Cozens (1993) illustrates some of the difference between clinical audit and research.

Research	Audit
Aims to establish what is best practice	Aims to evaluate how close practice is to best practice and to identify ways of improving the quality of health care provided
Is designed so that it can be replicated and so that its results can be generalized to other similar groups	Is specific and local to one particular patient group – results are not transferable to other settings
Aims to generate new knowledge/increase the sum of knowledge	Aims to improve services
May involve a completely new treatment	Never involves a completely new treatment
Is usually initiated by researchers	Is usually led by service providers
Is theory driven	Is practice-based
Is often a one-off study	Is an ongoing process
May involve administration of a placebo	Never involves a placebo treatment
May involve allocating service users randomly to different treatment groups	Never involves allocating patients randomly to different treatment groups

Clinical Audit Do's and Don'ts

Do's

Choose the area of most impact/concern.

Plan carefully step by step.

It is a team exercise; so involve other members.

Advertise in local meetings.

Accurate data collection and analysis.

Don'ts

Don't judge; the results are for improvement of care.

Don't mention names; anonymize and take non-confrontational approach.

Don't make it personal or investigate colleagues.

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Audit of Diagnosis and Management of Anaemia in Patients Referred to a Hospital Support Team for Palliative Care

Becky Hirst

Introduction

Anaemia is common in patients referred to palliative care though there have been no studies to quantify this. The cause can be multi-factorial and can include any or many of:

- Anaemia of chronic disease
- Acute or chronic haemorrhage
- Marrow suppression
- Malnutrition
- Haemolysis (unusual)[1]

There are guidelines for the diagnosis and management of anaemia in the general population[2] and also for patients with conditions such as chronic renal failure,[3] but there are no specific guidelines for the diagnosis and management of anaemia in the palliative care population. This group needs to be considered differently. Although they may have symptoms which might be attributable to anaemia such as fatigue or dyspnoea, this may also be due to their underlying disease and will not change significantly if their haemoglobin is normal. Consequently, management of these patients requires pragmatism when weighing up the benefits and burdens of investigation and treatment. The main challenge is the correct differentiation between anaemia of chronic disease (ACD) and iron deficiency anaemia (IDA). The most useful discriminator of this is the serum ferritin, although it needs to be kept in mind that this is also an acute phase protein and so will be elevated when there is inflammation. Studies show that a ferritin of <40 μg/L in the absence of inflammation, or a ferritin of <70 μg/L in the presence of inflammation are values highly predictive of iron deficiency anaemia.[4]

Aim

The purpose of this audit was to review the literature and develop criteria and standards against which to audit the current practice of diagnosis and management of anaemia. Patients were those referred to a Palliative Care Support Team in a large teaching hospital. The aim is to develop a guideline to improve the care of future patients.

Criteria and standards

- Criterion 1
  - All patients with a low haemoglobin will have further investigation and diagnosis of their anaemia
- Standards
  - Minimum – MCV (mean cell volume) (part of FBC)
  - May need B12, folate, TFTs or other Ix depending on above and history/examination
  - In light of the above, patients with normocytic anaemia will be assumed to have ACD and not given iron supplementation[5]
  - If iron deficiency is suspected despite normocytosis (for instance, because of history), ferritin will be measured
- Criterion 2
  - ACD may present as a microcytic anaemia. This will be differentiated from a microcytic anaemia caused by IDA before iron is started
- Standards
  - Patients with a low MCV will have ferritin measured
  - If this does not indicate iron deficiency they will be assumed to have ACD and not given iron supplementation
- Criterion 3
  - Patients with diagnosed IDA will have cause of deficiency and prognosis considered before treatment
- Standards
  - Chronic blood loss may benefit from treatment to reduce this
  - Asymptomatic patients will not be given iron
  - Symptomatic patients with a short prognosis will have option of transfusion considered
  - Symptomatic patients with a longer prognosis may be offered iron but should be monitored for side effects

Method

Data was collected from 100 consecutive referrals to the Hospital Support Team (providing palliative care outreach) in the Northern General Hospital in Sheffield. Demographic data included age and diagnosis. Anaemia was defined as haemoglobin below the laboratory's normal limits, namely 13.1 g/dL for males and 11 g/dL for females. MCV was also noted. For patients with anaemia, details of any further haematological investigations and any management were noted.

Results

Patients were equally distributed between male and female. The average age of the cohort was 75 and there were 73 malignant diagnoses (69%) and 33 non-malignant (31%), with three patients having more than one significant diagnosis. Three patients with a normal haemoglobin and MCV were on ferrous sulphate 200 mg three times a day. Forty-two patients were defined as anaemic. Of these, the mean haemoglobin for males was 10.2 g/dL with an MCV of 85.1 fl and for females 9.7 g/dL and 80.8 fl respectively.

Criterion 1

One hundred per cent of patients had an MCV measured. Of the 42 anaemic patients, nine (21%) had further investigations. Thirty-five had a normocytic anaemia and of these six were receiving iron. Consequently, this met the standard by 83% for patients appropriately not receiving iron. One patient with a normocytic anaemia had a ferritin of 20 μg/L indicating iron deficiency.

Criterion 2

Five patients had a microcytic anaemia. Of these, two (40%) had a ferritin measured. Neither had a ferritin below 70μg/L. Both of these patients were on iron in spite of their ferritin indicating they were more likely to have ACD so 0% was being treated appropriately with regard to the standard.

Criterion 3

As mentioned above, only one patient in this cohort had been investigated appropriately to diagnose IDA. This was a patient with a normocytic anaemia who had a haemoglobin of 6.1g/dL and was treated with a blood transfusion and an iron infusion. He had advanced metastatic prostate cancer and died a few days after the treatment of his anaemia. Given this, it is debatable whether he obtained any symptomatic benefit. Parenteral iron does not cause haemoglobin to rise any faster than oral preparations except in haemodialysis patients.[6]

At 3 months, only one of the 42 anaemic patients was still alive.

Discussion

Anaemia is present in just under half of patients referred to a hospital support team for palliative care. It is usually mild and is most likely to be normocytic or mildly microcytic. Further investigations are commonly not performed, and even when they are management does not always reflect the results or the patient's symptoms and prognosis.

ACD is the most common anaemia in hospitalised patients in developed countries. Seventy-five per cent of cases are caused by the 'big three' of infection, inflammation (including connective tissue disorders) and malignancy.[7] ACD is immune mediated, causing changes in iron homeostasis. An important part of this is that there is increased uptake and retention of iron into the reticuloendothelial system so it is less available for use by erythroid progenitor cells. This is also why serum iron measurements may be low in ACD.

Although the MCV can help guide as to the type of anaemia, a small proportion of people with a normocytic anaemia will have iron deficiency, and 20% of elderly patients with an MCV < 75fl will not be iron deficient. A serum ferritin can help distinguish between ACD and IDA after taking into account the fact that it may be raised due to also being an acute phase protein.[5] A summary of results of haematological investigations in ACD and IDA is shown in Table 1.

	ACD	IDA
Haemoglobin	7–11g/dL	↓
MCV	N or ↓	Usually ↓ (<76fl)
Ferritin	N or ↑	↓
Serum iron	N or ↓	↓
TIBC	N or ↓	↑
Transferrin receptor levels	N	↑
Bone marrow	Fe stores + +	No Fe stores

Table 1: Discriminating between IDA and ACD[8].

Why is it important to tell the difference between IDA and ACD?

Iron is not a risk free treatment. Side effects include nausea, epigastric pain and gastrointestinal disturbances even to the extent of faecal impaction. Palliative care patients frequently complain of nausea and constipation without iron tablets worsening the situation. Ingestion of iron tablets is a common cause of accidental overdose in children, causing bowel infarction and liver failure.[6]

Taking iron when stores are saturated causes overload, for instance in ACD which is primarily a disorder of iron utilisation. The body has no physiological mechanism to excrete the excess, and once all transferrin is saturated the surplus will be laid down in tissues. It causes potentially fatal tissue damage due to the formation of highly toxic hydroxyl radicals resulting in hepatic dysfunction, abdominal pain and cardiomyopathy with dysrhythmias and heart failure. It also increases the risk of bacteraemia.[9]

Summary and recommendations

- Most patients in the palliative care population will have ACD rather than IDA
- MCV and ferritin are the most useful tests to discriminate between IDA and ACD
  - Patients with an MCV > 75fl are unlikely to have IDA
  - Patients with an MCV < 70fl are likely to have an IDA
  - A ferritin of <40μg/L confirms IDA
  - A ferritin of >70μg/L in the presence of chronic inflammation means IDA is unlikely
- Iron should not be prescribed unless
  - Definite iron deficiency has been diagnosed
  - The patient has symptoms attributable to anaemia
  - The patient has a long enough prognosis to benefit (treatment should give a rise in haemoglobin of 1–2g/dL in about 3 weeks)

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Clinical audit

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Management of Malignant Pericardial Effusion

Rohit Malde and Amit Bahl

A 65-year-old woman presents to the medical admissions unit (MAU) with a 4-week history of progressive dyspnoea, weight loss, swelling over feet and a hacking cough worse on lying down and improving on leaning forward and sitting upright.

She was previously diagnosed as having metastatic breast cancer and had recently completed her course of palliative chemotherapy a few months back. She continues on endocrine treatment with tamoxifen. There was no other relevant past history.

Examination revealed an elevated jugular venous pulse. The heart sounds were faint and distant while there were no crepitations or rhonchi. There was subscapular dullness to percussion. The patient underwent a chest X-ray at the MAU which is shown in Figure 1.

What would you do next to confirm your diagnosis?

Transthoracic echocardiography (ECHO): This is the investigation of choice to confirm the diagnosis, quantify the amount of effusion and aid its drainage. Pericardial fluid appears normally as an echo-free space in both 2-D images and M-mode images. Mild effusions accumulate posterior to the left ventricle and are more apparent in systole, while large effusions are evident all around the heart and are seen throughout the cardiac cycle. At times, diastolic ventricular filling may be affected due to cardiac compression. Normally 15–50 mL of fluid may be present (Table 1).

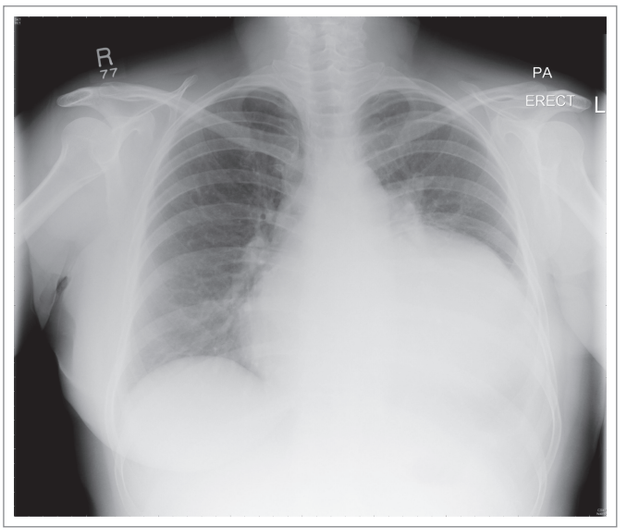

Figure 1: A chest radiograph showing a large cardiac shadow producing a characteristic water bottle heart.

Size	Small	Medium	Large
Volume (mL)	<100	100–500	>500
Localization	Localized	Circumferential	Circumferential
Width (cm)	<1	1–2	>2

Table 1: Sizing of pericardial effusion by echocardiography.

Computerized tomographic (CT) scan of the thorax: This can also detect pericardial effusions and in addition can provide additional information on the status of the vena cava, heart chambers, pleural changes and help in identifying any space occupying masses within the pericardium and adjacent mediastinum and lungs.

Electrocardiography: These changes are usually non-specific but might still be useful to support the diagnosis. Low voltage QRS complexes are usually seen. However at times, even ST segment elevation, T wave inversions may be seen. Electrical alterans and arrhythmias may also be associated in severe cases.

Other non-invasive investigations: FBC, U & E's, CRP, clotting profile (important before undertaking invasive procedures), tumour markers like CA 15-3 (relevant in this clinical scenario, although non-specific); CA 125, Serum alpha fetoprotein (depending on other scenarios) and blood cultures if appropriate.

The ECHO demonstrated a large pericardial effusion measuring 5.2 cm posteriorly and 4.5 cm anteriorly with slight right atrial diastolic compression but no significant right ventricular collapse. There was good left ventricular function.

What would you like to do next?

Pericardiocentesis

This is the modality of choice to determine the cause of the effusion, and to decompress the pericardial cavity which may be responsible for the haemodynamic insult. The pericardiocentesis procedure should be ideally done by a specialist in a coronary care unit or Cathlab with facilities of haemodynamic monitors and fluoroscopy/echocardiography/ultrasonography. Although traditionally fluoroscopic guidance has been commonly used in the past, echo guidance has gained much popularity due to obvious reasons of radiation exposure with the former. However, in emergency scenarios which are quite often the case with cardiac tamponades, this procedure could be carried out in the A & E under blind guidance to treat the haemodynamic compromise. Even a small amount drained can give significant benefit.

The patient is positioned in the sitting position at 30–45° head elevation to increase the pooling of fluid towards the inferior and anterior surface, thus maximizing fluid drainage. A 18–20 gauge cardiac needle or long central venous catheter is introduced under the xiphoid process and angled upwards and towards the left shoulder at an angle of 15–20° from the abdominal wall until the needle tip is posterior to the rib cage and fluid is aspirated in the syringe.

Malignant pericardial effusion

Malignant pericardial effusion often is undiagnosed in patients with cancer, although as many as 10–15% of patients with cancer will have some degree of pericardial effusion at autopsy.[1] Pericardial effusions in patients with cancer are malignant about one half of the time. The primary tumours most often associated with pericardial effusions are lung (40%), breast (23%), lymphoma (11%) and leukaemia (5%).[2] Non-malignant causes in a cancer

patient include radiation-induced pericarditis, doxorubicin- and daunorubicin-related pericarditis or myocardial dysfunction, infections, haemolytic uraemic syndrome after bone marrow transplantation, chronic graft-versus-host disease, malignant hepatic involvement with portal hypertension, microvascular tumour spread in lungs with secondary pulmonary hypertension and superior vena caval obstruction with infiltration of thoracic duct leading to a chylous pericardial effusion.

The pericardial fluid may be serous, serosanguinous or haemorrhagic. It may be distinguished from cardiac chamber blood because of its absence of clot formation and because its haematocrit is lower than that of venous blood. A red blood cell count >100,000/mm^3 is suggestive of trauma, malignancy or pulmonary embolism (rare). Chylous fluid implies injury to the thoracic duct by trauma or infiltration. The fluid should be sent for a cell count, Gram's stain and culture, cytology, acid-fast bacilli, glucose, protein, lactate dehydrogenase (LDH) and specific gravity. The parameters listed in Table 2 have a high sensitivity for differentiating exudates versus transudates. An elevated protein level >6.0 g/dL often indicates tuberculous, purulent or parapneumonic effusion. Isolated increased fluid LDH (>300 U/L) with normal serum LDH is most likely due to malignancy. Low pericardial fluid glucose level (<60 to 80 mg/dL) may be due to parapneumonic, rheumatoid, tuberculous or malignant effusion. However, no diagnostic test of pericardial fluid is specific for effusion associated with post-pericardiotomy syndrome, radiation or uraemic pericarditis, hypothyroidism or trauma. The overall diagnostic yield of pericardial fluid analysis and biopsy is low (about 20%), emphasizing the importance of clinical history and examination.

In patients with known malignancy, pericardial cytology has a good sensitivity and specificity with an overall cytological accuracy of 95% with a predictive value of the correct histological type of cancer of nearly 77%.[3] Potential complications of pericardiocentesis are laceration of the myocardium or coronary artery, haemorrhage, arrhythmia and cardiac arrest.

	Exudate	Transudate
Etiology	Malignancy Infectious/ parainfections Post-pericardiotomy syndrome Collagen vascular diseases	Radiation Uraemia Hypothyroidism Trauma
Specific gravity (g/mL)	>1.015	<1.015
Total protein (g/dL)	>3.0	<3.0
Fluid/serum protein ratio	>0.5	<0.5
Fluid/serum LDH ratio	>0.6	<0.6
Fluid/serum glucose ratio	<1.0	>1.0

Table 2: Effusion: exudate versus transudate.

The patient drained nearly 500 mL of serosanguinous fluid, which showed malignant cells on cytopathology examination. She underwent further staging investigations in the form of a CT scan of chest, abdomen and pelvis, which showed evidence of mediastinal lymphadenopathy and multiple liver metastasis. She had developed progressive disease on tamoxifen. Since her general condition at that point in time did not permit further systemic treatment in the form of aggressive chemotherapy, it was decided to change her hormones to an aromatase inhibitor (anastrozole).

The patient remained well for 2 months on anastrozole only to present later with similar symptoms and a recurrent moderate-sized pericardial effusion.

How will you manage her now?

The decision to treat a patient is more often based on physiologic issues and symptoms rather than on the size or appearance of an effusion. The standard management of patients with recurrent malignant pericardial effusion is undefined. Hence, her therapeutic options would differ from centre to centre and would range from repeat pericardiocentesis, creation of a pericardial window with the help of a balloon catheter, to percutaneous insertion of catheter (typically by subxiphoid route) in the pericardial space with instillation of sclerosing agents like talc or tetracycline or chemotherapeutic agents like thiotepa, doxorubicin, bleomycin, cisplatin, nitrogen mustard, fluorouracil, teniposide or even radionuclides.[4]

In some tertiary centres, more invasive procedures are employed at times in the form of a thoracoscopic partial pericardiectomy or subtotal pericardiectomy. Besides these, there are various non-invasive modalities including mediastinal irradiation and systemic chemotherapy that may be considered in the patients presenting with mild or slowly progressing symptoms. Mediastinal irradiation for malignant effusions has an overall 50–60% short-term response rate, with the best success seen in radiosensitive tumours such as lymphoma or leukaemia.[5]

This patient had a repeat pericardiocentesis to palliate her symptoms in the short term and is currently receiving systemic chemotherapy with oral capecitabine.

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Good clinical care

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Good clinical care assessment questions

Questions

1. All of the following clinical signs are usually seen in patients with pericardial effusion except?
   A. Subscapular dullness on percussion
   B. Faint heart sounds
   C. Decreased vocal fremitus
   D. Elevated jugular venous pulse
   E. Pulsus paradoxus
2. The characteristic finding with pericardial effusion seen on a chest X-ray is
   A. Bats wing shadow
   B. Water bottle shadow
   C. Positive silhouette sign
   D. Collar sign
   E. Inlet to outlet shadow
3. The investigation of choice to confirm pericardial effusion is
   A. Cardiac catheterization
   B. CT scan of thorax
   C. Transthoracic echocardiography
   D. Chest X-ray
   E. MUGA scan
4. Transthoracic echocardiography helps
   A. To diagnose pericardial effusion
   B. To quantify the amount of fluid
   C. To determine the effects of the effusion on the heart chamber functions
   D. To aid accurate drainage of the effusion
   E. All of the following
5. Which of the following is the correct statement during the procedure of pericardiocentesis?
   A. The needle is introduced into the 2nd intercostal space.
   B. The patient is positioned in the supine position at 30–45° head elevation.
   C. After percutaneous insertion the needle is angled upwards and towards the right shoulder at an angle of 15–20° from the abdominal wall.
   D. A 20G cardiac needle is introduced just under the xiphoid process.
   E. This procedure is always done under echocardiographic guidance.
6. Which one of the following tests is suggestive of a malignant pericardial effusion?
   A. Specific gravity of 1.012
   B. A red blood cell count of 1000/mm^3
   C. Serum LDH 300 U/L and fluid LDH of 210 U/L
   D. Serum glucose 5.8 mmol/L and fluid glucose of 6 mmol/L
   E. A fluid/serum protein ratio of 0.5
7. What percentage of patients with terminal cancer have evidence of pericardial effusion at autopsy?
   A. 35–45%
   B. 10–15%
   C. 1–2%
   D. 50–60%
   E. 75–80%
8. Which of the following is of least relevance in the consideration of a known cancer patient presenting with pericardial effusion?
   A. History of mediastinal radiotherapy in the past
   B. History of receiving doxorubicin chemotherapy in the past
   C. History of adjuvant hormone therapy with tamoxifen
   D. History of recent bone marrow transplant
   E. Patient with a superior vena cava obstruction with infiltration of the thoracic duct
9. Which of the following primary tumours is most often associated with pericardial effusions?
   A. Lymphoma
   B. Leukaemia
   C. Lung cancer
   D. Breast cancer
   E. Prostate cancer
10. Which of the following therapeutic options could be offered to a patient presenting with recurrent malignant pericardial effusion?
    A. Creation of a pericardial window
    B. Partial pericardiectomy
    C. Instillation of thiotepa
    D. Mediatinal radiation
    E. All of the above

Answers

1. **C**
2. **B**
3. **C**
4. **E**
5. **D**
6. **C**
7. **B**
8. **C**
9. **C**
10. **E**

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Pain Control in Palliative Care

Becky Hirst and Irene Lawrence

Peter is a 62-year-old man who has recently been diagnosed with locally invasive prostate cancer. He comes to see you complaining of pelvic pain. It is somewhat relieved by paracetamol, but is waking him up at night. What would you prescribe?

Pain is a common symptom – around 75% of patients with cancer will experience it during the course of their illness[1] as will a significant proportion of those with non-malignant disease.[2] In order to prescribe analgesics appropriately, we need to ascertain the most likely cause of the pain with a careful history, examination and investigations if required. A useful framework for this is to think of PQRST.

- Palliative factors – 'What makes it better?'
- Provocative factors – 'What makes it worse?'
- Quality – 'What exactly is it like?'
- Radiation – 'Does it spread anywhere?'
- Severity – 'How bad is it?' 'How does it affect what you can do?'
- Temporal factors – 'Is it worse at any particular time of the day or night?'[1]

Peter's pain is most likely to be caused by damage to tissues from the cancer or from treatments such as radiotherapy. A good foundation for pain relief prescribing is the WHO Analgesic Ladder, shown in Figure 1, although this has never been validated.[3]

Often patients have been taking paracetamol under their own initiative, so it would be sensible to prescribe a weak opioid such as codeine with this, or jump straight to step 3 if the pain is very severe. Patients whose pain is constant should take analgesics regularly – 'by the clock' – rather than waiting for the last dose to wear off and the pain to return.[1] It is common to prescribe a compound preparation of codeine and paracetamol such as co-codamol. This comes in several strengths, the most common being one with 8 mg of codeine combined with 500 mg of paracetamol and a stronger one with 30 mg of codeine.

Prescribing strong opioids

Pain may be unrelieved by a weak opioid, or the patient may be one of the 10% of Caucasians who do not metabolise codeine into its active form due to a genetic polymorphism of the Cytochrome p450 enzyme complex, and so get no analgesic benefit. These are reasons why you might want to move to a step 3 strong opioid. However, there are also genetic differences in the metabolism of strong opioids including oxycodone and fentanyl, with some patients being 'fast metabolisers' and others 'slow metabolisers'. This can lead to variation in response between patients on a dose-for-dose basis and is worth bearing in mind.[4] There is little benefit from changing to a different weak opioid. Strong opioids are useful and safe drugs, especially when prescribed orally. They have had an unjustifiably bad press since Harold Shipman. Patients are often anxious about the implications of taking these drugs. Careful counselling improves concordance.

Common questions patients ask are as follows:

Q. Won't this make me feel 'drugged up'?

A. Some people feel a little bit drowsy for the first few days when they start this type of medicine. We will start at low doses to help prevent this and it should wear off after a few days. If it doesn't, that tells me this might not be the best painkiller for your type of pain and we should re-assess the situation. Other painkillers may be more helpful. (It sometimes helps to switch a patient from morphine to another opioid such as oxycodone or fentanyl.)

Q. Won't I get addicted?

A. No. This is not a problem if people are taking these medicines for pain as the pain acts like an antagonist. It is true that your body develops what we call a physiological dependence, due to changes caused by the drug. We see this with lots of different kinds of drugs and all it means is

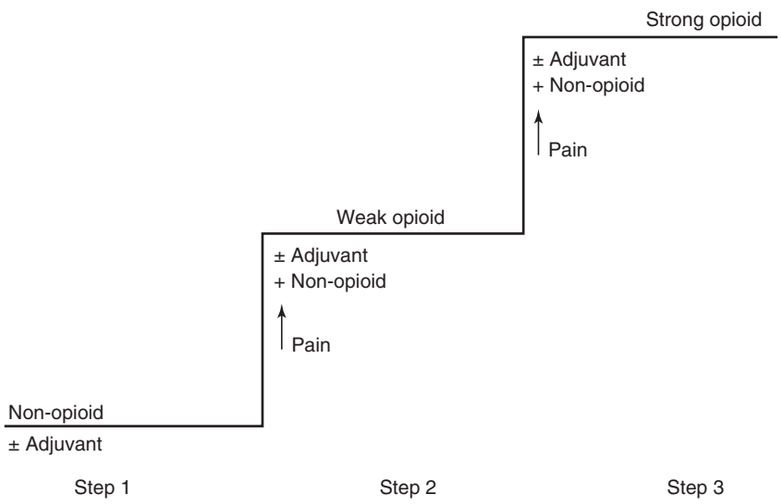

Figure 1: WHO pain ladder.

that we mustn't stop the medicine suddenly if you've been on it for sometime. It does not mean you are addicted.[5] Some patients do become pharmacologically tolerant – these need to be referred to a specialist.

Q. Won't my body get used to it and I'll need bigger and bigger doses?

A. This usually means that the pain is bigger, maybe as the disease is progressing, rather than that you've become tolerant to the drug.[6] I think of the pain as being like a sponge that soaks up the pain killer. A bigger sponge can soak up more.

Q. What about driving?

A. Legally, you do not need to tell the DVLA you are on these drugs.[7] However, we would recommend that you don't drive until we have got you on a stable dose and you are feeling ok. Then, go out with someone you trust, in daylight, on roads you know and see how it goes. Also, be aware that although having an alcoholic drink is not contra-indicated, it might make you sleepier after both.

Choosing and initiating a strong opioid

Which you choose depends on your familiarity with different drugs, cost and patient preference. You may need to take into account patient factors such as renal or hepatic function. There is an increasing choice (see Table 1). In the primary care setting, it would be reasonable to start with a small dose of a short acting opioid such as 2.5 mg of morphine immediate release solution (Oramorph®) every 4 h and as required. Review the patient after a couple of days and calculate a dose for a 12 hourly modified release preparation such as MST Continus® or a 24 hourly modified release preparation such as MXL®. Some patients will tolerate starting straight on a modified release preparation. If you choose to start a fentanyl patch, remember this will take up to 72 hours to get to steady state, so warn the patient that the amount of short acting analgesic they require may not decrease initially. When you increase the dose of a modified release preparation or patch do not forget to increase the breakthrough dose in proportion. The usual dose of immediate release opioid is usually a sixth of the daily dose.[8] For fast acting breakthrough pain relief, for instance if a patient with bone metastases gets pain on movement, then fentanyl lozenges (Actiq®) can be helpful.

Some patients will feel nauseated or vomit when starting opioids. As with the drowsiness, this will wear off for most but it is worth giving them an anti-emetic, such as domperidone 10 mg as required or haloperidol 1.5 mg at night, to take for the first few days, especially in the outpatient or primary care setting. Ninety per cent of patients taking opioids (weak or strong) will suffer from constipation,[9] so a laxative should be prescribed routinely unless there is a definite reason for not doing so such as in a patient with an ileostomy.[8] Patients experience significantly less constipation with fentanyl compared to other opioids.

Peter has been stable for some months on Zomorph® 30 mg twice a day with Oramorph® 10 mg as required. He comes to see you because he has developed a new pain in his back, radiating down his left leg associated with unpleasant numbness and tingling. The Oramorph® makes no difference and he complains it is making him drowsy and he is having vivid dreams. An MRI shows a metastasis of the second lumbar vertebra with compression of the nerve root, but no spinal cord compression. What would you prescribe now?

Opioid	Oral potency compared to oral morphine	Potency when given parenterally	Typical starting dose	Comments
Codeine	One-tenth as potent	N/A	16–60 mg four times a day (as co-codamol 8/500 or 30/500)	Very constipating. Not metabolised to active compound by everyone
Morphine		Twice as potent (10 mg oral = 5 mg s.c.)	Immediate release (IR) solution 2.5–5 mg 4 hourly and as required	Cheap. Avoid or reduce dose in renal failure
Oxycodone	Twice as potent (10 mg oral oxycodone = 20 mg oral morphine)	Twice as potent (10 mg oral = 10 mg morphine s. c. = 5 mg oxycodone s.c.)	1.25–2.5 mg 4 hourly and as required	Expensive. Reduce dose in renal failure
Diamorphine	Only given parenterally	Three times as potent (30 mg oral morphine = 10 mg diamorphine s.c.)	If opioid naïve, 5–10 mg/24 h via syringe driver. Otherwise, calculate from oral opioid	Relatively cheap. Dissolves in small volume of water so good if large doses required
Fentanyl patch	Twenty-five microgram/hour patch equivalent to 90 mg of morphine in 24 h	Seek specialist advice	12[a] – 25 μg/h patch	Expensive. Safe in renal failure. Remember to prescribe adequate breakthrough analgesia: i.e. morphine IR 15 mg for 25 μg/h patch, 30 mg for 50 μg/h patch, etc.

Table 1: Opioid analgesics.
[a]Licensed only for titration although commonly used for initiation.

Opioid toxicity

Oral opioids rarely cause respiratory depression. More commonly, patients complain of being drowsy and having vivid dreams or hallucinations. Classically they will say that they think there is someone standing behind them but when they turn there is no one there. Oxycodone is les prone to this side effect than morphine. Myoclonic jerks may cause the patient to complain that they keep throwing cups of tea. Development of opioid toxicity may indicate a change in renal function with reduced clearance of neuro-excitatory metabolites or it may indicate that a pain is not particularly opioid responsive. Haloperidol can help with these symptoms while you sort out the underlying cause.

Some patients' pain seems completely unresponsive to opioids, and may actually worsen with rapidly escalating doses of strong opioids. This may be caused by opioid-induced hyperalgesia and may be more common than previously thought. This is a palliative care emergency and advice should be sought urgently from either a specialist palliative care team or a pain team. Patients may benefit from a dose reduction, but they may also need a switch to an alternative opioid.[10]

Adjuvant analgesics

These are drugs which work alongside opioids at any step of the WHO ladder.

Non-steroidal anti-inflammatory drugs (NSAIDS)

Given the inflammatory nature of many cancers, there is strong argument for prescribing an NSAID early on in the patient's disease, unless there are contra-indications such as asthma or active peptic ulceration, or cautions such as renal impairment when NSAIDs should be avoided if at all possible. If they have not already been prescribed, they can be a useful adjunct in treating bone pain due to metastases.[1] Choice is determined by the balance of benefit versus possible harms of treatment, particularly gastrointestinal disturbance (see Table 2). All NSAIDs are associated with an increased risk of thrombotic events, particularly myocardial infarction, but the excess mortality is small and associated with high doses over a prolonged period, which is less of an issue in the population with advanced disease. It is also important to remember that NSAIDs interact with many other drugs, including warfarin.[9]

Agents for neuropathic pain

Common drugs used for neuropathic pain include anti-convulsants, such as gabapentin and carbamazepine, and tricyclic anti-depressant drugs such as amitriptyline. Although listed for use in this situation in the British National Formulary, amitriptyline is only licensed in trigeminal neuralgia. Amitriptyline is safe in renal failure, but is contra-indicted in patients with arrhythmias. Anti-muscarinic side effects can limit the dose tolerated, but slow titration can help reduce this (see Table 3).

Drug	Class	Typical starting dose	Comments
Amitriptyline	Tricyclic anti-depressant	10–25 mg at night, titrate up according to response to 100–150 mg at night	Safe in renal failure. Contra-indicated in arrhythmias. Anti-muscarinic side effects may be problematic
Gabapentin	Anti-convulsant	100 mg three times a day, titrate up to 600–900 mg three times a day	Reduce dose in renal failure. Can make some patients very sleepy

Table 3: Neuropathic agents.

Peter is admitted to the hospice. He is clearly terminal and although his pain has remained stable on his previous dose of opioid, he is now no longer able to swallow. How would you manage him?

The subcutaneous (s.c.) route is used to continue to provide analgesia once patients are unable to swallow. Because opioids given by this route are more potent, it is important to reduce the dose (see Table 1). A dose equivalent to the 24 h requirement can be given using a syringe driver, and then boluses can be given if breakthrough analgesia is required. This also means that you need to prescribe oral opioids separately from parenteral opioids on the drug chart. For instance, you cannot write morphine immediate release 10 mg oral/s.c. on a drug chart as the s.c. dose is the equivalent of giving 20 mg by mouth. If a patient is on a fentanyl patch, leave this *in situ* and add extra via a syringe driver.

NSAID	Typical dose	Comments
Ibuprofen	400–800 mg three times a day	Non-selective COX inhibitor. Low incidence of gastrointestinal events. No increased risk of myocardial infarction at doses up to 1200 mg/day
Naproxen	250–500 mg twice a day	Non-selective COX inhibitor. May be particularly good for paraneoplastic pyrexia. Some evidence of increased thrombotic risk
Diclofenac	50 mg three times a day	Preferential COX-2 inhibitor. Does not appear to effect platelet aggregation *in vivo*. Can be used s.c.
Etoricoxib	60–90 mg daily	Selective COX-2 inhibitor. Lower risk of gastrointestinal events. Contra-indicated in those with ischaemic heart disease, peripheral vascular disease or moderate/severe heart failure

Table 2: Non-steroidal anti-inflammatory drugs.

Prescribing

Summary

Pain is a common symptom, but much of it can be controlled with good prescribing based on the use of the WHO Pain Ladder. It is important to review the patient regularly. Patients often require several different analgesics because there may be different mechanisms underlying the pain. Strong opioids are safe and are there to be used. It is important to remember that there are many non-pharmacological methods of pain control, such as TENS and acupuncture. Using these in conjunction with drugs can enhance management. There are also more invasive measures such as spinal analgesia and nerve blocks. Hospital support teams and hospices provide advice and support for generalists providing palliative care in hospitals and the community, and their guidance should be sought at any time, either with respect to drugs or to access more specialist measures.

Top tips for prescribing opioids

1. Start low and titrate up
2. Warn patients about side effects
3. Prescribe laxatives and anti-emetics routinely
4. Increase the breakthrough dose whenever you increase the regular dose
5. On hospital drug cards, prescribe oral and injectable preparations separately due to differing potencies
6. Controlled drug prescriptions for FP10s and hospital discharge need to include the form of preparation (e.g. tablets, capsules, liquid), the strength (e.g. 10 mg/mL) and the total amount to be dispensed *in words and figures* 'like writing a cheque'
7. If in doubt, show the prescription to a pharmacist

Acknowledgement

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Advance Directives – A practical approach

Rajeena Ackroyd

Introduction

Patients are taking a more active role in decisions around what treatment they would or would not want. A competent patient cannot request a treatment that is thought to be not in their best interests but is permitted to refuse a treatment.[1] An advance directive allows this to be extended to a time in the future when the patient has lost their capacity to make decisions.

Definition

An advance directive (also known as a living will or an advance statement) is an instrument whereby a competent person can give instructions about what treatments they wish to receive under certain circumstances should they subsequently lose the capacity to decide for themselves at a future time.

Reasons for and against advance directives

Arguments for advance directives	Arguments against advance directives
Extends autonomy regarding treatment decisions when individual unable to express wishes	Open to misinterpretation or implemented in circumstances the patient had not foreseen
Individual retains control over decision-making	Language used in advance directive may be ambiguous
Promotes dignity in incompetent patient	There may be no information about what in the patient's view constitutes a good quality of life
Preserves well-being	
Protects individual from futile interventions	Written when patients healthy – can only hypothesise on future events

The Mental Capacity Act

The Mental Capacity Act provides a legal framework on how decisions should be made about patients who lack capacity.[2] Before this act came into effect in October 2007, advance directives were regulated by common law. Therefore, this is the first time the status of advance directives is defined in statute.[3]

Advance directives are legally binding if valid and applicable within the context of the act. Advance directives are referred to in the act as 'advance decisions to refuse life-sustaining treatments'. Therefore only advance directives that *refuse* treatment are legally binding. However, a request for specific treatments in an advance directive should be taken into consideration by the health care team with regard to making decisions on what are in the patient's best interests but are not legally binding.

In order that the advance directive is valid and applicable under the Mental Capacity Act, the following criteria must be met:

- The individual must have had capacity when the advance directive was produced.
- The patient must have been over 18 years of age when the document was made.
- The advance directive should be in writing.
- The advance directive should be signed by the patient (if the patient is unable to sign it, then another person can do it on behalf of the patient in the patient's presence and the presence of a witness).
- It must be signed by a witness in the presence of the patient.
- An individual cannot request a treatment or refuse the provision of basic care, defined as the maintenance of bodily cleanliness, relief of sustained and serious pain, and the provision of oral nutrition and hydration.[1]
- The document should be as specific as possible with regard to what treatments are to be refused, and in what situations. This can be in layman's terms.
- A statement needs to be included in the advance directive that the directive should apply 'even if life is at risk'.
- The patient must have lost capacity in order for the advance directive to come into effect.

The advance directive should have been made without duress and it is recommended that the patient's GP is aware of its existence and has a copy. The document should be regularly reviewed and patients who have advance directives that were compiled before the Mental Capacity Act should ensure that they meet the requirements of the act.

An advance decision to refuse treatment is not valid:

- If the patient still has capacity to make the decision
- If the patient has subsequently created a lasting power of attorney which is an individual who is given the authority to make health (and financial) decisions for the patient should they lose capacity and overrides any advance directive
- If since the advance directive has been made the patient has acted in a way that is inconsistent with the decision that was expressed in the directive
- If the treatment applicable to the current clinical situation is not specified in the advance directive
- If the document is not signed or witnessed

Professional's obligation

There should be an effective system for identifying patients with advance directives and their existence should be recorded in the patient's notes in conjunction with local policies and guidelines. It is important to ensure that the advance directive is valid and applicable to the situation at hand.

If the situation falls within the terms of the advance directive, the document should be respected.

If there is any doubt about the validity or reliability of an advance directive, then treatment should be provided if appropriate

and legal advice should be considered. The evidence of validity can sometimes be difficult to prove.

In England and Wales under the Mental Capacity Act, health professionals are protected from liability for providing treatment if there is doubt about the validity or applicability of an advance directive, and no liability is incurred for withholding or withdrawing treatment if those responsible for the patient's care reasonably believe that a valid and applicable advance decision exists.

Conclusion

Doctors are increasingly likely to be involved with patients who have advance directives and also may have a role in advising patients on the circumstances in which an advance directive may be appropriate, for example if the patient has a certain clinical condition e.g. dementia, or a terminal illness such as cancer or motor neurone disease,[4] it is then particularly important that patients understand fully the treatments that they are refusing at a future time and the implications. Therefore having a good legal and ethical understanding of advance directives and their application will prove invaluable to all health care professionals.

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Intercostal Chest Drain Insertion

Max Yates, Uta Hill and Tim Cotter

Insertion of an intercostal chest drain is an important procedure used in many different clinical settings both for diagnosis and symptom relief. Therefore, doctors of most specialities need to be capable of performing this procedure safely. Chest drain insertion is indicated in the treatment of patients with malignant pleural effusions, empyemas and traumatic haemopneumothoraces. In addition, it is a necessary therapeutic intervention in patients with tension pneumothorax (after initial decompression with a needle), persistent or recurrent pneumothorax after simple aspiration and large secondary spontaneous pneumothorax in patients over 50 years of age.[1-3]

In the following article, the insertion of small bore chest drains (8-14F) with the aid of a guidewire by a Seldinger technique is described, as these are indicated and widely used in the management of pneumothoraces and pleural effusions. In certain circumstances, the insertion of larger bore chest tubes (16-28F) may be necessary, but this should be performed under the supervision of respiratory physicians, thoracic surgeons or trauma consultants.[2]

It is essential that the doctor performing the procedure has been adequately trained. It is therefore recommended that they gain experience in chest drain insertion under appropriate supervision and guidance.

Preparation

It is important that the patient is fully informed about the procedure and that written consent is obtained except in the case of emergency situations where clinicians should act in the best interest of patients. Immediately before performing intercostal drainage, the site and side for insertion of the chest tube is checked by reviewing the clinical signs and the chest radiograph.

Materials

It is crucial to obtain all necessary equipment prior to starting the insertion of a chest drain:

- Sterile drapes, gown and gloves
- Skin antiseptic solution, e.g. iodine or 2% chlorhexidine solution
- Gauze swabs
- A selection of syringes and needles (21–25 gauge)
- 10–20 mL of 1% lidocaine solution
- Sterile scalpel and blade
- Suture (e.g. ‘1’‘silk)
- Adhesive dressing, e.g. Mefix or Mepore
- Large bore needle with curved end through which guide-wire can be passed
- Guidewire with dilators
- Seldinger chest drain
- Connector tubing
- Closed drainage system (including sterile water to allow for the underwater seal)

In most hospitals, this equipment is available in kit form.

Positioning the patient

The preferred position for the patient is on the bed with the arm on the side of the lesion behind the head (as illustrated in Figure 1). This will allow access to the ‘safe triangle’ which lies between the lateral border of the pectoralis major, the anterior border of the latissimus dorsi and a line superior to the horizontal level of the nipple (Figure 1). This area should now be examined and the optimal site for insertion, just above the superior border of the rib to avoid the neurovascular bundle, should be marked.

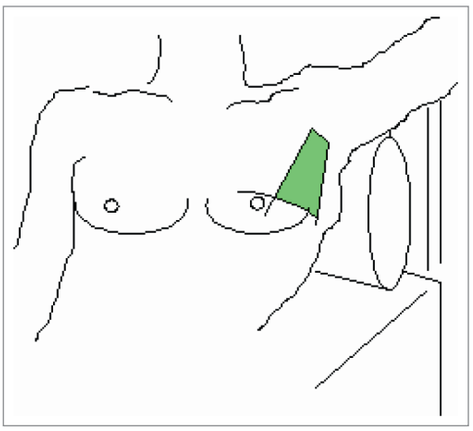

Figure 1: Safe triangle for chest drain insertion. Modified from D. Laws et al.[3]

Analgesia

This is a painful procedure and pain control is of paramount importance during insertion of the drain, whilst the drain is *in situ* and during drain removal. It is important that the requisite analgesia is written up at the start of the process. Prior to the procedure, it is necessary to assess the patient’s needs in relation to pain and anxiety control.

Morphine-based analgesia can be given 30 min prior to the procedure, e.g. liquid oral morphine (2.5–5 mg) or intravenous diamorphine (2.5 mg). In addition, local anaesthetic is infiltrated, as described below, to reduce pain sensation from the area of insertion.

Following placement of the chest drain, the patient should be offered paracetamol, non-steroidal anti-inflammatories or codeine phosphate, unless contra-indicated.

Performing intercostal drainage

In order to reduce the risk of wound infection or secondary empyema following chest drainage, preparation of the skin in a 20-cm radius around the site of insertion with antiseptic solution, e.g. 2% chlorhexidine and the use of sterile drapes, gowns and gloves is necessary.

A couple of minutes prior to drain insertion, the area should be infiltrated with up to 20 mL of 1% lidocaine. The intercostal space is palpated and a 25 gauge needle is used to raise an intradermal bleb of local anaesthetic just above the superior border of the rib to avoid the neurovascular bundle. A 20 or 22 gauge needle should then be used to infiltrate the deeper tissues down to the parietal pleura. Assessment

of the correct depth can be made by aspirating as the needle is advanced until fluid or air from the pleural space is obtained.

Next, the large bore needle with the curved ending (usually supplied along with the guidewire in the Seldinger chest drain kit) is attached to a syringe and inserted above the rib through the chest wall into the pleural cavity until air or fluid can be aspirated freely from the pleural cavity. The bevel of the needle should be pointing upwards in the case of a pneumothorax and downwards in the case of a pleural effusion. The needle is kept in position, but the syringe is removed, so that the guidewire can be passed through the needle into the pleural cavity. It is crucial to keep hold of the guidewire at all times. Once the guidewire is in position, the needle is removed and the dilator is inserted over the guidewire in a gentle rotating motion to a maximum of 6–7 cm. At this point, it may be necessary to make a small horizontal skin incision with the scalpel of 0.5–1 cm next to the insertion point of the guidewire to allow easier passage of the dilator. The dilator is then removed and the intercostal chest drain is inserted over the guidewire into the pleural cavity up to the 12–14-cm mark. The guidewire is now removed and the drain connected to the tubing of the underwater seal bottle. It should now be possible to observe the drainage of pleural fluid or the appearance of air bubbles in the case of a pneumothorax. The drain is now firmly sutured in place using a minimum of two anchor sutures, which have one loop through the skin and multiple ties around the tube. A dressing consisting of gauze should be applied on top of the skin around the drain and secured by adhesive tape.

Following the procedure, a chest radiograph should be obtained to ensure correct positioning of the intercostal drain.

Collection of pleural fluid

During the insertion of an intercostal drain, pleural fluid can be collected for diagnostic purposes if necessary. This is best performed with a 20 gauge needle and syringe following insertion of the local anaesthetic. Three samples of 5–10 mL should be collected into sterile tubes and sent for microbiological (microscopy, culture and sensitivity), biochemical (protein, glucose, pH, LDH, amylase) and cytological testing. If pulmonary tuberculosis is suspected, it is important to contact the microbiologist so that special stains and culture conditions can be undertaken. Light et al. used a fluid to serum total protein ratio >0.5, a fluid lactate dehydrogenase (LDH) value >200 U/L, or a fluid to serum LDH ratio >0.6 to diagnose exudates, with the remaining fluids being transudates. These criteria may be helpful when the protein level is close to that of the 30 g/L, Table 1 lists below.[4,5]

Protein	Glucose <3.3 mmol/L	pH <7.3	LDH ↑	Amylase ↑
Transudate <30	Empyema, malignancy,	Empyema, malignancy,	Empyema, malignancy,	Pancreatitis, carcinoma, bacterial pneumonia
Exudate >30	TB, RA, SLE	TB, RA, SLE	TB, RA, SLE	Oesophageal rupture

Table 1: Biochemical pleural fluid analysis.
Serum ranges for the above are as follows: protein 60–80 g/L, fasting glucose 3.5–5.5 mmol/L, pH 7.35–7.45.

Management of patients with intercostal chest drains

Patients with pleural drains should be cared for on wards where the medical and nursing staff are familiar with the equipment and have been trained in chest drain management. A number of points are particularly crucial in avoiding potentially harmful complications of chest drains.

It must be explained to the patient that the drainage bottle should always be below the level of his or her chest to avoid any back flow along the tubing.

Drainage of a large effusion should be done in a controlled manner to prevent re-expansion pulmonary oedema, e.g. following drainage of 1–2 L of pleural fluid the drain should be clamped for a minimum of 1 h prior to allowing further drainage.

It is important that a chest drain inserted for a pneumothorax is not clamped, as this may lead to tension pneumothorax if there is a continuing air leak allowing air into the pleural space which cannot escape. Clamping of chest drains for pneumothoraces may only be undertaken under special circumstances under very close supervision of a respiratory physician or thoracic surgeon.

The nursing staff caring for patients with intercostal chest drains should be asked to record the following observations on a regular (at least 4 hourly) basis:

1. the presence of oscillations of the fluid level in the chest drain tubing, as this indicates that the drain is patent;
2. the amount of fluid drained and the time point when drainage of pleural fluid ceases (in a patient with a chest drain for pleural effusion);
3. the presence of air bubbles escaping through the underwater seal (as this indicates continuing air leak in a patient with a pneumothorax) and the time point when the air bubbles cease;
4. the patients' temperature, blood pressure, respiratory rate and oxygen saturations; and
5. the presence of any discharge from around the chest drain site.

Removal of the drain

In the case of a pneumothorax, the intercostal drain can be removed safely when the lung is fully expanded (confirmed on chest radiograph) and there has been no further air leak (observed by bubbling in the drainage system) for 24 h. A drain inserted for an effusion may be removed once the effusion has resolved (confirmed on chest radiograph). It may, however, be indicated to perform a chemical pleurodesis at this point. (This is not covered in this article, but the reader is referred to the BTS guidelines on pleurodesis for further reading.)

It should be ensured that analgesia is given prior to removal of the chest drain. The patient should be asked to perform the Valsalva manoeuvre or exhale deeply, as this creates a positive intrathoracic pressure and thereby reduces the risk of air flow into the pleural space from outside whilst the chest drain is removed in a brisk, firm movement. An assistant should be at hand with an adhesive dressing to cover the insertion site. It is usually not necessary to place a suture to close the site when a Seldinger chest drain has been removed.

A chest radiograph should be performed following removal of the drain to ensure no new pneumothorax has been created and the lung is still fully expanded.

References

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Who's who

How to Get Ahead as a Foundation Year Programme Director!

Helen C. Underhill

Having just taken on the roles of Foundation Year Programme Director and Associate Clinical Tutor, I have found it an extremely steep learning curve establishing exactly what my role involves and the responsibilities that go with the job.

Emerging from the sheltered world of the paediatric department, I was vaguely aware that 'house jobs' as we know them had been transmogrified into a two year rollercoaster of 4 month aliquots of different specialties that unsuspecting trainees were subjected to in the name of 'MMC'. In paediatrics I had come across the new breed known as a 'FY2 trainee' and had had to make the adjustment to the frequent turnover of junior doctors needing inducting and supporting. I had also become *au fait* with the tongue twisting acronyms of DOPs, mini-CEX and CBDs and was using them regularly in my roles of educational supervisor and college tutor. But this whole 'Foundation Year Programme' thing and the actual requirements and processes involved still remained an enigma to me. I was keen to take this role on to further my knowledge and understanding – probably not a great reason to apply for the post but as valid as any other. More esoterically I also wanted to be able be constructively involved with medical education beyond the boundaries of training in paediatrics.

Firstly, to totally understand my role it was back to basics. What exactly is the point of a Foundation Training? From my reading and discussions it seems that the Foundation Programme is aimed at reinforcing the principles of good medical practice and establishing practical skills and competencies. The core curriculum provided for the trainees is designed to develop generic skills, knowledge, competencies and attitudes to ensure the highest professional conduct.[1] I would add that it also gives the opportunity for junior doctors to be exposed to a larger variety of specialties than perhaps they had been previously without having to commit to a full 'old style' SHO job. So tasters in specialties like paediatrics, anaesthetics, psychiatry, genito-urinary medicine and suchlike widen the trainees', foundation of knowledge and skills whatever specialty they go on to, but may also contribute to aiding decision making from the point of view of career progression – something several of the FY doctors have commented to me about.

So what does this role involve?

1. **Coordinating the programme including generic training**

   It is important that all the components of the core curriculum are covered and incorporated into the teaching programmes for each FY year and it is the responsibility of the Foundation Year Director to ensure that a suitable teaching programme is in place for both years covering the core skills and generic knowledge that is required.

2. **Ensuring that local processes for assessments are in place**

   The training is competency based and it is important that the trainees are kept informed and up to date about the necessary assessments that are required to undertake as well as how to record and document them. Trainers also need to be kept up to date with the appropriate skills required and for each attachment an educational supervisor must be allocated.

3. **Liaising with the Foundation School**

   This involves attending the appropriate meetings and also involvement in the recruiting and allocation process. Within our Deanery the FY1 trainees have to be graded for their application for their FY2 posts and as Foundation Programme Director this is an important part of the role.

4. **Pastoral care**

   This may take the form of mentoring a trainee through a difficult time or being able to give career advice or directing to the appropriate person if not able to. On taking the role I met up with most of the trainees individually for an informal chat which I found useful in putting names to faces but also to highlight any active issues sooner rather than later and also to make them aware that they could contact me if necessary.

5. **Identifying and supervising trainees in difficulty**

   As Foundation Year trainees the junior doctors rotate through specialties every 4 months and it is critical that processes are in place so that a trainee in difficulty is identified early and the information is passed on to the next specialty so that the necessary support and measures can be put in place. As the Programme Director you are the person who can have that overview of the trainee and support both the trainee and, if necessary, the educational supervisor.

6. **Working closely with the clinical tutor and post graduate medical team**

   In fact this really should be on the top of my list of roles as I could not have achieved the other goals over the last few months without the support, advice and input of the clinical tutor and the extremely knowledgeable and experienced post graduate manager. As my experience and knowledge grows, hopefully I will be more of a contributory member of the team!

I am sure that if I was asked to write this in a year's time the list would probably be more extensive but I am convinced that the basic underlying principles of the role will be much the same . . . unless the 'powers that be' change everything (again!).

Reference

[1] Academy of Medical Royal Colleges. Curriculum for the Foundation Years in postgraduate education and training, 2004.

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